The end of season electroencephalographic and neuropsychological status of a team of secondary school rugby players – a comparison between very mild traumatic brain injury, mild traumatic brain injury and a sedentary control group

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Declaration

I, HENDRIK PAULUS DIJKSTRA..., hereby declare that the work on which this dissertation is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

Signed by candidate

(Signature)

1-11-2004

(Date)

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List of abbreviations

ApoE (Apolipoprotein epsilon)

ATBI (Acute traumatic brain injury)

CSF (Cerebrospinal fluid)

CT (Computed Tomography)

CTBI (Chronic traumatic brain injury)

CTT (Colour Trial Test)

DAI (Diffuse axonal injury)

DSST (Digit Symbol Substitution Test)

EEG (Electroencephalogram)

GCS (Glascow coma scale)

IMPACT (Immediate Measurement of Performance and Cognitive Testing)

LD (Learning disability)

LOC (Loss of consciousness)

MRI (Magnetic resonance imaging)

MTBI (Mild traumatic brain injury)

PCS (Post-concussion syndrome)

PTA (Post traumatic amnesia)

ROS (Reactive oxygen species)

SAC (Standardized Assessment of Concussion)

SIS (Second impact syndrome)

SRT (Simple reaction time)

TBI (Traumatic brain injury)

TMT (Trial Making Test)

vMTBI (Very mild traumatic brain injury)

WMS-R (Wechler Memory Scale-Revised)

Abstract

Objectives- Although the incidence and consequences of mild traumatic brain injury (MTBI) in secondary school rugby has been well documented, little is known about the incidence and consequences of repetitive sub concussive injuries – the so called very mild traumatic brain injuries (vMTBI). The aim of this study was to compare the end of season neuropsychological and electroencephalographic (EEG) status as well as the academic performance of players in a secondary school rugby team who, during the course of the season sustained only repetitive vMTBI to those players who sustained MTBI and a sedentary control group.

Methods- A cohort of 20 secondary school male rugby players from a local secondary school's first rugby team, was followed for a full competitive season by a sports physician and trained biokineticists, who were present at all the games played. All vMTBI and MTBI and the severity of these injuries were documented. At the end of the season the players were divided into two study groups: group 1 (N=11, mean (SD) age 17.9 (0.54) years) sustained only vMTBI during the season; group 2 (N=9, mean (SD) age 17.5 (0.88) years) sustained one or more MTBI during the season. At the end of the season an EEG-test was done on each subject according to the international 10-20 system and on a third group, group 3 (N=9, mean (SD) age 17.3 (0.5) years) of age- and educational level matched controls who did not participate in any collision sport. Fast Fourier transformation of each EEG was used to obtain power spectrum areas in the δ (0-3Hz), τ (4-7Hz), α (8-13Hz) and β (14-25,30 Hz) frequencies. The mean amplitude for each of the 21 EEG positions and for the different wave types were calculated for each group and compared. End of season Neuropsychological tests (Colour Trial Test 1 and 2, Digit Symbol Substitution Test, Wechsler Memory Scale-Revised (WMS-R) Test for verbal-, visual-, general-, delayed and attention/concentration) were conducted on the 3 groups. The academic results of different subjects (Afrikaans, English, Science, Mathematics and Latin) were obtained from the school and compared for the year that the study was done and for the two years preceding the study.

Results- Ten MTBI's were sustained in the 9 players of group 2 during the season. In group 1 five players reported between 2 and 4 vMTBI's per game played, two reported between 5 and 9 vMTBI's per game played and four reported less than 2 vMTBI's per game played. The EEG test results showed that the mean amplitudes of the total, the β- and τ-waves' frequencies in the right temporal regions tested, were significant smaller ($p<0.05$) for the MTBI and the vMTBI groups when compared with the aged matched controls. The amplitudes for the other positions did not change significantly. Only one abnormal EEG, with suspicious theta wave activity in the right posterior region, was found in the study population in a player who sustained 2 MTBI's during the season. No stastically significant results were found for the neuropsychological tests and academic results of the different groups.

Conclusion- The findings of this study indicate subtle and EEG changes at the end of the rugby season, in the total, β- and τ-wave frequencies of the temporal region of secondary school rugby players who sustained vMTBI's and MTBI's during a single rugby season. This is the first evidence of possible temporary and sub clinical brain cortex abnormalities in the temporal region of rugby players who suffered only vMTBI's during a single rugby season. The abnormalities were the same as in the MTBI group. A multicentred prospective study, ensuring a big enough sample size with baseline, pre-season EEG and neuropsychological data is necessary to evaluate these abnormalities further.

Keywords: (Concussion, Minor head injury, Mild Traumatic Brain Injury, Very Mild Traumatic Brain Injury, Neuropsychological testing, EEG, Rugby)

Chapter 1

Introduction and scope of the thesis

Introduction

Public and professional concern about the number and the severity of head and neck injuries occurring in secondary school rugby is increasing, especially after the tragic death of four rugby players during the 2002 schools rugby season in South Africa. Collision injuries are inevitable in contact sports, like ice hockey, American football and rugby [1]. Specific situations of the game such as the "set scrum", the "rucks", the "maul" or the "tackle" leads to heavy bodily contact and increase the risks of injury, especially to the head and neck [2].

The types of brain injury in collision sports vary from scalp lacerations, intracranial haemorrhage to concussion [3]. Concussion has been defined in many ways over the years but the definition of concussion is confusing because different terminology is being used for the same injury[4]. The terms "concussion" and "mild traumatic brain injury" (MTBI) are used interchangeably in the medical literature[4;5]. "Very mild traumatic brain injuries (vMTBI)" are those injuries that are often not recognized by the off-the-field medical team as concussion. VMTBI is defined as an 'asymptomatic' knock against the head often causing no symptoms, very short term dizziness, headache and importantly *no amnesia or loss of consciousness*, or late symptoms and signs like headache or impaired cognitive functioning (like the so called sub-concussive blows to the head from impacts with the soccer ball – headers [6-9]). The player always continues to play and finishes the match. The vast majority of head injuries in rugby are minor head injuries [10-12] or vMTBI's. The problem with these kinds of injuries is that they are often repetitive in nature. The possible negative long-term consequences of these injuries are a further concerning factor.

In November 2001, the first International Symposium on Concussion in Sport was held in Vienna, Austria. Previous definitions for concussions had certain limitations because there was an inability to include relatively minor impact injuries that result in persistent

physical and/or cognitive symptoms. Seeking to transcend these limitations, the Concussion in Sport Group has developed the following definition of concussion:[13]

Concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features that incorporate clinical, pathological, and biomechanical injury constructs that may be used in defining the nature of a concussive head injury include:

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

Sports-related head injuries in the 1980's accounted for 19% of non-fatal injuries in American football[14] and 4.5% of all high school sports injuries[15].

One South African study on the epidemiology of schoolboy rugby injuries, reported an incidence of 29% head and neck injuries. The authors of this study also indicated that there was a significant underreporting of concussion by certain schools involved in the study[12].

VMTBI's are subtle and often overlooked and are referred to as a "silent epidemic". MTBI and vMTBI can be caused by the head being struck, the head striking an object, or the brain undergoing an acceleration and deceleration movement (i.e. whiplash; coup, contra-coup) without external trauma to the head[15-17]. Minor head injury can occur without the athlete ever suffering from loss of consciousness [18]. For this reason, it is sometimes difficult to recognize a vMTBI.

A matter of concern with brain injuries, is the incidence and consequences of repetitive vMTBI's in rugby. The injury is either not recognised by the off-the-field medical team, or the severity and potential consequences of the injury is downplayed by the player and the medical team, parent or coach.

This means a player gets a blow or blows to the head in different situations of the game, plays on with no definite or immediate signs or symptoms of MTBI. The long-term outcome may represent symptoms and signs of MTBI, for example headaches, inaccurate memory, poor concentration and possible brain damage[19]. If these repetitive vMTBI's carry long-term consequences, it might indicate a need for action, but research on this topic is lacking. Therefore neuropsychological testing can be of assistance to estimate the consequences of mild and very mild traumatic brain injuries [20;21]. From the above data and taking the revised definition of concussion in sport into account, it is clear that MTBI and especially vMTBI may be under reported in collision sports, including rugby [12].

Chapter two of this thesis will give an overview of MTBI's and vMTBI's in collision sport and in rugby in particular. The definition, pathophysiology and the metabolic basis of MTBI will be discussed. The clinical features and grading of MTBI will be discussed and reasons for the possible underreporting of vMTBI will be given. The thesis will also focus on the concern about the incidence, management and potential consequences of MTBI and vMTBI. This is particularly relevant to rugby because of the repetitive nature of these injuries in this sport.

Although studies have been done on the effects of repetitive heading in soccer, no study has been done to compare the end of season Electroencephalographic (EEG) and neuropsychological status to academic performance of secondary school rugby players with either vMTBI or MTBI [8;9;22]. In chapter three, the results of a study to determine these relationships are reported. This study could give some clarity about the incidence of very mild traumatic brain injuries in a secondary school rugby team and possible answers about the consequences it could have on the player in the medium to long-term. This is especially important since the final exams for secondary schools start shortly after the end of the rugby season.

The focus of the research was to examine the difference in the end of season EEG- and neuropsychological status of secondary school rugby players with very mild- and mild traumatic brain injuries and to compare that with a sedentary control group. The academic performance of the three groups was also evaluated.

Chapter 2

Mild and very mild traumatic brain injuries in rugby – a review

Introduction

Head injuries in sport have for many years been a topic of intensive debate and research especially when young sportsmen are involved. Two million persons suffer a head injury
each year in the United States [23]. Of these, between 300 000 and 350 000 are sports- and recreation related head injuries, with concussion being the most common sports-related head injury [23-25]. Approximately 7% of high-school football players in the United States could expect to suffer at least one concussion each playing season [26].

Definition

The exact definition of concussion is controversial and no universal agreement on the standard definition or nature of concussion exists [27]. This has certain implications for the player, coach and team physician and can affect clinical decisions on how to classify and manage the injury.

The Congress of Neurologic Surgeons definition states that concussion is: "A clinical syndrome characterized by the immediate and transient post-traumatic impairment of neural function such as alteration of consciousness, disturbance of vision or equilibrium etc. due to brainstem involvement" [27].

The American Orthopaedic Society for Sports Medicine recently defined concussion as "any alteration in cerebral function caused by a direct or indirect (rotation) force transmitted to the head resulting in one or more of the following acute signs or symptoms: a brief loss of consciousness, light-headedness, vertigo, cognitive and memory dysfunction, tinnitus, blurred vision, difficulty concentrating, amnesia, headache, nausea, vomiting, photophobia, or a balance disturbance. Delayed signs and

symptoms may also include sleep irregularities, fatigue, personality changes, an inability to perform usual daily activities, depression, or lethargy" [28].

At the first International Symposium on Concussion in Sport, held in Vienna in November 2001, the following revised definition of concussion was adopted:

"Concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features that incorporate clinical, pathological, and biomechanical injury constructs that may be used in defining the nature of a concussive head injury include:

1. Concussion may be caused by a direct blow to the head, face, neck, or elsewhere on the body with an "impulsive" force transmitted to the head.
2. Concussion typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously.
3. Concussion may result in neuropathological changes but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury.
4. Concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course.
5. Concussion is typically associated with grossly normal structural neuro-imaging studies" [13].

Terminology

Different terminology for the same injury in the published literature on concussion exists making clinical diagnosis and management of the injury even more difficult. The term "concussion" is derived from the Latin verb *concutere*, which means, "to shake violently". The term *commotio cerebri*, which was introduced by Pare in the 16th century, has a similar meaning [5].

The term mild brain injury can be defined in one of two ways. The term is either used interchangeably with concussion, using the same defining symptoms and sequelae, or it is viewed as a distinct state remaining after concussion has receded [29]. The term mild head injury as opposed to mild brain injury is also used interchangeably with concussion. It is however proposed to abandon the use of the word 'head' in sports-related research. The term "head" injury implies a broader definition that includes extra

cranial trauma and craniocerebral (concussive) trauma. The terminology 'minor' should also be reconsidered because no trauma to the brain should ever be considered as minor. For the purpose of this thesis the term 'mild traumatic brain injury' (MTBI) will be used implying concussion as has been defined by the International Symposium on Concussion in Sport [13]. The term 'very mild traumatic brain injury' (vMTBI) will refer to the sub concussive (or apparently sub concussive but on further evaluation changing to concussive) brain injuries in collision sport, those injuries causing either no symptoms or signs, or very mild and brief symptoms and/or signs, with no loss of consciousness. The player *always* continues to play/finish the match, for a number of potential reasons mentioned later in this thesis.

A sub concussive event has been defined as "an apparent brain insult with insufficient force to cause hallmark symptoms of concussion" [30]. It could be argued that most of these injuries are by definition, concussive injuries, but that they are not recognized and treated as such. Although no symptoms or signs may be apparent, subtle and short-lived neurological impairment may be detected using more sensitive testing, for example neuropsychological testing. MTBI and vMTBI may actually exist on a continuum of histologically based damage [30]. Therefore, one of the alarming problems concerning head injuries in rugby is the number of players that suffer from 'undetected', repeated, vMTBI's. These have also been referred to as sub concussive 'dings' [22;31], or "bell ringers" [32] in other sports.

These injuries have been described as acute traumatic brain injuries (ATBI) in boxing [33]. A number of these boxers may not fully recover from these injuries, and the continued insult may then account for the insidious manifestations of chronic traumatic brain injuries (CTBI), with the problem of potential cumulative cognitive damage. Chronic traumatic brain injury (CTBI) has primarily been described in boxers and are the result of the cumulative long-term neurological consequences of repetitive concussive and sub concussive blows to the head [6;33]. However, caution should be exercised when applying this model of repetitive head injuries to other sports, as boxing presents unique risks to the athlete because of high frequency of head trauma [20]. The ATBI of boxing can be defined as a concussive injury from the moment of injury because some symptoms and or signs, although of a very mild nature and short duration,

are immediately apparent. Repeated head trauma in boxing may give rise to syndromes characterized by pyramidal, extra pyramidal and cerebellar signs and sometimes cognitive impairment and personality change [22;34;35]. This has also been termed the posttraumatic encephalopathy of boxing [36]. In a study of 538 athletes who had sustained minor head trauma, 424 were examined 3 months post injury. 79% complained of persistent headaches and 34% were unemployed. Organic brain damage with impaired cognitive function were mentioned as one of the possible causes [37].

Repetitive head injuries in rugby may therefore be concussive where symptom(s) and sign(s) of concussion are immediately apparent, or sub concussive (at least initially sub concussive) where no symptoms or signs of concussion are immediately apparent. These sub concussive injuries may be analogous, as mentioned, to the repetitive blows to the head in boxing, but perhaps also to the sub concussive brain injury caused by heading in soccer. Recent studies, utilizing neuropsychological testing, provide evidence to suggest that cumulative effects of concussion are detectable in amateur soccer players [6;8;9;22;38-40]. Although methodological concerns limit some of the findings of these studies, it is still of interest to consider whether multiple very mild traumatic brain injuries (sub-concussive impacts) have longer negative effects on brain function [39].

Rugby at all levels in South Africa is increasingly competitive, involving strong sentiments of national pride and with vast sums of corporate and private money being invested annually. It frequently involves player contact, and therefore places players at risk for head and neck injuries [25]. Public and professional concern about the number and severity of MTBI's occurring in rugby is increasing, especially after the death of four secondary school rugby players during the 2002 season in South Africa. The function of organized sports, especially in school-aged children, should be to contribute to their physical and mental health, and therefore the risk of injuries must be reduced [34]. Injuries to the central nervous system account for the majority of sporting deaths, with the highest rate of severe head and neck injuries occurring in American football and rugby [36].

The well being of rugby players is regarded as one of the priorities by various schools, clubs, and rugby organizations. Rugby is a physical game, increasing the risk of injuries, including MTBI. Rugby players require a combination of agility, speed, stamina and strength. The lack of protective gear and the aggressive style increases the risk of injury - in particular injuries to the neck [25]. These risks need to be carefully considered when an athlete decides to play rugby.

The risk for traumatic brain injury is apparent, but it has been suggested that there is a greater epidemic of subtle concussive, and many times sub-concussive, repetitive head injuries in rugby – the so called very mild traumatic brain injuries (vMTBI). The experienced coach and intelligent observer of the game will agree that this is probably one of the most important unresolved issues concerning head injuries in rugby.

Epidemiology

Over the years a number of studies have reported on the incidence, diagnosis and management of MTBI in a variety of collision sports including: American football, basketball, wrestling, softball, soccer, baseball, boxing, rugby and ice hockey [10;11;16;34;38;39;41-45]. One South African study on the epidemiology of schoolboy rugby injuries, reported an incidence of 29% head and neck injuries. The authors of this study also indicated that there was a significant underreporting of concussion by certain schools involved in the study. The authors recommended direct personal contact between the researcher and the injured player to prevent underreporting of particularly MTBI [12].

Injuries in children and adolescents playing organized sports are estimated to occur at rates of 39 per 100 participants per season for boys. Half of all injuries and close to 100% of serious injuries occur in American football, rugby, and wrestling [36]. Sports- and recreation related injuries account for 3% of hospitalized persons with concussion in the United States. It was previously conservatively estimated that 300 000 sports related brain injuries occur per year in the USA of which 250 000 are seen in high school football alone [14;46]. Although this already seems alarmingly high, the data

presented was likely to be an underestimation of the true incidence due to underreporting of minor injuries [47].

The actual incidence of sports-related MTBI is underestimated because approximately 90% of sports-related MTBI are mild and may go unreported. Thus it is possible for an athlete to be concussed and still continue to play. It is also of concern that sports-related MTBI's occurs most frequently among people aged 5 to 24 years and the long-term effects of MTBI episodes are not well documented [35].

It is possible that under reporting, -diagnosis and -treatment of MTBI also occur in South African rugby [12] - at all levels. There may be a number of other reasons for this. Firstly, there is ongoing lack of uniformity in defining the injury and a number of different classification systems of severity and return-to- play guidelines for MTBI are used. Secondly, management decisions may be influenced by the player's desire to remain on or return to the field and a coach's or parents' will to win at all cost. Thirdly, there may be confusion about different terminology that is being used for the same injury in the published literature on concussion, as mentioned earlier.

It is important to have a good understanding of the pathophysiology and metabolic basis of mild traumatic brain injury in order to evaluate and manage the injured rugby player and to appreciate the potential negative consequences of vMTBI's - especially repetitive vMTBI's.

Pathophysiology and metabolic basis of mild traumatic brain injury

Recent developments regarding the pathophysiology of traumatic brain injury and spinal cord injury have yielded new targets for therapeutic intervention. Laboratory and clinical evidence has implicated excitotoxic, inflammatory, oxidative, bioenergetic, and apoptotic cascades in worsening primary injury in the hours and days after the initial traumatic insult [23].

No missile head injury results in either focal or diffuse injury. Contact or inertial forces typically cause focal injuries, characterized by intra- or extraparenchymal hematoma formation or cortical contusion, from a mechanical insult to the skull. In contrast diffuse injury, results from acceleration/deceleration forces and rotational strain on the brain, causing diffuse axonal injury (DAI). Diffuse injury is not dependent on a direct blow to the cranium [23].

It is clear that in collision sports like rugby, vMTBI's and MTBI's may result in either focal or diffuse injury. However, MTBI usually is a result of acceleration-deceleration forces that are applied to the freely moving head and brain. The brain is protected by the cerebrospinal fluid, skull, and scalp and indirectly by the neck muscles. The cerebrospinal fluid (CSF) acts as a shock absorber, cushioning and protecting the brain by converting focally external stresses to a more uniform compressive stress. The scalp also has energy-absorbing properties. It is further very important to note that an athlete's head can sustain far greater forces with less risk of brain injury if the neck muscles are contracted at the moment of impact [3;17].

It is important to understand the forces that produce skull and brain injuries. The following principles apply:

1. A forceful sudden impact to the resting movable head will produce a translational (linear) acceleration of the skull differentially compared with the brain that lags slightly after impact. This usually produces maximum brain injury at the point of cranial impact (coup injury) that is characterized by swelling or bleeding of the adjacent cortex. If these coup injuries involve areas of the brain that are not that easily evaluated, like the frontal lobes, they may not be clinically apparent.
2. As the head recoils from a translational impact against an unyielding object, the deceleration of the head causes the brain to "bounce" along the bony irregularities of the frontal and temporal skull base, producing contusion injuries remote from the site of cranial impact (countercoup injury).
3. If a skull fracture is present, the bone itself may directly injure brain tissue [3;17].

Forces, especially acceleration forces with rotation in a coronal plane, cause axonal shear injury [20], and this is the primary pathologic feature of concussion at all levels of severity [48].

There are three types of stresses that can be generated by an applied force: compressive, tensile and shearing. Uniform compressive stresses are fairly well tolerated by neural tissue, but shearing stresses are very poorly tolerated. Shearing stresses cause diffuse and widespread injury to myelin sheaths and axons [36]. It is the shearing strain at the cerebrum-brainstem junction, secondary to angular acceleration when the head and neck bends around a center of rotation in the mid to lower neck, that causes injury to the brain stem. When the reticular activating system in the dorsal midbrain is affected by the shearing force, sudden dysfunction and loss of consciousness follows [39;49]. Injury, secondary to rotational acceleration with shearing strain, to the corpus calossum, and structures around the third ventricle, such as the septum and fornix, may be responsible for transient or long lasting memory impairments which can occur following head injuries [39;49].

Initial increases in the extra cellular concentrations of potassium after concussion, occur in large brain regions and can last for 3 to 5 minutes. Following injury, increased levels of the excitotoxin glutamate, open ligand-gated channels, resulting in large ionic flux [5]. Inhibition of the action potential and loss of consciousness may occur when the extra cellular concentration of potassium increases beyond the normal upper limit (about 4-5mmol/L) to levels of 20-50mmol/L and greater. This accumulation of potassium above threshold levels may require several seconds or longer before very high levels are reached, and this may explain why athletes can sometimes walk of the field and collapse unconscious at the sidelines [5].

Hyperglycolysis occurs as a pathophysiological response to ionic and neurochemical cascades. This hypermetabolic state with an accompanying decrease in cerebral blood flow results in the accumulation of lactate which causes intracellular acidosis [5;50]. The reduction in cerebral blood flow after traumatic brain injury is likely due to an increased vasoconstriction caused by endothelial accumulation of calcium. This calcium induced vasoconstriction appears to involve protein kinase C as an intermediate step [28].

This initial hypermetabolic state is followed by a cerebral hypo-metabolism that may last for days to months. It is during this phase, characterized by decreased protein synthesis and reduced oxidative capacity, that the cells are more susceptible to death if a second traumatic insult of even lesser intensity occurs [5;50].

A further cause of neuronal death, frequently observed in cortical and hippocampal tissue after traumatic brain injury, is the influx of sodium and calcium ions that leads to organelle failure. This influx is precipitated, through receptor mediated neuro-excitation, by an elevation of excitatory amino acids [23].

Although the above mentioned cascade of neurochemical, ionic and metabolic changes have been described in moderate to severe brain injury it remains controversial, but possible, that the same processes occur in milder injuries [51]

Damage to the neuronal cytoskeleton in particular damage to the more vulnerable cholinergic neurons, and diffuse axonal injury are the anatomical common denominators in TBI. Three prominent mechanisms known to play important roles in worsening outcome in TBI are currently under intensive investigation [23]:

The generation of reactive oxygen species (ROS), following traumatic brain injury, may contribute to further damage in both focal and diffuse axonal injury. Anti-oxidants, which act as scavengers of ROS, and glucocorticoids (immunosuppressant, stabilizing membranes and inhibiting free-radical-mediated lipid peroxidation) have been used in laboratory settings to inhibit the effect of ROS. However, there is no proven benefit for these agents in the clinical situation, as yet.

The rise in intracellular calcium has been related to mitochondrial damage. Mitochondrial functioning under conditions of oxidative stress can be blocked in vitro by cyclosporin A. Cyclosporin A has been used by a number of investigators in different studies and is currently under investigation in clinical trials in humans to treat MBI [23].

Extrinsic (receptor dependent) and intrinsic (receptor independent) apoptosis, both being active in traumatic brain injury and studies are underway to investigate the potential use of caspase inhibitors as an interventional strategy for the treatment of MBI [23].

Clinical features of mild traumatic brain injury

The acute symptoms of mild traumatic brain injury are well described and include amnesia, loss of consciousness (LOC), headache, dizziness, blurred vision, attention deficit, and nausea [13;21;39;43;50]. As the brain itself is not pain sensitive, surrounding structures like the meninges, basal vessels, scalp, skull, temperomandibular joint, neck joints, ligaments, and muscles may cause headache. Other causes of exercise-related headaches, like exertional migraine needs to be considered in the differential diagnosis of post concussive headache, as well as the profound, sudden increase in headache due to an expanding mass, vasoparalysis or edema. Dizziness, nausea and imbalance are likely to be at least partially caused by injury to the vestibular system.

In a prospective survey of Australian rules footballers with acute concussion [19], the following were established as common symptoms: confusion, headache, dizziness, blurred vision, nausea, diplopia and/or photo/phonophobia. In this study, comparing recent memory and orientation in a concussed and a non-concussed control group, the concussed athletes were more likely to fail recent memory questions pertaining to simple game facts. Answers to orientation questions were not significantly different between the concussed group and the non-concussed control group [19].

Tables 2.1 and 2.2 lists the frequently observed symptoms and signs of concussion [13;21;39;43;50].

Table 2.1: Symptoms of MTBI

Symptoms of MTBI
Early (minutes to hours)
Headache
Dizziness or vertigo
Lack of awareness of surroundings
Nausea and vomiting
Late (days to weeks)
Persistent low-grade headache
Lightheadedness
Poor attention and concentration
Memory dysfunction
Easy fatigability
Irritability and low frustration tolerance
Intolerance of bright lights or difficulty focusing vision
Intolerance of loud noises, sometimes ringing in the ears
Anxiety and depressed mood
Sleep disturbance

Table 2.2: Signs of MTBI

Signs of MTBI
Vacant stare
Delayed verbal and motor responses
Inability to focus attention
Disorientation
Slurred or incoherent speech
Gross observable incoordination
Emotionality out of proportion to circumstances
Memory deficits
Any period of loss of consciousness

The consequences of MTBI are rarely limited to one set of symptoms, impairments or disability. The long-term neurological consequences of MTBI may vary and can include sensory, motor and autonomic nervous system function impairment. Seizures, headaches, visual deficits, sleep disorders and a variety of movement disorders may be some of the long-term sequelae of MTBI [13;35;52]. The frequency of post-MTBI headache has been reported to vary between 40% and 86% [42;53;54]. It has been suggested that high school athletes suffering from post-MTBI headache approximately 7 days after injury are likely to have persistent adverse effects as a result of MTBI. This includes the presence of other post-MTBI symptoms and attenuated neurocognitive functioning [53]. The apolipoprotein E (ApoE) epsilon-4 gene (ApoE4) appears to be associated with increased severity of chronic neurological deficits in boxers [20].

The cognitive consequences of MTBI are similarly broad and may include memory impairment, and difficulties in attention and concentration, deficits in language use and visual perception. Frontal lobe functions such as executive skills of problem solving, abstract reasoning, insight, judgment, planning, information processing and organization can also be associated with MTBI [13;31;35;52;55-57].

Some of the common behavioral deficits after MTBI include a decreased ability to initiate responses, verbal and physical aggression, agitation, learning difficulties, shallow self-awareness, altered sexual functioning, impulsivity, social disinhibition, mood disorders, personality changes, altered emotional control, depression and anxiety. In one study, referred patients to a neuropsychiatric brain injury unit showed evidence of neuropsychiatric symptoms in right-sided brain injury, while impairment of intellectual ability (cognitive impairment) was associated with injury to the left side of the brain [55].

The long-term effects on social function may include increased suicide risk, divorce, chronic unemployment, economic strain and substance abuse [13;35;52].

Classification of MTBI severity

There are currently at least 25 different published injury-grading systems for MTBI. Most of these are impractical for clinical use in sport [27]. The definition of the grades of severity of MTBI has also changed over the years. The definitions of a few organizations and authors that are widely used are listed in Table 2.3 [39].

Table 2.3: Definitions of MTBI grades [39]

Congress of Neurological Surgeons (1966)
Mild - no loss of consciousness (LOC)
Moderate - a loss of consciousness with retrograde amnesia
Severe - unconsciousness lasting for longer than 5 minutes
Parkinson (1977)
Normal somatic mobility with impaired performance
Normal visceral mobility with impaired somatic mobility
Return of irregular visceral mobility with continuing somatic immobility
Visceral (respiratory) immobility and somatic immobility
Cantu (1986)
Mild - no LOC; post-traumatic amnesia (PTA) less than 30 minutes
Moderate - LOC lasting less than 5 minutes or PTA lasting longer than 30 minutes
Severe - LOC for 5 minutes or more, or PTA lasting longer than 24 hours
Colorado Medical Society (1990)
Mild - confusion without amnesia; no LOC
Moderate - confusion with amnesia; no LOC
Severe - LOC

Most of these classification systems are impractical for clinical use in sport [27]. In the review by Johnston the grading scales were broken down into a number of broad groupings [27]:

1. Surrogate head injury scales (with the aim to avoid missing the more severe brain injuries like cerebral hemorrhages, that may mimic concussion in the early stages.) There is however no evidence that these scales achieve this goal.

2. Neurosurgical scales (LOC and PTA are important outcome predictors in severe brain injury. The extrapolation of such concepts to milder grades of brain injury remains speculative.)
3. Sport-specific scale (too much emphasis on LOC and PTA)
4. Sporting injury scales (aim is to distinguish the trivial or marginally significant injuries from those that are more severe.) No scientific validation has been attempted with any of these scales.
5. Unclassifiable scales (the scaling system proposed is more reflective of pathophysiological constructs rather than clinical management.)

An overemphasis of the importance of LOC has probably contributed to the general misconception of injury severity amongst players, coaches and others. In particular, the absence of LOC has led to the subsequent downplaying of injury severity/ diagnosis of MTBI. This is especially the case at school- and college level rugby where appropriate medical care isn't always available.

It is now known that LOC is not a prerequisite for cognitive dysfunction [49]. In a study on the immediate effect of concussion on neurocognitive function in high school football players, significant deficits could be detected even without LOC, PTA or physical neurological abnormalities [58]. In this study subjects who experienced a brief period of PTA after injury were more impaired after the injury than those who did not experience PTA. Subjects with observed LOC did however display the most severe neurocognitive impairment immediately after and 15 minutes after concussion. This impairment resolved within two days post injury [58]. In a new evidence-based grading of MTBI, more weight on PTA as a predictor for subsequent impairment severity has been placed [59]. Cantu suggested that the final management decision following MTBI is a clinical judgment in every case. It may be appropriate to deviate from a particular set of guidelines depending on the individual circumstances [60].

The practical dilemma that the well-informed sports physicians faces when treating MTBI patients in rugby, is that the vast majority of these injuries are subtle and that the symptoms and consequences are constantly being downplayed by patients, institutions and parents involved in rugby. Added to this is the fact that there appears to be no perfect practical and scientifically valid scale that satisfies the needs of the clinician.

More recently, neuropsychological assessment of cognitive function has been proposed as a valuable tool helping the clinician to make appropriate severity and management decisions in sportspeople with MTBI.

Neuropsychological assessment of cognitive function

The neuropsychological assessment of cognitive function in sportspeople with mild traumatic brain injury is now recognized as the most valuable method for assessing brain injury severity. It represents the most sensitive, practical and objective method for delineating cognitive and neurobehavioral sequelae associated with MTBI. It can track recovery and can help to assist physicians in determining a player's readiness to return to competition [13;28;61].

The reliability, validity, and sensitivity of neuropsychological tests in assessing the specific cognitive areas associated with MTBI has been evaluated [13;31;47;62-64]. Neuropsychological testing involves the application of neuropsychological test instruments that are sensitive to subtle changes in attention, concentration, memory, information processing and motor speed or co-ordination [13;31;47;52;62;63;65].

In the past, neuropsychological testing has been limited by poor psychometric properties for serial study, including a limited range of possible scores, floor and ceiling effects and poor test-retest reliability. Accessibility was (and still is) poor and tests required a neuropsychologist or trained technician for administration, scoring and interpretation [62].

New approaches using shorter "paper and pencil" test batteries, as well as computerized tests, have been developed to overcome the problems faced with conventional neurological and neuropsychological testing techniques [62]. The sensitivity of such tests can be enhanced by obtaining baseline performance measures before commencement of the sporting season [50;65]. In a study on concussed Australian Rules footballers using a 15 minute computerized cognitive test battery (CogState), Simple Reaction Time (SRT) showed an increase in response variability and latency after concussion in injured athletes. This was in contrast with a decrease in response variability and no change in latency on follow up of the control players [66]. The CogState test battery includes

measures of sustained and individual attention, learning and memory, problem solving, and decision-making. This performance based test battery uses playing cards as stimuli, and is designed to have almost infinite equivalent alternative forms. CogState may be self-administered.

The problem is that the vast majority of secondary school and college rugby players in South Africa have never had a neuropsychological assessment. The majority of rugby players (at least at school and social college level in South Africa) with MTBI appear to be medically fit to continue participation on the basis of the clinical judgment only, and then of clinicians not always (medically) qualified to do so. Such judgments are often made with reference to the player's subjective rating of his symptoms or other non-standardized assessments of recovery. It is not uncommon for a player to sustain up to 5 'trivial' knocks to the head during a single match – the so-called very mild traumatic brain injuries (vMTBI) as mentioned earlier. It is well known that early re-entry into match playing, possess potentially serious neurological and cognitive consequences.

The use of portable computerized neuropsychological testing techniques offer the theoretical advantages of a number of randomized forms, standardized self administration, rapid testing and centralized data storage, analysis and reporting [62;66]. It has also been suggested that computer based cognitive tests may be more sensitive to cognitive impairment after sports related head injury than conventional neuropsychological tests [62].

The Immediate Measurement of Performance and Cognitive Testing (IMPACT) is a microcomputer based test battery that has been developed specifically for use in sport [5]. The test battery was designed to evaluate multiple aspects of neuropsychological functioning among athletes, including attention span, sustained and selective attention, reaction time, and several dimensions of memory [5]. The test battery was designed to minimize training effects that have limited the usefulness of many paper-and-pencil procedures.

As previously mentioned, in order to maximize the value of clinical neuropsychological assessment, baseline testing must be performed [5;13;47]. Individual players can vary with

respect to their responses in neuropsychological tests. It can be difficult to assess whether deficits detected during testing are attributable to the concussion or to other factors such as previous concussions, learning disabilities, attention deficit disorders, substance abuse, and test anxiety.

It is also important to realize that no study has shown that, once neuropsychological test results have returned to baseline, the athlete is safe to return to contact or collision sports. The fact that this form of testing may hold athletes from play longer than necessary, is another unresolved issue [13;47].

The ease of use and the relatively low cost of computerized neurocognitive testing add to the potential to expand this form of testing to all the school- and college rugby teams in South Africa. This will contribute to an increased knowledge of the incidence and neuropsychological consequences of MTBI, particularly of repetitive vMTBI's.

Very Mild Traumatic Brain Injury (vMTBI)

It is the general experience of most sports medicine physicians that athletes suffering a single vMTBI have minimal of the classical symptoms and signs of MTBI, if any. These symptoms and signs, if present, usually resolve rapidly. Symptoms may however appear late and may persist for extended periods. It may be that the player suffering from one or multiple vMTBI's seldom reports these late symptoms and signs for a number of reasons. The player with vMTBI often continues training and playing matches without being fully evaluated. However, repeated incidents of vMTBI's, as defined above, may pose a greater threat to the well being of the rugby player. It may very well be that a number of players suffer from symptoms and signs of the so-called post concussion syndrome, due to repetitive vMTBI's as the season progresses.

In animal models when repetitive vs. single impact of MTBI were investigated, no behavioral or histological changes were observed after a single 'sub-concussive' (vMTBI) impact. In these studies, it has been shown that repetitive vMTBI episodes often resulted in permanent injury [67]. It is therefore possible that, in humans, repetitive vMTBI's may result in significant impairment of an individual's physical, cognitive,

and psychosocial functioning. This may have a negative affect on interpersonal relationships, work and academic performance at school or college [13;35].

Rugby players at all levels are at risk, but especially those at school- and college level rugby, where the incidence and severity of MTBI and vMTBI are downplayed for reasons mentioned in Table 2.4. This may have serious consequences. The chance for a second MTBI after a single MTBI has been found to be four [14] to six [16] times over the chances for an initial MTBI. The same may apply for vMTBI.

A proportion of young individuals may have sustained what could be a permanent cumulative reduction in brain function at an early age. It should be apparent that, like in soccer, there are a number of factors to be considered before identifying a single factor for diminished cognitive function in rugby players. In this regard the role of previous MTBI's, alcohol and learning disabilities (LD) should be considered in evaluating neuropsychological performance.

It has been shown in a study of a large multi-university sample of football players, that a history of MTBI and LD are independently related to lower baseline cognitive performance. In this study a history of two or more prior MTBI's in an individual, was significantly and independently associated with long-term deficits in executive functioning and speed of information processing, as well as an increase in self-reported symptoms such as headache, dizziness, and trouble falling asleep. LD resulted in impaired executive function, speed of information processing, speeded word fluency and memory at baseline neuropsychological testing. This study also suggested an additive effect of learning disability and multiple episodes of prior MTBI on impaired neuropsychological functioning [63]. However, there is no scientific evidence that vMTBI and repetitive vMTBI's affect neuropsychological performance in rugby players.

Table 2.4: Reasons for downplaying the severity of symptoms/signs suggestive of MTBI and vMTBI in rugby

1.	**Player reasons**
	Fear of losing position to another player [14;47;57]
	Personal ego/ position in school/ society. Passionate or social need to play a particular sport [5]. It become less acceptable for the player to complain about injuries as they get older [36]
	Parental pressure [5;14;47]
	Under estimating possible negative consequences of injury/ re-injury. In a 1983 study by Gerberich 69% of high school football players who reported a loss of consciousness and 81% of players who reported a loss of awareness were allowed to return to play the same day. Of those 60% made the decision to return to play themselves while 29% were sent back on the field by the coach. A further 3% and 2% were sent back by a physician or parent respectively [14]
	Lack of proper education. Players don't always realize the symptoms they have as actually being a concussion [14]
	Missing out on probable selection for a better team
	Poor player selection
2.	**Team reasons**
	Pressure from team mates, coach, school/ university [5;14;47]
	Importance of future games
	Vital player
	Not enough depth in selection of reserves in team/ school
	Too much emphasis on winning at all cost as opposed to playing a good game and enjoying the sport
3.	**Administrative reasons**
	Unqualified or under-qualified medical attendants
	A medical team that is influenced by individual- or team factors
	Lack of uniformity in the definition of the injury [14]
	Lack of uniformity in management / return-to-play guidelines [14]
	Underestimating the severity of the injury [14]

	Lack of proper medical services at school/college matches. Many schools can't afford proper medical care; coaches, trainers and parents need to treat sports injuries with the danger that significant injuries will be overlooked
	Ignorance about the importance of neuropsychological testing (pre-season and ongoing)
	The limited knowledge and availability of computerized neuropsychological testing
	Poor refereeing
	Inappropriate rules
4.	**Nature of injury**
	VMTBI with no symptoms or signs
	VMTBI with delayed onset of symptoms and/or signs of MTBI
	MTBI with no signs and symptoms lasting only a few seconds

Current acute and long-term management of MTBI and the return to play guidelines

A number of guidelines for the evaluation of the athlete with MTBI have been published. This probably reflects the lack of consensus, which results from the absence of evidence-based data [13;67;68]. Good clinical judgment and individualized care by qualified emergency care personnel, physiotherapists, sports physicians and neurosurgeons should be the goal. Most of the general principles of management guidelines include the following: [46]

1. Any athlete with a MTBI or potential MTBI should be removed from the field of play after initial emergency management for further examination and observation
2. Serial assessment is important
3. If the athlete shows evidence of deterioration, no matter how 'mild' the initial injury seemed, they should be admitted to hospital for further diagnosis and management and neurosurgical consultation
4. An athlete with LOC (even transient) or amnesia should not return to playing field

5. No athlete should return to play until completely asymptomatic, both at rest and with exertion, both clinical and neuropsychological. The resolution of symptoms has multiple individual variance
6. The effects of recurrent episodes of MTBI may be cumulative

Initial sideline evaluation

It is important for the attending medical staff to always begin the evaluation with basic life support (airway and C-spine, breathing, circulation) followed by an assessment of the level of consciousness [13;67;68]. An unconscious player or a player with a suspected cervical spine injury should urgently be transported to a hospital for further diagnosis and support.

In an injured player where no loss of consciousness has occurred and no injury to the cervical spine is suspected, the patient can be moved to the sideline. A detailed history, including an assessment of the athlete's long- and short-term memory should be obtained. A Standardized Assessment of Concussion (SAC) as a neuropsychological on-site examination, has been developed to identify and objectively document the presence and severity of cognitive sequelae in football players, particularly as a result of MTBI [61]. The SAC includes measures of orientation, immediate memory, concentration, and delayed recall, as well as a total score to derive a composite index of injury severity. Pre-season testing to establish a baseline score for each athlete is used. The SAC lacks a measure of speed of information processing as well as a means to measure the cognitive-linguistic processes that have been noted to be affected by concussion. Two types of verbal fluency tasks are traditionally used to asses cognitive-linguistic processes – semantic and phonemic and can be used in conjunction with the SAC [69].

Early management in the medical room of the sporting facility or an emergency department.

A comprehensive history, including the history of the current injury, mechanism of injury, symptoms of MTBI, previous injuries and general medical history should be taken.

After the history a physical examination including a complete neurological examination should be conducted to establish an accurate diagnosis. In case of MTBI being diagnosed serial monitoring of neurological status and neuropsychological testing at various intervals until recovery should be done.

Transportation to hospital

Transportation to a hospital for further evaluation is indicated when the patient has sustained *any* loss of consciousness, any focal neurological deficit, and an injury to the cervical spine is suspected or when the patient's clinical condition deteriorates [46;70]. The indications for urgent referral are depicted in Table 2.5. [70].

Table 2.5: Indications for urgent referral to hospital

Any player who has or develops the following:
Fractured skull
Penetrating skull trauma
Deterioration in conscious state following injury
Focal neurological signs
Confusion or impairment of consciousness > 30 min
Loss of consciousness > 5 min
Persistent vomiting or increasing headache post injury
Any convulsive movements
More than one episode of concussive injury in a match or training session
Any assessment difficulty (e.g., an intoxicated patient)
Head injuries in children
High-risk condition (e.g., hemophilia, anticoagulant use)
Inadequate post injury supervision
Injury that results from high-risk mechanism (e.g., high-velocity impact, missile injury)

Special investigations in MTBI and vMTBI

If a more serious injury is suspected the injured player should be referred for x-ray evaluation of the cervical spine including flexion and extension views and a computed tomography (CT) scan to rule out neck- and head injuries that pose immediate danger, like acute epidural or subdural hemorrhage, and a neck or a skull fracture [13;67;68]. It has been proposed that early CT scan in all patients with a Glasgow coma scale (GCS) score of 13-14 should be performed [71].

Magnetic resonance imaging (MRI) scans are used to detect chronic changes, minor structural changes or control examinations within the first week after trauma [71]. It is more sensitive in detecting lesions within the first week and for discovering chronic lesions [71]. The problem with both CT and MRI scans is the expense and availability.

Clinical electroencephalography (EEG) is a safe and cost-effective investigation and is still widely used to evaluate MTBI [71]. It is generally accepted that EEG examination can show cerebral discharges or convulsion potentials in the late phase of so-called mild head injuries. EEG recovery is usually fast in patients with mild or moderate brain injuries [72]. The slowing of alpha rhythm is usually considered to be the slightest degree of generalized disturbance after MTBI [72] and may only be found with repeated EEG. The mildest disturbance of consciousness seen, following MTBI and vMTBI is drowsiness and hypersomnia. These are accompanied by EEG changes of normal sleep - a generalized slowing of all frequencies and altered sleep patterns. Sensory stimulation will block the slowing and sleep [72]. The EEG changes in the majority of these cases are however very subtle or minimal, if any.

In a study comparing 608 mild head trauma patients (GCS 13-15) without subsequent deterioration, to age-matched normals, the head-trauma patients could be discriminated from the age-matched controls by means of EEG with high accuracy (>90%). Two components of cerebral damage have been revealed: localized dysfunctions specific to the areas of maximal injury and a global or generalized state of reduced information processing capacity [73]. In a study comparing the EEG changes in 94 male patients who suffered from mild head trauma (GCS 13-15) the group with at least one sign of MTBI

(amnesia, anisocoria, changes of vigilance or vegetative symptoms such as vomiting) had more pathologic EEG records than those without any symptoms of MTBI [71]. The EEG abnormalities found were generalized in 76.4% and localized in 23.4% of those with an abnormal EEG. The majority of the localized changes (90.9%) were in the temporal region. The pathologies found in this study were irregular α-waves, high frequent α-waves, finding of τ-waves, increased β-waves, general dysrhythmia, β-spikes, τ-groups with spikes, slow α-waves and monophasic α-waves. The authors concluded that an EEG can be used for detecting pathologic unspecific alterations with high accuracy in mild head trauma, but that it is not useful in specifying an exact diagnosis [71]. Structural damage to the frontal lobes and temporal poles is more common than any other region of the brain. These regions are most important for psychopathological changes like concentration, orientation and cooperation. The vast majority of local changes have been found at the temporal pole of the brain [71]. In patients with postconcussional syndrome, focal EEG abnormalities strongly indicate the presence of brain damage, with the τ-band representing direct cortical damages [74].

In a study on the 'electrophysiological evidence for the cumulative effects of concussion' significantly longer P3 latencies were observed in individuals who had three or more episodes of a MTBI, compared to those who had never experienced a MTBI [67]. The cognitive event-related potential, the N2/P3 response, is a negative/positive potential elicited when a rare target is detected or counted in the midst of more prevalent non-target stimuli. The P3 amplitude is believed to index allocation of attention, while the latency is related to stimulus evaluation and categorization time, transfer of information to 'consciousness' and memory systems, and stimulus saliency [67]. In one study measuring the immediate neurocognitive effects of concussion in high school football players, who experienced "ding" injuries (concussion without LOC or PTA) exhibited significant deterioration from their pre-injury baseline levels of cognitive functioning [58].

Pharmacotherapy for concussion remains controversial and there is no evidence based data to support the use of any pharmacological treatment for concussion at present [51].

Return to play guidelines following MTBI

The criteria for readiness of an athlete to continue playing or to return to play following MTBI are a major and ongoing problem in sport [13;52;62;63]. A number of return-to-play grading scales have been published of which the Colorado Medical Society-, Cantu- [60], and the American Academy of Neurology guidelines have been widely used by clinicians all over the world. These three systems (Table 2.6) all grade MTBI severity on a scale of one to three using early post-traumatic signs, with a higher grade representing a more severe MTBI [75].

Table 2.6: Management of MTBI based on grade

Guideline	Frequency	Grade 1	Grade 2	Grade 3
Cantu [60]	1st concussion	Athlete may return to play if asymptomatic for a week (if athlete is totally asymptomatic return to play on the same day may be considered)	Atlete may return to play if asymptomatic for one week	Athlete may not return to play for at least one month, athlete may return to play if asymptomatic for a week
	2nd concussion	Athlete may return to play in 2 weeks if asymptomatic for a week	Athlete may not return to play for at least one month; athlete may then return to play if asymptomatic for a week	Terminate season
	3rd concussion	Terminate season	Terminate season	
Colorado Medical Society [64]	1st concussion	Athlete may return to play if asymptomatic for 20 minutes	Athlete may return to play if asymptomatic for a week	Athlete should be transported to a hospital emergency department; athlete may return to play one month after injury if asymptomatic for two weeks

	2nd concussion	Athlete may return to play if asymptomatic for a week	Athlete may return to play if asymptomatic for one month	Terminate season
	3rd concussion	Terminate season	Terminate season	
American Academy of Neurology [76]	1st concussion	Athlete may return to play if asymptomatic for 15 minutes	Athlete may return to play if asymptomatic for a week	Athlete should be transported to a hospital emergency department; if athlete had brief loss of consciousness (i.e. seconds), may return to play when asymptomatic for a week; if athlete had prolonged LOC (i.e. minutes), may return to play when asymptomatic for two weeks
	2nd concussion	Athlete may return to play if asymptomatic for one week	Athlete may return to play if asymptomatic for 2 weeks	Athlete may return to play if asymptomatic for one month or longer
	3rd concussion	No recommendation	No recommendation	No recommendation

The current guidelines for management and return-to-play issues have been controversial because of the lack of a scientific basis for constructing the management guidelines. The guidelines further assume a standard use for all groups (sport and non-sport MTBI) and all the playing levels of the individual sport. Individual variability in symptom presentation and the differing vulnerabilities to neurological injury at different ages, are not accounted for. There also appears to be an overemphasis on loss of consciousness compared with other symptoms and signs of concussion [52]. This means that it is possible for athletes, coaches and team physicians to find an injury scale and return-to-play guidelines that suit their sporting needs but which may not be the best objective medical management for the injury.

In a more recent, "Summary agreement statement of the first International Conference on Concussion in Sport, Vienna 2001", the aim was to 'provide continuing leadership in the continued development and updating of guidelines and maintenance of the pursuit of a high standard of care in concussion' [13]. Consensus was reached that an athlete with a MTBI should not return to the field of play until *completely* symptom free at rest and exercise [13;67;68;77]. However, it is not always possible to determine when an athlete is fully asymptomatic, and this may be problematic as symptoms can be minimal [75].

Guidelines published in 1986 stressed, "The final decision is a clinical judgment in every case" [60]. This may well be true today especially concerning the differences in the various guidelines. All the guidelines agree though that no athlete should return to competition while still symptomatic with post concussion symptoms. The athlete must be free of symptoms at rest and exercise and have a normal neurological examination [13;60].

Current management and return-to-play guidelines for very mild traumatic brain injury

Currently, there are no return-to-play guidelines for athletes with single or repetitive episodes of vMTBI. There is a possibility that a player with vMTBI or even repetitive vMTBI's may complete a match without having been evaluated properly. These injuries may fulfill the diagnostic criteria of MTBI [13] but may go unnoticed by the referee, coach, player, parent and attending medical team because of their subtle nature. The possible cumulative neurological damage, especially of repetitive injuries of this nature poses a threat to the well being of the young rugby player and for any athlete participating in a collision sport. The current state of management and return-to-play guidelines combined with the current lack of scientific data, probably mandates stricter control of the situation by the South African Rugby Football Union, at least at school and college level rugby and until more clarity emerges from good scientific studies. The possibility of short and long-term complications of MTBI may very well be apparent for vMTBI.

Complications of MTBI

Post concussion syndrome

The so-called post-concussion syndrome (PCS) is defined as persistent cognitive deficits following MTBI. Persistent cognitive deficits lasting longer than the generally believed recovery period of 3 months, suggest a biological and psychological interaction as a possible cause [67]. The syndrome is characterized by fatigue, dizziness, nausea, headaches, blurred vision, noise and light sensitivity, sleep disturbance, anxiety, hallucinations, loss of appetite, equilibrium disturbances or difficulty in concentrating [27;68]. Risk factors for PCS include returning to competition prematurely and repetitive injuries. If a second injury occurs before complete recovery of function is provided by dendritic arborization or spouting, further damage of neurons in the same system may reduce their capacity for recovery.

Chronic encephalopathy

Chronic encephalopathy can be defined as a progressive and chronic degeneration of brain structure and is characterized by among other axonal shear injuries. The patient suffers from memory loss, problems with executive function, Parkinsonian features including tremor, bradykinesia and dysarthria as well as ataxia and spastic tone. There has been concern that soccer players may develop a brain syndrome after years of exposure to episodes of MTBI and vMTBI as has been reported in boxers [39].

In boxers, the risk factors for chronic encephalopathy have been identified as the length of the career, later age at retirement, number of bouts (but not knock-outs), and possibly the total number of punches absorbed in a career (exposure index) [39]. Recent investigations suggest that patients with the apolipoprotein E4 genotype, have an increased risk to develop delayed boxer's encephalopathy [39].

The second impact syndrome

The so-called second impact syndrome (SIS) occurs when an athlete who has sustained an initial head injury, most often a concussion, sustains a second head injury before symptoms of the first injury have fully resolved [5;47;68;78-80]. The aetiology of SIS is thought to be a disruption of the auto regulatory system of the blood supply to the brain. A marked increased intracranial pressure results in herniation of the medial surface of the temporal lobe or lobes below the tentorium, or of the cerebellar tonsils through the foramen magnum. This rapidly develops into respiratory failure and death [78]. There is however some doubt if SIS really exists [79;80]. In the literature, 13 reports of sport-related catastrophic brain injury associated with unexplained cerebral swelling exist. In many of these cases, no second impact injury has been reported [79]. In these cases an "extremely rare catastrophic complication of single brain impact, namely diffuse cerebral swelling" is proposed as the cause. This complication of a single brain impact is more common in children and adolescents, and is typically but not exclusively associated with structural brain injury (e.g. cerebral contusion or subdural hematoma). Its causes are unknown, but may involve disordered cerebral vascular auto regulation [79].

Concussive or impact convulsions

Concussive convulsions can be defined as a form of convulsions that occur within 2 seconds of impact and are not associated with structural brain injury. The MTBI itself is thought to create a transient functional decerebration [70]. Late seizures do not occur, anti-epileptic therapy is not indicated and it is not necessary to withdraw from collision sport [70;81-83].

Improvements in the management of MTBI

Recent data suggest that MTBI in sport is an under-recognized but important entity, with the potential for long-term adverse sequelae deserving special attention [6;45;50]. Adverse long-term sequelae may be prevented by improved medical care, the use of protective equipment, rule- and competition changes at school- and college level for certain teams, better coaching, improved education and the implementation of cognitive and behavioral rehabilitation programs for affected individuals.

1. Improved medical care

Improved medical care, including an understanding of the assessment and management of MTBI, directly affects the long-term outcome of the injured individual [68;84]. A routine protocol for assessing athletes with head injuries, including the presence of adequately trained personnel, appropriate equipment and an emergency evacuation plan should be part of the management plan at the site of play.

- *At the site of play.*

On field emergency care should be available and a thorough sideline investigation, including a formal mental status examination should be done on each injured player. The proposed sideline evaluation includes a comprehensive history (memory of accident, questions pertaining to the hours before, questions about the game itself etc) and a physical examination (pupil size and response to light, cranial nerves, gross motor movement, consciousness, sensory function, cerebellar function with finger-to-nose testing and tandem gait, deep tendon reflexes) repeated every 10 minutes [36]. The standardized assessment of concussion (SAC) [61] by trained coaches/medical support staff has proven to be a sensitive and specific means of immediately detecting even mild grades of MTBI in the absence of observable neurological signs of injury, and is a gross predictor of post concussion injury [61]. A baseline preseason score with retesting at regular intervals immediately-, 15 minutes and 48 hours post injury, is recommended. It is important to understand that standardized screening instruments are not intended as a substitute for formal neurological evaluation, neuropsychological testing, and medical follow up of the injured individual. Nor is it intended to be a stand-alone method for clearing the athlete to return to competition [61]. The sum total of all the instruments available to the clinician should rather dictate return-to-play guidelines.

- *At the sports medicine clinic or hospital*

A complete history, mental status, and neurological examination are the gold standard. X-rays, CT scan, MRI scan or EEG examination should be considered for the patient at risk as mentioned above.

- *General medical principles*

The management team should enforce strict return-to-play guidelines based on regular assessment of the neurological status of the individual injured player by appropriately qualified clinicians.

Compulsory medical screening for all prospective collision sport players focusing on detecting potential risk factors may reduce the risk of permanent disability.
The child at risk and his parents should be counseled, including the option to pick another, safer sport. Potential risk factors for participating in collision sports are one previous episode of unconsciousness lasting longer than 5 minutes or two episodes of amnesia as a result of head trauma in one season, learning disabilities and previous severe head trauma like intracranial bleeding or a skull fracture [36].
The clinician should perform regular follow up evaluations of players after MTBI with early detection and treatment of complications. Clinical depression is fairly common after MTBI and may lead to significant psychosocial and functional impairment. Up to 77% of TBI patients suffer from a major depressive disorder and up to 35% of MTBI patients [85]. In a study two-thirds of mild TBI patients with major depression met remission criteria after an 8-week treatment trial of sertraline [85].

2. Protective equipment in rugby

- *Mouth guards*

The mouth guard has been defined "as a resilient device or appliance which is placed inside the mouth to protect against injuries to the teeth, lacerations to the mouth and fractures and dislocations of the jaw" [86]. There is no clear support in the scientific literature for the use of mouth guards to protect against head and spinal injuries in contact sports such as rugby. In a study of first-team rugby players in a residence league at Stellenbosch University, no significant differences in head and neck injuries (including MTBI, mouth, lip and tooth injuries) were found between players who wore and those who didn't wear mouth guards [87]. There is no convincing evidence to support a protective effect for mouth guards against any type of sporting injury [54]. Until randomized controlled trials of sufficient power to answer the question of the protective

role of mouth guard use are published, it is difficult for clinicians to accurately advise athletes on this issue. It would be safe though to advise on the use of mouth guards from an early age to give possible protection against injuries to the teeth, jaw and surrounding structures. In a study on the impact of face shield use on MTBI in intercollegiate ice hockey players, the use of a full-face shield compared with half face shield significantly reduced playing time lost because of MTBI. For both the full face shield and the half face shield those players who didn't wear a mouth guard at the time of injury missed significantly more playing time than players wearing a mouth guard [84].

The literature further provides some, though not convincing, evidence that mouth guards are effective in protecting against neck injury and MTBI through the repositioning of anatomical structures of the head and neck. It is recommended that the mouth guard in children be replaced every six months to allow for changes in the size and shape of the gums [11;84;86].

- *Headgear*

Recent studies of protective headgear have found that the role of protective light-weight and soft headgear to reduce MTBI in soccer, rugby and Australian football is minimal [88-90]. In sports where missile injuries (like in baseball) or falls on hard surfaces, like in cycling and ice hockey, is possible, there is evidence that helmets specific for the sport reduce head injuries [84;91;92].

The main role of headgear is protecting against lacerations and abrasions of the skull [81;93]. The belief that headgear is providing head protection may lead to higher player confidence. The risk of injury may thus be increased, especially in combination with inappropriate or badly designed headgear. Further research is needed to clarify this hypothesis. In a study on under 15 year old schoolboy rugby players to assess their perceptions on protective headgear, the two most common reasons given for wearing headgear were related to safety and injury prevention. They played more confidently but 63% said that their head was hotter [93].

As previously mentioned, the use of full-face shields in ice hockey players was associated with a significantly reduced risk of sustaining facial and dental injuries without an increase in the risk of neck injuries, concussion, or other injuries [84]. The use of full-face shield compared with half face shield by intercollegiate ice hockey players,

significantly reduced the playing time lost because of MTBI. This finding suggests that MTBI severity may be reduced by the use of a full face shield [84]. In cycling, helmet use reduces the risk of head injury by 85%, brain injury by 88% and severe brain injury by at least 75% [92].

3. Rule- and competition changes at school- and college level for certain teams

- Seven-a-side rugby may reduce the injury risk of players because the relative exposure of players is less - the duration of seven-a-side matches is shorter than the duration of a conventional fifteen-man rugby match. However, while the duration of matches is shorter, it may be required of players to compete repeatedly on the same day. This will increase the physiological demands, the onset of fatigue and may predispose to injury [41].
- Computerized neuropsychological evaluation - pre-season and ongoing in season, is the most sensitive clinical tool available to the clinician to identify injured players and to assist in return-to-play guidelines. Although the use of neuropsychological testing in high school and middle school-age athletes has not been adequately studied [47], it is an important tool if used individually and in conjunction with regular neurological evaluation.
- Protective equipment should also be worn during practice sessions.
- Penalties should be introduced during games for not wearing proper protective equipment, like in ice hockey [84;86].
- The forced participation of children in collision sports should be discouraged [36].

4. Coaching factors

Coaches should enforce the use of protective equipment, spend more time on skill training and development and reduce physical contact time in practice sessions.
Close communication between coaching and medical staff, not only during match play but also after the match may reduce the chance of an injured player returning to the playing field too early.

A change of position for injury-prone players may reduce the risk of further injury [16].
The teenage athlete should not be a pawn to be used by the coach, school or parent to produce a winning season.
Effective pre-season conditioning for example general fitness, conditioning of neck, abdominal and hip flexor muscles may reduce the chance of MTBI in collision sport. Weak neck muscles will result in more of a neck snapping action after impact with more rotational shearing strain to the intracranial structures [39].

5. Improved education

The education of players, parents, coaches and medical support teams on injury types, symptoms and risks may contribute to early recognition and appropriate management of the injured player. Many players don't recognize the symptoms and signs of MTBI. This makes it unlikely that a player will seek medical attention [38].
Counseling and educating prospective players and parents about the absolute and relative risks involved in rugby may reduce the injury risk by self-selecting the most appropriate player for the individual team position. Factors like pre-existing head trauma and learning disabilities have to be considered.

6. Cognitive and behavioral rehabilitation programs for affected individuals.

Cognitive and behavioral rehabilitation programs including cognitive exercises, compensatory devices and psychotherapy may improve the recovery of the player with MTBI. Access to rehabilitation services may be improved by public and private funding and the various National Rugby and Football Unions should be actively involved in this. Families, schools, and colleges should be involved and the educational needs of the young and school-aged children with MTBI and repetitive vMTBI should be addressed [35].

Conclusion

Head injuries in collision sports like rugby are inevitable. The nature of the game and the risks involved makes informed consent and medical clearance on an ongoing basis but especially before starting to play rugby, mandatory.

The precise incidence and the long-term consequences of MTBI and vMTBI in sport and in rugby in particular, require further investigation.

The possible consequences of these injuries, especially on the young rugby-playing population make inadequate clinical- and management guidelines not only inexcusable but also irresponsible. It is the duty of the medical community, with the help of the sport governing authorities like the National Rugby Football Unions, to initiate and enforce better care, including education on the risks involved. More data need to be collected in the form of large, prospective research studies to improve the diagnosis and clinical management of MTBI in rugby, and in collision sport in general. This is particularly important for very mild traumatic brain injuries.

Chapter 3

The end of season electroencephalographic and neuropsychological status of a team of secondary school rugby players – a comparison between very mild traumatic brain injury, mild traumatic brain injury and a sedentary control group

Abstract

Objectives- Although the incidence and consequences of mild traumatic brain injury (MTBI) in secondary school rugby has been well documented, little is known about the incidence and consequences of repetitive sub concussive injuries – the so called very mild traumatic brain injuries (vMTBI). The aim of this study was to compare the end of season neuropsychological and electroencephalographic (EEG) status as well as the academic performance of players in a secondary school rugby team who, during the course of the season sustained only repetitive vMTBI to those players who sustained MTBI and a sedentary control group.

Methods- A cohort of 20 secondary school male rugby players from a local secondary school's first rugby team, was followed for a full competitive season by a sports physician and trained biokineticists, who were present at all the games played. All vMTBI and MTBI and the severity of these injuries were documented. At the end of the season the players were divided into two study groups: group 1 (N=11, mean (SD) age 17.9 (0.54) years) sustained only vMTBI during the season; group 2 (N=9, mean (SD) age 17.5 (0.88) years) sustained one or more MTBI during the season. At the end of the season an EEG-test was done on each subject according to the international 10-20 system and on a third group, group 3 (N=9, mean (SD) age 17.3 (0.5) years) of age- and educational level matched controls who did not participate in any collision sport. Fast Fourier transformation of each EEG was used to obtain power spectrum areas in the δ (0-3Hz), τ (4-7Hz), α (8-13Hz) and β (14-25,30 Hz) frequencies. The mean amplitude for each of the 21 EEG positions and for the different wave types were calculated for each group and

compared. End of season Neuropsychological tests (Colour Trial Test 1 and 2, Digit Symbol Substitution Test, Wechsler Memory Scale-Revised (WMS-R) Test for verbal-, visual-, general-, delayed and attention/concentration) were conducted on the 3 groups. The academic results of different subjects (Afrikaans, English, Science, Mathematics and Latin) were obtained from the school and compared for the year that the study was done and for the two years preceding the study.

***Results*- Ten MTBI's were sustained in the 9 players of group 2 during the season. In group 1 five players reported between 2 and 4 vMTB injuries per game played, two reported between 5 and 9 vMTBI's per game played and four reported less than 2 vMTBI's per game played. The EEG test results showed that the mean amplitudes of the total, the β- and τ-waves' frequencies in the right temporal regions tested, were significant smaller ($p<0.05$) for the MTBI and the vMTBI groups when compared with the aged matched controls. The amplitudes for the other positions did not change significantly. Only one abnormal EEG, with suspicious theta wave activity in the right posterior region, was found in the study population in a player who sustained 2 MTBI's during the season. No stastically significant results were found for the neuropsychological tests and academic results of the different groups.**

***Conclusion*- The findings of this study indicate subtle EEG changes at the end of the rugby season, in the total, β- and τ-wave frequencies of the temporal region of secondary school rugby players who sustained vMTBI's and MTBI's during a single rugby season. This is the first evidence of possible temporary and sub clinical brain cortex abnormalities in the temporal region of rugby players who suffered only vMTBI's during a single rugby season. The abnormalities were the same as in the MTBI group. A multicentred prospective study, ensuring a big enough sample size with baseline, pre-seasonal EEG and neuropsychological data is necessary to evaluate these abnormalities further.**

Keywords: (Concussion, Minor head injury, Mild Traumatic Brain Injury, Very Mild Traumatic Brain Injury, Neuropsychological testing, EEG, Rugby)

Introduction

Head injury in sport is a growing concern worldwide. In the United States alone, approximately 350 000 athletes suffer from sports- and recreation related head injuries each year, the majority of these being mild traumatic brain injuries (MTBI) [23-25]. Public and professional concern about the number and the severity of MTBI's occurring in secondary school rugby is increasing, especially after the tragic death of four rugby players during the 2002 schools rugby season in South Africa due to head and neck injuries. At least one of them died from the so-called second impact syndrome. Injuries are inevitable in contact sports like rugby [1]. In fact rugby is considered to be one of the most dangerous sports being played. Specific situations of the game such as the "set scrum", the "rucks", the "maul" and the "tackle" leads to heavy bodily contact and increase the risks of injury, especially to the head and neck.

Concussion has been defined as a trauma induced alteration in mental status that may or may not involve loss of consciousness. In this chapter the terms concussion and mild traumatic brain injury (MTBI) are used interchangeably as is consistent with the medical literature [5].

In November 2001, the first International Symposium on Concussion in Sport was held in Vienna, Austria. Previous definitions for concussions had certain limitations; there was an inability to include relatively minor impact injuries that result in persistent physical and/or cognitive symptoms. Seeking to transcend these limitations, the Concussion in Sport Group (CISG) has developed the following definition of concussion: Concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces [13]. Several common features that incorporate clinical, pathological, and biomechanical injury constructs that may be used in defining the nature of a concussive head injury include:

1. Concussion may be caused by a direct blow to the head, face, neck, or elsewhere on the body with an "impulsive" force transmitted to the head.
2. Concussion typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously.

3. Concussion may result in neuropathological changes but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury.
4. Concussion results in a graded set of clinical syndromes that may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course.
5. Concussion is typically associated with grossly normal structural neuro-imaging studies.

Sports-related head injuries in the 1980's accounted for 19% of nonfatal injuries in football [14] and 4.5% of all high school sports injuries [15]. In a study reporting the epidemiology of schoolboy rugby injuries during one 18 –week season, in which players from 26 high schools in South Africa played 3350 rugby matches, 29% of all the injuries were to the head and neck. The incidence of concussion in this study was 12%. There was a marked underreporting of concussion by the schools that were monitored only by correspondence. In the more closely monitored schools the incidence was 18.4% [12].

From the above data, and taking the revised definition of concussion in sport into account, it is clear that MTBI are under diagnosed and possibly under treated, in contact sports, including rugby.

It has been suggested that the vast majority of head injuries in rugby are minor head injuries [10] or the so called very mild traumatic brain injury (vMTBI) and may be similar to the sub-concussive blows to the head from impacts during heading of the soccer ball[6].

VMTBI's are those injuries that are often not recognized by the off-the-field medical team as concussion because these injuries are often 'asymptomatic', subtle - a sub-concussive knock against the head. These injuries often cause only very short-term dizziness, headache and importantly there is no apparent amnesia or loss of consciousness. The player always continues to play and most of the time he finishes the match. The incidence and consequences of vMTBI's in rugby have not been studied well.

VMTBI's and MTBI's can be caused by the head being struck, the head striking an object, or the brain undergoing an acceleration / deceleration movement (i.e. whiplash) without external trauma to the head – the so called coup – contra-coup injury [16;16;17]. A minor head injury can occur without the athlete ever suffering a loss of consciousness [18]. For this reason, it is sometimes difficult to recognize a sports-related head injury.
The incidence and consequences of concussion in rugby can be reduced through the enforcement of rules, attention to technique, team skills and perhaps by the use of personal protective equipment like protective headgear [93].

A matter of concern thus with head injuries in rugby, is the incidence of repetitive blows to the head (minimal trauma to the head), or the so-called very mild traumatic brain injuries as mentioned earlier. The player with vMTBI suffers an impact injury to the head with only slight symptoms and/or signs. There is no loss of consciousness or amnesia and the player continues to play the match. This is the case with the current status of the diagnosis and treatment of vMTBI. The injury is either not recognized by the off-the-field medical team, or the player and the parent or coach downplay the severity and potential consequences of the injury.

That means a player sustains a blow or blows to the head in different situations of the game, continues to play with no definite signs of MTBI, but the outcome on the long-term may represent possible symptoms of MTBI including headaches, inaccurate memory, poor concentration and possible brain damage [19]. If these minimal blows to the head carry long-term consequences, it might indicate a need for action, but research on this topic is lacking. Neuropsychological testing can be of assistance to estimate the consequences of subtle head injuries [5;6;49;66;94].

However, there is no study that shows that, once neuropsychological testing has returned to baseline, the athlete is safe to return to contact or collision sports, nor has it been confirmed that this testing is not holding athletes longer than necessary.

Previous research has outlined the reliability, validity and sensitivity of neuropsychological tests in assessing the specific cognitive areas associated with MTBI in the general population [5;63]. Individuals may vary considerably in their performance on

many neuropsychological tests. It is thus difficult, if no baseline scores are available, to assess whether any deficits detected during testing are attributable to the effects of the injury or to previous unrelated factors [5;66].

Electroencephalography (EEG) is still widely used as a diagnostic tool, especially in the late phase of so-called mild head trauma. The EEG measures the background electrical activity of the brain and is recorded with scalp electrodes through the unopened skull. The electrodes are placed on the scalp according to the International 10-20 system of electrode placement. This placement is standard in many EEG laboratories and is supported by anatomic studies (Figure 3.1).

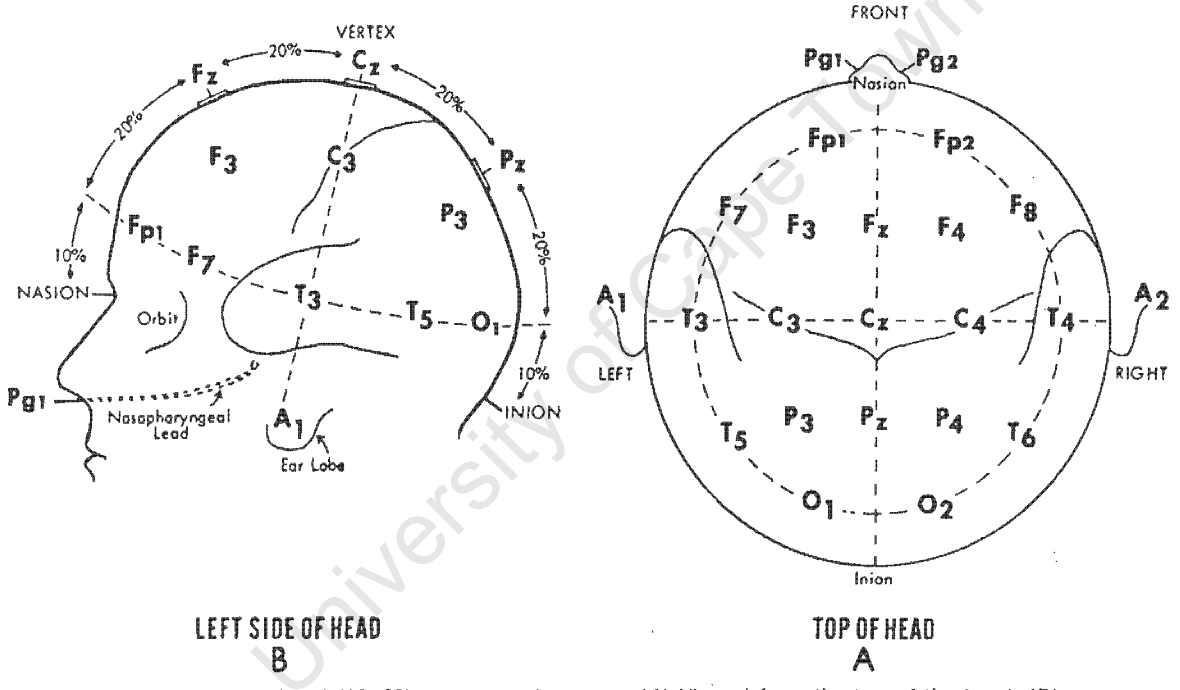

Fig. 3. International (10–20) electrode placement. (*A*) Viewed from the top of the head. (*B*) Viewed from the left side of the head. Odd numbers are on the left, even on the right; subscript *Z* means midline. A, ear (auricular); C, central; F, frontal; F_p, frontal polar; O, occipital; T, temporal; F_7, T_3, T_5, respectively, are inferior frontal, mid-temporal, and posterior temporal; F_7 and F_8 are commonly used as anterior temporal placements; P, parietal; Pg, nasopharyngeal. (Courtesy Grass Instrument Co.)

Figure 3.1: International (10-20) electrode placement

The cells in the cortex that give rise to the EEG are largely the pyramidal cells. These cells are arranged vertically in the cortex, with dendrites more superficial, and the cell bodies in the deeper cortical layers. They therefore give rise to vertically orientated cortical dipoles, which are negative on the cortex surface at the dendrites, and positive in the deeper cell bodies. The current flow about these dipoles constitutes the EEG.

EEG records may be bipolar or univocal. Bipolar records show fluctuations in potential between two cortical electrodes; univocal records show potential differences between a cortical electrode and a theoretically indifferent electrode on some part of the body distant from the cortex [72;95;96]. In an adult human at rest and with mind wandering and eyes closed, the most prominent component of the EEG is a fairly regular pattern of waves at a frequency of 8-12/s and amplitude of about 50μV when recorded from the scalp. This is the alpha rhythm. It is most marked in the parieto-occipital area. In addition to the dominant rhythm, 18-30/s patterns of lower voltage are sometimes seen over the frontal regions – the beta rhythm. A pattern of large 4-7/s waves called the theta rhythm occurs in children and is generated in the hippocampus in experimental animals. It may take up to 25 years for the EEG to mature. Large slow waves with a frequency of less than 4/s are the so called delta waves [72;95;96].

In an EEG study on 94 male patients who sustained mild head trauma (GCS 13-15) comparing those with at least one sign of cerebral concussion with those without any sign of concussion, more pathologic EEG records were found in the group with signs of concussion. In this study it was concluded that the EEG is safe and a low cost investigation that can be used for detecting pathologic nonspecific alterations with high accuracy [71]. The pathologies found were irregular α-waves, high frequent α-waves, finding of τ-waves, increased β-waves, general dysrhythmia, β-spikes τ-groups with spikes, slow α-waves and monophasic α-waves [71]. A generalized wave slowing was also found in the first hours after a head injury [97].

Although studies have been done on the effects of repetitive heading in soccer, no study has been done to compare the end of season EEG or neuropsychological status of secondary school rugby players with either vMTBI or MTBI, with a non-injured control group [8;9;22;71]. A study such as this could provide some insight in the incidence of very mild traumatic brain injuries in a secondary school rugby team and possibly outline the consequences of these injuries on the player in the medium to long-term. Symptoms of vMTBI like headache, dizziness, irritability, anxiety, blurred vision, insomnia, easy fatigability, and concentration and memory difficulty have been described [31]. This is especially important since the final academic examinations for secondary schools start

shortly after the end of the rugby season, while the equally important pre matric (final school examination) examinations are written during the rugby season.

The aim of this study was to investigate the end of season EEG- and neuropsychological status as well as academic results over three years of secondary school rugby players who sustained very mild- and mild traumatic brain injuries when compared with a non-injured and aged matched control group.

Methods

Subjects and study design

A secondary school rugby team (n=20) was monitored during one rugby season documenting and managing all MTBI's and vMTBI's. At the beginning of the rugby season 20 male players of a local secondary school rugby team enrolled in the study. A qualified sports physician and athletic trainer were present at all the practice sessions and the matches played. They monitored all the injuries sustained throughout the season. At the end of the season, the injured players were divided into two groups: Group 1 (n=11) consisted of players who sustained only vMTBI's and Group 2 (n=9) consisted of players who sustained MTBI's. These two groups were compared with a control group (n=9) of individuals who were not participating in any form of collision sport and who were without a history of previous head injury, alcohol abuse, epilepsy or any form of diagnosed learning disability. The control group was randomly selected from the same school and matched for gender, age- and education level. After the study was discussed with the participants and their parents, all the participants, parents and/or guardians gave informed consent. The Ethics Committee of the University of Cape Town approved the study.

At the end of the rugby season (7-10 days after the last rugby match), all the subjects underwent a single 20-minute standard EEG test while lying on a bed with their eyes closed. To test the reactivity of the alpha rhythm, they were asked to open their eyes twice. Each subject performed vigorous hyperventilation for 3 minutes. Photo stimulation was done on each subject with a probe held at a distance of 30cm from the

eyes. Typically flashes were given at varying rates including 1, 3, 6, 9, 11, 15, 20 and 30 Hz with each lasting 10 seconds and separated by 10 seconds from the next train of flashes. The EEG recordings were done in an EEG laboratory using a 32 channel portable Nihon Khoden machine (Nihon Kohden Corporation, Tokyo, Japan) and the EEG 2100 version 2.4 program (Nihon Kohden Corporation, Tokyo, Japan). Inter-electrode impedance was routinely checked, and did not exceed 5 KOhms. The EEG's were done and interpreted blinded by a qualified Clinical Technologist based on conventional EEG criteria. EEG Beckmann Ag-AgCl electrodes (Nihon Kohden Corporation, Tokyo, Japan) were affixed to the scalp with Elefix® conductive electrode paste (Nihon Kohden Corporation, Tokyo, Japan) after every position has been cleaned with Skinpure® (Nihon Kohden Corporation, Tokyo, Japan). Skinpure is a skin preparation gel for bioelectrical measurement and is designed to clean electrode sites and reduce skin surface impedance. The electrodes were positioned according to the international 10-20 system [96]. It is supported by anatomic studies. Although the actual distance between electrodes varies with the size and shape of the skull, these electrodes reflect the electrical activity over similar areas in different patients. The 10-20 system is so named because it divides each of three lines connecting skull landmarks into segments the length of which is 10% and 20% of the whole line. The baselines are the saggital distance from nasion to inion, the horizontal distance from the frontal polar midline (Fpz) to the occipital midline (Oz), and the transverse distance from the left to the right pre-auricular depression. Electrodes are located along each line at intervals of 10% and 20% of the entire length of the line, giving a total of 21 positions. Each position is named; it is given both an abbreviation, such as Fp for frontal polar, and a subscript number, such as Fp1 (left frontal polar). Odd numbers designate electrodes placed over the left hemisphere, and even numbers those placed over the right hemisphere. The subscript z indicates a midline position. The left and right ears are respectively labeled A1 and A2 [96]. Sensitivity for the EEG was set in the range of 5-10 μV/mm of open deflection. The low frequency filter was set at > 1 Hz and the high frequency filter at < 70 Hz. A paper speed of 3 cm/s was used. For the routine recording standard montages were used: longitudinal bipolar, transverse bipolar and referential. It is the convention in South Africa to place "right before left" when displaying the deviation of montages, opposite to the USA, but similar to Europe. The data analysis of the EEG was done by fast-Fourier transformation, made with a Focus version 2 software

package (Megis Software GmbH, Graefelfing, Germany), using steps of 0.2Hz for the power spectrum.

All the subjects underwent a battery of neuropsychological tests and two trained neuropsychologists interpreted these. The following neuropsychological tests were done: The Colour Trial Test 1 (CTT1), the Colour Trial Test 2 (CTT2), the Digit Symbol Substitution Test (DSST), and the Wechsler Memory Scale-Revised (WMS-R) subtests: Verbal Memory, Visual Memory, General Memory, Attention /Concentration and Delayed Memory.

The Digit Symbol Substitution Test (DSST) [98] consists of four rows containing in all, 100 small blank squares, each paired with a randomly assigned number from one to nine. Above these rows is a printed key that pairs each number with a different nonsense symbol. Following a practice trail on the first seven (WAIS-R) squares, the task is to fill in the blank spaces with the symbol that is paired to the number above the blank space as quickly as possible for 90 seconds. The score is the number of squares filled in correctly [99]. The DSST has been shown to be a sensitive and robust measure of cognitive function after concussion in Australian Rules football [66]. Several equivalent forms of the test exist, which minimize the practice effects that occur with repeated test administration.

In the Colour Trail Test-1 (CTT) [100] subjects are given a page with scattered circles numbered from one to 25, with even-numbered circles coloured yellow and the odd-numbered ones coloured pink. The task requires the subject to draw a line following the number sequence. Colour Trail Test-2 also presents the subject with a page containing 25 circles, but on this sheet each colour set is numbered: to 13 for the yellow odd numbers, to 12 for the pink even ones. The task is to follow the number series with a pencil but alternating between the two colours as well (1Y-1P-2Y etc.) The CCT was designed with psychometric properties similar to those found in the standard Trail Making Test (TMT), but it eliminates the use of alphabet letters. It instead relies on the universal concepts of colour and numbers. The CTT reduces the potential confounding effect of language and is, therefore, more applicable to cross-cultural research as well as to the clinical assessment of adults with limited education, English as a second language

(as were the subjects in this study), and reading and language disorders [100]. Additional scoring criteria that empirically capture evidence of subtle cognitive slippage that occurs following mild brain injury, were developed to assess these patients with acquired mild brain injury, particularly those whose premorbid functioning was high [100]

The Wechsler Memory Scale–Revised (WMS-R) [99] comprises a series of brief subtests, each measuring a different facet of memory [99]. It is an individually administered, clinical instrument for appraising major dimensions of memory functions in adolescents and adults. The functions assessed include memory for verbal and figural stimuli, meaningful and abstract material, and delayed as well as immediate recall [99]. The WMS-R is intended principally for detecting poor memory functioning, and most of its subsets therefore have relatively low "ceilings". The following subtests with their different measuring facets were used:

- *Information and Orientation Questions:*

  This subtest contains simple questions covering biographical data, orientation and common information from long-term memory. Questions 1 through 7 ask the examinee's name and other relative information. Questions 8 through 14 concern the individual's orientation in time (date, time of day) and place (Locality, place of testing). The last two questions concern the individual's hand preference and whether he or she has any impairments of hearing or vision that could influence performance on some of the WMS-R subtests [99].

- *Mental control:*

  The three items of this subtest are: 1) Counting backward from 20 to 1 (recording the time in seconds) with a time limit of 30 seconds, 2) Name the alphabet (recording the time in seconds) with a time limit of 30 seconds, and 3) Counting by 3's (recording the time in seconds) with the time limit of 45 seconds [99].

- *Figural memory:*

  This subtest measures memory for figural stimuli. Figural memory involves showing the examinee a set of abstract designs. Then, after each set of designs is removed, the examinee is asked to identify the designs within a larger set of designs [99].

- *Logical memory I (Immediate recall):*

  This subtest consists of two brief stories that are read to the examinee. After each story, the examinee retells the story from memory [99].

- *Visual paired associates I (Immediate recall):*

  This subtest requires the examinee to learn the colour associated with each of six abstract line drawings. In order to minimise the role of verbal mediation in memorising and responding to the figure-colour pairs, the colour names are not used either in presenting the items or in responding to them. The pairs are presented until the examinee answers all six items correctly; however, only the first three presentations are scored, and no more than six presentations are given. Using six presentations ensures that nearly all-nonimpaired examinees learn the material to the criterion of one perfect repetition [99].

- *Verbal paired associates I (Immediate recall):*

  In this subtest, the examinee is read a group of eight word pairs, then is read the first word of each pair, and is then asked to supply the second word from memory. Only the first three presentations are scored, although six presentations are given[99].

- *Visual reproduction I (Immediate recall):*

  The examinee looks at a geometric design and is then asked to draw it from memory [99].

- *Digit span:*

  The two parts of the Digit Span subtest are Digits Forward and Digits Backward, and these are administered separately. On Digits Forward, the examinee is read number sequences of increasing length, and after each sequence the subject is asked to repeat it from memory. On Digits Backward the examinee is read a similar number sequences, and after each sequence the subject is asked to repeat it backwards. The Digits backward is administered even if the examinee performs poorly on Digits Forward [99].

- *Visual Memory Span:*

  The two parts of the Visual Memory Span subtest, are Tapping forward and Tapping backward and these are administered separately. On Tapping Forward the examinee watches the examiner touch the red squares on Card 1 in sequences of increasing length, and after each sequence, the subject is asked to repeat the performance from memory. On Tapping Backward the examinee watches the examiner touch the green squares on Card 2 in sequences of increasing length, and the subject is asked to repeat the performance in reverse. The Tapping Backward is administered even if the examinee performs poorly on Tapping Forward [99].

- *Logical Memory II (Delayed recall):*

  This subtest is administered at least 30 minutes after completion of Logical Memory I test. If necessary a pause is allowed after the Visual Memory Span to ensure that at least 30 minutes have elapsed [99].

- *Visual Paired Associates II (Delayed recall):*

  This subtest was administered at least 30 minutes after completion of Visual Paired Associates I test [99].

- *Verbal Paired Associates II (Delayed recall):*

  This subtest was administered at least 30 minutes after completion of Verbal Paired Associates I test [99].

- *Visual Reproduction II (Delayed recall):*

  This subtest was administered at least 30 minutes after completion of Visual Reproduction I test [99].

Statistical analysis

For the statistical analysis, the software package STATISTICA (Version 6.0, Stat Soft, Tulsa, OK) was used. Descriptive statistics were used on each group to obtain the means and standard deviation on the neuropsychological tests (CTT1, CTT2, DST, WMS-R) and on the EEG data (mean amplitude in μV) as shown in Table 3.2 and 3.3 respectively. Two-way repeated measures ANOVA's were used to compare the

academic results of Afrikaans, English, Science and Mathematics and Latin combined. One way ANOVA's were used to compare the different group's performance in the neuropsychological battery of tests. One way ANOVA Post-hoc Tuckey HSD analysis were applied to examine significant differences between pairs of means in each group on the results of all the neuropsychological tests, the amplitudes of the different waves in all 21 EEG-positions and on the academic results. The p-value for significance was set at < 0.05.

The relationships between the EEG-, neuropsychological-, and academic variables were analysed using Pearson's product moment correlation and statistical significance was set at $p < 0.05$.

Results

The age of the rugby players in group 1 (N=11, mean (SD) age, 17.9 (0.54) years), group 2 (N =9, mean (SD) age, 17.5 (0.88) years), and the control group (N = 9, mean (SD) age, 17.3 (0.5) years) did not differ significantly, with a mean age of 17.6 ± 0.68 years in the entire group.

The nine players in group 2 sustained a total number of ten MTBI's. Two of these injuries were associated with post traumatic amnesia of more than 30 minutes and loss of consciousness of more than 5 minutes. Both players were hospitalized and discharged after a period of observation. The average time out of competition for the two players was 16.8 days. The MTBI players also reported vMTBI's: of the nine players in the MTBI-group, six reported vMTBI's of 2-4 per game played and three less than two per game played.

Of the eleven players in group 1, two sustained MTBI's during the previous rugby season. Four players reported less than two vMTBI's per game during the season; five reported 2-4 vMTBI's per game played and two between 5-9 vMTBI's per game played. Group 1 and 2 sustained, conservatively measured, an average total of 31 and 21 vMTBI's respectively per game played.

Electroencephalographic tests results

Only one of the subjects had an abnormal EEG in the form of suspicious theta waves in the right posterior region (Figure 3.2). The patient with the abnormal EEG sustained two MTBI's during the season and was out of competition for a total of 21 days. When the normal mean amplitudes of the different waves in the different positions were analyzed, there were smaller amplitudes of the theta waves (4 – 8 Hz) in the right temporal region (T4-T6) in group 2 when compared to group 3 (p=0.015) and in group 1 when compared to group 3 (p=0.01) (Figure 3.2). Differences in mean amplitude in this region between group 1 and 2 were not statistically significant. The same observations were made for the theta wave frequencies on the left temporal region (T3-T5) in group 1 when compared with group 3 (p=0.03). The mean amplitudes of the beta waves (13 – 30 Hz) in the right temporal region (T4-T6) were statistically significant smaller in group 2 vs. group 3 (p=0.015) and in group1 vs. group3 (p=0.01) (Figure 3.2). Differences between groups 1 and 2 were not statistically significant. The mean amplitudes of the total waves (1-30Hz) were statistically significant smaller in the right temporal area (T4-T6) of both groups 2 (p=0.004) compared with group 3, and for group 1 (p=0.006) compared with group 3 (Figure 3.3).

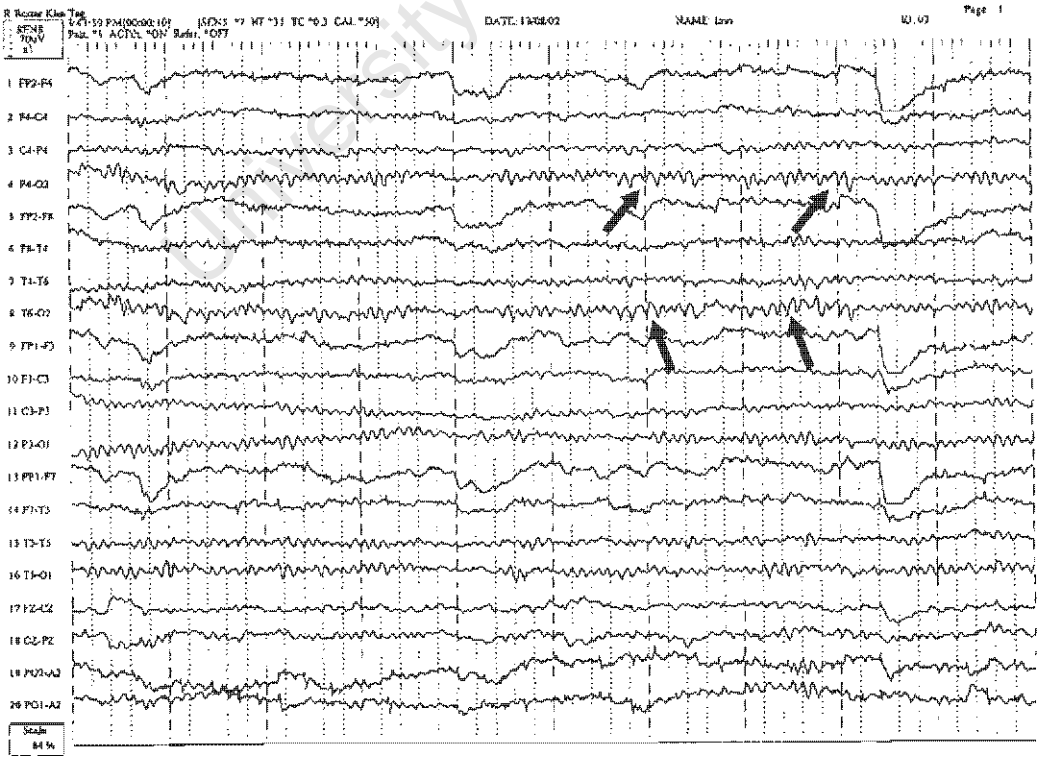

Figure 3.2: Abnormal EEG; suspicious theta waves in the right posterior region

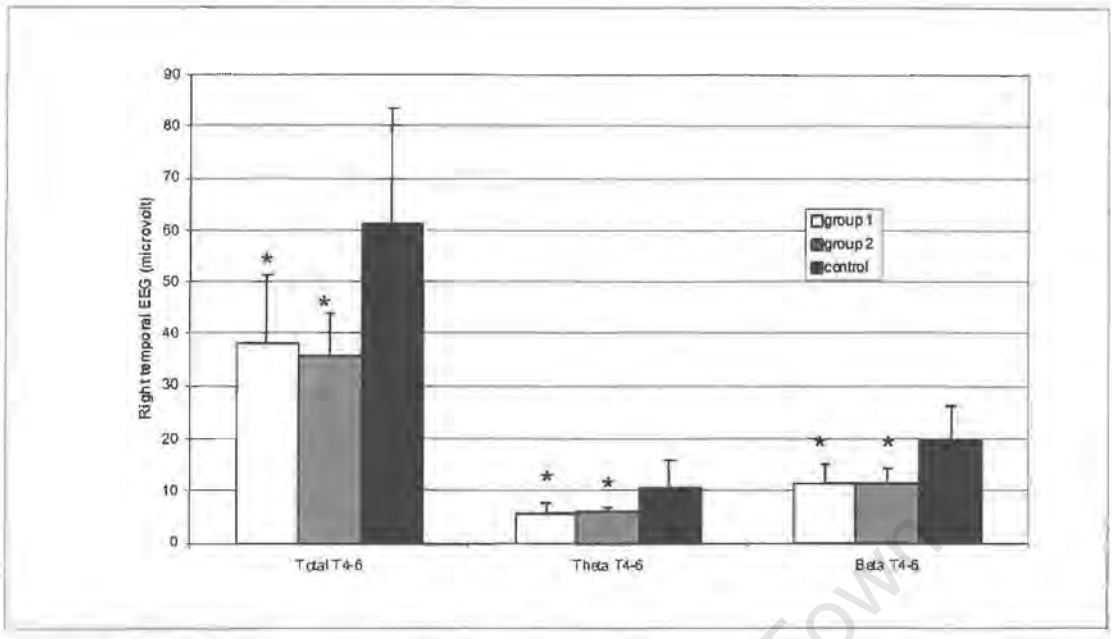

Figure 3.3: Mean micro volt in the right temporal area of different groups. Values are mean ± SD. * p<0.05.

Table 3.1 gives descriptive information on the EEG performed on the subjects. The positions on the head that detect the total, theta, alpha2 and beta waves, are described in all the groups towards mean amplitude in μV and SD. Statistically significant p-values are printed in italics.

Table 3.1: Descriptive information on the mean μV for each frequency wave in the different positions of the EEG

Group 3:									
Variable		C4-P4	P4-02	T4-T6	T6-O2	C3-P3	P3-O1	T3-T5	T5-01
Total	**N**	9	9	9	9	9	9	9	9
[1.0-30.0 Hz]	**Mean**	45.6	57.9	61.3	52.6	44.5	55.5	53.3	51.3
	SD	16.9	22.3	22	18.8	16.6	22.7	21.7	18.2
	p-value (a)	0.16	0.69	*0.004*	0.98	0.28	0.51	0.07	0.89
Theta	**N**	9	9	9	9	9	9	9	9
[4.0-8.0 Hz]	**Mean**	8.5	10.7	10.4	9.9	8.1	10.2	9.1	9.7
	SD	3.7	5.2	5.3	4.4	3.1	4.7	4.5	4.2
	p-value (a)	0.08	0.42	*0.015*	0.71	0.14	0.26	0.11	0.55

Beta	**N**	9	9	*9*	9	9	9	9	9
[13.0-30.0 Hz]	**Mean**	12.6	14.8	19.7	14.3	11.4	13.7	16.9	13.1
	SD	4.4	5.2	8.3	4.7	3.9	5.1	8.6	4
	p-value (a)	0.18	0.66	*0.015*	0.89	0.44	0.61	0.16	0.89
Alpha2	**N**	9	9	9	9	9	9	9	9
[8.0-13.0 Hz]	**Mean**	15.6	22.1	19.8	18	15.4	21.8	17.6	18.8
	SD	7.8	11.9	11.3	9.2	8	12.7	10.5	9.9
	p-value (a)	0.4	0.83	0.057	0.97	0.51	0.64	0.17	0.97

Group 1:									
Total	**N**	11	11	11	11	11	11	11	11
[1.0-30.0 Hz]	**Mean**	39.4	58.7	37.9	59.5	39.5	50.6	38.9	51.9
	SD	13.7	35.1	13.1	35.2	12.2	23.9	10.2	25.6
	p-value (b)	0.550	0.990	*0.006*	0.820	0.650	0.860	0.078	0.990
Theta	**N**	11	11	11	11	11	11	11	11
[4.0-8.0 Hz]	**Mean**	6.6	9.4	5.7	9.1	6.5	8	5.9	8.3
	SD	2.4	5.3	1.9	4.8	1.9	3.8	1.8	4.2
	p-value (b)	0.25	0.77	*0.01*	0.89	0.24	0.38	*0.03*	0.65
Beta	**N**	11	11	11	11	11	11	11	11
[13.0-30.0 Hz]	**Mean**	10.7	14.4	11.3	15.4	11.0	12.6	11.9	12.9
	SD	3.9	8.6	5.1	8.3	3.3	6.0	2.5	6.2
	p-value (b)	0.60	0.98	*0.01*	0.92	0.92	0.87	0.12	0.99
Alpha2	**N**	11	11	11	11	11	11	11	11
[8.0-13.0 Hz]	**Mean**	14	24.6	13.1	24.8	13.9	20.5	13.2	21.1
	SD	6.9	17.8	6.5	18.8	6.4	11.9	5.9	12.8
	p-value (b)	0.86	0.91	0.17	0.51	0.85	0.96	0.38	0.87

Group 2:									
Total	**N**	9	9	9	9	9	9	9	9
[1.0-30.0 Hz]	**Mean**	33.8	47.8	35.6	50.4	35.2	44.6	37.9	47
	SD	7.2	12.5	8.2	14.5	7.7	12.9	6.6	11.2
	p-value (c)	0.62	0.63	0.94	0.71	0.74	0.79	0.98	0.85
Theta	**N**	9	9	9	9	9	9	9	9
[4.0-8.0 Hz]	**Mean**	5.8	8.1	5.8	8.4	6.1	7.5	6.4	7.9

	SD	0.9	1.7	1.1	1.99	1.2	1.8	1.3	1.6
	p-value (c)	0.75	0.80	0.99	0.93	0.90	0.94	0.91	0.97
Beta	**N**	9	9	9	9	9	9	9	9
[13.0-30.0 Hz]	**Mean**	9.2	12.3	11.3	13.0	9.6	11.5	12.1	12.1
	SD	1.6	3.4	3.5	3.5	1.6	3.3	3.2	2.7
	p-value (c)	0.62	0.73	0.99	0.67	0.63	0.88	0.99	0.92
Alpha2	**N**	9	9	9	9	9	9	9	9
[8.0-13.0 Hz]	**Mean**	11.5	18.4	10.7	19.2	11.9	17.1	11.3	17.7
	SD	5.1	7.5	4.7	8.2	4.8	6.9	3.9	6.7
	p-value (c)	0.68	0.57	0.77	0.64	0.80	0.77	0.82	0.75

p value (a) = group 2 versus group 3 p value (b) = group 1 versus group 3 p value (c) = group 1 versus group 2

C4 - P4 = Central 4 – Parietal 4 (right)

P4 - O2 = Parietal 4 – Occipital 2 (right)

T4 - T6 = Temporal 4 – Temporal 6 (right)

T6 - O2 = Temporal 6 – Occipital 2 (right)

C3 - P3 = Central 3 – Parietal 3 (left)

P3 - O1 = Parietal 3 – Occipital 1 (left)

T3 - T5 = Temporal 3 – Temporal 5 (left)

T5 - O1 = Temporal 5 – Occipital 1

For group 1 (vMTBI) correlations (r) between the mean amplitudes of the beta frequencies in the right temporal region (beta (T4-T6)) and the mean amplitudes of the beta (T3-T5) (0.61), theta (T4-T6) (0.85), theta (T3-T5) (0.78) and total (T4-T6) (0.78) frequencies were significant ($p < 0.05$). In group 1 correlations between mean amplitudes of beta (T3-T5) frequencies and the mean amplitudes of the theta (T4-T6) (0.64), theta (T3-T5) (0.67) and total (T3-T5) (0.66) frequencies were significant ($p < 0.05$). Correlations between the mean amplitudes of theta (T4-T6) frequencies and the mean amplitudes of the theta (T3-T5) (0.95), total (T4-T6) (0.97), total (T3-T5) (0.79), alpha2 (T4-T6) (0.75) and alpha2 (T3-T5) (0.65) frequencies were significant ($p < 0.05$). Correlations between the mean amplitudes of theta (T3-T5) frequencies and the mean amplitudes of the total (T4-T6) (0.96), total (T3-T5) (0.92), alpha2 (T4-T6) (0.83) and alpha2 (T3-T5) frequencies were significant ($p < 0.05$). Correlations between the mean amplitudes of the total (T4-T6) frequencies and the mean amplitudes of the total (T3-T5) (0.86), alpha (T4-T6) (0.86) and alpha2 (T3-T5) (0.77) frequencies were significant ($p < 0.05$). Correlations between the mean amplitudes of the total (T3-T6) frequencies and the mean amplitudes of the alpha (T4-T6) (0.86) and alpha2 (T3-T5)

(0.92) frequencies were significant ($p < 0.05$). Correlations between the mean amplitudes of the alpha2 (T4-T6) frequencies and the mean amplitudes of the alpha2 (T3-T5) (0.94) frequencies were significant ($p < 0.05$).

For group 2 (MTBI) correlations between the mean amplitudes of the right temporal beta frequencies (beta (T4-T6)) and the mean amplitudes of the beta (T3-T5) (0.95) and the total (T4-T6) frequencies were significant ($p < 0.05$). Correlations between the mean amplitudes of the theta (T4-T6) frequencies and the mean amplitudes of the theta (T3-T5) (0.70), total (T4-T6) (0.87) and the total (T3-T5) frequencies were significant ($p < 0.05$). Correlations between the mean amplitudes of the theta (T3-T5) frequencies and the mean amplitudes of the total (T3-T5) (0.81) frequencies were significant ($p < 0.05$). Correlations between the mean amplitudes of the total (T4-T6) frequencies and the mean amplitudes of the total (T3-T5) (0.77) and the alpha2 (T4-T6) (0.83) frequencies were significant ($p < 0.05$). Correlations between the mean amplitudes of the total (T3-T5) frequencies and the mean amplitudes of the alpha2 (T3-T5) (0.70) frequencies were significant ($p < 0.05$). Correlations between the mean amplitudes of the alpha2 (T4-T6) frequencies and the mean amplitudes of the alpha2 (T3-T5) (0.81) frequencies were significant ($p < 0.05$). Correlations between the difference in academic performance in Science, Mathematics and Latin combined for year 1 and 3 (3 = year of study) and the mean amplitudes of both beta (T4-T6) (-0.86) and beta (T3-T5) (-0.75) frequencies were significant ($p < 0.05$), indicating that those players with smaller mean amplitudes in the beta frequency waves of the temporal area, performed worse in these 3 subjects combined in the year the study was done (Figure 3.4).

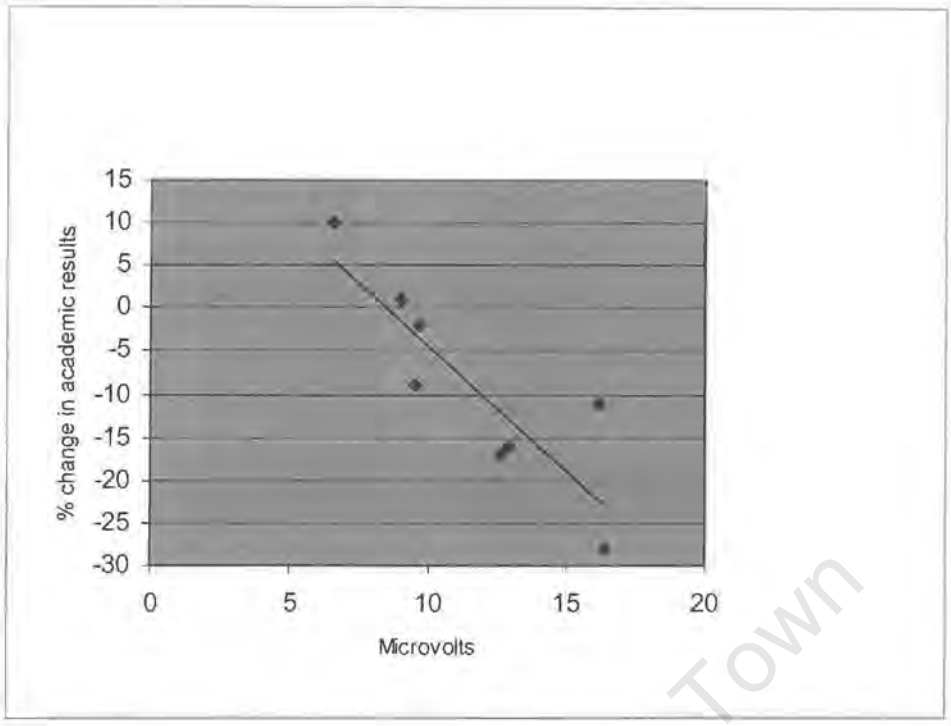

Figure 3.4: Correlation between amplitudes of beta frequencies in the right temporal area of group 2 and % change in academic results of Mathematics, Science and Latin combined between year 1 (2 years before study) and year 3 (year of study); r=-0.86; p=0.006.

Academic results

There were no statistically significant differences in the academic performance of the 3 groups over three years (year of study and 2 preceding years).

Neuropsychological test results

In Table 3.3 descriptive information on the results of the neuropsychological tests used in this study is depicted. The mean score on each neuropsychological test is based on a Z score (Table 3.2) to classify the ability level of the subjects. The Z score on CTT1 and CTT2 are reversed (for instance a score of -0.50 is normally classified as low average, but here it is classified as high average).

No statistically significant differences were observed between the 3 groups studied.

The CTT1 was average in all the groups but CTT2 was high average in all the groups.

Classification on DST, attention or concentration, delayed memory, general memory and verbal memory in all the groups were average. Visual memory was average in both group 1 and group 2, and was high average in the control group.

Table 3.2: Classification of ability levels

Classification	*Z*-Score
• Very superior	+2.0 and above
• Superior	+1.3 to 2.0
• High average	+0.6 to 1.3
• Average	±0.6
• Low average	-0.6 to -1.3
• Borderline	-1.3 to –2.0
• Retarded	-2.0 and below

Table 3.3: Descriptive information on the neuropsychological tests

Variable	Group	N	Mean	SD	p value
CTT 1 (Colour Trial Test 1)	1	11	-0.51	0.83	a 0.97
	2	9	-0.22	0.90	b 0.90
	3	9	-0.33	1.11	c 0.78
	Total	29	-0.37	0.91	
CTT 2 (Colour Trial Test 2)	1	11	-0.85	0.43	a 0.85
	2	9	-0.87	0.49	b 0.90
	3	9	-0.74	0.59	c 0.99
	Total	29	-0.82	0.49	
DSST (Digit Symbol Substitution Test)	1	11	0.17	0.43	a 0.51
	2	9	0.26	0.52	b 0.29
	3	9	0.57	0.78	c 0.93
	Total	29	0.33	0.59	

WMS-R					
Verbal memory	1	11	0.18	0.95	a 0.94
	2	9	0.08	0.75	b 0.83
	3	9	-0.05	0.88	c 0.97
	Total	29	0.08	0.84	
Visual memory	1	11	0.35	0.96	a 0.49
	2	9	0.29	0.98	b 0.56
	3	9	0.75	0.45	c 0.98
	Total	29	0.45	0.83	
General memory	1	11	0.29	0.96	a 0.99
	2	9	0.21	0.71	b 0.97
	3	9	0.21	0.83	c 0.97
	Total	29	0.24	0.82	
Attention/ Concentration	1	11	-0.31	0.95	a 0.54
	2	9	-0.29	0.60	b 0.49
	3	9	-0.08	0.58	c 0.99
	Total	29	-0.18	0.75	
Delayed memory	1	11	0.11	0.93	a 0.88
	2	9	0.14	0.79	b 0.83
	3	9	0.35	0.97	c 0.99
	Total	29	0.19	0.88	

Group 1 = Sub-concussive blows against the head.
Group 2 = Concussion.
Group 3 = Control group.
WMS-R = Wechsler Memory Scale-Revised.

p value (a) = group 2 versus group 3
p value (b) = group 1 versus group 3
p value (c) = group 1 versus group 2

Discussion

The finding of this study is that there are subtle but statistically significant reductions in the amplitudes of the theta wave frequencies (4 – 8Hz) of both the left (T3-T5) and right (T4-T6) temporal regions in the group of players with repetitive vMTBI's during a

single rugby season when compared with a normal age matched control group. The same results were obtained in the right temporal region (T4-T6) of the group of players with MTBI's during a single rugby season when compared with the control group. The amplitudes of the theta waves in the temporal region were on average smaller in the injured groups than in the age-matched control group.

The amplitudes of the beta waves (13 – 30 Hz) were on average significantly reduced in the right temporal (T4-T6) region of both the groups with MTBI and vMTBI when compared with the average beta wave amplitudes of the control group in the right temporal region.

The average amplitudes for the total wave frequencies (1-30Hz) were reduced in the right temporal region of both the players with MTBI and those with vMTBI's when compared to the average amplitudes of the total wave frequencies of the control group in the right temporal region. The fact that the mean amplitudes of the different waves in different positions correlated statistically significant for both groups 1 and 2 indicated that the smaller amplitudes were a diffuse phenomenon in the affected players.
Although drowsiness is a problem during the EEG tests and this may confound EEG data, the Clinical Technologist observed no apparent difference in drowsiness between the groups. The mildest disturbance of consciousness seen, following MTBI and vMTBI is drowsiness and hypersomnia. These are accompanied by EEG changes of normal sleep – a generalized slowing of all frequencies and altered sleep patterns [72]. In one study on 94 male patients who suffered from mild head injury, the majority of localized EEG changes (90.9%), were in the temporal region [71]. Structural damage to the frontal lobes and temporal poles is more common than any other region of the brain. These regions are most important for psychopathological changes like concentration, orientation and cooperation [71]. In patients with postconcussional syndrome, focal EEG abnormalities that strongly indicate the presence of brain damage, are in the τ-band and represent direct cortical damage [74]. It has been shown that cortical damage, including neuronal death secondary to organelle failure and somatic cytoskeletal damage, is caused by excitatory amino acids, that precipitate the influx of sodium and calcium ions [23]. The localized cell death may very well lead the to subtle EEG changes observed in the temporal poles of the subjects in this study.

To our knowledge the relationship between EEG data, neuropsychological- and academic performance in secondary school rugby players has never been investigated. The fact that the difference in academic performance in Science, Mathematics and Latin combined between year one and year 3 (3= year of study) correlated significantly with the mean amplitudes in both the right (-0.86) and left (-0.75) temporal beta wave frequencies of players who sustained MTBI during one rugby season, may indicate that subtle cortical brain damage negatively impacts on the performance of rugby players in analytical subjects. This may be aggravated by the fact that many important examination papers are written during and shortly after the end of the rugby season.

The value of neuropsychological testing in determining persistent and continuing cerebral effects of head injuries sustained during athletic endeavors has been illustrated at many levels of competition (including high school, collegiate, and professional) and across many sports [64].

In a study on concussed Australian Rules footballers using a 15 minute Computerised cognitive test battery (CogState), Simple Reaction Time (SRT) showed an increase in response variability and latency after concussion in the injured players. This was in contrast with a decrease in response variability and no change in latency on follow up of the control players [66]

Groups of individuals with mild head injuries presenting at emergency service departments in hospitals, have shown statistically significant differences in measures of many aspects of cognitive functioning including attention, concentration, short-term memory, and information processing speed, compared with matched controls [94;101-103]. Other studies failed to find any differences between mild head-injured groups at one month post-injury and controls [104;105]. Research focusing exclusively on the contact sport of football has suggested that recovery to baseline usually occurs within 5 to 10 days of injury [106]. Different reasons for such rapid recovery in athletes like less severe injuries, greater motivation to recover and the younger age of the sports sample compared to head-injured samples in the general population, have been proposed [57]. The fact that the neuropsychological testing in this study was done only at the end of the

rugby season may have resulted in some players having recovered from their injuries by the time of testing. The lack of pre-season neuropsychological testing further compromised the validity of the neuropsychological data.
Pre-season testing is essential and the sooner neuropsychological testing is implemented post injury, the better [64].

In conclusion, this study shows that there is a shift in the EEG power distribution with a general reduction in amplitudes over a wide frequency spectrum, mainly in the temporal region of secondary school rugby players, who suffered MTBI and repetitive vMTBI's during a single rugby season. These observations suggest that there is a sub clinical and temporary dysfunction in the electrical activity of the brain's temporal region, probably due to some form of cortical damage that is caused by the repetitive insults of MTBI's and vMTBI's to the brain.

Recommendations

The main limitation of this study is the lack of pre-season testing and the small sample size. It is recommended that a larger, multicentred, prospective study with pre-season baseline testing be conducted in future in secondary school and college rugby players to confirm our finding of EEG abnormalities associated with possible cognitive impairment due to repetitive vMTBI's in rugby.
It is further possible that more widespread cortical damage existed, but due to the time delay before EEG testing at the end of the season, most of these could have resolved. It is therefore recommended that EEG testing of injured players should be done within hours after finishing a rugby game and compared with pre season EEG tests.

Chapter 4

Summary and conclusion

It is likely that the incidence and consequences of MTBI and vMTBI in secondary school rugby may be under-reported [12]. The potential long-term sequelae pose a direct threat to the neuropsychological well being of the young rugby player with MTBI and vMTBI. Our study of a secondary school rugby team has shown subtle and sub-clinical abnormalities in the amplitudes of the end of season EEG's over a broad frequency spectrum in the temporal regions in both the players with MTBI and vMTBI. The neuropsychological status and academic performance in the groups were the same. This may point to subtle and probably up to now unrecognized abnormalities in the temporal cortex of rugby players with vMTBI. Our study sample size was small and we were not able to conduct pre-season evaluations. A further study with a larger subject group and with pre-season evaluation is recommended. In the meantime it may be argued that stricter management guidelines for vMTBI, especially at school- and college-level rugby, is needed.

It has been acknowledged that the science of concussion is at the early stages and therefore management and return to play decisions is largely in the realm of clinical judgment on an individual basis [13].

It is very difficult for the team physician and medical personnel to make clinical decisions without clear diagnostic and management guidelines, especially at school and college level rugby in South Africa. On site informed and specialized medical care is at this point in time more the exception than the rule. The majority of clinicians still prefer some sort of clinical management and return-to-play guideline.[107]

Based on the results of our study and the review of the literature, we propose the following clinical, management and return to play guidelines for traumatic brain injuries in rugby players, especially at school and college level. These guidelines are proposed to protect young players from potential cumulative neurocognitive damage. It will be irresponsible to ignore the possibility of cumulative damage with even vMTBI and until

research proves otherwise it would be reasonable to adopt more strict guidelines for the vMTBI group.

Table 4.1: Recommended clinical and management model for MTBI and vMTBI in collision sport:

1. Each player suffering from a MTBI or more than two episodes of vMTBI's should be evaluated by a qualified physician.
   a) Preseason medical, Standardized Assessment of Concussion (SAC)[61] and computerized neuropsychological evaluation is mandatory. Results send to the governing body and to be kept on central database. Any subsequent SAC and/or neuropsychological test results be sent to governing body.
   b) No athlete with any form of a learning disability, epilepsy, abnormal neuropsychological testing or serious previous head injury is allowed to play rugby.

Type	1st incident	2nd incident	3rd incident
Grade 1:(very mild traumatic brain injury) - sub concussive impact to the head without any apparent symptoms or signs of concussion	Remove from play and do sideline evaluation. If no symptoms or signs of MTBI return to play after 10 minutes	Remove from play for the day. Perform a SAC. Alternative training & initiate rehabilitation programme. * Return to contact if asymptomatic and when SAC is normal, compared to baseline test.	Remove from play for the day. Perform a SAC. Alternative training and initiate rehabilitation programme. * Refer for neuropsychological testing. Return to play only if asymptomatic and when SAC and neuropsychological assessment is normal, compared to baseline test.
Grade 2: (low grade traumatic brain injury) - concussive impact with symptoms and/or signs of concussion but no PTA or LOC	As for grade 1, 3rd incident.	As for grade 1, 3rd incident.	As for grade 1, 3rd incident. Terminate season.
Grade 3: (moderate grade traumatic brain injury) - concussive impact with symptoms/signs of concussion including PTA of any length but without LOC	As for grade 1, 3rd incident.	As for 1st episode. Consider terminating season.	As for grade 3,1st episode but terminate season.

Grade 4: (high grade traumatic brain injury) - concussive impact with symptoms/signs of concussion including PTA and LOC of any length	As for 1st episode grade 3 and consider to terminate season.	As for grade 3, 1st episode but terminate season	As for grade 3, 1st episode, terminate season and consider quitting collision sport.
Grade 5: (Very high grade traumatic brain injury) - concussive impact with symptoms/signs of concussion including PTA and LOC of any length and any complication(s): associated neck injury, skull fracture, hemorrhage,	Terminate season and consider terminating collision sport.	Terminate season and collision sport.	

*Alternative training & rehabilitation programme follows a stepwise process. Proceed to next level once asymptomatic at the current level. If any symptoms occur after concussion, the patient should drop back to the previous asymptomatic level and try to progress again after 24 hours. [13]:

1. No activity, complete rest.
2. Light aerobic exercise such as walking or stationary cycling.
3. Sport specific training – for example, skating in hockey, running in soccer/ rugby.
4. Non-contact training drills.
5. Full contact training drills.
6. Game play.

Head injuries in a collision sport like rugby are inevitable. The nature of the game and the risks involved makes optimal medical care, including informed consent and medical clearance on an ongoing basis but especially before starting to play rugby, mandatory.

The proposed new guidelines are in agreement with the current knowledge and should assist the clinician to improve on his clinical and return-to-play management.

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Appendices

Appendix 1

Pasient inligtingstuk

Geagte.....

Baie dankie dat jy bereid is om aan die studie oor breinbeserings in skolerugby deel te neem. Die rede vir die studie is om die invloed en behandeling van breinbeserings in skolerugby beter te verstaan. Meer spesifiek wil ons die EEG- en neuropsigologiese status van skole-rugbyspelers met baie minimale traumatiese breinbeserings (ligte kopstampe) en minimale traumatiese breinbeserings (sg harsingskudding/konkussie) vergelyk met 'n sedentêre kontrolegroep, aan die einde van die rugbyseisoen.

Die volgende is 'n basiese uiteensetting van hoe die toetsings sal verloop:

Dag 1:

- Voltooi van 'n vraelys
- EEG studie deur 'n gekwalifiseerde neurofisioloog te Mooimed Hospitaal (Von Weillich straat, Potchefstroom), waarvan die aard en duur verduidelik is.
- Die nodige tydreelings sal telefonies met u bevestig word

Dag 2:

- Neuropsigologiese toetse, waarvan die aard en duur verduidelik is
- Die toetse sal deur die Departement Sielkunde, PU vir CHO afgeneem word by die Potchefstroomse Sentrum vir Sportgeneeskunde, Tomstraat 70 Die Bult, Potchefstroom
- Die nodige tydreelings sal telefonies met u bevestig word

Belangrikheid van die studie

Die resultate van die studie sal ons help om minimale traumatiese breinbeserings in skolerugby in die toekoms beter te hanteer.

Resultate

U sal na afloop van die studie, sodra die inligting verwerk is, die geleentheid kry om die resultate privaat of telefonies met die navorser te bespreek. Die nodige tydreelings sal met u bevestig word.

Appendix 2

INGELIGTE TOESTEMMINGSVORM

VERKLARING DEUR OUER/VOOG

Ek, die ondergetekende ouer/voog.....

van.....(naam van kind) bevestig hiermee dat:

1. My seun uitgenooi is om deel te neem aan 'n navorsingsprojek onderneem deur die *Department of Human Biology* van die Universiteit van Kaapstad.
2. Dit is aan my verduidelik dat die rede vir die studie die vergelyking van die EEG (Elektro-enkefalogram – meet die elektroniese brein funksie) en neuropsigologiese status van hoërskool rugbyspelers met baie minimale breinbeserings en minimale breinbeserings met 'n nie-kontaksport kontrolegroep, is. Die inligting verkry sal ons help om die konsekwensies van breinbeserings in skolerugby beter te verstaan en te behandel.
3. Geen indringende prosedures (trek van bloed / inspuitings) sal gedoen word nie. Geen geneesmiddels sal toegedien word nie. Die aard van die EEG en neuropsigologiese toetse is aan my verduidelik; spesifiek dat die toetse absoluut geen newe-effek het nie en dat die normale voorsorgmaatreëls by die afneem van sulke toetse getref sal word:
    - EEG: dit is 'n veilige, nie-indringende en vinnige toets om die aard van die brein se elektriese golwe te toets. Elektrodes word volgens 'n standaard metode op die kop geplaas (die sg 10/20 elektrode posisie). Geen risiko bestaan vir bv elektriese skok of enige snaakse gevoel spesifiek agv die EEG-toets nie. Die toets sal deur 'n opgeleide neurofisioloog (Me Retha Bester) gedoen word. Sekere elektriese breingolwe word abnormal na herhaalde breinbeserings.
    - Neurofisiologiese toetse: dit is 'n veilige, nie-indringende en vinnige reeks breintoetse wat 'n goeie aanduiding gee van die konsentrasie- en geheue-vermoë van die persoon wat getoets word. Die reeks toetse word deur gekwalifiseerde sielkundiges verbonde aan die Potchefstroomse Universiteit se sielkunde departement afgeneem en geïnterpreteer. Die resultate word absoluut konfidensieel hanteer.
4. Dit is aan my verduidelik dat deelname aan die projek heeltemal vrywillig en gratis is. Ek sou my kind enige tyd aan die projek kon onttrek.
5. Ek is ingelig dat die inligting verkry deur hierdie projek konfidensieel hanteer sal word en dat die resultate in 'n navorsingsjoernaal gepubliseer sal word.

6. Elke deelnemer sal ingelig word oor die aard van die resultate. Enige abnormale resultaat sal dan ook in detail mat die betrokke ouers en kind bespreek word om oor verdere stappe te besluit. (bv. Verwysing na 'n neuroloog)
7. Enige breinbesering tydens 'n wedstryd sal volgens die normale potokol vir die hantering van sodanige beserings, langs die veld en vir die tydperk daarna hanteer word. Die navorser is geregistreer by die Suid-Afrikaanse Mediese raad en is gedek deur professionele versekering.
8. Bg inligting is in Afrikaans aan my verduidelik deur:.....
9. Ek is die geleentheid gebied om vrae te vra en al die vrae is na wense beantwoord.

Ek gee hiermee toestemming dat my kind aan die studie mag deelneem.
Geteken te ..... op .....20.....

Ouer/voog..... Getuie.....

VERKLARING DEUR NAVORSER
Ek, ..., verklaar dat ek:

1. Die inligting in die dokument verduidelik het aan.....;
2. Hom/haar versoek het om vrae te vra oor enige onduidelikheid;
3. Die gesprek in Afrikaans plaasgevind het.

Geteken te .....op.....20.....

Navorser..... Getuie.....