Maastricht University

Brain injuries in boxing and soccer: a neuropsychological approach

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Brain injuries in boxing and soccer:

a neuropsychological approach

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Niets immers kan mij het leven vergoeden, niet al wat aan rijkdom,
naar wordt verteld, het bezit is geweest van het prachtige Troje,
vroeger in dagen van vrede, nog voor de komst van de Grieken,
noch wat de tempel van Foibos Apollo, de feilloze schutter,
achter zijn stenen drempel verbergt, in het rotsachtige Python.
Meester kan men zich maken van runderen en vlezige schapen
en het bezit van ketels en rosbruine paarden verkrijgen,
maar het leven kan men door koop noch door roof zich verwerven;
't keert niet terug, nadat het de heining der tanden ontsnapt is.
Mij heeft mijn moeder verteld, de zilvervoetige Thetis,
dat me het noodlot langs tweeërlei wegen het eind van de dood brengt:
als ik hier voortga de strijd om de stad der Trojanen te voeren,
valt me geen thuiskomst ten deel, maar wel onsterfelijke krijgsroem;
als daarentegen ik naar mijn vaderland weerkeer,
valt me geen krijgsroem te beurt, maar wel, tot in lengte van jaren,
zal ik het leven behouden en niet de dood mij verrassen

(Illias, boek negen)

Ter nagedachtenis aan
Cor Kanters, de vader van Marly,
een vader die te weinig jaren heeft gekregen.

Opgedragen aan:
Marly, Lize, Rolf en Myré,
de kleuren van mijn leven.

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Aim of the study

Head injuries are not uncommon in many sports. However, few sports include blows to the head or with the head as part of the accepted way of playing the game and boxing in particular of winning. Our growing knowledge concerning the detrimental effects on cognitive functioning of blows to the head especially of repeated blows to the head raises serious questions about the cognitive consequences of deliberately inflicting repeated blows to one's own head (i.e., doing headers in soccer) or to the opponents head (i.e., striving for a knock out in boxing). Since many young people throughout the world engage in these two sports, it is important to find out whether their participation in these sports can adversely affect their mental abilities in the present and for their futures. The aim of these studies has been to determine whether athletes risk cognitive impairment when they participate in sports in which blows to the head are an integral part of the game.

Background of the study

In recent years, brain injuries in athletes have captured many news headlines as several elite athletes have retired due to the effects of concussion. Depending on the nature of the sport these injuries are many times viewed as just part of the game. While many of these injuries are minor, some can be quite serious with long term consequences.

In 1928 an American neurologist published a paper in which he described a peculiar condition in prize fighters. Martland documented abnormal emotional responses, motor abnormalities and severe cognitive problems in relatively young people. In 1969 Johnson copied his research and reported that 20% of the professional boxing population he studied showed dementia pugilistica. MRI studies performed in the 90's showed brain abnormalities in 45% of professional boxers and in the 80's and 90's several studies showed that 90% of the professional boxing population showed mild to severe cognitive problems. Besides boxing noncontact and collision sports were studied concerning the risk for traumatic brain injury and cognitive impairments. Tysvaer demonstrated in Norway that 33% of retired amateur soccer players showed ventricular dilation and a high percentage (81%) of these players demonstrated memory problems compared to age matched controls. Most of these studies published were retrospective.

In 1993 in America, the Pittsburgh Steelers project was the first long term followup project in which athletes (American football players) were followed during several seasons. This study involved systematic testing of athletes at set times. Formal evaluation of each player prior to the beginning of the season provided the basis for comparison in the event of a concussion during the season. Testing was repeated 24 hours after a suspected concussion, and again approximately five days after injury. The study showed memory complaints after concussion in athletes and that 15% of the athletes showed these problems longer than a year. After this study was carried out the Quality Standards Committee of the American Academy of Neurology (AAN) developed in 1997 a formal practice parameter for the management of concussion in sports. These guidelines included recommendations for the development of a valid, standardized sideline evaluation for the immediate assessment of concussion in athletes.
Momentarily a sideline assessment of concussion is developed in the Netherlands focused on soccer. In the USA guidelines are recommended to protect American football/ ice hockey players after an acute traumatic brain injury.

Relevance of the study

A main problem in the diagnosis of acute MTBI is that both CT and MRI show no abnormalities in athletes and patients with these traumas as evaluations of MRI scans of amateur boxers directly after a knock out showed. Even in demonstrating chronic brain trauma, CT and MRI scanning techniques have limited value. Post traumatic atrophy can be detected by MRI and CT, especially in athletes with *dementia pugilistica*, but at the time the morphological abnormality is detected the athlete suffers already profound cognitive impairments. The introduction of neuropsychological assessment has made it possible to demonstrate subtle cognitive impairments due to MTBI. These findings are important because we will be able to identify brain injury earlier and more accurate. Neuropsychological assessment should add to our ability to identify athletes at risk for MTBI and the future development of more severe brain injury. In addition, neuropsychological assessment can be a very sensitive tool in detecting acute and the development of chronic traumatic brain injury in athletes. Finally, according to scores on neuropsychological tests return to play decisions can be made.

Chapter 1

Introduction*

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Summary

The studies represented in this dissertation are an attempt to describe and explain the many difficult issues that exist regarding diagnosis and outcome of sports related concussion, acute traumatic brain injury (ATBI) and chronic traumatic brain injury (CTBI).
In recent years, sportsneurological injuries have captured attention as several professional football-, hockey- and soccer players have retired due to the effects of sports related traumatic brain injury. Even in the amateur section millions of people sustain one or more sports-related traumatic brain injuries each year, causing mild to severe impairments. In typical contact- and collision sports like boxing, American football and soccer these injuries frequently occur.
Single sports-related TBI can result in neurologic- and cognitive deficits. Cumulations of sports related TBI in an extended period (weeks, months) can result in aggravation of neurologic- and cognitive symptoms. Repeated mild brain injuries occuring within a short period (i.e., minutes) can have dramatic effects regarding cognitive functioning and can be life threatening (diffuse cerebral brain swelling, second impact syndrome). Therefore, early detection through a thorough knowledge of symptoms and specific documentation of the injury is critical to the management of sports related traumatic brain injury.
Most of the impairments documented in our studies of contact- and collision sports athletes are in the cognitive domains like memory, planning, visuo-perception, attention and concentration. However, at the end of this century and at the beginning of a new millennium there is still a lot of work to do for designing areas of agreement and disagreement in the detection and management of sports related traumatic brain injury.

Introduction

Recreational, amateur, and professional sports occupy a high level of importance in present day European- and American society. The motivation to participate in contact or collision sports increases the risk for incurring all kinds of injuries. This is especially significant when one considers traumatic brain injuries (TBI) and a variety of other disabling neurological injuries. Although the majority of the neurological injuries tend to be mild, more serious and life threatening injuries can also occur. The impetus for writing a dissertation on sports neurological injuries has come

largely from clinical practice. In clinical practice and working with athletes it became clear that there was a gap between the overlapping territories, neuropsychology and neurology. Clearly, as with any borderland zone, there is a risk of relative neglect as each separate discipline has proceeded on its specialised way, leaving an uneasy interface between. Neurology deals directly with the apparatus of mind. Neuropsychology on its part deals essentially with mental functioning and has less to do with the hardware upon which cognition depends. One aspect of this uneasy interface is the belief of some that 'what you can't see don't exist', denying the complexity of human behaviour and the factors which can shape and distort it. Apart from the denial of complexity of behaviour there is a simple question 'why anyone should be interested in cognitive impairment in athletes'. One answer is denial of complexity, the other is that until quite recently many people were not interested. For example, in psychology many professionals see cognition as a non-issue. For them. behaviour is simply a function of habits, or response probabilities. Other psychologists translate perception, organisation and memory e.g., in behaviour ignoring the problems of cognitive impairment. This dissertation tries to provide information to narrow the gap between neurology and neuropsychology and that it gives a comprehensive view of the cognitive, behavioural and emotional consequences of cerebral injuries. This dissertation is based on the belief that determinants of complex human behaviour (coordination and control of thoughts) are fundamentally important. The other goal of this dissertation is to present studies and theoretical knowledge to assist medical and paramedical personel in diagnosing and recognizing symptoms of traumatic brain injury due to athletic activity.

The past decade has seen a growing interest in sportsneuropsychology. The interest is founded by ideas regarding the importance of cognitive processes and by the way brain injuries affects learning, memory, language, perception and attention. In addition to the more traditional role of neurology other approaches will be required to assess brain functioning, for instance neuropsychological assessment. Furthermore, there is some overlap between cognitive function loss and pathogenic mechanisms seen in brain injured athletes due to recurrent traumatic brain injuries (dementia pugilistica) and Alzheimers patients. From the convergence of neurology, neuropsychology and sports, we are gaining a new persepective on behaviour and cognitive functioning.

Since the beginning of the 1990's several sportsneuropsychological programs have emerged, mostly in Europe and the USA. One of the main sources of fosterments for sportsneuropsychological programs is the growing medical and political concern regarding TBI incurred in sports. This served in the USA as a stimulus for the

American Academy of Neurology to establish in 1997 guidelines for diagnosis and evaluation of the brain injured athlete. In the Netherlands, England, Norway, Iceland and Sweden there were governmental debates concerning the aftermath of professional boxing, professional soccer and questions were asked concerning medical guidelines to stop the detrimental neurologic- and cognitive effects of the sports mentioned above. The results: professional boxing is banned in Norway, Sweden and Iceland and money is invested in scientific programs with the aim to stop neurological injuries and cognitive impairment caused by sporting activities.
In addition, recently two articles have been published in which a neurologist and neuropsychologist working with athletes provided information about treatment of brain-injured athletes (1-2). In these articles the authors showed their concern about treatment and follow-ups of athletes who sustained TBI and who are at risk for severe brain injuries.The articles highlighted three main issues that should be addressed by any guidelines for the management of TBI in sport: (a) appropriate management of TBI in sport at the time of the injury, (b) prevention of a catastrophic outcome related to acute brain swelling, and (c) avoidance of cumulative brain injury caused by repeated concussions and the accumulations of trivial traumas.
Moreover, coaches and trainers may not be aware of the impact of TBI on cognition and emotional behaviour. Frequently an athlete's physical recovery from a TBI creates expectations for adequate functioning. However, a normal physical appearance can severely mask underlying cognitive deficits associated with TBI.

In this study the term head injury refers to any traumatic injury to the head and face. The brain may be, but is not necessarily, injured. Damage may be restricted to soft tissue, vasculature, and peripheral nerves. Head injury should not be used interchangeably with TBI because head injury, by definition, does not imply damage to the brain. TBI is a consequence of head injury in which damage to the head results in damage to the brain. Damage may result in symptoms that are transient or permanent. Long-term impairment may be absent, trivial or severe (3).
Furthermore, focal and generalized brain traumas will be discussed. A focal injury is one that often results from a direct blow or penetration of the brain and tearing of cerebral substance or vessels. This results in a localized area of bleeding or lesion of a localized brain area against bony irregularities of the inner surface of the skull. In diffuse brain injury, in contrast to focal brain injury, there has been a global effect on cerebral functioning and/or structure, usually resulting in a widespread dysfunction (4).

Epidemiology of traumatic brain injury (TBI)

Today, traumatic brain injury (TBI) is one of the leading causes of disability and death among individuals under 45 years in the industrialised nations (5-6). TBI account for half of the deaths and most cases of permanent disability after injury (7). Epidemiological studies suggests that between 250 and 300 hospital admissions a year/100.000 of the population in Britain involve a head injury (8). A rate that is at last three times greater than that of schizophrenia and a prevalence that is greater than the prevalence of for panic disorder (13). Furthermore, disorders arising from traumatic brain injuries are more common than any other neurological disease, with the exception of headaches (14).
Other epidemiological studies vary between 152-430/100.000 of annual hospital admitted TBI (9, 11).
Of all hospitals admissions in Britain 7% sustained severe TBI, 18% incurred moderate TBI and 75% sustained mild TBI (8). In addition, Rutherford (1979) (10) showed that 50% with mild brain injuries (MTBI) experienced a range of post concussion symptoms including headache, dizziness, irritability, reduced concentration, memory dysfunction and emotional changes like anxiety and depression. These symptoms resolved usually within 3-6 months after injury. But it was calculated that 15% of the total MTBI population still experienced complaints one year after injury.

Sports neuroepidemiology

The exact incidence of traumatic brain injury related to sports can only be roughly estimated. World wide estimates of the incidence of TBI of all causes range from 152 to 430 cases per 100.000 population per year, which is consistent with the results of the British study (8). The percentage of brain injuries related to sports ranges from 3% to 25%. Based on these figures, the rates of sports-related brain injuries could theoretically range from 5 to 68 cases with a median value of 42 per 100.000 person years (11). Other studies show that of all traumatic brain injuries, 10% to 22% are treated in hospitals and it is estimated that 11% of all hospital treated TBI in children and adolescents are sports related (15-17). These numbers are in accordance with the estimation made by Kraus (11). Studies designed to identify all individuals who seek medical attention after TBI report that only 16% to 20% are admitted suggesting significantly higher incidence rates than those cited above (18-19).

Since 1975, the National Athletic Injury/Illness Reporting System (NAIRS) has been following all athletic injuries in various sports, including concussion the most common head injury in sports. In the NAIRS system, a reportable concussion is any incident of disorientation caused by trauma that requires cessation of play to examine the athlete. The NAIRS report estimated the number of 250.000 concussions incurred in contact sports per year. With a population in the USA of 265 million people this means a number of 96 concussions in 100.000 person years, which is considerable higher than Kraus his figures. The average annual incidence of reportable concussion in selected college sports 1975-1978 showed (NAIRS report):

Table 1

	cases per 10.000 athlete exposures*	cases per 100 athletes**
Amercian football	11.1	2.2
Ice hockey	3.5	3.7
Wrestling	3.0	2.5
Soccer	2.7	1.6

* exposure is matches and training;
** case in whole sports career

Severe TBI is incurred in boxing (20) and sports with a high velocity like skiing. In professional boxing 20% of the boxers is developing boxing dementia, 45% show brain abnormalities seen on CT- and MRI scans and 90% develop severe to moderate chronic neuropsychological impairments (21-22). Even soccer players are vulnerable for traumatic brain injury. In the Netherlands 54% of the professional soccer players incur one or more concussions during their career (23). American studies show a concussion rate of 59% in male amateur players in two soccer season's (24). In professionals 45% of the soccer players show chronic neuropsychological impairments (23). Even in adolescents specific chronic neuropsychological problems are documented caused by playing soccer (25). In retired amateur soccer players 33% of the players show CT-scan abnormalities, and 81% show chronic neuropsychological impairments (26-28).
The incidence of injury to peripheral nerve in sports is largely unknown. Peripheral nerve injuries incurred in sports are relatively uncommon and are not typically detected by injury surveillance systems (29).

Causes of TBI in sports

Initial mechanical damaging factors

A change in the velocity of the brain caused by a blow to the cranium can produce linear and rotational movements of the brain within the skull (30). When the skull and the brain within it are accelerated coup-contrecoup injuries can occur. The bruise spots at the impact side are called coup lesions; contrecoup lesions are located in the area opposite the blow (31).

After a rapid rotation of the head (for example, when an athlete is hit by a blow coming in at an angle) diffuse axonal injury (DAI) and stretching and snapping of blood vessels may occur. The rotation of the brain within the cranial vault tends to be delayed relative to the initiating change in velocity, because of the brain's intrinsic inertia. This lagging movement of the brain leads to stretching and shearing forces on the fibers (DAI) and blood vessels (subdural and intracerebral hematomas) (32-34). Common locations of DAI after closed head trauma include: cerebral hemispheric gray-white matter interface, subcortical white matter, body and splenium of corpus callosum, basal ganglia, dorsolateral aspect of brainstem and cerebellum (35). Subdural hematomas (acute, subacute, and chronic) and intracerebral hematomas have effects that are specific to their locations, extend and degree of neuronal damage. The brain can also be injured when the moving head suddenly stops as in falls or collisions with other players. The moving head and the brain within it are stopped, followed by a sudden deceleration until the brain's movement stabilizes. Coup-contrecoup injuries and shearing injuries can result. As an example, falls on the back of the head may result in fracture of the skull and contrecoup contusions on the orbital surface of the frontal lobes and the tips of the temporal lobes. In falls and sudden stops of the moving head, the accelerating-decelerating brain slides around within the cranial cavity over bony structures who surround the orbital and frontal areas along the base of the skull (36). This is why most traumatic brain injuries are located in this region. Pudenz & Shelden (37) observed in 1946 the brains of monkeys after replacing their skull caps with transparant material during and directly after a head strike. Swirling movements of brain tissue could be witnessed after acceleration injury, both at the point of impact and elswhere.

In general, TBI is often a result of an interaction between linear and rotational forces and is seldom due to pure rotational or linear acceleration (20).

Brain damage can also be induced by hypoxia. Strangulation techniques in martial arts, for example, in which the blood supply to the brain is blocked by pressure on the carotid arteries, can produce hypoxia and unconsciousness if the victim fails to submit and subclinical electroencephalographic abnormalities may be induced (38-39). During hypoxia, free radicals and excitotoxic neurotransmitters, such as glutamate, are released and result in further damage to neurons (40-41).

Secondary brain damage

TBI occurs in different ways, involves different mechanisms, and results in different types of clinical disorders. The velocity, localization, and direction of mechanical force at impact as mentioned before are not the only specific factors that contribute to TBI. Cerebral injury can also start after the primary injury and results of a series of biochemical steps that are set in motion by the primary injury (4). It can be concluded that TBI should be seen as a process and not as an event (32).

Physiological changes after mechanical impact to the head

The early experimental studies by Dennie-Brown & Russel in the 1940's (42) showed a number of transient pathophysiological changes immediately after a concussive blow to the head. They observed depression of cardiac-, vasomotor- and respiratory functions as well as paralysis of brain stem reflexes. Remarkable is the fact that loss of brain stem reflexes could only be produced if the head was free to move when struck (42). This may explain why, for example, concussion is common when the head is in motion at the time of injury, but relatively rare in static crushing injuries. Further studies in animal models have confirmed these observations (43-44).

Biochemical changes after mechanical impact to the head

There have been several studies of neurochemical changes after TBI. Secondary neurologic injuries are partly due to ischaemic-hypoxic injury. The secondary injury, and cell death are mediated by progressive increases in intracellular CA2+ concentration due to activation of Ca2+ permeable glutamate receptor (i.e., N-methyl-D-aspartate (NMDA) receptors and, to a lesser extent, activation of voltage-gated CA2+ channels. Pathological increases in K+ concentration facilitate intracellular Ca2+ accumulation by causing massive membrane depolarization. Elevated K concentrations depolarizes synaptic terminals, facilitating the release of glutamate. Moreover, the activation of postsynaptic NMDA glutamate receptors

also requires membrane depolarization. Finally the membrane depolarization associated with these uncontrolled increases in K+ concentrations activates damaging CA2+ entry by opening voltage-gated Ca2+ channels (4).
From other studies it is evident that TBI can affect the neurotransmitter systems that mediate mood and affect, including norepinephrine, serotonin, dopamine and acetylcholine (45-46). Other studies show enhanced concentrates in serum and cerebrospinal fluid of proteins synthesised in astroglial cell or neurons as markers of cell damage in the central nervous system. Increased levels of these proteins in cerebrospinal fluid (CSF) and serum have been observed in TBI victims (47-48). Philips et al (49) found in 1980 raised serum concentrations of of creatine kinase isoenzyme BB (CK-BB) in 14 of 22 patients with concussions. Nordby et al (50-51) reported increased serum and CSF levels of CK-BB in 23% of their concussed patients. Raise in serum levels of S-100 could possibly predicts outcome after injury (51).

The studies mentioned above demonstrate that the sequelae of TBI depend not only on the primary mechanical damage but also on the complex of interaction of pathophysiologic and biochemical events that follow the initial distortion of brain tissue.

Classification of head injury

The Glasgow Coma Scale

The Glasgow Coma Scale (GCS) is designed for assessing the level of consciousness by establishing a rating concerning three aspects of behavior: motor responsiveness, verbal performance and eye opening (53).

Table 2 Glasgow Coma Scale

Eye Opening Response		
Spontaneously	4	Reticular activating system is intact; patient may not be aware
To verbal command	3	Opens eyes when told to do so
To Pain	2	Opens eyes in response to pain
None	1	Does not open eyes to any stimuli
Verbal Response		
Oriented-converses	5	Relatively intact CNS Aware of self and environment
Disoriented-converses	4	Well articulated, organized, but patient is disoriented
Inappropriate Words	3	Random, exclamatory words
Incomprehensible	2	Moaning, no recognizable words
No response	1	No response or intubated

Motor response		
Obeys verbal commands	6	Readily moves limb when told to
Localizes to painfull stimuli	5	Moves limb in an effort to remove painfull stimuli
Flexion withdrawal	4	Pulls away from pain in flexion
Abnormal flexion	3	Decorticate rigidity
Extension	2	Decebrate rigidity
Normal	15	
GCS score = E+M+V	None	

The patient's clinical state is charted regularly on the functions mentioned above. Numerical scores are summated for the best responses obtained under each category at a defined point in time. By observing and classifying these responses, one can quantify the level of consciousness. The scores have a range from 3 (totally irresponsive) to 15 (fully awake and alert). In this way useful predictions can be made, often within 24 hours of injury and more
certainly within the first week. The GCS has reached general acceptance as a reliable measure of the level of consciousness for clinical and research purposes and is now in routine use in many countries, in units caring for acute head injured patients.
Stein & Spettel (54) presented in 1995 (Table 3) the Head Injury Severity Scale (HISS) based on GCS scores. The HISS is designed to seperate minimal injuries with negligable risk of complications from mild and severe injuries requiring medical attention.

Table 3 The Head Injury Severity Scale (HISS)

HIIS category	Clinical characteristic
Minimal	GCS 15
	No loss of consciousness or amnesia
Mild	GCS 14 or 15
	Brief (<15 minutes) loss of consciousness or amnesia or impaired alertness or memory
Moderate	GCS 9-13
	or LOC > 5 minutes, or focal neurological deficit

Amnesia in relation to severity of traumatic brain injury

On recovery of consciousness, events often fail to be recorded in memory for a period of time. Often patients report a post-traumatic amnestic gap after head injury. The importance of the duration of amnesia as a guide to the severity of injury and

prognosis for recovery was already shown in the 1930's (55). Post traumatic amnesia (PTA) has now become a widely used part of clinical practice to predict severity of injury. The lenghts of PTA may be defined as the time from the moment of injury to the time of resumption of normal continuous memory and can be used as a guide to quantify the degree of severity (Table 4).

Table 4 **PTA** (classification according to Bigler, 1990) (56)

PTA duration		Severity
<5	minutes	Very mild
5-60	minutes	Mild
1-24	hours	Moderate
1-7	days	Severe
1-4	weeks	Very severe
More than 4	weeks	Extremely severe

Concussions

Concussions are often incurred in sports. In boxing and martial arts, concussions are common and inevitable because victory is direct by rendering the opponent unconscious by a grade three concussion (according to the AAN classification, 1997) (56). In collision sports like soccer a high frequency of concussions due to collisions between players are documented (23). Even in non-collision sports like horse back riding, biking, skiing, ice skating, roller skating, skateboarding, and in-line skating concussions are often reported as a result of collisions and falls (58-62).
Kelly and Rosenberg (1) define concussion as a trauma-induced alteration in mental status that may or may not involve loss of consciousness. Some frequently observed features of concussion are vacant stare, inability to focus attention, disorientation (walking in the wrong direction, being unaware of time, data, place), slurred or incoherent speech, incoordination, emotional lability, memory deficits, problems with abstract reasoning, slowed reaction time, slowed mental- and motor speed, problems with planning and judgment, and difficulties in processing novel or complex visual spatial stimuli (63-68).
Symptoms of concussion can be classified as early and late. ***Early symptoms*** (minutes to hours after injury) are headache, dizziness or vertigo, lack of awareness of surroundings, nausea, and vomiting. ***Late symptoms*** (days to weeks after injury) are persistent low-grade headache, light headedness, poor attention and concentration,

memory dysfunction, inefficient information processing, inadequate perceptual processing, problems with planning, and slowed reaction time, intolerance of bright lights or difficulty focusing vision, intolerance of loud noises, sometimes ringing in the ear, easy fatiguability, irritability and low frustration tolerance, anxiety, depressed mood, sleep disturbances (1).
Although, many concussions seems mild at the time of injury, the range of symptoms can be wide and long-lasting.

Concussions are graded in three categories: according to AAN norms (1997) (grading according to the *Quality Standards Subcommittee: Practice Parameter*) (57).

Grade 1 Concussion: Transient confusion, no loss of consciousness, and a duration of mental status abnormalities of < 15 minutes

Grade 2 Concussion: Transient confusion, no loss of consciousness, and a duration of mental status abnormalities of > 15 minutes

Grade 3 Concussion: Loss of consciousness (LOC), either brief (seconds) or prolonged (minutes or longer).

Cumulative effects of concussion

Cumulative effects of concussions such as, disabling headache, classic migraine, and delayed recovery of brain functioning (after a second concussion) have been reported in studies of concussion victims (67, 69). Repeated mild brain injuries occuring over an extended period (i.e., months or years) can result in cumulative neurologic- and cognitive deficits. Gronwall and Wrightson (70) found that deficits in information storage and retrieval were exacerbated by successive injuries occurring over an extended period (i.e., months or years). Repeated mild brain injuries occurring within a short period (i.e., hours, days, weeks) when someone is still symptomatic from the former insult can be catastrophic or fatal (71-72).

Postconcussion syndrome

The postconcussion syndrome is often reported in athletes, especially those with repeated or successive concussions during the course of one season (4). Carlsson and co-workers (73) found a strong correlation between the degree and the extend of post-concussive symptoms and the number of previous head injuries, and conjectured that the cumulative effects of repeated head injury play a significant role in this syndrome.

The postconcussion syndrome consists predominantly of deficits in cognition, the sense of physical well being, and mood (Table 5). A multitude of complaints can occur with post concussion syndrome including persistent headache, irritability, concentration problems, memory problems, vertigo and generalized fatigue. Often these symptoms occur in the absence of any neurological abnormalities on examination. Most symptoms are typically short-lived and resolve spontaneously; however, in a small number of athletes, problems may become persistent (74). The aetiology of the post concussion syndrome has long been debated. Psychological factors are frequently evoked to explain such symptoms in an otherwise 'normal' individual. As an example, Cook (75) studied at the end of the sixties 300 amateur rugby players suffering at least 1 concussion. Over 60% of the rugby amateurs reported headaches after injury but in three-quarters the headaches resolved within 48 hours. Only 17% were unable to work, with 80% returning in less than 1 week. Cook attributed the rapid recovery in athletes to a high motivation. However, Barth et al (76) demonstrated neuronal loss and and microscopic lesions in the brainstem in post concussion syndrome patients. In addition, as many as 50% of patients with post-concussion syndrome have abnormalities on brainstem evoked potential studies (77). Moreover, MRI has shown a variety of abnormalities in the frontal and temporal regions in post concussion syndrome patients with normal CT-scans (78). Such information, taken in combination, would indicate that at least in some athletes with post-concussion syndrome symptoms underlying neuropathological or structural abnormalities in the brain are induced by (repeated) mild brain injuries.

Table 5 Postconcussion syndrome

Cognitive	Personality	Somatic
Memory	Depression	Headaches
Attention	Mood changes	Dizziness
Problem solving	Motivation	Photophobia
Word finding	Anxiety	Difficulty sleeping
Fluency	Irritability	
Tiredness (psychic and somatic)		

Dementia pugilistica

Athletes can develop a severe chronic progressive traumatic encephalopathy after being exposed to a cumulation of (mild) traumatic brain injuries. The syndrome follows a three-stage clinical pattern. The first stage is manifested by affective disturbances and mild motor incoordination. In the second stage, affective symptoms increase; paranoid ideas and mild dysarthria and tremor may appear.

The third stage is characterised by a decrease in general cognitive functions (memory deficits, dysarthria, motor incoordination), impaired hearing, hyperreflexia, and intention tremor. Furthermore, dementia, personality change (immaturity, aggressiveness, suspiciousness), and social instability have been described. (79-81).

Diffuse brain swelling/second impact syndrome (SIS)

It has been well documented that a fatal outcome may result from repeated minor head injuries occurring in short succession (1). Saunders and Harbaugh (82) reported in 1984 a case of a 19 year old college football player who was involved in a fist fight and received a blow to the head resulting in a brief loss of consciousness. Four days later he was involved in in a football game and, despite receiving no unusual head impact he collapsed on the field. He died 4 days later with diffuse and uncontrollable cerebral edema.

Diffuse cerebral swelling is a rare but well recognized cause of delayed catastrophic deterioration resulting in death or persistent vegetative state after an apparently minor head injury. Risk factors for this post traumatic complication have not been established, although most cases occurred in children and adolescents (71, 83). This phenomenon has also been observed in collision sports (84).

It has been postulated that a specific form of cerebral swelling may be the consequence of a repeated minor head injury. The 'second impact syndrome' (SIS) results from acute, usually fatal, brain swelling that occurs when a second head blow is sustained before complete recovery from a previous concussion. Brain swelling apparently results from a failure of autoregulation of cerebral circulation causing vasoparalysis which in turn causes increased intracranial pressure, which may be difficult or impossible to control. This complication can result in brainstem herniation and death (72, 85). The second impact syndrome has been reported more frequently since it was characterized in 1984 (72, 82). Combined data from four states in the USA (Colorado, Missouri, Oklahoma and Utah) indicates an annual rate of 2.6 cases per 100.000 population of sports related TBI resulting in hospitalization or death. Although rare, sports related diffuse cerebral swelling has a high mortality. If SIS represents a real clinicopathologic entity, then there are potential major consequences for the management of minor head injuries in sports and implications for the innumerable amateur- and professional athletes who sustain such injuries. Since all previously reported cases of second impact syndrome victims show the common feature of an athlete who remains symptomatic (e.g., headaches, dizziness) after the first injury, a number of guidelines have been established for allowing a return to competition after a concussion. Many of the published case reports (82, 86)

and most notably the guidelines of the Colorado Medical Society and the American Academy of Neurology suggest that prevention of this syndrome should be based on a policy of not allowing individuals to return to sports until postconcussive symptoms have fully resolved (57, 85, 87).

Why is the brain so vulnerable for second injuries?

Metabolic disturbances

Essential for understanding the vulnerability of the brain for second injuries is the fact that the minutes to few days following concussive injury, cells which are not irreversibly destroyed, remain alive but exist in a vulnerable state. Experimental animal studies have identified metabolic dysfunction as a major post-concussive physiological event which produces and maintains this state of vulnerability. This period of enhanced vulnerability is characterized by both an increase in the demand for glucose and an inexplicable reduction in cerebral blood flow. The result is an inability of the neurovascular system to respond to increasing demand for energy in order to re-establish its normal chemical and ionic environments. In animal studies, the concept of injury-induced vulnerability have indicated that up to three days following a concussion, a reduction in cerebral blood flow, which would be normally well-tolerated, now produces extensive neuronal cell loss (88-92). Moreover, some studies using animals conclude that the mismatch between glucose demand and fuel availability can even be seen following mild concussive brain injury (93-94).
Whatever the mechanism, it seems to be clear that a concussion can produce a state of vulnerability for surviving nerve cells. This state can be characterized in terms of a metabolic dysfunction which can last for days. When the brain is sustaining another concussion in this critical period, massive neuronal cell loss can be the result.

References

Chapter 1

1. Kelly JP, Rosenberg JH. Diagnosis and management of concussion in sports. Neurology 1997; 48: 575-580.
2. McCrea M. Use of standardized assessment of concusion (SAC) in the immediate sideline examination of football players. Neurology 1997; 48: 586-588.
3. Kay T, Newman B, Cavallo M, Ezrachi O, Resnick M. Toward a neuropsychological model of functional disability after mild traumatic brain injury. Neuropsychology 1992; 6: 371-384.
4. Bailes JE. Head injuries in sports. In: Jordan BD, Tsairis P, Warren RF, Eds. Sports Neurology, 2nd edition. Philadelphia: Lippincott-Raven, 1998.

5. Goldstein M. Traumatic brain injuries: a silent epidemic. Annuals of Neurology 1990; 27: 327.
6. Jenett B. Epidemiology of head injury. Journal of Neurology, Neurosurgery and Psychiatry 1996; 60: 362-369.
7. Kraus JF. Epidemiology of head injury. In: Cooper PR, ed. Head injury, 3rd edition. Baltimore: William Wilkins, 1993.
8. Jennett B, MacMillan R. Epidemiology of head injury. British Medical Journal 1981; 282: 101-104.
9. Klauber MR, Barret-Connor E, Marshall LR, Bowers SA. The epidemiology of head injury: a prospective study of an entire community – San Diego County, California, 1978. American Journal of Epidemiology 1981; 113: 500-509.
10. Rutherford WH, Merrett JD, McDonald JR. Symptoms at one year following concussion from minor head injuries. Injury 1979; 10: 225-230
11. Kraus JF, Epidemiologic features of injuries to the central nervous system. In: Anderson DW, ed. Neuroepidemiology, a tribute to Bruce Schoenberg. Boca Raton, FL: CRC Press 1991; 333-357.
12. National Athletic Injury Reporting System. Pennsylvania State University, 1979.
13. Silver JM, Hales RE, Yudofsky SC. Psychiatric consultation to neurology. In: Tasman A, Goldfinger SM, Kaurmann CA, Eds. American Psychiatric Press Review of Psychiatry, vol 9. Washington DC: American Psychiatric Press, 1990; 433-465.
14. Kurtzke JF. Neuroepidemiology. Ann Neurol 1984; 16: 265-277.
15. Lehman LB. Nervous system sports-related injuries. American Journal of Sports Medicine 1987; 15: 494-499.
16. Frenquelli A, Ruscito P, Sicciolo G, Rizzo S, Massarelli N. Head and neck trauma in sporting activities:Review of 208 cases. Journal of Craniomax. Surgery 1991; 19: 178-181.
17. Hansen TB, Pless S, Bravvers M. Cranial injuries among children in the county of Ringkobing. Ugeskr Laeger 1991; 15: 2947-2479.
18. Strang I, MacMilian, R, Jennett B. Head injuries in accident and emergency departments at Scottisch hospitals. Injury 1978; 10: 154-159.
19. Fife D. Head injury with and without hospital admission: comparison of incidence and short-time disability. American Journal of Public Health 1987; 77: 810-812.
20. Jordan BD. Neurologic aspects of boxing. Archives of Neurology 1987; 44: 453-459.
21. Casson RI, Sham R, Campbell Eam et al. Neurological and CT evaluation of knocked out boxers. Journal of Neurology, Neurosurgery and Psychiatry 1982; 45: 170-174.
22. Casson IR, Seigel O, Sham R, Campbell EA, Tarlau M, Didomenico A. Brain damage in modern boxers. JAMA 1984; 251: 2663-2667.
23. Matser JT, Kessels AGH, Jordan BD, Lezak MD, Troost J. Chronic traumatic brain injury in active professional soccer players. Neurology 1998; 51: 791-796.
24. Boden BP, Kirkendall GT, Garret WE jr. Concussion incidence in elite college soccer players. American Journal of Sports medicine 1998; 26: 238-241.
25. Witol A, Webbe F. Neuropsychological deficits associated with soccer play. Archives of Clinical Neuropsychology 1994; 9: 104-105.
26. Tysvaer AT, Storli O, Bachen NI. Soccer injuries to the brain. A neurologic and electroencephalographic study of former players. Acta Neurologica Scandinavica 1989; 80: 151156.
27. Tysvaer AT, Lochen EA. Soccer injuries to the brain. A neuropsychological study of former soccer players. The American Journal of Sports Medicine 1991; 19: 56-60.

28. Sortland O, Tysvaer AT. Brain damage in former association football players. An evaluation by cerebral computed tomography. Neuroradiology 1989; 31: 44-48.
29. Clarke KS, Jordan BD. Sports Neuroepidemiology. In: BD Jordan, P Tsairis, RF Warren, Eds. Sports Neurology, 2nd edition. Philiadelphia: Lippincott-Raven, 1998; 3-13.
30. Holbourn AHC. Mechanics of head injuries. Lancet 1943; 2: 438-441.
31. Zomeren van AH, Burg van den W. Residual complaints of patients two years after severe head injury. Journal of Neurology, Neurosurgery and Psychiatry 1985; 48: 21-28.
32. Gennarelli TA. The pathobiology of traumatic brain injury. Neuroscientist 1997; 3: 73-80.
33. Strich SJ. Shearing of nerve fibers as a cause of brain damage due to head injury. A pathological study of twenty cases. Lancet 1961; 2: 443-448.
34. Lampert PW, Hardman JM. Morphological changes in brains of boxers. JAMA 1984; 251: 26762679.
35. Ozsarlak PM, Goethem van JW, Hauwe van den L, Dillen C, Verlooy J, Cosyns P, Schepper de AM. Imaging findings in diffuse axonal injury after closed head trauma. European Radiology 1998; 8: 960-965.
36. Courville, CB. Pathology of the Nervous System, 2nd edition. Mountain View CA: Pacific Press, 1945.
37. Pudenz RH & Shelden CH. The lucite calvarium-a method for direct observation of the brain. Journal of Neurosurgery 1946; 3: 487-505.
38. Owens RG, Ghadiali EJ. Judo as a possible cause of anoxic brain damage. Journal of Sports Medicine and Physical Fitness 1991; 31: 627-628.
39. Rau R, Raschka C, Brunner K, Banzer W. Spectral analysis of electroencephalography changes after choking in judo (juji-jime). Medicine & Science in Sports & Exercise 1998; 30: 1356-1362.
40. Becker DP, Verity MA, Povlishock J, et al. Brain cellular injury and recovery: horizons for improving medical therapies in stroke and trauma. West J Med 1988; 148: 670-684.
41. Faden AI, Demediuk P, Panter S, et al. The role of exitatory amino acids and NMDA receptors in traumatic brain injury. Science 1989; 244: 798-800.
42. Denny-Brown D, Russell WR. Experimental cerebral concussion. Brain 1941; 64: 93-164.
43. Elson LM, Ward C. Mechanisms and pathofysiology of mild head injury. Semin Neurol 1994; 14: 8-18.
44. Gennarelli TA, Thibault LE, Adams Jh. Diffuse axonal injury and traumatic coma in the primate. Ann Neurol 1982; 12: 564-574.
45. Clifton GL, Ziegler MG, Grossman, RG. Circulating catecholamines and sympathetic activity after head injury. Neurosurgery 1981: 8: 10-14.
46. Hamill RW, Woolf PD, McDonald JV, et al. Catecholamines predict outcome in TBI. Ann Neurol 1987: 21: 438-443.
47. Vecht CJ, van Woerkom TCAM, Teelken AW, et al. Homovanillic acid and 5-hydroxyindoleacetic acid cerebrospinal fluid levels. Archives of Neurology 1975; 32: 792-797.
48. Woerkom van TCAM, Teelken AW, Minderhout JM. Difference in neurotransmitter metabolism in fronto-temporal-lobe contusion and diffuse cerebral contusion. Lancet 1977; 1: 812-812.
49. Philips JP, Jones HM, Hitchcock R. Radioimmunoassay of serum creatine kinase BB as index of brain damage after head injury. British Medical Journal 1980; 281: 777-779.
50. Nordby HK, Urdal P. The diagnostic value of measuring creatine kinase BB activity in cerebrospinal fluid following acute head injury. Acta Neurochirurgica 1982; 65: 93-101.
51. Nordby HK, Urdal P, Bjornaes H. The prognosis of patients with concussion and increased creatine kinase BB in cerebrospinal fluid. Acta Neurochirurgica 1984; 71; 205-215.

52. Woertgen C, Rothoerl RD, Holzschuh. Comparison of serial S-100 and NSE measurements after severe head injury. Acta Neurochirurgica 1997; 139: 1161-1165.
53. Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet 1974; 2: 81-84.
54. Stein SC, Spettell C. The head injury severity scale (HISS): a practical classification of closed head injury. Brain Injury 1995; 9: 437-444.
55. Russel WR. Cerebral involvement in head injury. Brain 1932; 55: 549-603.
56. Bigler ED. Neuropathology of traumatic brain injury. In: Bigler ED, Ed. Traumatic brain injury. Austin, Texas, 1990.
57. American Academy of Neurology. Practice parameter: the management of concussion in sports (summary statement). Neurology 1997; 48: 581-585.
58. Hamilton MG, Trammer BI. Nervous system injuries in horse back riding accidents. Journal of Trauma 1993; 34: 227-232.
59. Frank E, Frankel P, Mullins RJ, Taylor N. Injuries resulting from bicycle collisions. Acad Emerg Med 1995; 2: 200-203.
60. Mellion MB. Common cycling injuries. Management and prevention. Sports medicine 1991; 11: 52-70.
61. Calle SC, Eaton RG. Wheels-in-line skating injuries. Journal of Trauma 1993; 35: 946-951.
62. Schieber RS, Branche-Dorsey CM, Ryann GW. Comparison of in-line skating injuries with rollerskating and skateboard injuries. JAMA 1994; 271: 1856-1858.
63. Dencker SV. A follow up study of 128 closed head injuries in twins using co-twins as controls. Acta Psychiatr Neurol Scan 1958; 33: 123-125.
64. Jacobsen J, Baadsgaard SE, Thomsen S, henriksen PB. Prediction of post concussional sequelae by reaction time tests. Acta Neurologica Scandinavia 1987; 75:341-345.
65. Rimel RW, Giordani B, Barth JT, Boll TJ, jane JA. Disability caused by minor head injury. Neurosurgery 1981; 9: 221-228.
66. Klein M. Cognitive aging, attention and mild traumatic brain injury. Thesis, Department of Neuropsychology University Maastricht, 1997.
67. Rutherford WH, Merret JD, Mc Donald JR. Symptoms at one year following concussions from head injuries. Injury 1979; 10: 225-230.
68. Miller JD. Head injury. Journal of Neurology, Neurosurgery and Psychiatry 1993; 56: 440-447.
69. Wrightson P, Gronwall D. Time of work and symptoms after minor head injury. Injury 1980; 12: 445-454.
70. Gronwall D, Wrightson P. Cumulative effect of concussion. Lancet 1975; 2: 995-997.
71. Bruce DA, Alavi A, Bilaniuk L, Dolinskas C, Obrist W, Uzell B. Diffuse brain swelling following head injuries in children: the syndrome of 'malignant brain edema". Journal of Neurosurgery 1981; 54: 170-178.
72. Cantu RC, Voy R. Second impact syndrome: a risk in any contact sport. The Physician and Medicine 1995; 23: 27-34.
73. Carlsson GS, Svardsodd K, Welm L. Long term effects of head injury sustained during life in three male populations. Journal of Neurosurgery 1987; 67: 197-205.
74. Wilberger JE. Minor head injuries in American football: prevention of long term sequelae. Sports Medicine 1993; 15: 338-343.
75. Cook JB. The effects of minor head injury sustained in sport and the post concussional syndrome. In: Walker et al., eds. Delayed effects of head injury. Thomas, Springfield, 1969; 408-413.

76. Barth JT, Macciocchi SN, Giordani B. Neuropsychological sequelae of minor head injury. Neurosurgery 1983; 13: 529-533.
77. Rowe MJ, Carlsson C. Brainstem auditory evoked potentials in post concussion dizziness. Archives of Neurology 1980; 37: 379-683.
78. Levin HS, Amparo E, Eisenberg HM. MRI and CT in relation to neurobehavioral sequelae of mild and moderate head injury. Journal of Neurosurgery 1987; 66: 706-713
79. Robberts AH. Brain damage in boxers. London: PitmannMedical and Scientific, 1969.
80. Robberts GM, Allsop D, Bruton CJ. The occult aftermath of boxing. Journal of Neurology, Neurosurgery and Psychiatry 1990: 53: 221-228.
81. Hollnagel P. Punch drunk syndrome. Ugeskr Laeg 1974; 136: 2871-2874.
82. Saunders RL, Harbaugh RE. The second impact in catastrophic contact-sports head trauma. JAMA 1984; 252: 538-539.
83. Pickles W. Acute general edema of the brain in children with head injuries. New England Journal of Medicine 1950; 242: 607-611.
84. McCrory P, Berkovic SF. Second impact syndrome. Neurology 1998; 50: 677-684.
85. Kelly JP, Nichols JS, Filley CM, Lillehei KO, Rubinstein D, Kleinschmidt-DeMaster BK. Concussion in sports: guidelines for the prevention of catastrophic outcome. JAMA 1991; 266: 2867-2869.
86. McQuillen JB, McQuillen EN, Morrow P. Trauma, sport and malignant cerebral edema. American Journal of Forensic Medical Pathology 1988; 9: 12-15.
87. Colorado Medical Society. Report of the sports medicine comittee: guidelines for the management of concussion in sport (revised). Denver: Colorado Medical Society, 1991.
88. Jenkins LW, Moszynski K, Lyeth BG, et al, Increased vulnerability of the mildly traumatized rat brain to cerebral ischemia: The use of controlled secondary ischemia as a research tool to identify common or different mechanisms contributing to mechanical and ischemic brain injury. Brain res 1989; 477: 211-224.
89. Jenkins LW, Marmarou A, Lewelt W, Becker DP. Increased vulnerability of the traumatized brain to early ischemia. Baetmann A, Go GK. Unterberg (eds): 273-282, 1996.
90. Lee SM, Lifshitz J, Hovda DA et al. Focal cortical impact injury produces immediate and persistent deficits in metabolic auto-regulation (Abstract). J Cereb Blood Flow Metab 1995; 15: 722
91. Lifshitz J, Pinanong P, Le HM, et al. Regional uncoupling of cerebral blood flow and metabolism in degenerating cortical areas following a lateral cortical contusion. Journal of Neurotrauma 1995; 12: 129.
92. Sutton RL, Hovda, DA, Adelson PD, et al. Metabolic changes following cortical contusion. Relationships to edema and morphological changes. Acta Neurochir 1994; 60: 446-448.
93. Hovda DA, Le HM, Lifshitz J, et al. Long term changes in metabolic rates for glucose following mild, moderate and severe concussive head injuries in adult rats. Soc Neurosci 1994; 20: 845.
94. Katayama Y, Becker DP, Tamura T, et al. Massive increases in extracellular potassium and the indiscriminate release of glutamate following concussive brain injury. Journal of Neurosurgery 1990; 73: 889-900.

Chapter 2

Acute traumatic brain injury in amateur boxing*

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Abstract

Objective: Acute traumatic brain injury (ATBI) represents the neurological consequence of concussive and subconcussive blows to the head in millions of athletes annually. Evidence suggests that ATBI may be associated with boxing and collision sports like American football and soccer. The objective of this study was to determine the presence and nature of cognitive impairment due to ATBI. Moreover, all boxers in this study used head gear during their fights and the second objective was to determine if head gear could reduce the risk for ATBI in amateur boxing.
Design: In this inception cohort study thirty-eight amateur boxers were tested before and shortly after a boxing match and were compared to a control group of 28 amateur boxers who were tested before and after a comparable physical test. The participants in this study underwent neuropsychological examination. The main outcome measures were neuropsychological tests proven to be sensitive to cognitive changes incurred in contact and collision sports.
Results: The boxers who competed exhibited an ATBI pattern of impaired performance in planning, attention, and memory capacity when compared to controls.
Conclusions: Participation in amateur boxing matches may adversely affect neurocognitive functioning despite the use of headgear. The neurocognitive impairment resembles cognitive symptoms due to concussions. Guidelines are recommended to reduce the risk for cumulations of ATBI, second impact syndrome (SIS) and difuse cerebral swelling (brain edema).

Keywords: acute traumatic brain injury, boxing, collision sports, cognitive impairment

Introduction

Moderate to high incidences of acute traumatic brain injury (ATBI) are reported in sports with millions of participants world wide e.g., American football, soccer, basketball, baseball, rugby, martial arts and ice-hockey (1-4). ATBI incurred in contact and collision sports can result in functional alterations which vary from transient cognitive impairments to death (5). Evidence of ATBI in contact and collision sports appears as temporary cognitive function loss, knock-outs, amnesia, postconcussion syndrome, subdural hematomas, headaches, and convulsions (5-9).

Off all sports, boxing is associated with a large number of deaths and ATBI (7). Considering the objective of boxing competition to disable ones' opponent and render him nonfunctional, it is no surprise that ATBI can occur in boxing matches and sparring sessions. Jabs and blows to the head at an angle deliver acceleration-deceleration, linear, and rotatory energy vectors that are likely to result in ATBI.
In addition to ATBI, chronic traumatic brain injury (CTBI) is of concern in boxing. CTBI due to contact and collision sports is often an insidious process which might start as mild subclinical dysfunction in the beginning of a career, detected only through neuropsychological investigation (10-12), evolving into the slowed motor performance, tremors, cognitive deficits, personality change as seen in the 'punch drunk syndrome' (PDS), and psychiatric changes (13-17). The amount of ATBI received during the boxing career seems to be directly related to the severity of CTBI (5). This conforms to the model of cumulative effect which states that repeated blows to the head have an additive deleterious effect (18-19).
To get more understanding of ATBI symptomatology the current investigation was undertaken to determine the presence and nature of ATBI, as evidenced by cognitive impairment.

Methods

Subjects

The inception cohort of this investigation consisted of 38 Dutch male amateur boxers and 28 male controls. The boxers who competed were selected at fight nights taking from every boxing bout at random one boxer. The boxers who served as controls were selected from four licensed Dutch boxing teams and frequency matched on boxing level, weight, age and education. All participants were native Dutchmen and educated in the Dutch educational system. Potential participants with a learning disorder, an alcohol consumption of more than five intakes a week, concussions not related to boxing, drug use (soft and hard drugs) and medical conditions which might affect cognitive functioning as reported by their general practitioner were excluced. Data on age, weight, boxing skill and education were obtained in interviews. Weight was classified into three categories: light (40-65kg), middle (65-75kg), and heavy (>75 kg). Education was classified according to the Dutch Secondary Schooling System: LBO (lower-, 10 years), MBO (middle-, 10 years), HBO (higher-, 11 years). Boxing skill was ranked according to the system used by the Dutch Boxing Federation (N, C, B and A ranking). A ranked boxers are the most experienced fighters and won more than 12 matches, N ranked boxers won

fewer than 3 matches. The number of punches to the head was counted by reviewing the score cards of the ringside referees and then averaged. All the competitors used protective headgear during their fights conform to Dutch Boxing Federation regulation.
Comparisons were made between the amateur boxers who competed and the boxer controls who performed a bag punching task (3 times for 3 minutes each time). Trauma index variables regarding the number of matches fought previously, the time after the boxing match before the boxer was tested, impact (weight in combination with the total number of head punches incurred in the match), win - draw - or loss, ranking and special circumstances (knock-outs or technical knock-outs) were also documented. The technical knock outs scored in this study were situations that boxers showed symptoms of distress after being hit by head blows and were stopped by the referee. Distress by hits on the liver or for example stops for eyebrow cuts were not classified as technical knock outs in this study.

Neuropsychological Testing

In the current investigation, neuropsychological tests that had proven sensitivity to cognitive impairment incurred in contact and collision sports (4, 11, 19) were given. Each participant received before and after a boxing match or the bag punching task the same battery of neuropsychological tests administered by a trained psychometrician and an interview by a registered neuropsychologist. All tests were administered according to standardized instructions and procedures. Test responses were scored by a registered neuropsychologist who was blinded to the status (competitor or control) of the participant. The test battery included the following: Categorization Test (20), Paced Auditory Serial Addition Task (21), Digit Symbol (Wechsler Adult Intelligence Scale) (22), Trailmaking Test (A and B) (23), Stroop Test (interference trial, 24), Wechsler Memory Scale (WMS) subtests: Visual Reproduction and Logical Memory (25), and the Puncture Test (26). The Wechsler Memory Scale subtests were administered with an immediate and a 20 minutes delayed interval recall. One of the two subversions of the Logical Memory test (WMS) was administered and for the visual memory part we selected the most complex form of the Visual Reproduction Test of the WMS. The competitors and boxer controls used the same version of these tests before and after competition or physical task. The Categorization Test is a word learning test which contains 9 different elements which can be classified into three groups (fruit, vegetables, trees). The test can be divided in two different cognitive functions: a) word learning, b) planning/organization.

Statistical analysis

Comparisons between the changes in neuropsychological testscores were performed by calculating the differences between the means and with the Student's t-statistic their confidence intervals and one-tailed p values were determined. These comparisons were also performed with a linear regression analysis, adjusting for age, boxing skill, level of education, weight and time interval after excercise. To determine the association between boxing-related variables and change in test scores within the group of boxers who competed, adjusted regression coefficients and their p values were calculated using a mutivariate linear regression model adjusting for the same variables.

Results

Population Characteristics

No significant differences between the two groups was found regarding the level of education ($p=0.67$) and age ($p=0.37$) (see Table 1). The control boxers had a shorter time interval after exercise than did the boxers after their fight ($p=0.053$), this was not a significant difference (Table 1). Most of the boxers who competed were light (47%) and middle weight (32%) boxers. Twenty-one percent of the boxers in this study were heavy weight fighters. Most of the boxers were skilled fighters (N-level 24%, C-level 26%, A-level 50%). Characteristics of the controls are presented in Table 1.

Table 1 Characteristics of boxers who competed and controls.

	Competing boxers N=36	Controls N=28	P-value
Distribution in % over 3 levels of education:			
low - middle - high	40 - 42 - 18	41 - 30 - 30	0.67
Distribution in % over 3 weight categories:			
light - middle - heavy	47 - 32 - 21	44 - 33 - 22	0.83
Distribution in % over 4 levels of boxing skill:			
N - C - B - A	24 - 26 - 0 - 50	26 - 19 - 15 - 41	0.75
Median age (range)	22 (18-30)	22 (16-30)	0.37
Median time after intervention in min (range)		5 (1-20)	4 (1-15) 0.053

Mann-Whitney test

Traumatic Exposure in an Amateur Boxing Match

The median number of head punches sustained during an amateur boxing match was 8 (range 0-31). Sixty-five percent of the boxers received 10 or fewer head punches and 35% of the boxers incurred more than 10 head punches in a match. Concussions with or without loss of consciousness were a frequent occurence in amateur boxing matches. Thirteen percent of the fights ended in a knock-out (concussive state with loss of consciousness) or referee-stops-contest-head (RSC-H) decision (concussive state without loss of consciousness, e.g., when the boxer was not able to continue the fight due to mental impairment).

Neuropsychological testing

Table 2 shows the means of the testscores and their unadjusted differences. All the tests were also analysed using a multivariate linear regression model adjusting for time after match/physical task, level of education, weight category and level of boxing skill. The results differed only marginally. Boxers who competed exhibited more cognitive impairments on tests of planning, attention, and memory compared to controls. On a number of tests significant differences were found e.g., Categorization Test ($p=0.04$), Logical Memory STM ($p<0.001$) , LTM ($p< 0.001$) Visual Reproduction STM ($p<0.001$), LTM ($p=0.003$) and Digit Symbol ($p=0.02$).
No significant differences were found in tests of speed of information processing. (PASAT (3.2 condition, $p=.413$) or attentional efficiancy (Stroop Interference Card: speed $p=0.07$, number of mistakes $p= 0.5$).
Tests requiring fine motor behavior proved to be invalid for this study due to the very large intersubject variability on these tests. Both controls and competitors punched with both hands. The heaviest blows in matches and training are delivered by the dominant hand, which introduced greater intersubject variability in dominant hand performance. As a consequence the standard deviation of the Puncture test (fine motor behavior) is an example. The standard deviation was high for the dominant hand but not for the non dominant hand.

Table 2 Mean change in test score in boxers who competed and in control boxers who performed a physical task, the difference of the changes with its 90% Confidence Interval and one tailed p-value if $p<0.05$.

	Change in scores in competing boxers	controls	Difference in change p-value (90%-CI)
Categorization task			
numbers remembered	- 0.05	0	- 0.05 (-0.67 to 0.57)
number of categories	- 0.08	+0.19	- 0.27 (-0.54 to-0.003) 0.047
PASAT	- 1.22	- 1.17	- 0.06 (-2.27 to 2.16)
Puncture test			
dominant hand	+3.00	+5.31	- 2.31 (-6.41 to 1.80)
non-dominant hand	+5.24	+6.76	- 1.52 (-6.38 to 1.76)
Stroop Interference Card			
number of seconds	- 7.16	- 7.65	+0.91 (-6.48 to 7.46)
number of mistakes	- 0.24	- 0.62	+0.43 (-0.42 to 1.17)
Digit Symbol	+5.66	+9.38	- 3.73 (-6.63 to -0.82) 0.02
Trailmaking Test			
Form A	- 4.42	- 3.63	- 0.79 (-3.24 to 1.66)
Form B	- 9.45	-11.27	+1.82 (-3.59 to 7.23)
Logical Memory (stories)			
subtest WMS, STM	- 1.19	+1.52	- 2.71 (-3.75 to -1.66) < 0.001
subtest WMS, LTM	- 1.17	+1.70	- 2.87 (-3.87 to -1.86) < 0.001
Visual Reproduction			
subtest WMS, STM	- 1.39	+0.04	- 1.43 (-1.49 to -0.88) < 0.001
subtest WMS, LTM	- 1.05	- 0.11	- 0.94 (-1.49 to -0.40) 0.003

Intra-group comparison in competitors

Some traumatic exposure variables have a negative impact on cognitive function. Impact (weight in combination with the number of punches) is reducing the number of categories ($p=0.03$) and number of elements reproduced in a visual memory STM task ($p=0.02$). Knock outs and technical knock outs (RSC-H) reduced the number of elements remembered in a verbal memory task ($p=0.03$). Lower ranked boxers (N and C skilled boxers) showed a slower response in the Stroop reading card ($p=0.01$) compared to A ranked boxers. Remarkable is a better performance of the non-dominant hand in boxers who sustained a knock out ($p=0.04$). No statistical differences in test performance was found between winners, losers or boxers with a draw.

Discussion

There is a high risk for ATBI in amateur boxing competition. Despite the use of protective headgear, the brain is still susceptible to ATBI in amateur boxing. ATBI typically involves impairments in planning, attention, and memory. Our findings are in agreement with those of Critchley (27) who reported memory problems in boxers who sustained an ATBI (knock-out or technical knock-out). Furthermore, Kelly et al (7) documented attention and memory problems as acute symptoms in athletes who sustained concussions with or without loss of consciousness.

In this inception cohort study, several methodological precautions were taken to minimize the problems associated with group comparisons. Cultural and educational differences were eliminated; drug use, more than five alcohol intakes a week, learning disorders and medical conditions that could affect cognitive functioning served as exclusion criteria. Controls were frequency matched on age, boxing skill and weight category thus implicitly controlling for variables concerning previous boxing experience as concussions sustained in previous fights and the number of fights. Furthermore, the influence of possible confounders was limited by using the individual *changes* in cognitive functioning as outcome. Physical activity variables were essentially equated for the two groups of boxers. Assessment and scoring procedures were controlled for expertise and bias. Difference in mental, physical and emotional status could also explain these results. Boxers who competed fought a real match which evokes a 'fight' response in physical, cognitive and emotional systems. It proved to be difficult to control for these arousal variables which would be unlikely to affect the bag punchers.

ATBI in amateur boxing appear to be related to concussions, subconcussive blows, and trivial traumata associated with the impact of head punches. The amateur boxers in the current investigation sustained a median of 8 head punches during the match and 13% of the matches ended in concussions with or without loss of consciousness. Impact (weight in combination with the number of punches) of the blows delivered to the opponent in the match, knock outs and technical knock outs were predictors for cognitive functioning after a match (impairment in memory and planning abillity).

Depression (losing the match) was not an important predictor, this study showed no significant differences in neuropsychological test scores between winners, losers or boxers who boxed a draw. Moreover, cognitive symptoms of ATBI (planning, attention, and memory problems) resemble those of CTBI. Thus it is plausible that athletes who incur repeated ATBI do not fully recover from these injuries and that

this is what might account for the insidious manifestations of CTBI in contact and collision sports athletes.

This study showed a lower level of semantic clustering demonstrating frontal lobe dysfunction. Levin et al (28) showed that patients with frontal lobe lesions revealed a lower level of clustering as compared with both nonfrontal patients and normal controls. In our study the strategy of clustering items according to their semantic relations was underutilized in the competitors.

Our findings, that amateur boxing competition is associated with neuropsychological impairments, supports the hypothesis that ATBI is a serious health problem for amateur boxers. Accordingly, prevention measures should be taken to maximize safety. The referee should stop the match at the first sign of distress and the attending physician should have mandate to stop fights immediately when a boxer shows ATBI symptoms. Concussion programs as advocated by the AAN (29) should be applied on a mandatory basis by the AIBA in amateur boxing to reduce the cumulative effect of ATBI in amateur boxers and to reduce the risk for difuse cerebral swelling and second impact syndrome (SIS). SIS results from acute, usually fatal brain swelling due to recurrent head injuries when symptomatic from a previous ATBI (30-31). Although rare, sports related SIS has a mortality approaching 100% (32-33). If SIS represents a real clinicopathologic entity, then there are potential major consequences for the management of acute brain injuries in amateur and professional boxing. The Standardized Assessment of Concussion (SAC) as proposed by McCrea (34) and the test strategy to detect symptoms of concussion as proposed by Maddocks (35) et al (1995) are reasonable procedures for assessment of ATBI in amateur and professional boxing. As our study shows visual memory tests should be added in the SAC. Visual memory tests are easy to administer and beside visual memory performance it gives the physician an impression of planning capacity.

Conclusion

Participation in amateur boxing matches may adversely affect cognitive functioning (i.e., planning, attention and memory) despite the use of protective headgear. This impairment in neurocognitive function appears to be attributable to the impact and number of head punches and concussions with or without loss of consciousness causing ATBI. Guidelines are recommended to reduce cumulations of ATBI and the risk for SIS.

The duration and course of ATBI symptoms (e.g., attention, memory and planning) could not be documented in this study but are important issues for further research. Whether our findings can be extrapolated to other contact sports like soccer and American football remains to be determined in future investigations.

References

Chapter 2

1. Jordan B. Sports Injuries. In: Proceedings of the mild brain injury in sports Summit. Dallas, TX: National Athletic Trainers' Association, Inc., 1994; 43-46.
2. Ommaya A, Ommaya A and Salazar A. A spectrum of mild brain injuries in sports. In: Proceedings of the mild brain injury in sports Summit, Dallas, TX: National Athletic Trainers' Association, Inc., 1994; 72-80.
3. Boden B, Kirkendall D, Garrett W. Concussion incidence in elite college soccer players. American Journal of Sports Medicine 1998; 26: 238-241.
4. Matser J, Kessels A, Jordan B, Lezak M, Troost J. Chronic Traumatic Brain Injury in Professional Soccer Players. Neurology 1998; 51: 791-796
5. Jordan B. Boxing. In: Jordan B, Tsairis P, Warren R, eds. Sports Neurology, second edition. Philadelphia: Lippincott-Raven 1998; 351-367.
6. McCunney R, Russo P. Brain injuries in boxers. The Physician and Sportsmedicine 1984; 12: 5367.
7. Kelly JP, Rosenberg JH. Diagnosis and management of concussion in sports. Neurology 1997; 48: 575-580.
8. Evans R. The post concussion syndrome and the sequelae of mild head injury. Neurology Clinics 1992; 10: 815-847.
9. McCrory P, Bladin P, Berkovic S. Retrospective study of convulsions in elite Australian rules and rugby league footballers: phenomenology, etiology and outcome. British Medical Journal 1997; 24: 25-38.
10. Drew R, Templar D, Schuyler H, Newell T, Cannon W. Neuropsychological deficits in active licensed professional boxers. Journal of Clinical Psychology 1986: 42: 520-525.
11. Matser JT, De Bijl MAO, Luijtelaar ELJ. Is amateurboxing dangerous? De Psycholoog 1992; 12: 515-521.
12. Gronwall D, Wrightson P. Delayed recovery of intellectual function after minor head injury. Lancet 1974; 14: 605-609
13. Roberts A. Brain damage in boxers. London:Pitman Pubishing, 1969
14. Martland H. Punch drunk. JAMA 1928; 91: 1103-1107.
15. Johnson J. Organic psychosyndromes due to boxing. British Journal of Psychiatry 1969; 115: 4553.
16. Haglund Y, Eriksson E. Does amateur boxing lead to chronic brain damage? A review of some recent investigations. American Journal of Sports Medicine 1993; 21: 97-109.
17. Mendez M. The neuropsychiatric aspect of boxing. Int J Psychiatry Med 1995; 25: 249-262.
18. Gronwall D, Wrightson P. Cumulative effect of concussion. Lancet 1975; 22: 995-997.
19. Jordan B, Matser J, Zazula T. Sparring and cognitive functioning in professional boxers. The Physician and Sportsmedicine 1996; 24: 87-98.
20. Categorization Task. In: Kolb B, Whishaw I. Fundamentals of Human Neuropsychology. New York: Freeman and Company 1990; 451.
21. Gronwall D. Paced Auditory Serial Addition Task: A measure of recovery from concussion. Perceptual and Motor Skills 1977; 44: 367-373.
22. Wechlser D. WAIS-R Manual New York: The Psychological Corporation, 1981
23. Spreen O, Strauss E. A compendium of neuropsychological tests. New York: Oxford University Press, 1991.

24. Golden CJ. Stroop Color and Word Test. Chicago: Stoelling, 1978.
25. Wechsler D. Wechsler Memory Scale Manual. San Antonio: The Psychological Corporation, 1974.
26. Van der Vlugt H. Lateralisaties van hersenfuncties: een neuropsychologisch onderzoek naar de relatie tussen handvoorkeur en de relatie van de taalfunctie. Lisse: Swets & Zeitlinger, 1979.
27. Critchley M. Medical aspects of boxing, particularly from a neurological standpoint. British Medical Journal 1957; 1: 357-362.
28. Levin H, Goldstein, F, Williams D, Eisenberg H. The contribution of frontal lesions to neurobehavioral outcome of closed head injury. In: Levin H, Eisenberg H, Benton A, eds. Frontal lobe function and dysfunction. New York: Oxford University Press, 1991; 318-338.
29. Quality Standards Subcommittee. Practice Parameter: The management of concussion in sports. Neurology 1997; 48: 581-585.
30. Kelly JP, Nichols JS, Filley CM, Lillehei KO, Rubinstein D, Kleinschmidt-DeMaster BK. Concussion in sports: guidelines for the prevention of catastophic outcome. JAMA 1991; 266: 2867-2869.
31. Cantu RC, Voy R. Second impact syndrome: a risk in any contact sport. The Physian and Sportsmedicine 1995; 23: 27-34.
32. McQuillen JB, McQuillen EN, Morrow P. Trauma, sport and malignant cerebral edema. American Journal of Forensic Medical pathology 1988; 9: 12-15.
33. Saunders RL, Harbaugh RE. The second impact in catastrophic contact-sports head trauma. JAMA 1984; 252: 538-539.
34. McCrea M, Kelly MD, Kluge J, Ackley B, Randolph C. Standardized assessment of concussion (SAC) in football players. Neurology 1997; 48: 586-588.
35. Maddocks DL, Dicker GD, Saling MM. The assessment of orientation following concussion in athletes. Clin J Sports Med 1995; 5: 32-35.

Chapter 3

Neuropsychological impairment in amateur boxers*

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* Modified from Matser JT, De Bijl MAO, Luijtelaar G. Is amateurboxing dangerous? De Psycholoog 1992; 12: 515-521.

Introduction

There is little empirical evidence that amateur boxing can cause brain damage. The few studies in amateur boxers published yet (1-3) show no association between boxing and neurologial dysfuctions. However, the research done on professionals and ex-professionals suggest a positive correlation between the duration of the boxing career and the intensity of cerebral damage, particularly temporal and frontal brain damage (4-7). Furthermore, most research lacks a neuropsychological evaluation specific for symptoms associated with traumatic brain injury. In the past few years it is becoming clearer that these subtle cognitive defects, which can not always be recognized by means of a neurological examination, can be detected with neuropsychological assessment (8). In the present research it will therefore be examined, by means of neuropsychological research, if amateur boxers show signs of brain damage. It will in particular be examined whether a dose (number of matches)-response (intensity of impairment) relation can be detected. The choice of tests is particularly motivated by the literature on closed head injury (9).

Methods

Subjects

All boxers participating in this study, were members of a boxing club affiliated to the Dutch Boxing Federation (NBB). A doctor member of the Medical Commission of the NBB approached a number of coaches with the request to ask their pupils to participate. The coaches were also asked to select boxers who fought from 5 to 16 matches (16 boxers) and equal to or more than 30 matches (17 boxers). The boxers were asked if they could bring a non-boxing friend on the day of the tests, who were used as a control (n=6). The other controls (n=10) were recruited from the new members of the local boxing club who had never sparred before. None of the controls had a prehistory of brain damage or drug abuse. The three groups were matched on age and education. All participants were native Dutchmen and educated in the Dutch system. Potential participants with a learning disorder, an alcohol consumption of more than 5 intakes a week, concussion not related to boxing, medical conditions which might affect cognitive functioning as reported by their general practitioner were excluded.

Of all participants age, education (1 stands for highschool only and a 6 for academic or higher vocational education level) and if applicable number of matches (lost, draw or won) and duration of boxing career, were registered.

Neuropsychological tests

The test battery consisted of a few subtests of the WAIS (intelligence test) (10), the Wisconsin Card Sorting Test (measures a form of abstract thinking, particularly the exchange of solving strategies) (11), the word fluency test of the Groninger Intelligence Test (measures the speed and facility of verbal production and the long-term memory) (12), the Wechsler Memory Scale I WMS) (with which various aspects of memory can be tested and word associations can be learned and memorized) (13). The Complex Figure Test (short and long-term memory for visual-spacial information) (14), attention and concentration tasks (the Trail Making A and B (15) and the Stroop Color Word Test) (15), the Figure Detection test of the Groninger Intelligence test (perceptual observation and memory) (12) and the Puncture test (fine motor behavior) (16). The Complex Figure Test consists of three phases. First the boxer is asked to redraw a map, this is to measure the perceptual organization. After completing the task, unexpectedly, the boxer is asked to reproduce the figure, however without the original (STM visual memory). Twenty minutes later the boxer is asked again to draw the figure (LTM visual memory). The majority of the tests used are explained in Lezak (9).

Statistical analysis

The differences between the three groups have been tested with variance analysis with the number of matches (0, from 5 to 16 and more than 29 matches) as factor. If a significant effect was found, the differences between the three groups were tested with a post hoc test according to Scheffé. Also, the 25th percentile score of each test was calculated.

Table 1 Boxing experience, age and education.

Number of boxing matches	0	5-15	>30
Age	24.0 ± 4.8	22.8 ± 4.6	24.1 ± 4.5
Number of boxing matches			
Won	-	6.5 ± 2.5	36.5 ± 8.6
Lost	-	3.2 ± 2.2	13.7 ± 8.6
Draw	-	0.6 ± 0.9	4.2 ± 2.6
Boxing experience in years	-	5.2 ± 2.4	8.6 ± 3.0
Education (according to Verhage)	5.5 ± 1.8	5.1 ± 2.2	5.2 ± 2.2

Results

The three groups did not differ significantly in age and education. The Complex Figure Test (immediate and delayed recall) showed differences in memory performance between the three groups. Nil, four and nine controls from respectively the control group, the 5 to 16 matches group and the more than 29 matches group performed under the 25th percentile score; boxers who boxed more than 29 matches performed significantly worse on these tests than the control group. Also on the Stroop card 2 (name colors) boxers with more than 29 matches scored significantly worse than the control group. The ANOVA showed significant differences in the Trail Making A test scores.

Discussion

These findings suggests that participating in amateur boxing may be associated with neuropschological impairment. A relationship was (found between an increasing number of boxing matches and a decline in memory and attention performance.
In our study, several methodologic precautions were taken to minimize the problems associated with group comparisons. All the participants in this study were native Dutchmen and educated according to the Dutch educational system therefore eliminating misinterpretations caused by educational differences. In addition, the three groups were matched on age and education and potential participants with a history of drug and alcohol abuse, learning disorders and neurological complaints not caused by boxing were excluded.
The Complex Figure Test (immediate and delayed recall), Trailmaking A and the Stroop test (the colour naming card) detected symptoms of neurocognitive impairment in amateur boxers. An increasing number of boxing matches was negatively associated with scores on these tests. These findings are in agreement with those of Matser et al (17) who showed a higher degree of memory impairment in association with a greater exposure time in sparring. This memory impairment can be related to diffuse brain damage as well as to the right posterior temporal lobe pathology (18). In addition, the copying score on the Complex Figure Test did not differ significantly between the groups suggesting that the visual organisation of the boxers was not abnormal.

Table 2 Psychometric performance of controls, boxers with 5 up to 16 matches and boxers > 29 matches. The mean with the standard deviation is shown.

Number of boxing matches	0	5-15	>30
WAIS subtest			
Digit symbol	59.6 ± 11.4	54.2 ± 6.2	52.7 ± 8.6
Similarities	19.9 ± 4.2	17.1 ± 6.2	16.7 ± 5.6
Block Design	20.9 ± 6.1	20.3 ± 6.1	17.9 ± 5.5
Vocabulary	34.3 ± 9.3	32.9 ± 12.1	27.8 ± 12.2
Complex figure test			
Copy	35.4 ± 0.8	34.2 ± 3.2	34.0 ± 1.7
Immediate recall	23.3 ± 3.3	20.3 ± 7.1	15.8 ± 7.4
Wechsler memory scale*			
Visual memory	15.4 ± 0.8	17.5 ± 1.0	17.3 ± 0.8
Logical memory A	7.2 ± 1.8	8.0 ± 2.6	8.1 ± 3.6
Logical memory B	6.1 ± 2.7	6.5 ± 3.1	8.2 ± 3.5
Digits (forward and backwards)	11.1 ± 1.2	10.9 ± 1.0	9.4 ± 1.1
Mental control	8.2 ± 0.4	7.5 ± 0.5	7.6 ± 0.5
Associate learning	16.2 ± 3.3	17.5 ± 3.4	16.9 ± 3.2
Fluency (GIT)			
Animals	29.8 ± 8.1	27.4 ± 6.2	26.3 ± 5.4
Professionals	23.2 ± 5.0	23.2 ± 6.2	22.4 ± 6.6
Trailmaking			
A	20.6 ± 5.3	26.1 ± 8.8	26.7 ± 7.4
B	58.8 ± 29.6	64.2 ± 16.1	66.8 ± 15.0
Stroop			
Card 1 (reading)	40.7 ± 7.1	43.7 ± 5.9	44.7 ± 4.7
Card 2 (color naming)	52.7 ± 9.3	60.2 ± 8.6	61.9 ± 9.1
Card 3 (interfence)	86.5 ± 21.9	93.1 ± 15.6	100.5 ± 18.5
Wisconsin card sorting test			
Number of shifts	4.7 ± 1.4	5.2 ± 1.2	4.1 ± 1.5
Figure detection score	15.9 ± 2.4	16.2 ± 3.0	15.4 ± 2.7
Puncture test			
Dominant hand	98.5 ± 5.3	96.5 ± 5.6	94.1 ± 7.9
Non-dominant hand	74.9 ± 31.7	76.4 ± 22.3	72.4 ± 21.1

* Total sum of all scales (included delayed recall)
† significant difference according to Scheffé between >29 and control group ($p<.01$)
‡ significant difference according to Scheffé between >29 and control group ($p<.05$)
§ significant experience effect according to the ANOVA analysis.

It was remarkable that boxers with a high number of matches only performed badly on the complex memory tasks (Complex Figure Test) and not on the simple memory tasks (WMS). This seems to correspond with the results published by Brooks (19). This research group did not find significant differences between amateur boxers and a control group on scores of the WMS subtests.
The results of the Trailmaking A and the Stroop Card 2 indicate that boxers with more than 29 matches are obstructed by attention and concentration problems. However, these results can also be interpreted as a starting problem indicative for frontal lobe pathology or brain damage in general. In this explanation the organization of a cognitive schedule is delayed (20) and the patient needs more time to organize responses that are needed for the realization of a neuropsychological test.
This study demonstrates that amateur boxers, in the long run, sustain neuropsychological impairments. Prevention guidelines should be developed by the official boxing bodies (EABA, AIBA) to protect amateur boxers for cognitive deterioration.

References

Chapter 3

Neuropsychological impairment in amateur boxers

1. Erikson E. Does amateurboxing cause brain damage? Circular nr 12 of the European Amateur Boxing Association (EABA), 1984.
2. Levin HS, Lippold SC, Goldman A. Neurobehavioral functioning and magnetic resonance imaging in young boxers. Journal of Neurosurgery 1987; 67: 657-667.
3. Jordan BD, Zimmerman RD. Magnetic resonance in amateur boxers. Archives of Neurology 1988; 45: 1207-1208.
4. Jordan BD, Zimmerman RD. Computer tomography and magnetic resonance imaging comparisons in boxers. Journal of the American Medical Association 1990; 263: 1670-1674.
5. Casson IR, Siegel O, Sham R, Campbell EA, Tarlau M, Dimomenico A. Brain damage in modern boxers. Journal of the American Medical Association 1984; 251: 2663-2667.
6. Ross RJ, Cole M, Thompson JS, Kim KH. Boxers, computed tomography, EEG and neurological evaluation. Journal of teh American Medical Association 1983; 249: 211-213.
7. Robberts AH. Brain damage in boxers. London: Pitman Medical Scientific Publishing Co, 1969.
8. Van Zomeren AH. Reaction time and attention after closed head injury. Lisse: Swets & Zeitlinger, 1981.
9. Lezak MD. Neuropsychological Assessment. New York: Oxford University Press, 1983.
10. Wechlser D. WAIS-R manual New York: The Psychological Corporation, 1981.
11. Berg EA. A simple objective treatment for measuring flexibility in thinking. Journal of General Psychology 1948; 39: 15-22.
12. Luteyn F. een nieuwe verkorte GIT. Dutch Journal of Psychology 1966; 2: 675-682.
13. Wechlser D. Wechlser Memory Scale manual. San Antonio: The Psychological Corporation, 1974.

14. Osterieth PA. Le test de copie d'un figure complexe. Archives de Psychologie 1944; 30: 206-356.
15. O, Strauss E. A compendium of neuropsychological tests. New York: Oxford University Press, 1991.
16. Van der Vlugt H. lateralisaties van hersenfuncties: een neuropsychologisch onderzoek naar de relatie tussen handvoorkeur en de relatie van de taalfunctie. Lisse: Swets & Zeitlinger, 1979.
17. Matser JT. de Bijl MAO, Luijtelaar ELJ, Jordan BD. Neuropsychological functioning in active amateur and professional boxers. Paper, 20th INS meeting, San Diego, USA, 1992.
18. Kolb B, Whisaw IQ. Fundamentals of Human Neuropsychology. New York: Freeman and Co, 1985.
19. Brooks N, Kupshik G, Wilson L, Galbraith S, Ward R. A neuropsychological study of active amateur boxers. Journal of Neurology, Neurosurgery and Psychiatry 1987; 50: 997-1000.
20. Tromp E, Mulder T. Slownesss of information processing after traumatic head injury. Journal of Clinical and Experimental Neuropsychology 1991; 13: 821-830.

Chapter 4

Neuropsychological impairment in amateur soccer players*

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Abstract

Context: Soccer players incur concussions during matches and training sessions, as well as numerous subconcussive blows to the head from impacts with the soccer ball (headers). The combination of soccer-related concussions and the number of headers may be a risk for chronic traumatic brain injury (CTBI).
Objective: To determine whether soccer players have evidence of CTBI.
Design, Setting, and participation: Cross-sectional study of 33 amateur soccer players and 27 amateur athletes involved in swimming and track (controls) in the Netherlands who underwent interviews and neuropsychological testing.
Main Outcome Measures: Performance of soccer players vs controls on 16 neuropsychological tests having 27 outcomes.
Results: Compared with control athletes, amateur soccer players exhibited impaired performance on tests of planning (39% vs 13%; P=.001) and memory (27% vs 7%; P=.004). Among soccer players, 9 (27%) had incurred 1 soccer-related concussion and 7 (35%) had had 2 to 5 concussions during their career. The number of concussions incurred in soccer was inversely related tot the neuropsychological performance on 6 of the neuropsychological tests.
Conclusions: Our results indicate the participation in amateur soccer in general and concussion specifically is associated with impaired performance in memory and planning functions. Due to the worldwide popularity of soccer, these observations may have important public health implications.

Key words: Chronic Traumatic Brain Injury, CTBI, Soccer

Introduction

Chronic traumatic brain injury (CTBI) is the cumulative long-term neurologic consequence of repetitive concussive and subconcussive blows to the head, and has been described primarily among professional boxers (1-4). Evidence suggests that CTBI also may be associated with professional soccer (5-6). Neuropsychological impairment and neurological abnormalities have been observed in retired amateur and active professional soccer players (7-11). A survey of active soccer players reported that neurological symptoms correlated with reported history of acute head injury received during play (12). To date, only 1 preliminary investigation evaluated

neuropsychological functioning in nonprofessional high school soccer players (13) The current investigation was undertaken to determine if neuropsychologica dysfunction occurs in amateur soccer players.

Methods

Between September 1997 and May 1998, athletes from 3 complete teams of different regional-league amateur soccer clubs (n=33) were compared with 2 control athletes. The control athletes were middle-distance runners (2 complet premier-league track teams) and middle distance swimmers (1 complete premier league team) from the same region as the soccer clubs. All participants completed ar interview that inquired about age, education, lifetime number of general anesthesias occupational history, learning disorders, life-time number of concussions (sports and nonsports-related), alcohol intake, drug use, and medical conditions that migh affect cognitive functioning. The primary physicians for all participants were interviewed regarding the number of concussions athletes had incurred and medica conditions that might interfere with cognitive functioning. All participants in this study were native to the Netherlands and educated in the Dutch educational system Athletes also excluded for history of drug use, learning disorders, epilepsy, and othe medical conditions that might affect cognitive functioning. Education was scored or a 7-point scale and because all participants completed their secundary schoo education, only the level 3 (technical and vocational training for ages 12-16 years) to level 7 (academic) were used. This study was approved by the St. Anna Hospita Medical Ethical Committee and oral informed consent was obtained from al particiapants.

Each participant was interviewed by a neuropsychologist and receivec neuropsychological tests administered by an experienced psychometrician. All tests were administered according to standardized instructions and procedures. The test data were scored by a neuropsychologist who was blinded to the status of the participant. The neuropsychological tests included the Raven Progressive Matrices (14), Wisconsin Card Sorting Task (15), Paced Auditory Serial Addition Task (16). Digit Symbol Modalities test (17), Trail-making A and B (18), Stroop test (19). Bourdon-Wiersma test (20), Wechsler Memory Scale (21); (Associate Learning Logical Memory, and Visual Reproduction subtests), Complex Figure Test (22), 15-Word Learning Test (23), Benton Facial Recognition Test (24), Figure Detection Test (25), Verbal Fluency Test (25) and the Puncture Test (26).

The means of the neuropsychological test scores were compared using the t test, with 1-tailed P values. The differences, their confidence intervals, and 1-tailed P values were determined with a linear regression analysis adjusting for level of education, alcohol intake, number of general anesthesias, and number of concussions not due to soccer play.
Multiple end point testing was controlled for by clustering the 27 test scores into 8 cognitive functions as described by Lezak (27). For each cluster, a global null hypothesis stating that all differences of the included test scores are 0 was tested using the ordinary least squares (OLS) test (28-29). For the 8 resulting one-tailed P values, a Bonferroni correction for the significance level was applied. In addition, the means of the neurocognitive tests scores among the soccer players were analyzed. Soccer players with and without soccer-related concussions were compared using the t test.

Results

Soccer players were similar to controls in age, education, nonsoccer related concussions, and number of times they had recieved general anesthesia (Table 1). The soccer players exhibited a higher frequency of alcohol intake per month compared with controls.

Table 1 Characteristics of soccer players and controls.

	Soccer players	Controls	2 tailed P value
Number	33	27	...*
Age: mean (sd)	24.9 (4.2)	24.5 (4.5)	0.95'
Level of education: median (range)'	5 (4-6)	5 (3-7)	0.53'
Number of alcohol intakes per month: median (range)	50 (0-99)	28 (0-99)	0.02'
Number of general anaesthesias: median (range)	1 (0-4)	0 (0-4)	0.11'
Percentage of persons with one concussion not sustained in soccer"	27.3	14.8	0.55^

* Ellipses indicate data not applicable. ' Data were calculated using the t test.
' Education level is based on a Dutch 7-point scale.
' Data were calculated using the Mann-Whitney test.
^ Data were calculated using the Fisher exact test.
" No subject experienced more than 1 concussion in their lifetime.

The average duration of an amateur career was 17 years. Thirty-three percent played 5 to 15 years and 67% played more than 16 years. The average time spent in training practices was 3.6 h/wk and the median of the number of matches played annually was 36 (range 20-70). The median number of balls headed in a match was 8.5 (range, 0-20). Nine players (27%) incurred 1 soccer-related concussion; 7 players (23%) reported 2 to 5 concussions in a career.

Amateur soccer players exhibited impairment in planning (OLS P=.001) and memory (OLS P=.004) (Table 2). These scores remained statistically significant after Bonferroni correction. After adjusting for concussions unrelated to soccer, alcohol intake, level of education, and number of general anesthesias, soccer players performed significantly poorer (1-tailed P<.05) on the Complex Figure Test (Immediate Recall [P=.03]); Digit Span Test (Forward [P=.01] and Backward [P=.01]); and Logical Memory (P=.01), Visual Reproduction (P=.04), and Associate Learning (P=.03) subtests of the Wechsler Memory Scale.

Concussions incurred in soccer were inversely correlated with performances on Digit Span Test (Forward) (P=.004), Facial Recognition Task (P=.001), Complex Figure Test (Immediate Recall) (P=.04), Complex Figure Test (Delayed Recall) (P=.03), Digit Symbol Modalities Test (P=.03), and Logical Memory subtest of the Wechsler Memory Scale (P=.006).

Table 2 Psychometric performance of controls and amateur soccer players*.

Neuropsychological test	Mean controls	Mean soccer players	Adjusted difference (95%-CI)
Planning *OLS-test: P=.001*[†]			
Wisconsin Card Sortin Task			
number of perseverations	4.26	6.09	3.35 - 1.04 to 7.74
number of shifts	4.61	3.76[‡]	-0.51 - 1.67 to 0.65
Complex Figure Test copy	35.30	34.61[‡]	-0.57 - 1.50 to 0.36
Mental Speed *OLS-test: P=.08*			
Bourdon-Wiersma test:			
Number of seconds	602.12	617.61	17.98 - 41.2 to 77.2
PASAT	3.68	5.67	2.14 - 1.61 to 5.88
Attention *OLS-test: P=.10*			
Bourdon-Wiersma test:			
Number of mistakes	6.00	5.03	- 1.63 - 5.19 to 1.92
Stroop I	39.64	37.91	-1.65 - 5.38 to 2.08
Stroop II	51.40	50.30	-0.18 - 4.08 to 3.73
Stroop III	77.76	82.24	4.09 - 4.82 to 13.00
Trail-making A	20.85	19.52	-2.14 - 6.34 to 2.06
Trail-making B	49.68	40.45	-11.52 - 19.39 to -3.65
Digit Symbol Modalities Test	68.47	69.42	-4.02 - 13.98 to 5.93
Digits Backwards	5.38	5.00	-0.72[‡] - 1.34 to -0.11
Digits Forward	6.50	5.79[‡]	-0.68[‡] - 1.28 to -0.08
Memory *OLS-test: P=.004*[†]			
Complex Figure Test STM	26.37	23.58[‡]	- 3.52[‡] - 7.18 to 0.13
Complex Figure Test LTM	24.93	22.82[‡]	- 2.56 - 5.76 to 0.64
Wechsler Memory Scale			
Associate Learning	27.48	26.38	- 2.14[‡] - 4.43 to 0.15
Logical Memory	19.04	16.64[‡]	- 2.80[‡] - 6.08 to 0.49
Visual Reproduction	11.00	10.00[‡]	- 0.92[‡] - 1.96 to 0.18
15-Word Learning Test	51.46	53.67	0.07 - 7.61 to 7.75
Abstract Reasoning *Student's t-test: P=.48*			
Raven Progressive Matrices	53.56	54.09	-0.21 - 2.82 to 2.40
Fine Motor Behavior *OLS-test: P=.01*			
Puncture Test nondominant hand	75.26	82.61	5.07 - 1.43 to 11.56
Puncture Test dominant hand	93.26	96.49	0.36 - 3.18 to 3.89
Visual Perceptional Skills *OLS-test: P=.22*			
Benton Facial Recognition Test	22.96	22.73	-0.98 - 2.35 to 0.39
Figure Detection test	16.46	16.03	-0.90 - 2.23 to 0.43
Verbal Fluency *OLS-test: P=.13*			
Verbal Fluency Test, animals	29.11	30.23	2.43 - 1.27 to 6.12
Verbal Fluency Test, professions	21.26	22.90	0.60 - 3.16 to 4.35

* CI indicates confidence interval; OLS, ordinary least squares; PASAT, Paced Auditory Serial Addition Task; STM, short-term memory; and LTM, long-term memory.

† Tailed P< .05. ‡ 1-Tailed P<.006 (Bonferroni correction).

Comment

These findings suggests that participation in amateur soccer may be associated with mild CTBI, as evidenced by impairment in cognitive functioning based on tests of memory and planning. Concussions incurred in amateur soccer may play a fundamental role in the development of cognitive impairment observed in these players.

As reported in our previous study of professional soccer players (11), amateur soccer appears to be associated with impairments in planning and memory functions. The Complex Figure Test immediate recall (memory) and the Wisconsin Card Sorting Task (planning) were sensitive in detecting symptoms of neurocognitive impairment in amateur soccer players. According to standards used in clinical practice (27), 7% of controls and 27% of amateur soccer players showed moderately to severely impaired scores on the memory test and 13% of controls and 39% of amateur soccer players showed moderately to severely impaired scores on the planning test. The neurological effects of amateur soccer appear to be related to soccer-related concussions caused by contact trauma. Most of the memory tests that showed a significant difference between the soccer players and the controls were inversely related with concussions incurred in soccer in the intragroup analysis.

In conclusion, participation in amateur soccer is associated with decreased performance on tests of memory and planning. Although the cognitive impairment appears to be mild, it presents a medical and public health concern, with 200 million Federation International Football Association-registered soccer players worldwide. Methods for suveillance and prevention should be developed and adopted to maximize safety.

References

Chapter 4

1. Critchley M. Medical aspects of boxing, particularly from a neurological standpoint. British Medical Journal 1957; 1: 357-362.
2. Robberts AH. Brain Damage in Boxers, London: Pittman Medical and Scientific Publishers Company LTD, 1969.
3. Jordan BD. Neurologic Aspects of Boxing. Archives of Neurology 1987; 44: 453-459.
4. Mendez MF. The neuropsychiatric aspects of boxing: International Journal of Psychiatry in Medicine 1995; 25: 249-62.
5. Matser JT. Sponzen en aspirines, voldoende bij hoofdletsels in sport?. Geneeskunde en Sport 1996; 29: 66-73.
6. Corsellis JA. Brain damage in sport. Lancet 1976; 1: 401-402.
7. Tysvaer AT, Lochen EA. Soccer injuries to the brain. A neuropsychological study of former soccer players. The American Journal of Sports Medicine 1991; 19: 56-60.
8. Sortland O, Tysvaer AT. Brain damage in former association football players. An evaluation by cerebral computed tomography. Neuroradiology 1989; 31:44-489
9. Tysvaer AT, Storli O, Bachen NI. Soccer injuries to the brain. A neurologic and electro-encephalographic study of former players. Acta Neurologica Scandinavica 1989b; 80: 151-156.
10. Tysvaer AT, Storli, O. Association Football Injuries to the Brain, A Preliminary Report. British Journal of Sports Medicine 1981; 15: 163-166.
11. Matser JT, Kessels AGH, Jordan BD, Lezak MD, Troost J. Chronic Traumatic Brain Injury in Professional Soccer Players. Neurology 1998;51:791-796.
12. Jordan SE, Green GA, Galanty H., Mendelbaum BR, Jabour AJ. Acute and Chronic Brain Injury in United States National Team Soccer Players. The American Journal of Sports Medicine 1996; 24: 205-210.
13. Witol A, Webbe F. (1994). Neuropsychological Deficits Associated with Soccer Play. Archives of Clinical Neuropsychology 1994; 9: 104.
14. Raven JC. Guide to the Standard Progressive Matrices. London: HK Lewis, 1960.
15. Berg EA. A simple objective treatment for measuring flexibility in Thinking. Journal of General Psychology 1948; 39: 15-22.
16. Gronwall DMA. Paced Auditory Serial Addition Task: A measure of recovery from concussion. Perceptual and Motor Skills 1977; 44: 367-373.
17. Wechsler D. WAIS-R Manual. New York: The Psychological Corporation, 1981.
18. Army Individual Test Battery. Manual of Directions and scoring. Washington DC: War Department Adjutant General's Office, 1944.
19. Golden CJ. Stroop Color and Word Test> Chicago, IL: Stoelling, 1978.
20. Van de Loo L. Enkele beschouwingen over de Bourdon-Wiersma test. Tijdschrift van de psychologische kring van de Nijmeegse Universiteit 1956; 2: 33-46.
21 Wechsler D. Wechsler Memory Scale manual. San Antonio, TX: The Psychological Corporation, 1974.
22. Osterieth PA. Le test de copie d'une figure complexe. Archives de Psychologie 1944; 30: 206356.
23. Deelman BG, Brouwer WH, van Zomeren A.H. et al. Functiestoornissen na trauma capitis. In: Jennekens-Schinkel A, et al., Eds. Neuropsychologie in Nederland. Deventer: Van Loghum Slaterus, 1980.

24. Benton AL, Hamsher K, Varny NR, Spreen O. Contributions to neuropsychological assessment. New York: Oxford University Press, 1983.
25. Luteyn F. Een nieuwe verkorte GIT. Dutch Journal of Psychology 1966; 2: 675-682.
26. Van der Vlugt H. Lateralisaties van hersenfuncties: een neuropsychologisch onderzoek naar de relatie tussen handvoorkeur en de relatie van de taalfunctie. Dissertatie, Lisse, Swets & Zeitlinger, 1979.
27. Lezak MD. Neuropsychological Assessment 3rd ed. New York: Oxford University Press, 1995
28. O'Brien PC. Procedures for comparing samples with multiple endpoints. Biometrics 1984; 40: 1079-1087.
29. Läuter J. Exact T and F tests for analyzing studies with multiple endpoints. Biometrics 1996; 52: 964-970.
30. Kelly JP, Rosenberg JH. Diagnosis and management of concussion in sports. Neurology 1997; 48: 575-580.

Chapter 5

Chronic traumatic brain injury in professional soccer players*

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Abstract

Objective: To determine the presence of chronic traumatic brain injury in professional soccer players.
Methods: Fifty-three active professional soccer players from several professional Dutch soccer clubs were compared with a control group of 27 elite noncontact sport athletes. All participants underwent neuropsychological examination. The main outcome measures were neuropsychological tests proven to be sensitive to cognitive changes incurred in contact and collision sports.
Results: The professional soccer players exhibited impaired performances in memory, planning, and visuoperceptual processing when compared with control subjects. Among professional soccer players, performance on memory, planning, and visuoperceptual tasks were inversely related to the number of concussions incurred in soccer and the frequency of "heading" the ball. Performance on neuropsychological testing also varied according to field position, with forward and defensive players exhibiting more impairment.
Conclusion: Participation in professional soccer may affect adversely some aspects of cognitive functioning (i.e., memory, planning and visuoperceptual processing).

Key words: Soccer - Chronic Traumatic Brain Injury - Cognitive Impairment - Heading

Introduction

Worldwide, soccer is the most popular and frequently played sport with at least 200 million registered participants (1). Although soccer is considered safe by the general public, the American Academy of Pediatrics has classified soccer as a contact/collision sport (2). Accordingly, acute traumatic brain injury (ATBI) is a concern in soccer. It is notable that the concussion rates per 1,000 athlete exposures in American football and men's soccer are equal (3). Furthermore, fatal neurologic injuries in soccer have also been reported. During the period from 1931 to 1974, 26 English soccer players experienced fatal traumatic brain injuries, including 8 attributed to "heading" the ball (4). In the United States, from 1979 to 1993, 18 fatal accidents were reported from players running into goalposts (5). Another four fatal

accidents in high school soccer have also been documented between 1980 and 1988 (6).

In addition to ATBI, chronic traumatic brain injury (CTBI) is of concern in soccer. CTBI, which represents the long-term cumulative neurological consequence of concussive and subconcussive blows to the head, has been described primarily among professional boxers (7-10). However, it has been speculated that CTBI can occur in soccer (11). In a survey of British neurologists, a traumatic encephalopathy resembling that encountered in boxing has been reported in soccer players (12). Tysvaer and Lochen (13), Sortland and Tysvaer (14), and others have observed neuropsychological impairment (13), CT pathology (14), and EEG abnormalities (15) among retired soccer players in Norway. In contrast, a more recent investigation (16) of active soccer players on the United States national team failed to demonstrate any significant neurological symptomatology (MRI) when compared with control subjects. There are two distinct mechanisms by which CTBI can occur in soccer. First, because the cognitive impairment from concussion can be cumulative (17), CTBI can occur from repeated, relatively mild concussions. In soccer, concussions may occur from typical contact sport collisions: head-to-head, head-to-goalpost/ground, and head-to-body (e.g., elbow, foot, knee) collisions. However, unlike some other contact sports (e.g., American football), soccer players do not wear protective headgear.

The second mechanism for potential CTBI is a distinguishing feature of soccer: repetitive heading of the ball. It has been estimated that professional soccer players play approximately 300 division games and head the ball more than 2,000 times during their career (18). This is compounded by the fact that a plastic-coated soccer ball weighs 396 to 453 g, and the speed of the ball can reach 60 to 120 km/h (19). It has been calculated that a ball kicked with half power from a distance of 10 m travels 83.2 km/h and hits the head with an impact of 116 kPa. Kicked with full power the ball hits the head with 200 kPa (20). Together these mechanisms suggest that soccer players are particularly vulnerable to head injuries with consequent CTBI.

The current investigation tested the hypothesis that CTBI is associated with professional soccer as evidenced by impairment on neuropsychological testing. This study also sought to identify possible risk factors for CTBI (e.g., soccer-related concussions, heading the soccer ball) and to evaluate their contributions to neurocognitive dysfunction in soccer players.

soccer-related concussions, heading the soccer ball) and to evaluate their contributions to neurocognitive dysfunction in soccer players.

Methods

Subjects

The volunteer group of this investigation consisted of 53 active Dutch male professional soccer team members and 27 male control subjects. The control subjects were members of elite swimming and track teams in the Netherlands. All participants in this study were interviewed and examined using an extensive neuropsychological test battery. Soccer players and control subjects participated in an extensive interview that inquired about age, education, number of general anaesthesias, occupational history, number of concussions (sports and nonsports related) in a lifetime, alcohol intake, drug use and medical conditions that might affect cognitive functioning (table 1). Education was scored on a seven-point scale and, because all participants completed their secundary school education, only the levels 3 (technical and vocational training for 12- to 16-year-olds) to 7 (academic level) were used. In addition, the team physicians of the professional soccer players were interviewed concerning the number of concussions (sports and nonsports related) and medical conditions that might interfere with cognitive functioning. All the participants in this study were native Dutchmen who were educated in the Dutch educational system. Nonnative Dutchmen and people who were not educated in the Dutch school system were excluded. Other exclusion criteria included history of drug abuse, epilepsy, and medical conditions that might affect cognitive functioning.

Table 1 Characteristics of soccer players and controls, and p-values.

	Soccer players	Controls	P value (2 tailed)
Number	53	27	
Age: mean (sd)	25.4 (4.1)	24.5 (4.5)	0.30[1]
Level of education: median (range)	6 (5-7)	5 (3-7)	0.05[2]
Number of alcohol intakes per month: median (range)	12 (0-99)	28 (0-99)	0.02[2]
Number of general anaesthesias: median (range)	1 (0-7)	0 (0-4)	0.09[2]
Percentage of persons with concussions not sustained in soccer:	11.3	14.8	0.74[3]

[1] Student's t-test. [2] Mann-Whitney U test. [3] Fisher's exact test.

Education levels are scored on a 3 (3 = technical and vocational training for 12 to 16 years old) to 7 (7 = academic level) scale.

Group comparisons were made between professional soccer players and control subjects for level of cognitive functioning. Intergroup comparisons were made in soccer players who do many "headers" and these who do not (non-headers). Midfield players and goalkeepers were classified as nonheaders, and forward and defensive players were classified as headers. Trauma index variables regarding the number of concussions with or without loss of consciousness (soccer related), duration of professional soccer career, the number of headers produced each match, and the number of matches in one season were also determined for the professional soccer players. By multiplying the number of headers each match with the number of matches, we estimated the total number of headers in one season.

Neuropsychological testing

In the current investigation, neuropsychological tests that have proven to be sensitive in the detection of cognitive impairment in contact and collision sports (13, 21-23) were used. Each participant in this study received a battery of neuropsychological tests that were administered by a trained psychometrician. The interview was performed by a qualified neuropsychologist. All tests were administered according to standardized instructions and procedures. The test data were scored by a qualified neuropsychologist who was blinded to the status (professional soccer player or control subject) of the participant. The neuropsychological test battery included the following: Raven Progressive Matrices Test (RPM) (24), Wisconsin Card Sorting Task (WCST) (25), Paced Auditory Serial Addition Task (PASAT; slowest version) (26), Digit Symbol Test (27), Trail Making A and B (28), Stroop test (29), Bourdon-Wiersma Test (30), subtests of the Wechsler Memory Scale (WMS; Associate Learning, Logical Memory, and Visual Reproduction (31), Complex Figure Test (Copy, Immediate Recall, and Delayed Recall) (32), 15-Word Learning Test (33), Benton's Facial Recognition Task (34), Figure Detection Test (35), Verbal Fluency Test (35) and the Puncture Test (36).

Statistical analysis

Comparisons of the neuropsychological test scores were performed by calculating the differences between the means using the Student's t-test. With a linear regression analysis these differences, their CIs, and one-tailed p values were determined, adjusting for the level of education, alcohol intake, number of general anesthesias, and number of concussions not sustained in soccer. To determine the association between heading-related variables and test scores, unadjusted and adjusted regression coefficients and their p values were calculated using a univariate and a multivariate linear regression model, respectively.

Because 27 outcome variables are compared, we performed a Bonferroni correction on the significance level. Furthermore, using the ordinary least squares (OLS) test as described by O'Brien (37) and modified by Läuter (38), we tested a global null hypothesis.

Results

Population characteristics

Characteristics of the study population are presented in table 1. The average age of the soccer players was 24.4 ± 4.1 (SD) years compared to 24.5 ± 4.5 years in the control group (p=0.3). There was no significant difference between the percentage of people sustaining concussions unrelated to soccer (11.3% in the professionals and 14.8% in the control subjects, p = 0.7). None of the professional soccer players in this study abused drugs. Higher alcohol consumption was noted in the control subjects (p=0.02). Soccer players achieved a higher educational level (p=0.05) and experienced a greater number of general anesthesias (p=0.09). Among the 53 professional soccer players, 54% experienced one or more soccer associated concussions with or without loss of consciousness. During their professional careers 79% of the players sustained one or more head-to-head collisions (with or without post concussive symptoms).

Professional soccer players reported a median of 800 (range, 50 to 2,100) headers during competitive matches in one soccer season. Headers experienced in training sessions were excluded from this approximation. The median of the number of headers during a match was 16 (range, 1 to 42). Approximately, 47% of the players headed the ball 0 to 10 times, 36% headed the ball 11 to 20 times and 17% headed the ball 21 or more times during an average match. Thirty percent of the players also participated in heading drills during training. These drills consisted of repetitive heading of the ball for 30 minutes once a week.

The median of the number of soccer matches played annually was 50 (range, 25 to 70), and the median of the number of practices per week was six (range, 4 to 9). Ninety-two percent practiced 4 to 7 times a week and 7.5% practiced more than seven times a week. An average training session lasted 2 hours. Twenty-four percent of the players in this study were forward players, 45% were midfield players, and 30% were defense players.

Soccer players experienced extensive amateur and professional careers. The median of an amateur soccer career was 12 years (range, 4 to 23 years). Thirty percent of the players played 4 to 10 years, 57% played 10 to 16 years and 13% played 16 years or

longer as amateurs. The median of the duration of a professional soccer career in this study was 5 years (range, 1 to 18 years). Professionally, 43% of the players participated 1 to 5 years, 30% played 6 to 12 years and 27% played more than 12 years.

Neuropsychological testing

We examined the distributions of the test scores on outliers and skewness. The skew of the distributions necessitated a log transformation for six scores - namely, the Bourdon-Wiersma test (number of mistakes), the PASAT, Facial Recognition Test, the Complex Figure test (Copy), the Puncture test (dominant hand), and the total Raven score - to symmetrize and normalize the distribution. The analysis was done with these transformed scores, but in the following the inversely transformed values are presented.

Table 2 shows that professional soccer players exhibited more cognitive impairments when compared to control subjects. Professional soccer players performed poorer on verbal and visual memory, planning, and visuoperceptual processing tasks compared to control subjects. On a number of tests, the differences between the two groups (i.e., Figure Detection Groninger Intelligence Test [GIT] subtest, The Complex Figure Test [Copy, Immediate, and Delayed recall], Logical Memory [WMS], and Visual Reproduction [WMS]) remained significant after adjustments for confounding variables (the number of concussions not related to soccer, alcohol consumption, level of education and the number of general anesthesias). These psychometric test scores also remained significant after a Bonferroni correction. Furthermore, the one-tailed p value based on the OLS test statistic was 0.00006 (Z_{n-1}=3.84). Evidence for a dose-reponse relation between selected soccer-related exposure variables and the neuropsychological test scores was demonstrated (Table 3). An increasing number of headers and concussions incurred during soccer participation were associated negatively with memory, visuoperceptual, and planning capacity. The number of headers was inversely associated with test scores on Complex Figure Test Immediate ($p=0.048$) and Delayed Recall score ($p=0.01$). The number of concussions sustained in soccer was inversely associated with the test score on the Complex Figure Test Copy ($p=0.01$). Field position also influenced performance on neuropsychological testing. Forward and defensive players performed significantly poorer on Figure Detection ($p=0.01$), Complex Figure Test Immediate ($p<0.001$), Delayed Recall ($p<0.001$), Logical Memory (WMS, $p=0.02$), and Visual Reproduction (WMS, $p=0.02$) compared with midfield players and goalkeepers.

Table 2 Comparison of psychometric performance between control subjects and professional soccer players.

Test	Mean controls scores	Unadjusted difference (1-tailed p value)	Adjusted difference (1-tailed p value)	99.63% CI
Part I				
Bourdon-Wiersma test:				
Number of mistakes	6.00	2.20 (0.03)	1.47	-1.65 to 7.12
Number of seconds	602.12	5.77	-9.79	-88.2 to 68.4
PASAT	3.68	1.06	1.38	-1.62 to 5.19
Stroop I	39.64	-1.07	-1.59	-5.80 to 2.64
Stroop II	51.40	-0.74	-1.71	-7.72 to 4.30
Stroop III	77.76	1.41	2.86	-6.82 to 12.54
Trailmaking A	20.85	1.46	1.33	-4.02 to 6.68
Trailmaking B	49.68	1.38	-0.03	-12.14 to 12.08
Wisconsin: number of perseverations	4.26	3.63 (<0.001)	3.93 (0.002)	-0.12 to 7.98
Part II				
Wisconsin: number of shifts	4.61	-0.68 (0.026)	-0.69 (0.04)	-1.88 to -0.50
Digits Backwards	5.38	-0.21	-0.48	-1.34 to 0.40
Digits Forward	6.50	-0.41	-0.52 (0.03)	-1.36 to -0.32
Facial Recognition Test	22.96	0.10	-0.28	-2.44 to 1.15
Figure Detection	16.46	-3.34 (<0.001)	-3.66 (<0.001)	-6.14 to -1.18
Complex Figure Test Copy	35.30	-1.73 (<0.001)	-1.25 (<0.001)	-2.45 to -0.48
Complex Figure Test STM	26.37	-8.09 (<0.001)	-7.55 (<0.001)	-12.32 to -2.80
Complex Figure Test LTM	24.93	-7.15 (<0.001)	-5.87 (<0.001)	-9.94 to -1.80
Fluency, animals	29.11	-0.11	-0.69	-5.94 to 4.56
Fluency, professions	21.26	-0.52	-1.20	-5.46 to 3.06
Puncture Test nondominant hand	75.26	-0.90	1.29	-7.80 to 10.38
Puncture Test dominant hand	93.26	-0.23	-0.41	-7.96 to 5.42
Total Raven score	53.56	-1.99 (0.03)	-2.31 (0.003)	-6.15 to 0.12
Digit Symbol	68.47	-3.81	-5.03	-15.92 to 5.88
Associate Learning	27.48	-1.32 (0.01)	-1.49 (0.01)	-3.34 to 0.36
Logical Memory	19.04	-4.38 (<0.001)	-4.60 (<0.001)	-8.60 to - 0.60
Visual Reproduction	11.00	-1.89 (<0.001)	-2.02 (<0.001)	-3.50 to -0.52
15-Word Learning Test	51.46	-2.22	-2.95	-9.66 to 3.76

* To facilitate the interpretation of the differences we divided the table in two parts. Pn part I all test scores are included that are correlated positively with cognitive impairment. In part II all test scores are correlated negatively.

† Controls are elite swimmers and elite 800-meter runners.

In accordance with the Bonferroni correction.

PASAT = Paced Auditory Serial Additon Tast; STM = short term memory; LTM = long term memory.

Table 3 Associations between heading-related variables and test scores in the group of soccer players.

	Unadjusted score (p value)	Adjusted score (p value)
Field position, headers vs non-headers		
Figure Detection	-1.84 (0.02)	-2.09 (0.01)
Complex Figure Test copy	-1.25 (0.01)	-1.30 (0.01)
Complex Figure Test STM	-6.34 (<0.001)	-6.08 (<0.001)
Complex Figure Test LTM	-5.36 (<0.001)	-5.17 (<0.001)
Logical Memory	-2.88 (0.02)	-3.02 (0.02)
Visual Reproduction	-1.24 (0.01)	-1.19 (0.02)
Number of concussions in a lifetime		
Complex Figure Test copy	-0.20 (0.03)	-0.25 (0.02)
Number of headers in one season		
Complex Figure Test STM	-0.16 (0.048)	-0.17 (0.048)
Complex Figure Test LTM	-0.20 (0.008)	-0.19 (0.01)

Adjusted and unadjusted regression coefficients and their p values are shown for the associations with one-tailed p values >0.05 only. STM = short term memory; LTM = long term memory.

Discussion

These findings suggest that professional soccer may be associated with neurocognitive impairment. The observed impairment in neuropsychological function preferentially involves memory, planning and visuoperceptual processing. Forward and defensive players tend to be more vulnerable to cognitive impairment because they are more likely to head the ball and experience a higher frequency of soccer-related concussions. Our findings are in agreement with those of Tysvaer and Lochen (13), who showed a higher degree of neuropsychological impairment in headers than in nonheaders (20% versus 8%). In their study, 73% of the former professional soccer players showed neurocognitive impairment (3% severe, 38% moderate, and 32% mild). The neuropsychological differences between the retired players and the control subjects were documented in tasks regarding perception, learning, and mental speed. The players in the study of Tysvaer and Lochen (13) had an average age of 48.6 years (range, 35 to 64 years) and were retired.

In a neurological evaluation of 20 soccer players on the United States national team (16), no association was found between heading the soccer ball and neurological

symptoms, including MRI-detected abnormalities. The discrepancy between this investigation and our findings may be related to methodological issues. This study did not use the more sensitive neuropsychological techniques nor did it distinguish between different styles of soccer play in training and competition.

The Complex Figure Test proved to be sensitive to detecting symptoms of neurocognitive impairment in professional soccer players. The sensitivity of the Complex Figure Test may be explained by the fact that this test requires memory, planning, and visuo-spatial ability (i.e., cognitive functions that are sensitive to deterioration after concussion and multiple trivial impacts). According to the standard of Osterieth (32), as used in clinical practice, 7% of the control subjects and 45% of the professional soccer players showed moderate to severe impaired scores. Surprisingly, the PASAT Test did not differentiate between socccer players and control subjects. In this study we utilized the slowest PASAT version, which could explain the lack of sensitivity.

In our cross-sectional study, several methodologic precautions were taken to minimize the problems associated with group comparisons. All the participants in this study were native Dutchmen who were educated according to the Dutch educational system, therefore eliminating misinterpretations caused by educational differences. In addition, drug users and participants with medical conditions that might interfere with cognitive functioning were excluded from this investigation.

Other potential biases associated with physical exertion and exercise in soccer were excluded by using elite swimmers and runners as control subjects. To ensure reliability, tests and interviews were administered by a certified neuropsychologist and trained psychometricians. The test scores were scored by a neuropsychologist who was not informed about the status (professional soccer player or control subject) of the participants. Confounding variables such as alcohol use, the number of concussions not sustained in soccer, the level of education, and the number of general anesthesias were adjusted by using multivariate regression analysis. The problem with multiple end point testing was controlled by a Bonferroni correction of the significance level and a global null hypothesis testing procedure.

Difference in mentality and test attitude could also explain these results. However, soccer players had a higher level of education, and all participants were tested by a trained psychometrician. Nevertheless, there may be inherent differences between those who choose to play soccer and those who choose to swim or run that may affect neuropsychological test performance and that may not be captured adequately in the variables we controlled. Our findings that professional soccer is associated with neuropsychological impairment supports the hypothesis that CTBI is a health

problem for soccer players. Although the neuropsychological impairment is milder than what might be anticipated in boxing, the dose-reponse relation between traumatic brain injury (i.e., concussions and heading the ball) and cognitive impairment presents a medical and public health concern. Accordingly, surveillance and prevention measures should be undertaken to maximize safety. For example, soccer players should have a medical evaluation after sustaining a concussion. Baseline neuropsychological testing (performed in preseason) and postconcussion sideline follow-up evaluations may be useful in determining the effects of concussion and the appropriateness for return to competition. To limit the adverse effects of heading the ball, precautionary measures need also be established. In other contact sports with similar concussion rates (e.g., American Football (3)), these preventive measures are recommended.

Participation in professional soccer may affect adversely some aspects of cognitive functioning (i.e., memory, planning, and visuoperceptual processing). This impairment in neurocognitive function appears to be attributed to increased heading of the ball and soccer-related concussions experienced by forward and defensive players. Whether our findings can be extrapolated to amateur or lower exposure soccer players remains to be determinated in future investigations.

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References

Chapter 5

1. Statistics on the 150 affiliated National Associations of Fifa. Fifa News 1982; 235: 538.
2. American Academy of Pediatrics Policy Statement. Recommendations for participation in competitive sports. The Physician and Sportsmedicine 1988; 16: 165-167.
3. Kelly JP, Rosenberg JH. Diagnosis and management of concussion in sports. Neurology 1997; 48: 575-580.
4. Hughes R. Head damage. A warning to all players. The Sunday Times, nov 10, 1974.
5. DeMarco J, Reeves, C. Injuries associated with soccer goalposts, United States, 1979-1993. JAMA 1994; 271: 1233.
6. Mueller FO, Cantu RC. Catastrophic injuries and fatalities in high school and college sports, 1982-1988. Medicine and Science in Sports and Exercise 1990; 22: 737-741.
7. Critchley M. Medical aspects of boxing, particularly from a neurological standpoint. British Medical Journal 1957; 1: 357-362.
8. Robberts AH. Brain Damage in Boxers. London: Pittman Medical and Scientific Publishers Company LTD, 1969.

9. Jordan BD. Neurologic Aspects of Boxing. Archives of Neurology 1987; 44: 453-459.
10. Mendez MF. The neuropsychiatric aspects of boxing: International Journal of Psychiatry in Medicine 1995; 25: 249-262.
11. Matser JT. Sponzen en aspirines, voldoende bij hoofdletsels in sport? Geneeskunde en Sport 1996; 29: 66-73.
12. Corsellis JA. Brain damage in sport. Lancet 1976; 1: 401-402.
13. Tysvaer AT, Lochen EA. Soccer injuries to the brain. A neuropsychological study of former soccer players. The American Journal of Sports Medicine 1991; 19: 56-60.
14. Sortland O, Tysvaer AT. Brain damage in former association football players. An evaluation by cerebral computed tomography. Neuroradiology 1989; 31: 44-48
15. Tysvaer AT, Storli O, Bachen NI. Soccer injuries to the brain. A neurologic and electro encephalographic study of former players. Acta Neurologica Scandinavia 1989b; 80: 151-156.
16. Jordan SE, Green GA, Galanty H., Mendelbaum BR, Jabour AJ. Acute and Chronic Brain Injury in United States National Team Soccer Players. The American Journal of Sports Medicine 1996; 24: 205-210.
17. Gronwall DMA, Wrightson P. Cumulative effect of concussion. Lancet 1975; 2: 995-997.
18. Tysvaer AT, Storli, O. Association Football Injuries to the Brain, A Preliminary Report. British Journal of Sports Medicine 1981; 15: 163-166.
19. Keller D, Hennemann MC, Allegria J. Fussball, der Elfmeter. Leistungsport 1979; 9: 394-398.
20. Von Schneider PG, Lichte H. Untersuchungen zur Grösse der Krafteinwirkung beim Kopfballspiel des Fussballers. Sportartz und Sportmedizin 1975; 26: 222-223.
21. Witol A, Webbe F. Neuropsychological Deficits Associated with Soccer Play. Archives of Clinical Neuropsychology 1994; 9: 104.
22. Matser JT, de Bijl MAO, Luijtelaar ELJ. Is amateur boxing dangerous? De Psycholoog 1992; 12: 515-521.
23. Jordan BD, Matser JT, Zazula T. Sparring and cognitive function in professional boxers. The Physician and Sportsmedicine 1996; 24: 87-98.
24. Raven JC. Guide to the Standard Progressive Matrices. London: HK Lewis, 1960.
25. Berg EA. A simple objective treatment for measuring flexibility in thinking. Journal of General Psychology 1948; 39: 15-22.
26. Gronwall DMA. Paced Auditory Serial Addition Task: A measure of recovery from concussion. Perceptual and Motor Skills 1977; 44: 367-373.
27. Wechsler D. WAIS-R Manual. New York: The Psychological Corporation, 1981.
28. Spreen O, Strauss E. A compendium of neuropsychological tests. New York: Oxford University Press, 1991.
29. Golden CJ. Stroop Color and Word Test. Chicago: Stoelling, 1978.
30. Van de Loo L. Enkele beschouwingen over de Bourdon-Wiersma test. Tijdschrift van de psychologische kring van de Nijmeegse Universiteit 1956; 2: 33-46.
31. Wechsler D. Wechsler Memory Scale Manual. San Antonio: The Psychological Corporation, 1974.
32. Osterieth PA. Le test de copie d'une figure complexe. Archives de Psychologie 1944; 30: 206356.
33. Deelman BG, Brouwer WH, van Zomeren AH. Functiestoornissen na trauma capitis. In: Jennekens-Schinkel A, et al., eds. Neuropsychologie in Nederland. Deventer: Van Loghum, Slaterus, 1980.

34. Benton AL, Hamsher K, Varney NR, Spreen O. Contributions to neuropsychological assessment. New York: Oxford University Press, 1983.
35. Luteyn F. Een nieuwe verkorte GIT. Dutch Journal of Psychology 1966; 2: 675-682.
36. Van der Vlugt H. Lateralisaties van hersenfuncties: een neuropsychologisch onderzoek naar de relatie tussen handvoorkeur en de relatie van de taalfunctie. Lisse: Swets & Zeitlinger, 1979.
37. O'Brien PC. Procedures for comparing samples with multiple endpoints. Biometrics 1984; 40: 1079-1087.
38. Läuter J. Exact T and F tests for Analyzing Studies with Multiple Endpoints. Biometrics 1996; 52: 964-970.

Chapter 6

Contribution of headers and soccer related concussions to cognitive impairment in professional soccer players

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Abstract

Objective: To determine the effect of headers and soccer related concussions to cognitive impairment in professional soccer players.
Methods: Eighty-four active professional soccer players from several professional premier league soccer clubs underwent neuropsychological evaluation. The number of headers in one professional season and the number of soccer related concussions was investigated concerning cognitive functioning.
Results: Soccer related concussions were inversely related to results of tests measuring sustained attention, planning (organisation)and visuo-perception. The number of headers in one season was inversely related to results of tests measuring focused attention and visual/verbal memory.
Conclusion: It seems that concussions produce a more diffuse profile of brain injury whereas headers seems to deteriorate fronto-temporal functions.

Introduction

Soccer players are vulnerable for chronic traumatic brain injury (CTBI) (1-7). There are two main distinct mechanisms by which CTBI can incur in soccer: concussions and headers (1).
In soccer, concussions may occur from typical contact sport collisions; head-to-head, head-to-goalpost/-ground and head to body (elbow, foot, knee) collisions (8-10). It is notable that the concussion rates per 1,000 exposures in American football and men's soccer are equal (9). In a recent published prospective study, 59% of american amateur soccer players sustained one or more concussions during two seasons (11). Most of the concussions resulted from contact with an opponent's head (28%) or elbow (14%). Dutch studies show a concussion rate of 54% in professional players and 50% in amateur soccer players during a soccer career (1,12).
The second mechanism for potential CTBI is repetitive heading of the ball, a unique aspect of soccer(13-14). Matser et al (1998) documented a median of 800 headers (range 50-2,100) in professional soccer players in a single season (1).
Although, two previous studies showed neuropsychological impairment in amateur and professional soccer players, it remained unclear whether this impairment was caused by the number of headers or concussions. This study sought to identify the specific contribution of soccer related concussion and headers to neurocognitive dysfunction in professional soccer players.

Methods

Subjects

The volunteer group of this investigation consisted of 84 active Dutch male premier league professional soccer players. All participants in this study were interviewed and examined using an extensive neuropsychological test battery. Soccer players participated in an extensive interview that inquired about age, education, number of general anaesthesias, occupational history, number of concussions (sports and nonsports related) in a lifetime, alcohol intake, drug use, medical conditions that might affect cognitive functioning and the number of headers in the last season (the number of headers in a single match multiplied with the number of matches played in the last season). Education was scored on a sevenpoint scale and, because all participants completed their secondary school education, only the levels 3 (technical and vocational training for 12 to 16 year-olds) to 7 (academic level) were used. In addition, the team physicians of the players were interviewed concerning the number of concussions (sports and nonsports related) and medical conditions that might interfere with cognitive functioning. All the participants in this study were native Dutchmen who were educated in the Dutch educational system. Furthermore, soccer players with a history of drug abuse, epilepsy, and medical conditions that might affect cognitive functioning were excluded.

Neuropsychological testing

In the current investigation neuropsychological tests that have proven to be sensitive in the detection of cognitive impairment due to soccer were used (1,12). Each participant in this study received a battery of neuropsychological tests that were administered by a trained psychometrician. The interview was performed by a qualified neuropsychologist. All tests were administered according to standardized instructions and procedures. The test data were scored by a qualified neuropsychologist who was blinded to the status of the participants.

The neuropsychological test battery included the following: Raven Progressive Matrices Test (RPM) (15), Wisconsin Card Sorting Task (16), Paced Auditory Serial Addition Task (PASAT); slowest version) (17), Digit Symbol Test (18), Trailmaking A and B (19), Stroop test (20), Bourdon-Wiersma Test (21), subtests of the Wechsler Memory Scale (WMS; Associate Learning (all scores added), Logical Memory, and Visual Reproduction) (22), Complex Figure Test (Copy, Immediate Recall, and Delayed Recall) (23), 15-Word Learning Test (24), Benton's Facial Recognition Task (25), Figure Detection Test (26), Verbal Fluency Test (26), and the Puncture Test (27).

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Statistical analysis

The association of headers and soccer related concussions with the test scores was simultaneously determined, using a multivariate linear regression model. With this model their regression coefficients and one-tailed p-values were determined, adjusting for age, the level of education, alcohol intake, the number of general anaesthesias and the number of concussions not sustained in soccer.

Results

Population Characteristics

The characteristics of the soccer players are shown in Table 1. Most of the professional players showed a high education level: level 5 (44%), level 6 (41%), level 7 (15%). The studied cohort consisted of forward players (26%), midfield players (40%), defense players (32%) and 2% of the players were goalkeepers. Nine players (11%) incurred concussions unrelated to soccer in a lifetime. The median of concussions sustained in professional soccer is 1 and the professionals reported a median of 500 headers in a single season. The number of headers and the number of soccer related concussions showed a low correlation (Spearman's rank correlation =.18).

Table 1 Characteristics of 84 professional soccer players.

	Median (10th and 90th percentile)
age in years	24 (19- 32)
number of alcohol intakes per month	5 (0- 40)
number of general anaesthesias	1 (0- 5)
duration of professional soccer career (yrs)	4 (1- 12)
number training hours weekly	10 (9- 14)
number of matches in last season	50 (40- 65)
number of headers in last season	500 (70- 1,260)
number of soccer related concussions in professional career	1 (0- 12)

Neuropsychological Testing

In table 2 the results of the regression analysis are shown for the associations with one-tailed p-values <0.05 only.

Headers: The number of headers was inversely related to scores on the Complex Figure Test STM (p=0.02) and LTM (p=0.01), 15 Word Learning Test (p=0.03) and Trailmaking A (p=0.048)

Concussions: The number of concussions was inversely related to scores on: Complex Figure Test Copy (0.01), Facial Recognition Test (0.04) and Bourdon-Wiersma Test (0.04).

Table 2 Associations between neuropsychological test scores and the number of headers, soccer related concussions respectively Adjusted regression coefficients and their p-values are shown for the associations with a one-tailed p-value less than 0.05 only.

	Impairment per 1000 headers (p-value)	Impairment for each soccer related concussion (p-value)
Complex Figure Test STM	-3.24 (0.02)	
Complex Figure Test LTM	-3.34 (0.01)	
Trail Making A	+2.57 (0.048)	
15-Word Learning Test	-3.76 (0.03)	
Complex Figure Test copy		-0.11 (0.01)
Facial Recognition Test		-0.09 (0.04)
Bourdon Wiersma Test (sec)		+3.00 (0.04)

Discussion

These findings suggests that neurocognitive impairment in professional soccer is caused by two distinct mechanisms e.g., concussions and headers. It seems that concussions produce a more diffuse profile of brain injury whereas headers seems to deteriorate fronto-temporal brain functions.

In this study, several methodological precautions were taken to minimize interpretation mistakes. All the participants in this study were native Dutchmen who were educated according to the Dutch educational system, therefore eliminating misinterpretations caused by educational differences. In addition drug users and participants with medical conditions that might interfere with cognitive functioning were excluded from this investigation. To ensure reliability, tests and interviews were administered by a certified neuropsychologist and trained psychometricians. Confounding variables such as alcohol use, the number of concussions not sustained in soccer, the level of education and the number of general anaesthesias were adjusted using multivariate regression analysis.

The number of sports related concussions and the number of headers as used in this analysis are based on interviews with the soccer players and their physicians. This way of collecting data is susceptible to misclassification.

We expect that this misclassification is nondifferential and consequently, the misclassification bias will be toward the null (28). This means that if there is an association of concussion and/or headers with cognitive functioning the coefficients as presented in table 2 are an underestimate of the real values.

The aim of this study is not to prove that soccer is dangerous, as we did in the two previous studies, but to explore whether soccer related concussion and/or headers contribute to neurocognitive dysfunction. Therefore we present the results with no adjustment of the signicance level for multiple endpoint testing.

Neuropsychological function loss associated to soccer related concussion involved tests measuring sustained attention, planning abbility and visuo-perceptual functioning characteristic of diffuse brain injury.

Cognitive function loss associated to heading the ball involved tests measuring focused attention and verbal/visual memory capacity. This is characteristic of fronto-temporal impairments as can be caused by linear movements of the head.

Our finding that CTBI in professional soccer is partly due to the number of concussions is in agreement with studies of concussion victims (29-31). The association between heading the ball and cognitive impairment is rarely documented in (sports) traumatic brain injury literature. Although, our results are in agreement

with a recent MRI-study which compares MRI-abnormalities in soccer players and in American football players. Soccer players showed more abnormalities although both groups sustained the same number of concussions (7). The cumulative effect of headers can be an explanation for the differences found.
To limit the adverse effects of heading the ball, precautionary measures need to be established. Coaches can be instructed to limit the number of heading drills in adults and headers can be banned in youth soccer.

References

Chapter 6

1. Matser JT, Kessels AGH, Jordan BD, Lezak MD, Troost J. Chronic Traumatic Brain Injury in Professional Soccer Players. Neurology 1998; 51: 791-796
2. Sortland O, Tysvaer AT. Brain damage in former association football players. An evaluation by cerebral computed tomography. Neuroradiology 1989; 31: 44-48.
3. Tysvaer AT, Storli O, Bachen NI. Soccer injuries to the brain. A neurologic and electro encephalographic study of former players. Acta Neurologica Scandinavica 1989b; 80: 151-156.
4. Tysvaer AT, Lochen EA. Soccer injuries to the brain. A neuropsychological study of former soccer players. The American Journal of Sports Medicine 1991; 19: 56-60.
5. Tysvaer AT. Head and neck injuries in soccer. Impact of minor trauma. Sports Medicine 1992; 14: 200-213.
6. Witol A, Webbe F. Neuropsychological Deficits Associated with Soccer Play. Archives of Clinical Neuropsychology 1994; 9: 104-105.
7. Autti T, Sipilä L, Autti H, Salonen O. Brain lesions in players of contact sport. Lancet 1997; 349: 1144.
8. American Academy of Pediatrics Policy Statement. Recommendations for participation in competitive sports. The Physician and Sportsmedicine 1988; 16: 165-167.
9. Kelly JP, Rosenberg JH. Diagnosis and management of concussion in sports. Neurology 1997; 48: 575-580.
10. Barnes BC, Cooper L, Kirkendall DT, McDermott TP, Jordan BD, Garret WE Jr. Concussion history in male and female soccer players. American Journal of Sports Medicine 1998; 26: 433-438.
11. Boden BP, Kirkendall DT, Garrett WE Jr. Concussion incidence in elite college soccer players. American Journal of Sports Medicine 1998; 26: 238-241.
12. Matser JT, Kessels AGH, Jordan BD, Lezak MD, Troost J. Neuropsychological Impairment in Amateur Soccer Players. JAMA 1999; 282: 971-973.
13. Keller D, Henneman MC, Allegria J. Fussball, der Elfmeter. Leistungsport 1979; 9: 394-398.
14. Von Schneider PG, Lichte H. Untersuchungen zur Grosse der Krafteinwirkung beim Kopfballspiel des Fussballers. Sportartzt – Sportmedizin 1975; 26: 222-223.
15. Raven JC. Guide to the Standard Progressive Matrices. London: HK Lewis, 1960.
16. Bouma A., Mulder J, Lindeboom J. Handboek Neuropschychologische Diagnostiek. Lisse Swets & Zeitlinger B.V. 1996, Section H, page 2-3.
17. Gronwall DMA. Paced Auditory Serial Addition Task: A measure of recovery from concussion. Perceptual and Motor Skills 1977; 44: 367-373.

18. Wechlser D. WAIS-R manual New York: The Psychological Corporation, 1981.
19. Spreen O, Strauss E. A compendium of neuropsychological tests. New York: Oxford University Press, 1991.
20. Golden CJ. Stroop Color and Word Test. Chicago: Stoelling, 1978.
21. Van de Loo L. Enkele beschouwingen over de Bourdon-Wiersma test. Tijdschrift van de psychologische kring van de Nijmeegse Universiteit 1956; 2: 33-46.
22. Wechsler D. Wechsler Memory Scale Manual. San Antonio: The Psychological Corporation, 1974.
23. Osterieth PA. Le test de copie d'une figure complexe. Archives de Psychologie 1944; 30: 206-356.
24. Deelman BG, Brouwer WH, van Zomeren AH. Functiestoornissen na trauma capitis. In: Jennekens-Schinkel A, et al, Neuropsychologie in Nederland. Deventer: Van Loghum, Slaterus, 1980.
25. Benton AL, Hamsher K, Varney NR, Spreen O. Contributions to neuropsychological assessment. New York: Oxford University Press, 1983.
26. Luteyn F. Een nieuwe verkorte GIT. Dutch Journal of Psychology 1966;2:675-682.
27. Van der Vlugt H. Lateralisaties van hersenfuncties: een neuropsychologisch onderzoek naar de relatie tussen handvoorkeur en de relatie van de taalfunctie. Lisse: Swets & Zeitlinger, 1979.
28. Sanford W. Applied linear regression.John Wiley & Sons, New York. 1985 pp 76-78.
29. Parasuraman R, Mutter SA, Molloy R. Sustained attention following mild closed head injury. Journal of Clinical and Experimental Neuropsychology 1991; 13: 789-811
30. Gronwall D, Wrightson P. Memory and information processing after closed head injury. Journal of Neurology, Neurosurgery & Psychiatry 1981; 44: 889-895.
31. Bohnen N, Twijnstra A, Jolles J. Neuropsychological deficits in patients with persistent symptoms sicx months after mild head injury. Neurosurgery 1992; 30: 692-696.

General Comment

Summary Statement

The serious questions about the cognitive consequences of (repeated) blows to the head due to sports activities remained not fully unanswered. In our studies, boxers and soccer players showed long-lasting neurocognitive changes (memory and planning impairments) due to concussive blows to the head. This dissertation also demonstrated that acute symptoms resemble chronic sequels of concussion and that a number of repeated subconcussive blows to the head can cause detrimental effects concerning neurocognitive functioning. Looking at the number of brain injuries in soccer, a huge number of people suffer short or long term effects, now and in the future.

Test Selection

After an ample study of the literature concerning mild traumatic brain injury (MTBI), we made a first selection of the test instruments, which were applicated in these patients. Tests were chosen which were validated and generally used in clinical practice. A third consideration was the limitation regarding time that was available for testing athletes. An exposure of 2 hours work for the athletes was the best what could be done. Computerised time saving versions of neuropsychological test are now under development and should result in increased access to testing. In addition, after talks with the researchers of the Pittsburgh Steelers project some tests were added in our battery (the Pittsburgh Steelers project was a large nation-wide prospective study in the USA in American football players in the beginning of the 90's). In this way it was possible to compare our results with theirs.
In making choices between several tests for a new research field it is possible that we missed some opportunities to detect more defects in athletes. However, our American colleagues with, more or less, the same test batteries showed the same cognitive impairments after recurrent head/brain injuries in different kinds of athletic activities.

Validity of our test results

One important factor contributing to the validity of neuropsychological testdata is the motivation of the persons taking the tests. The possibility of secondary gains, such as the lure of a big insurance settlement or the opportunity for early retirement

from unpleasant work may lead subjects to exaggerate deficits that are present or invent new ones. Although the issue of questionable motivation may be raised about any set of neuropsychological test data, it is typically not relevant when the subjects may actually comprise future opportunities by performing poorly. This would appear to be the situation for the athlete participants in these studies: a record of poor performance could reduce their value when competing for places on a team, and could follow them throughout their lives when they seek employment or to try raise their level of responsibility on the job.

However, should the question of motivation, as it relates to test validity, be raised, the group data provide keys to the validity of the athletes' neuropsychological test performances in several ways: (a) Some persons whose motivation is something other than the desire to do their best perform poorly across the board (1), but such a test pattern in someone under the age of 50 with a good school or work history makes no neuropsychological sense and can be accounted for by no known neuropathologic entity (2). This was not how the athletes at risk for brain injury due to blows to the head performed, as all of their performances contained instances of average or better mental abilities. (b) Neuropsychologically naive persons are likely to believe that brain disorders will usually cause memory problems and motor slowing, so that abnormally poor performances on tests that seem to measure memory ability and slowed responding on timed tests are also common features of malingering (3,4). Yet the athletes at risk for head injury did not have problems with every kind of speeded test, and few displayed problems involving the memory system per se. (c) Perhaps the most telling characteristic of the test performances of these athletes was the way in which they sorted out into distinctive pattern reflecting the effect of headers and, in contrast, concussive effects. It would not have been possible for these neuropsychologically naive athletes to anticipate how or why to pretend they had incurred brain damage.

Future Directions

The Quality Standards Committee of the American Academy of Neurology (AAN) developed in 1997 a sideline evaluation designed for the immediate assessment of concussion in athletes. An objective quantifiable initial assessment of the injury is in our opinion essential for evaluating a player's readiness to return to competition (5). Close observation and reliable neuropsychological assessment of the injured athlete are critical to the prevention of a more serious injury and cumulative neuropsychological impairment. The AAN practice parameter and the studies shown in this dissertation both emphasise the importance of objectively assessing

cognitive functioning as part of a screening procedure of athletes susceptive of mild traumatic brain injury and cumulations of these injuries.

Recommendations

In general, if an athlete has any symptoms related to MTBI, they should not be allowed to continue to play. Additionally, athletes with concussions should always be evaluated by a neuropsychologist prior to return to athletic play. Most importantly, any athlete who is symptomatic following a MTBI requires serial neuropsychologic evaluations till the athlete is performing on his own base-line level. Therefore we specifically promote the establishment of databases on contact and collision sport athletes. If similar neuropsychological instruments are utilised at all levels, longitudinal analysis of the test results for specific athletes will be possible as the athlete progresses from one level to the next when he/she incurred MTBI. This type of information would be particularly useful to athletes and physicians to assess the risk of future injury and further difficulties.

References

General comment

1. Bernard LC, Houston W, Natoli L. Malingering on neuropsychological memory tests: potential objective indicators. Journal of Clinical Psychology 1993; 49: 45-53
2. Lezak MD (1995). Neuropsychological Assessment. New York: Oxford University Press
3. Reitan RM & Wolfson D. The question of validity of neuropsychological test scores among headinjured litigants: Development of a Dissimulation Index. Archives of Clinical Neuropsychology 1996; 11: 573-580
4. Sweet JJ, Wolfe P, Sattlberger E et al. Further investigation of traumatic brain injury versus insufficient effort with the California Verbal Learning test. Archives of Clinical Neuropsychology 2000; 15: 105-113.
5. Parkinson D. Evaluating cerebral concussion. Surg. Neurol 1996; 45: 459-462.

Summary

The researches which are described in this thesis, started about ten years ago. The main question was: do athletic activities cause cognitive impairment and can this be detected by neuropsychological research. Moreover, can sports specific conditions be linked with this impairment.

At the beginning of our researches there was little to no literature that could give us an idea about the brain damages and the gravity of these damages which were caused by sports. Sport neurological subjects were not presented at congresses and/or symposiums until the beginning of the nineties. The interest in this subject has grown because of the attention that the media paid to some fatal sport accidents and to the deplorable condition some famous athletes were in after their sport career. Politics got interested in the subject and money was invested in research. At the moment there is data concerning the percentages of brain damage in sports such as soccer, American football, ice hockey and rugby, and because of the increasing number of publications, it is becoming clearer that brain damage often causes permanent symptoms. In response to this the American Academy of Neurology (AAN) issued recommendations in 1997 to protect athletes against (cumulative) brain damage caused by sports. Since 1995 the AAN also organizes a sportsneurological congress every year in which numerous researches concerning damages of the central and peripheral nerve system are presented, and since 1995 the yearly conferences of the European Neurological Society (ENS) also present sportsneurological subjects.

Numerous (top-class) athletes consisting of boxers, amateur and professional soccer players, swimmers and middle distance runners participated in the researches described in this thesis. They were interviewed extensively and underwent extensive neuropsychological testing. The aim of the interview was to document factors that could be related to the gravity and the frequency of the sustained brain injuries (including the number of concussions, the frequency of matches and practices, and the number of traumas). The factors that are not related to sports but that could have a negative effect on the brain functions (such as concussions sustained outside the practice of sports, number of anaesthesias, diseases, educative/wage forming status etc.) were also documented. The neuropsychological tests were considered to be able to objectify mental changes due to brain damage.

Chapter 1 describes the epidemiology concerning brain damage caused by boxing, soccer and sports in general. Furthermore the mechanical factors that can lead to brain damage and the physiological and neurochemical processes, which can be observed in these damages are described. Rating scales to determine the gravity of brain damage are also shown and discussed. Extensive attention is paid to the symptoms of a concussion and how concussions can be rated in measurements of gravity. Furthermore the cumulative effects of concussions and the risks of metabolic changes in the brain when sustaining cumulative brain damage are discussed.

Chapter 2 describes a study, which was conducted with the cooperation of 66 amateur boxers. Thirty-eight boxers were interview and tested neuropsychologically before and after a boxing match and 28 boxers were interviewed and tested neuropsychologically before and after a practice task (without head contact). The boxers who had boxed a match performed less well on the memory, planning and attention tasks than the boxers who had practiced without head contact. Especially the combination weight class (the heavier the boxer), the number of blows to the head and knockouts appeared to correlate negatively with the memory and planning performance.

Chapter 3 describes a study conducted with the participation of 49 amateur boxers who were classified in three groups according to their boxing experience (new members, 5 to 15 matches and more than 30 matches). All of them were interviewed and underwent neuropsychological testing in order to find out whether there was a difference in the neurocognitive functioning. The boxing experience had a negative influence on memory and attention. These attention disorders can be explained by the so-called "starting problem". A starting problem is a relative inactivity while commencing a mental action, a characteristic of frontal patients as well as patients with brain damage in general.

Chapter 4 describes a study in which the neurocognitive functioning of 33 amateur soccer players was compared with that of 27 controls (swimmers and athletes). All of them were interviewed extensively and they all underwent neuropsychological testing. The test results of the two groups were compared, corrected by age, education, anaesthesias and alcohol. The soccer players preformed less well on the memory and planning tasks. Concussions were reported relatively often with the soccer players. Fifty per cent of the amateur soccer players had sustained one or

more concussions during a match or during practice. The concussions appeared to correlate negatively with the memory tasks. Thirty-nine per cent of the amateur soccer players performed under the norm on the planning tasks, in comparison, only 7 per cent of the controls performed under the norm.
The results of the researches suggest that practicing amateur soccer can be risky with regard to sustaining mild chronic brain damage. Especially the contact traumas (concussions) that occur during soccer matches and practices seem to cause neurocognitive problems in amateur soccer players.

Chapter 5 describes a research conducted on 53 professional soccer players who were compared to 27 controls (swimmers and middle-distance runners). All of them were interviewed extensively and underwent neuropsychological testing. The test results of the two groups were compared, corrected by age, education, anaesthesias and alcohol. In comparison with the controls, the professional soccer players performed less well on the memory, planning and visual perceptual tasks.
Of the professional soccer group 54 per cent of the participants had sustained one or more concussions during a soccer match and an average of 800 as registered per season. The number of headers and concussions related negatively to memory, planning and visual perceptual performances. The field position (defenders/strikers were compared with midfielders/goalkeepers) was also a factor that correlated negatively with performances on visual perceptual and memory tasks.
The results suggested that practicing professional soccer has a negative influence on memory, planning and visual perceptual performances. A dose-response relation was ascertained between these functions and the number of headers and concussions. Even though the neurocognitive damages are milder than the shortages observed in boxing, it could be concluded the preventive measures are needed to protect soccer players against brain injuries.

Chapter 6 describes a study among 81 professional soccer players in which it was examined which part of the neurocognitive damage can be ascribed to headings and which part can be ascribed to concussions. This was done by a simultaneous analysis of the role of both factors. There was a low correlation between the number of headings and the number of concussions. Neuropsychological function loss associated to soccer related concussion involved tests measuring sustained attention, planning abbility and visuo-perceptual functioning characteristic of diffuse brain injury.
Cognitive function loss associated to heading the ball involved tests measuring

focused attention and verbal/visual memory capacity. This is characteristic of fronto-temporal impairments as can be caused by linear movements of the head. Thus, headers and concussions seem both to be risk factors for neurocognitive impairment.

From the results of the 5 demonstrated studies it can be concluded that boxing as well as soccer leads to relevant neurocognitive changes. It can be advised, following the guidelines drawn up by the AAN in 1997, to examine contact and collision sport athletes neurpsychologically every year. Through this the athlete can compare oneself and stop in time after detection of a cognitive decline. It can also be advised to let athletes undergo a simple neuropsychological test following a head injury. After showing deviant test scores a mandatory rest of at least one week without symptoms is recommended.

Samenvatting

Ongeveer 10 jaar geleden begonnen wij met de voorbereidingen eerste instantie stelden we ons de vraag of de symptomen van hersenletsels die werden veroorzaakt door sportbeoefening aangetoond konden worden middels neuropsychologisch onderzoek. Ook waren we geïnteresseerd of sportspecifieke condities gerelateerd konden worden aan neuropsychologische afwijkingen en of het mogelijk was dosis-respons relaties aan te tonen.
Bij het begin van de onderzoekingen was nagenoeg geen literatuur voor handen om ons een beeld te kunnen vormen van de hersenletsels en de ernst van deze letsels die werden veroorzaakt door sportbeoefening. Momenteel zijn gegevens bekend betreffende de hersenletsel percentages bij de 'grote' sporten zoals voetbal (soccer), American football, ijshockey en rugby en wordt het steeds duidelijker door een toenemend aantal publicaties wat de neurocognitieve veranderingen zijn die hersenletsels kunnen veroorzaken. Ook heeft de American Academy of Neurology (AAN) in 1997 preventieve aanbevelingsmaatregelen uitgevaardigd om atleten te beschermen tegen cumulatieve hersenschade door sportbeoefening. Tevens wordt sinds 1995 jaarlijks een sportneurologisch congres georganiseerd waarbij tal van onderzoeken worden gepresenteerd betreffende letsels aan het centrale en perifere zenuwstelsel veroorzaakt door sportbeoefening. Ook bij de jaarlijkse conferenties van de European Neurological Society (ENS) staan sinds 1995 elk jaar sportneurologische onderwerpen op het programma.

Aan de onderzoeken zoals beschreven in dit proefschrift hebben tal van (top)sporters meegewerkt waaronder amateurboksers, amateur- en professionele voetballers, zwemmers en atleten. Bij allen die hebben geparticipeerd, is een uitgebreide anamnese en een neuropsychologisch onderzoek afgenomen. De anamnese had tot doel factoren te documenteren die een relatie konden hebben met de ernst van de neuropsychologische afwijkingen. Hierbij kan gedacht worden aan het aantal hersenschuddingen opgelopen bij trainingen of wedstrijden, de frequentie van wedstrijden en trainingen, en bij voetballers het aantal kopballen. Ook had de anamnese tot doel factoren te verzamelen die van invloed zijn op de mentale status van de sporters zoals het aantal hersenschuddingen opgelopen buiten de sportbeoefening, het aantal narcose's, de doorgemaakte ziekten en de opleiding. Bij alle onderzoeken is in grote lijnen gebruik gemaakt van dezelfde neuropsychologische tests. De neuropsychologische testbatterij was door de

onderzoeksgroep na literatuurstudies dusdanig samengesteld dat deze in staat geacht moest worden om veranderingen van mentale prestaties door hersenletsels te kunnen objectiveren.

Hoofdstuk 1 beschrijft de epidemiologie betreffende hersenletsels veroorzaakt door sport met name boksen en voetbal. Verder worden mechanismen beschreven die leiden tot hersenletsels en wordt aandacht besteed aan fysiologische en neurochemische veranderingen door voornoemde letsels. Classificatieschalen om de ernst van hersenletsels te beoordelen worden getoond en besproken. Uitgebreide aandacht wordt geschonken aan de symptomen van een mild traumatisch hersenletsel en hoe deze letsels in te delen in mate van ernst. Verder worden cumulatieve effecten van mild traumatische hersenletsels besproken in de acute en chronische fase.

Hoofdstuk 2 beschrijft een studie die verricht is bij 66 amateurboksers. Achtendertig boksers die een wedstrijd boksten en 28 boksers die een fysieke taak uitvoerden werden neuropsychologisch getest voor en na de wedstrijd respectievelijk na de fysieke taak. Participanten met leerstoornissen, een alcoholconsumptie van meer dan 5 eenheden per week, soft- en harddruggebruikers en participanten met een medische aandoening die het cognitieve functioneren kon benadelen werden uitgesloten van het onderzoek. De mediaan van het aantal treffers op het hoofd tijdens wedstrijden was 8 en 35% van de boksers incasseerden meer dan 10 treffers per wedstrijd. Dertien procent van de wedstrijden eindigden in een knock out. De voor-na testscores tussen de beide groepen werd vergeleken en gecontroleerd werd voor leeftijd, opleiding, niveau van boksklasse, gewicht en tijdsinterval na de wedstrijd. Boksers die een wedstrijd hadden gebokst verschilden van de controles op plannings-, geheugen- en aandachtstaken. Uit de groep die een wedstrijd gebokst hadden bleek dat gewicht in combinatie met het aantal geïncasseerde treffers een negatieve correlatie toonde met het plannings- en het visueel herinneringsvermogen. Knock-outs reduceerden het aantal elementen dat gereproduceerd kon worden op een verbale leertaak. Geconcludeerd kon worden dat het participeren aan bokswedstrijden acute neurocognitieve veranderingen veroorzaakt.

Hoofdstuk 3 beschrijft een studie die verricht is met medewerking van 33 amateurboksers. Zeventien van de boksers hadden meer dan 30 wedstrijden gebokst, 16 boksers hadden tussen de 5 en 15 wedstrijden gebokst. Ook participeerden 16 personen die geen wedstrijd gebokst hadden. De drie groepen waren gematched op

leeftijd, etniciteit, opleiding en lichaamsgewicht. Bij allen werd een interview en een neuropsychologisch onderzoek afgenomen. Tussen de boksers die meer dan 30 wedstrijden hadden gebokst en de overige boksers werd een verschil geconstateerd betreffende het onthouden van complexe geometrische informatie op zowel de korte en lange termijn. Op eenvoudige geheugentaken werd geen verschil geconstateerd. Ook op gerichte aandachtstaken en kleurbenoemingstaken werd een significante relatie met het aantal wedstrijden aangetoond. De gegevens suggereren dat amateurboksers met een hoge bokservaring gehinderd worden door aandachtsstoornissen en visuele geheugentekorten.

Hoofdstuk 4 beschrijft een studie die is uitgevoerd bij 33 amateurvoetbalspelers en 27 controles (zwemmers en atleten). De voetballers waren afkomstig van 3 complete voetbalelftallen en de controles waren middenafstandslopers (2 complete atletiek teams) en zwemmers (1 compleet team) Van allen werd een uitgebreid interview en 27 neuropsychologische testen afgenomen, die geclusterd werden in 8 neurocognitieve functiegroepen.. De voetballers verschilden niet van de controles betreffende leeftijd, opleidingsniveau en aantal narcose's. Wel gebruikten de voetballers meer alcohol. Ook na correctie voor deze factoren werden verschillen in neurocognitief presteren tussen voetbalspelers en controles geconstateerd. Dit betrof de planningscapaciteit (coördinatie van het denken) en de inprentingscapaciteit (het onthouden van zowel verbaal als visueel materiaal).
Hersenschuddingen werden relatief vaak gemeld in de voetbalpopulatie. Vijftig procent van de amateurvoetbalspelers had een of meerdere hersenschuddingen opgelopen tijdens het spel cq. de training. De hersenschuddingen bleken negatief te correleren met testscores behaald op verbale en visuele geheugentaken.
De onderzoeksbevindingen suggereren dat het spelen van amateurvoetbal risicovol kan zijn wat betreft het verwerven van mild traumatisch hersenletsel. Vooral de contacttrauma's (hersenschuddingen) die tijdens het voetbalspel en trainingen worden opgelopen lijken de neurocognitieve problemen bij amateurvoetballers te veroorzaken.

Hoofdstuk 5 betreft een onderzoek uitgevoerd bij 53 professional voetbalspelers die werden vergeleken met 27 zwemmers en middenafstandslopers van Olympisch niveau. Al de participanten werd een uitgebreid interview en een neuropsychologisch onderzoek afgenomen. De profvoetballers verschilden niet van de controles wat betreft het aantal hersenschuddingen wat buiten de sport was opgelopen. De voetballers hadden een hogere opleiding en waren vaker onder

narcose geweest dan de controles. Geen van de profvoetballers gebruikten soft cq. harddrugs. In de profvoetbalgroep had 54% van de participanten een of meerdere milde hersenletsels opgelopen tijdens het voetbalspel. Gemiddeld werden 800 kopballen per seizoen genoteerd en werden gemiddeld 50 wedstrijden per seizoen gespeeld.
In vergelijking met de controles toonden de profvoetballers een significant verschil betreffende scores op verbale en visuele geheugentaken, planningstaken en visuo-perceptuele tests, ook na correctie voor alcoholgebruik, aantal narcose's, opleidingsniveau en hersenschuddingen opgelopen buiten het voetbal. Het aantal kopballen en milde hersenletsels beïnvloedden negatief de prestaties op verbale- en visuele geheugentaken, planningstaken en visuo-perceptuele tests. Veldpositie (verdedigers en spitsen vergeleken met middenvelders) was eveneens een factor die negatief associeerde met scores op visuo-perceptuele- en geheugentaken.
De resultaten suggereren dat door het spelen van profvoetbal neurocognitieve functies verslechteren. Om deze negatieve effecten te beperken kan base-line neuropsychologisch onderzoek en neuropsychologisch onderzoek direct na het letsel (side-line testing) aangeraden worden, zoals gepropageerd door de American Academy of Neurology (AAN, 1997).

Hoofdstuk 6 betreft een studie bij 81 profvoetballers waarbij het effect van koppen en hersenschuddingen op het neurocognitieve functioneren afzonderlijk is bekeken. Uit de analyse bleek dat er geen correlatie was tussen kopballen en het aantal hersenschuddingen. Hersenschuddingen die waren opgelopen tijdens het spelen van voetbal bleken planningscapaciteit, visuo-spatieel vermogen en het vasthouden van de aandacht negatief te beïnvloedden. Een toenemend aantal kopballen bleek het vermogen om de aandacht te richten en visuele en verbale geheugenprestaties te benadelen.
De resultaten suggereren dat hersenschuddingen neuropsychologische afwijkingen veroorzaken die duiden op diffuus hersenletsel en de afwijkingen door het aantal kopballen op fronto-temporale letsels wijzen.

Uit de resultaten van de 5 gepresenteerde studies kan geconcludeerd worden dat boksen en voetbal een negatieve invloed hebben op neurocognitieve prestaties. Het is te overwegen om de richtlijnen zoals voorgesteld door de American Academy of Neurology (1997) in deze sporten toe te passen.

Dankwoord

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