CUMULATIVE MILD HEAD INJURY IN RUGBY: COGNITIVE TEST PROFILES OF PROFESSIONAL RUGBY AND CRICKET PLAYERS

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To my Safta, who at age 90, continues to inspire with her zest for life, her warmth, compassion and tremendous capacity to love.

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ABSTRACT

This study investigates the effects of cumulative concussive and subconcussive mild head injury on the cognitive functioning of professional rugby players. A comprehensive battery of neuropsychological tests was administered to 26 professional rugby players and a non-contact sport control group of 21 professional cricket players. The test performances of the rugby players were compared to those of the cricket players. Within the rugby group, forward and backline players were compared. An analysis of *mean* score differences between the rugby and cricket group failed to support the presence of brain damage effects in the rugby group. However, there was significantly increased *variability* of scores for the rugby players compared with the cricket players on tests particularly sensitive to cognitive deficit associated with mild head injury. This invalidates the null indications of average effects, indicating that a notable proportion of rugby players' performances were falling off relative to the rest of the rugby players on tests vulnerable to the cognitive effects of diffuse brain damage. Mean score comparisons within the rugby group indicated that it was the subgroup of forward players, in particular, whose test performances revealed deficits suggestive of cerebral damage. Specifically, deficits were found in working memory, visuoperceptual tracking, verbal memory and visual memory, a pattern of deficits commensurate with cumulative mild head injury. The theoretical perspectives of Satz's (1997) *Brain Reserve Capacity Theory* and Jordan's (1997) '*Shuttle' model of variability* are drawn upon in order to elucidate research findings and suggestions for future research are provided.

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CHAPTER ONE
INTRODUCTION

1.1 RESEARCH CONTEXT

This study is a component of a larger research project comprising a long term prospective study of the effects of cumulative mild head injury (MHI) in the contact sport of rugby. The project sought to elicit baseline data of the cognitive functioning of members of a South African professional rugby team. Accordingly, a battery of neuropsychological tests and questionnaires were utilized and the data were analysed against that derived from a control group of professional sportsmen not exposed to repeated subconcussive or concussive head injury (a team of professional South African cricket players).

1.2 RESEARCH QUESTION

The research question arose out of the findings from a relatively new body of research in the field of MHI in contact sport. There are concerns relating to the possible cumulative effects of concussive and subconcussive MHI in contact sports such as boxing, soccer and rugby. The pivotal question is whether or not players of contact sports exhibit the cognitive or behavioural impairments typical of MHI.

There is a paucity of empirical research on the cumulative effects of repeated MHI. In 1989 Barth et al. suggested that because contact sports players are vulnerable to repeated head injuries, they can be used as a 'laboratory' model for acceleration/deceleration MHI in the general population. More recently however, Binder (1997) has suggested that due to the tremendously high number of blows to the head that contact sports players are exposed to, they represent a special case of MHI. In most sports great efforts have been made to assess and minimize the potential for severe head trauma. Clearly these efforts would impact on injuries of all severities. However as Barth et al. asserted, by 1989 there had been no significant focus to reduce the incidence of MHI. Boll (1983) refers to MHI as a 'quiet' or 'silent' head injury as this form of injury can so easily remain deceptively invisible. He points out that while effects are subtle and difficult to establish on objective testing, this does not necessarily mean that the consequences on an individual will be trivial and that the lack of obvious visibility of effects in itself can be particularly pernicious

While researchers generally agree on the presence of neuropsychological sequelae immediately following MHI, the course of recovery is still under contention. Research has generally relied on a collection of empirical findings derived from an examination of relatively short term group MHI effects, and has tended to ignore more complex issues such as future and delayed symptomatology. In addition, the empirical nature of research into MHI outcome has typically been devoid of theoretical elaboration. Shuttleworth-Jordan (1999) addresses this limitation of past research by proposing the use of Satz's (1993) Brain Reserve Capacity (BRC) threshold concept together with Jordan's (1997) 'Shuttle' model of variability, in order to identify patterns that might otherwise not be apparent. Satz's BRC model postulates a threshold factor which exists prior to the manifestation of symptoms caused by disease in the central nervous system. Central to this model is the notion that individual differences exist in relation to brain reserve capacity. These differences account for variable instances of protection from, or vulnerability to, symptom onset and so alter the symptom threshold between individuals in pre-clinical stages of disease. Jordan's 'Shuttle' model of inter-individual variability (developed within the context of Satz's BRC theory) provides highly specific indications regarding the variability of symptom onset likely to occur with normal ageing. While the 'Shuttle' model of variability was developed with respect to normal ageing, Jordan recognises its potential applicability to research on outcome following cumulative MHI .

Noting firstly, the special risk posed to contact sports players due to the extraordinarily high number of mild head injuries they are exposed to and secondly, Shuttleworth-Jordan's concerns regarding the limitations of studies which preclude theoretical elaboration, this study aims to understand the sequelae of cumulative mild closed head injury in contact sport, from a theoretically informed perspective. Specifically, the research examines whether there are any statistically significant differences between contact sport players and non-contact sport players on neuropsychological testing. The theoretical perspectives of Satz's BRC threshold concept and Jordan's 'Shuttle' model of variability are drawn upon in order to elucidate research findings.

CHAPTER TWO
LITERATURE REVIEW

2.1 MILD HEAD INJURY (MHI): DEFINITION AND CLASSIFICATION

The field of MHI has evoked many controversial issues surrounding its definition and classification of severity. The lack of a single, universally accepted system of severity classification has severely limited both research and clarification of this area. As Satz et al. (1997) assert "the determination of head injury classification, particularly in the mild to moderate category, represents one of the most fundamental problems confronting researchers of head injury" (p. 126).

2.1.1 MAJOR TYPES OF HEAD INJURY

The focus of this study is closed head injuries which differ from penetrating head injuries in terms of external causes as well as the patterns of neurological and neuropsychological impairments that tend to result. Penetrating head injuries which are typically produced by gunshot or fragments from exploding shells, cause lacerations of the scalp, perforation or fracture of the skull, and laceration of brain tissue in the path of the foreign body. A shower of bone fragments often penetrates the brain at the point of impact, the depth of dural penetration and loss of brain tissue representing indices of injury severity (Levin, Benton & Grossman, 1982). Penetrating head injuries account for less than 10% of all documented head trauma in the civilian population (Richardson, 1990).

Lezak (1995) asserts that most head injuries are closed, in that the skull remains intact and the brain is not exposed. Closed head injury involves blunt trauma to the head, resulting either from impact by a moving object upon a stationary or slower moving head (acceleration), or when the head and body are decelerated by a stationary or slower moving object (Gualtierri, 1995). Linear and rotational acceleration typically coexist or follow one another in closed head injury. Blunt trauma to the head may injure the scalp, deform the skull with or without fracture, and shift the intracranial contents. Intracranial pressure appears to increase briefly on impact. Although blunt trauma associated with acceleration/deceleration forces to the head is the primary mechanism of impact in closed head injury, bone fragments can penetrate the brain tissue in cases of depressed skull (Levin et al., 1982).

Focal lesions may occur at the point of impact (coup) and in an area opposite the blow (centre coup) which is caused by the brain rebounding off the opposite side of the skull. These coup- and contrecoup lesions account for the localizable changes that accompany closed head injury (Lezak 1995). Diffuse damage in closed head injury arises from rotational shear stresses within the brain (Lishman, 1987). Evidence suggests that this diffuse cerebral damage arising at the moment of impact is the primary mechanism of brain damage in closed head injury and that the severity of this damage is a more important prognostic indicator than the presence of focal lesions in closed head injury (Adams, Mitchell, Graham & Doyle, 1977; Levin et al., 1982). The spectrum of closed head injury severity, depending on the amount of diffuse damage incurred, ranges from mild to moderate to severe (Levin et al., 1982).

2.1.2 HEAD INJURY CLASSIFICATION

Any discussion of head injury must consider severity as a defining characteristic and predictor of outcome. Head trauma exists on a continuum with mild bumps causing no overt symptoms representing one end of the continuum and very severe head injuries causing prolonged coma or vegetative states at the other (Lezak, 1995). The measures most frequently used to make attributions regarding the severity of head injury include loss of consciousness (LOC), the Glasgow Coma Scale (GCS) and the duration of Posttraumatic Amnesia (PTA).

Loss of Consciousness (LOC)

According to Lezak (1995) LOC alone is a poor predictor of outcome for the many patients with brief periods of coma (up to 20-30 minutes) but is a good predictor for more severe injuries. Lishman (1987) notes that the longer the duration of LOC, and the deeper the level of coma, the more probable it is that permanent brain damage will have been sustained. Prolonged unconsciousness is more likely to be accompanied by evidence of brain damage on neurological examination, by evidence of raised intracranial pressure and by the presence of blood in the cerebrospinal fluid. It is also more likely to be followed by a considerable period of posttraumatic confusion and by both physical and mental long term sequelae. In severe cases unconsciousness may persist for weeks or even months and permanent after effects may be expected.

Glasgow Coma Scale (GCS)

The development of the GCS (see Table 1 below) by Jennett and Bond (1975) and its widespread use remains a major milestone in head injury evaluation, prognosis and clinical research (Kelly, 1991). The GCS has proved to be of considerable predictive value in pointing to the long-term outcome in terms of both survival and ultimate levels of disability (Jennett, 1984; Jennett & Bond, 1975). The GCS is based on the presence, degree and duration of coma. The patient's clinical state is charted regularly on a number of graded parameters: motor responsiveness, verbal performance and eye opening. Numerical scores are summated for the best responses obtained under each category at a defined time. In this way useful predictions can be made often within 24 hours of injury and more certainly within the first week (Dacey, Bollmer, & Dikmen, 1993). Patients whose GCS score is less than 8 on admission (or 6 after injury) are considered to have sustained severe head injuries. Patients with GCS scores of 9 to 12 are generally designated as having sustained moderate head injuries and patients whose initial GCS score is 13 to 15 are designated as having MHI. The GCS has the benefit of low inter-observer variability and high reliability when applied by medical, nursing or paramedical personnel (Lishman, 1987). The validity of the GCS with respect to differentiating levels of head injury severity is supported by the finding that length of hospital stay is inversely proportional to GCS admission scores (Dacey et al., 1993).

Lezak (1995) points out that despite its demonstrated usefulness, the GCS has certain inherent problems. For instance, some trauma patients are lucid in the first hours after head injury but may deteriorate afterwards. Others may be anaesthetized and intubated at the scene of the accident and should they go to surgery before regaining consciousness, an early GCS score cannot be obtained. In addition intoxicated patients may produce unreliable GCS scores with impaired consciousness attributed inappropriately to head trauma severity in some cases, to alcoholic stupor in others. Also drugs and metabolic alterations due to injuries not directly involving the brain can also affect level of consciousness resulting in a misleading GCS score. Additional concerns regarding the GCS are that one or more examination modality may not be measurable during the first few days that the patient is under observation. For example, intubated patients cannot talk, swollen eyes will not open and immobilization or paralysis precludes limb movement.

Table 1. Severity Classification Criteria for the Glasgow Coma Scale

Classification	CGS		Coma Duration
Mild	≥13	or	≤20 minutes
Moderate	9-12	or	No longer than within 6 hours of admission
Severe	≤8*	or	≥6 hours after admission

* Patients with GCS ≤ 8 are considered to be in coma
(In Lezak, 1995, p. 755).

Posttraumatic Amnesia (PTA)

Some investigators rely on the duration of PTA to classify the severity of closed head injury. PTA refers to the patient's inability to form new memories of a particular period after recovering consciousness. The length of the PTA may be defined as the time from the moment of injury to the time of resumption of normal continuous memory (Lishman, 1987). According to Lishman (1987) the importance of the duration of amnesia as a guide to the severity of injury and prognosis for recovery was first demonstrated by Russell in 1932. This has not become a well-attested part of clinical practice. The PTA has proved to be more valid and useful than the Retrograde Amnesia (the time between the moment of injury and the last clear memory from before the injury which the patient can recall). In general, the Retrograde Amnesia tends to increase in length with the PTA but since the great majority of Retrograde Amnesias are extremely brief they are less valuable as a guide to severity. The PTA also emerges as more valid for this purpose than the duration of unconsciousness or overt confusion, as the latter depends more closely on secondary complications. The PTA is, moreover, a permanent index of severity, and available to the clinician who enquires long after the injury (Lezak, 1995).

A detailed classification based on the duration of PTA was proposed by Teasdale and Jennett (1974) (see Table 2 below). Both Evans (1992) and Levin et al. (1982) found that the duration of PTA correlates well with GCS ratings. Lezak (1995) explains that estimates of severity of

injury on the basis of PTA tend to parallel the GCS severity range except for some finer scaling at the extremes. Difficulties in defining, and therefore determining, the duration of PTA have made its usefulness as a measure of severity questionable in some cases (Lezak, 1995). For example, many patients with relatively MHI are discharged while still in PTA, leaving it up to the examiner to establish the duration of PTA at some later stage based on often unreliable patient and family reports. In light of these considerations Brooks (1989) and Jennett (1979) asserted that "fine tuned accuracy of estimation is not necessary as judgements of PTA in the larger time frames of hours, days or weeks will usually suffice for clinical purposes " (in Lezak, 1995, p. 174).

Table 2. Estimates of Severity of Injury based on duration of Posttraumatic Amnesia

PTA duration	**Severity**
≤5 minutes	Very mild
5-60 minutes	Mild
1-24 hours	Moderate
1-7 days	Severe
1-4 weeks	Very severe
> 4 weeks	Extremely severe

(In Jennett & Teasdale, 1981).

The classification and measures of severity of head injury are not entirely clear cut. As suggested above, various researchers have drawn on differing criteria such as LOC, GCS and PTA, to the extent that authors such as Satz et al. (1997) have suggested that the categories are so arbitrary they cease to become useful.

Satz et al. (1997) claim that definitions of mild and moderate closed head injury should be based on empirically determined variables. Advocating a multidimensional approach, Satz et al. (1997) argue that researchers should seek to determine the severity of injury along multiple dimensions as opposed to a simplistic continuum ranging from mild to severe. Despite their assertion that subjective terminology be replaced by operationalizeable terms which define severity in terms of outcomes, these categories have thus far endured in research.

2.1.3 DEFINITION OF MILD HEAD INJURY

In their meta-analytic review of neuropsychological studies of MHI, Binder, Rohling and Larrabee (1997) acknowledge that no definition of MHI has achieved widespread knowledge. The term MHI is broadly understood to refer to head injuries in which LOC and/or PTA is relatively brief and in which there is an absence of any structural pathology of the skull (Binder, 1986). As mentioned previously, criteria used in defining MHI are usually based on alterations in consciousness (GCS), changes in orientation and memory (duration of PTA) and length of unconsciousness (Satz et al., 1997). However while these symptoms are successfully used to define the more severe range of head injuries, they become unreliable or not applicable in the mildest range of head injury severity (Satz et al.). While criteria used in defining MHI have varied considerably in the literature (Binder et al., 1997; Evans, 1992; Levin, Eisenberg & Benton, 1989), Evans,1992, has stressed that strict criteria for defining MHI are essential to ensure the exclusion of confounding variables in such studies. He recommends that a duration of LOC of 30 minutes or less without further neurological complications is a reasonable defining criterion to adopt for research into MHI.

Satz et al. (1997) delineate the varying, and at times idiosyncratic, ways in which researchers have defined MHI. They conclude with a description of how the 'Mild Traumatic Brain Injury Committee' (of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine) expanded the spectrum of mild injury to include different grades of injury severity. It was recommended that MHI be defined by the measure of at least one of the following : (a) any period of LOC for less than 30 min, with a GCS of 13-15 following the LOC; (b) any loss of memory for events immediately before or after the accident with a PTA of less than 24 hours; (c) any alteration in the mental state at the time of the accident (e.g., double vision, loss of balance, taste, or smell) that may or may not be transient. This definition encompasses a broader range of injury severity than is traditionally used which arguably not only increases the prevalence rate of MHI but also encourages investigation of participants without hospitalization stays (Satz et al., 1997). Although this approach has much clinical appeal Satz et al. warn that it also suffers from the use of arbitrary and a priori cut points to designate grades of severity along the distribution of head injury that lack empirical verification at this time.

Evans' (1992) criterion for classifying MHI in terms of the duration of loss of consciousness (30 minutes or less without further neurological complications) is commensurate with the broader recommendations of the Mild Traumatic Brain Injury Committee (as reported by Satz, 1997). Consequently for the purposes of this study these criteria were adopted. In sum, qualification for classification of MHI include LOC less than 30 minutes; PTA less than 24 hours and any alteration in mental state at the time of the accident. As this was not a hospital based study GCS scores were not attainable and thus were not used.

2.1.4 DEFINITION OF CONCUSSION

There is considerable degree of semantic inconsistency with the terms 'cerebral concussion', 'MHI' and 'concussive head injury' leading to virtually interchangeable usage in the literature. However, not all concussion is synonymous with MHI as an examination of these terms will reveal.

Rutherford (1989) defines concussion as an acceleration/deceleration injury to the head almost always associated with a period of amnesia and followed by a characteristic group of symptoms such as headache, poor memory and vertigo. Thus impairment of consciousness, however transient, follows all but the mildest blow in closed head injuries, especially when the head is free to move at impact. In occasional cases there may be no detectable impairment of consciousness, but merely transient dizziness, or blurring of vision, or there may be no outward sign of impairment of consciousness to onlookers, yet the patient is without recollection of the injury, showing that at least the memorising functions of the brain were out of action for a time (Lishman, 1987).

Diffuse brain injuries are associated with widespread disruption of neurological function. These injuries result from the shaking of the brain within the skull and are thus lesions that are caused by the inertial or acceleration effects of a mechanical input to the head. Both theoretical and experimental evidence point to rotational acceleration as the primary injury mechanism for diffuse brain injuries.

Bruno, Gennarelli and Torg (1987) delineate three categories of diffuse brain injury:

(1) Mild concussion: several specific concussion syndromes that involve temporary disturbances of neurologic function without LOC.
(2) Classic cerebral concussion: a temporary, reversible neurologic deficiency caused by trauma that results in temporary LOC.
(3) Diffuse axonal injury: prolonged traumatic brain injury with LOC lasting more than 6 hours. Residual neurologic, psychologic or personality deficits often result because of structural disruption of numerous axons in the white matter of the cerebral hemispheres and brain stems.

Relating the above classification of concussion to MHI it is clear that diffuse axonal injury would be regarded as a 'severe head injury' on the GCS. Only the descriptions of the mild concussion syndromes and classical cerebral concussion can be referred to as mild closed head injuries. As concussion is not necessarily synonymous with MHI, it does not appear appropriate to use the terms interchangeably. Rather, greater clarity is achieved by providing the qualifier of *mild* concussion to be used as a term synonymous with MHI.

2.1.5 INCIDENCE OF MILD HEAD INJURY

Evans (1992) asserts that head injuries are one of the most important public health problems. There does not appear to be any South African research into the epidemiology of MHI, hence for the purpose of this review it has been necessary to utilize figures for the United States. In a study conducted by the United States National Centre for Health Statistics, the total number of all head injuries was estimated to be in excess of 8 million per year. MHI is much more common than severe head injury and accounts for most of the head trauma in the United States and other industrialized countries (Kraus & Nourjah, 1989). In fact, Caveness (1977) reported that 89%of the 9,760,000 head injuries recorded in 1975 by the US Department of Health, Education and Welfare were considered mild, as determined by the duration of hospitalization. Hospitalisation for MHI occurs with an incidence of 131 cases for every 100 000 population. It has been estimated that 20% - 40% of all patients with MHI in the United States do not seek medical care (Evans, 1992).

Dacey et al. (1993) delineate some of the reasons why the exact incidence of MHI is difficult to determine:

1. Most health surveys concentrate on patients who have been hospitalized. However, many patients who sustain MHI are not hospitalized but are evaluated in emergency rooms or doctors' offices. In a hospital survey in England and Wales in 1972, Field (in Dacey et al., 1993) found that head injury occurred at a rate of approximately 4 320 per 100 000 population. This confirms the impression that a significant number of patients with MHI are managed without hospitalization and that the more severely injured patients who are hospitalized represent only a fraction of all patients affected. Thus the total number of people at risk for sequelae after head injury is somewhere between the number of patients hospitalized and the millions of people who suffer a MHI of unknown severity each year (Rimel, Giordani, Barth, Boll & Jane, 1981).
2. The International Classification of Diseases (ICD) and other descriptive tools are often applied ambiguously to injuries (such as maxillofacial fractures and scalp lacerations) by including them in the overall statistics for injuries to the head.
3. Patients who have sustained multiple injuries that include a MHI may be classified according to their most severe or complex injury, and the occurrence of head injury may be ignored.

Despite these difficulties it has become apparent, as a result of several population based studies, that the incidence of hospitalization for head injury of all degrees of severity is approximately 200 per 100 000 per year (Dacey et al., 1993). In Virginia, 52% of this group of hospitalized patients were noted to have sustained MHI (Kraus & Nourjah 1989). Similarly in the National Head and Spinal Cord Injury Survey (HSCIS), a population based study of the incidence of head injury in the United States in 1974, 44.3% of all patients hospitalized for head injury had a length of stay of less than three days (Dacey et al., 1993). Thus it appears that the incidence of MHI requiring hospital admission is approximately 100 per 100 000 per year. A 1988 study, including patients who were dead at the scene of impact, found that the incidence of MHI in 3358 injuries was 72% (Kraus & Nourjah 1989).

An additional factor confounding estimates of the incidence of MHI is the lack of a universal definition of head injuries, as mentioned previously. This is illustrated by the data from the 1975 Health Information Survey (Kraus & Nourjah, 1989) in which 74.5% of all patients were said to have sustained cerebral concussion. However, a review of data regarding the length of hospitilization in this population indicated that 65.7% of the patients were hospitalized for longer than 48 hours. This supports the notion discussed previously that the term 'concussion' is not, in all cases, synonymous with a MHI and that if taken to be synonymous can lead to an overestimation of MHI.

2.1.6 DEMOGRAPHIC CHARACTERISTICS

The causes of MHI are similar to those for more severe head injury (Dacey et al., 1993). These authors reported that vehicular accidents caused 46% of the MHI, whereas falls, sports, accidents and assaults caused 23%, 28% and 10% respectively. They note that this is similar to the relative frequency of the causes of injuries noted in the HSCIS study (Dacey et al., 1993). Rimel et al. (1981) found that head injury also seems to occur primarily in patients of lower socio-economic status, predominantly young and a predominance of males over females. They reported that 66% of their patients were male, and in a separate group of patients, Dacey et al. (1993) reported that 65% were male, distributions which do not appear to differ significantly from that for head injury of all degrees of severity.

2.1.7 SYNOPSIS

While universal agreement on the definition and classification of MHI does not exist, the term MHI is generally understood to refer to closed head injuries in which LOC and/or PTA is relatively brief and does not involve any structural pathology of the skull. Based on recommendations of the Mild Traumatic Brain Injury Committee of the American Congress of Rehabilitation Medicine, for the purposes of this study the following defining criteria for MHI were adopted: LOC less than 30 minutes; PTA less than 24 hours and any alteration in mental state at the time of injury. Estimates suggest the incidence of MHI requiring hospital admissions is approximately 100 per 100 000 per year. The exact incidence of MHI is difficult to determine however, as a significant number of patients are managed without hospitalization.

2.2 THE SEQUELAE OF MILD CLOSED HEAD INJURY

2.2.1 PATHOPHYSIOLOGY OF MILD HEAD INJURY

The pathological changes following head injury in general have been carefully documented and have helped to illuminate some of the principal mechanisms by which clinical symptoms follow head injury and indicate the kind of pathology that may be expected to follow from different types of trauma (Lishman, 1987). The pathology and pathophysiology of MHI in particular are however very poorly understood (Dacey et al., 1993; Gualtieri, 1995). This is undoubtedly because very few patients who have had a documented MHI will come to post-mortem examination (Dacey et al., 1993). Yet these are the changes about which the most understanding is needed for routine clinical practice. The pathophysiology of MHI is based on the study of animal models, for example the angular acceleration model and the fluid percussion model (Gualtieri, 1995). Since animal models of severe brain trauma show neuropathological and neurochemical changes that are similar to those observed in severely injured humans it has been assumed that MHI in experimental animals is a valid model for what happens in mild head injured patients (Ommaya & Gennarelli, 1974). Lishman (1987) cautions that the experimental paradigm in animals is unsatisfactory as the margin is extremely narrow between the blow which barely concusses, and the blow which kills the animal instantly. Lishman thus welcomes the development of pathophysiological studies during life which may ultimately help to answer some of the outstanding questions.

Most elusive of all has been firm evidence concerning the basis for the brief LOC which follows all but the mildest blow to the head. In this regard the most notable advance has come from the conception of rotational shear stresses within the brain (Lishman, 1987). Russell (1963) found that unconsciousness was produced immediately after impact when the skull was subjected to a sudden change in velocity, i.e., 'acceleration concussion'. They reported that a much greater force was necessary to produce unconsciousness when an animal's head was in a fixed position as compared to when it was free to move when struck, that is when forces of acceleration or deceleration acted upon the brain within the cranium (Levin et al., 1982). Lishman (1987) describes experiments conducted by Pudenz and Shelden in 1946 in which they directly observed the brains of monkeys after replacing their skull caps with transparent material; swirling movements of brain tissue could be witnessed after acceleration injury, both at the point of impact and elsewhere. In spite of Lishman's caution about extrapolating from animal studies to humans,

he suggests that such animal observations may explain why concussion is common when the head is in motion at the time of injury, but relatively rare in static crushing injuries.

An additional problem is whether concussion depends on diffuse or focal damage to brain tissue. Lishman expounds on this issue in great detail. Swirling movements within the rigid skull stand to produce diffuse effects throughout the brain, yet the cardinal symptoms of concussion point to a major involvement of brain stem centres (Lishman, 1987). There is a lack of clarity regarding the physiology of amnestic defects which accompany concussion. These defects are contingent upon whether focal or diffuse brain changes have occurred. There is inadequate clinical data to determine this. The fact that current registration and retrograde recall are affected together would suggest that the special neural mechanisms in the hippocampus or diencephalon have been temporarily incapacitated. The temporal lobes are tightly encased within a bony framework and the medial temporal lobe structures may thus be vulnerable to disproportionate damage when the brain is subject to sudden motion. However, the duration of PTA is closely related to measures of the overall severity of brain damage in closed head injuries and it is widely assumed that this at least depends on diffuse, rather than focal, disturbance of neurological function. Significantly substantial amnesia is less likely in cerebral trauma precipitated by either crushing injuries or penetrating injuries as shearing stresses are unlikely to occur throughout the brain. It has even been suggested that amnesia after head injury might be due to widespread disturbances of the macromolecular pattern of proteins and lipoproteins within the nerve cells themselves (Dixon, 1962 in Lishman).

It is likely that there is an interplay of both diffuse and focal effects across various injuries. The hippocampal or diencephalic mechanisms may particularly be affected in instances marked by long and persistent retrograde amnesias. In contrast to this, a generalised effect on the brain may produce brief retrograde amnesia (a more common occurrence) compatible with the time required for registration regardless of the length of the posttraumatic amnesia. In the instance of a patient suffering from permanent memory deficits, yet without gross disturbance of other cognitive functions, it is a fair assumption that the focal damage in question has acted on the memory regions of the brain (Lishman, 1987).

2.2.2 MILD HEAD INJURY AND THE 'POSTCONCUSSIVE SYNDROME' (PCS)

A number of symptoms have been observed to occur subsequent to MHI and are collectively referred to as PCS (Anderson, 1996). As noted earlier, concussion is not necessarily equivalent to MHI, however, the term PCS does definitively refer to MHI. While the number and combination of symptoms vary from patient to patient, there is a remarkable consistency in the early symptoms displayed by these patients. The range of possible symptoms that may be experienced is summarized in Table 3, although it is noted that considerable inter-individual variability exists (Boll, 1983, DSM-IV, 1994, Shuttleworth-Jordan, Puchert, Balarin 1993).

Table 3. Range of symptoms associated with Postconcussive Syndrome

Somatic	headache, dizziness, vertigo, insomnia, vomiting, fatigue and weakness, loss of appetite, drowsiness, blurred vision, sleep irregularities, decreased noise tolerance, sensitivity to medications and alcohol, restlessness, clumsiness and postural changes (associated with disturbed sensorimotor syndrome).
Neurocognitive	impaired attention and concentration, memory and learning disorders, reduced mental flexibility, slowed reaction time, impaired decision making, cognitive impulsivity, speech difficulties, mental fatigue.
Neuropsychiatric	depression, anxiety, emotional lability, irritability, lowered frustration tolerance, somatization and hypochondriasis, denial of symptoms, apathy or lack of spontaneity, personality change.

(In Anderson, 1996, p. 24).

A review of the current literature indicates that the PCS is not necessarily a short term phenomena and that close to 50% of affected individuals demonstrate symptoms for at least three months. Jones (1974) studied 3500 victims of MHI and found that headaches and dizziness persisted in 57% for up to two months (in Szymanski & Linn, 1992). The definition used for MHI was not given. Rimel et al. (1981) found that out of 500 mild head injured victims, 78% complained of headaches and 59% of memory disturbances three months after injury (1981). The definition of MHI was LOC for 20 minutes or less, a GCS of 13-15 and hospitalization of 48 hours or less. Of those who were employed prior to injury, 34% were not working three months later. Levin, Gary, et al. (1987) reported that among MHI patients, 47% complained of headaches, 22% of diminished energy and 22% of dizziness three months after trauma. Gronwall and Wrightson (1981) found that 60% of mild head injured victims reported symptoms interfering with work three months after injury. As many as 59 of 66 subjects had a PTA of six hours or less. The definition of MHI was not otherwise specified. Lidvall et al. (1974) studied 38 patients with MHI, defined as acceleration trauma which caused immediate LOC and/or amnesia (in Szymanski & Linn, 1992). They reported that over time anxiety increased, so that by three months post-injury, anxiety and headache were the most prominent symptoms.

An additional report from the three centre study collaborative study (Ruff et al., 1989) with respect to postconcussional symptoms in head injured patients revealed that approximately 90% of patients reported postconcussional symptoms on the first assessment. The authors note the lack of correspondence between the patients' subjective complaints, which remained virtually unchanged at one month and the significant gains that they showed on the neuropsychological tests. Thus, although the test performance of subjects improved, they continued to complain of postconcussional symptoms after one month and in some cases these symptoms had increased. The authors suggest that the reason subjective complaints may increase over time is that in the early phases of recovery, patients typically are not challenged by their environment. When they begin to resume previous activities they are confronted by new hurdles and difficulties which may result in an emergence of subjectively experienced difficulties.

Variability in postconcussive symptom presentation has focused debate on the cause of neuropsychological symptoms after MHI. Several factors such as undetected parenchymal lesions, history of psychiatric illness or substance abuse, pending litigation, pain and exaggeration of symptoms or malingering are presumed to be causative, particularly in determining symptom duration (Jacobson, 1995; Macciocchi, Barth & Littlefield, 1998). Controversy regarding the origin of postconcussive symptoms has been exacerbated by methodological limitations. Macciocchi et al. criticize studies of MHI as tending to suffer from various threats to internal and external validity, including imprecise injury definition, selection bias, subject attrition, reliance on self report measures, lack of baseline neuropsychological data, diverse outcome measures, variable test time sequences and limited documentation or control of potentially influential subject factors.

2.2.3 NEUROPSYCHOLOGICAL STUDIES INTO THE COGNITIVE EFFECTS OF MILD HEAD INJURY

While a major research focus of the past was the postconcussional symptoms and why they persisted in some cases (Binder, 1986; McLean, Temkin, Dikmen & Wyler, 1983) later investigations attempted to identify additional factors such as objective neuropsychological and psychosocial sequelae and determine the time course for natural recovery from such consequences. There has been a strong interest in neuropsychological consequences based on the belief and the hope that they would provide objective evidence of impaired brain functions and therefore explain patients' subjective complaints and disruptions in psychosocial functioning (Dikmen, McLean & Temkin, 1986).

As noted in the literature, MHI per se was not studied in detail until the 1980s when an increased recognition of the public health implications of less severe injury spurred researchers to develop better understanding of this portion of the injury spectrum (Barth et al., 1983; Beers, 1992; Marshall and Marshall, 1985). With recent improvements in technology, physiological (Rutherford, 1989; Gentilini, Nichelli & Schoenhuber, 1989) and histological (Gennarelli, 1987) studies are beginning to identify specific neuropathological contributions to the postconcussion symptoms that accompany MHI.

2.2.3.1 Specific areas of Functional Deficit

Evidence for highly specific areas of deficit following MHI emerging from research include impairments in information processing, attention and memory.

Based on a series of studies using the Paced Auditory Serial Addition Test (PASAT), Gronwall and Wrightson propose that the principal dysfunction in MHI is slowed information processing (1974, 1975, 1981). They have shown that after MHI some patients demonstrate an inability to process information as rapidly as normal, as evidenced by their performance on the PASAT. They suggest that dysfunction of the attentional control system reduces the rate of information after head injury. Individuals frequently have difficulty with aspects of attention when they are required to analyse more items of information than they can deal with simultaneously. They often appear to be slow, easily distracted, forgetful and inattentive because of the extra effort required to process information. Alternatively, Chadwick (1985) concluded that after head injury rather than there being a disruption of attention, there is actually less attention available due to the increased time needed for information processing. Boll (1983) also found clinical evidence for information processing deficits in head-injured persons, who have problems learning under complex conditions, functioning efficiently in novel circumstances and coping with stress. Moreover, he suggests that these problems tend to exist in the absence of all other measurable difficulties and persist for several years.

Beers (1992) points out that later research concluded that attention is *not* a unitary concept and should not be investigated as such. This is illustrated in a study conducted by Gentilini et al. (1985) who used case-control pairing, incorporating only the Digits Forward Test, a Word Recognition test, Buschke's Selective Reminding Verbal Learning Test and the Raven Progressive Matrices. The authors found no significant differences between the MHI group and their matched controls on global measures of attention. In contrast, when different aspects of attention (selected, sustained, divided and distributed) were explored using the same sampling criteria and carefully matched controls, significant differences were found (Gentilini, et al., 1989). Commensurate with this is a study by Stuss et al. (1985) using a series of simple and complex choice reaction time tasks. Results suggest that head injuries of varying levels of severity result in deficits in both elements of attention and information processing rate of adults. A deficit of divided attention occurred in mild head injured or apparently recovered individuals

as well as those having more severe injuries. This study identified an impairment of focussed attention and performance inconsistency across the groups, but this effect was observed only with repeated assessments using a focussed attention task. Thus optimal performance was achieved but could not be maintained by head injured adults. A study by Ewing, McCarthy, Gronwall and Wrightson et al. (1980) investigating attention and memory in college students exposed to stress, found that under stressful conditions students who had sustained a MHI at least one year previously performed significantly below matched controls on tests of vigilance and memory.

Thus in sum, deficits in information processing, attention and reaction time are amongst the most overriding effects of head injury of any severity (Barth et al., 1989; Boll, 1983; Ewing et al., 1980; Gentilini et al., 1985, 1989; Gronwall and Wrightson, 1981). Often referred to as 'mental stamina' these deficits may be the cornerstone of other cognitive impairments. Attention and concentration are domains that researchers suggest should be investigated more fully (Barth et al., 1983, Boll, 1983).

2.2.3.2 Permanency of Cognitive Sequelae following single uncomplicated Mild Head Injury

Although numerous investigators have come to agree upon the presence of the neuropsychological sequelae of MHI, there is less consensus regarding the course of recovery (Binder, 1986; Klonoff & Lamb, 1998). Some researchers suggest recovery is rapid and occurs within days or weeks (Dikmen et al.,1986; Gentilini et al., 1985; Gronwall & Wrightson, 1974; Levin, Gary, et al., 1987; McLean et al., 1983) while other studies have demonstrated evidence of continued cognitive impairments in patients many months and even years post-injury (Leininger, Grammling, Farrell, Kreutzer & Peck, 1990). Finally some recent major reviews suggest that there is little support for permanent effects following MHI (Binder et al., 1997; Satz et al.,1997).

Evidence of Symptom Resolution within three months post Mild Head Injury

Gronwall and Wrightson (1974) found that while patients with MHI demonstrated impaired information processing abilities in the initial phase after their injury, their performance returned to normal within 30-35 days (Gronwall and Wrightson, 1974). McLean et al. (1983) found that patients with MHI demonstrated distractibility and difficulties in remembering and learning new

information at three days post-injury, but this was resolved within a month. Gentilini et al. (1985) found that while patients with MHI demonstrated a trend towards lower performances, there were no significant differences between head injured patients and the controls. The authors conclude that if there is structural damage after MHI, it generally recovers within one month of injury. Dikmen et al. (1986) found significant differences between patients with MHI and controls on tests measuring sustained attention and ability to retain new information over time. While symptoms were evident within the first month post-injury, there was no evidence of these symptoms at the follow up examination a year later. In a three centre collaborative study reported by Levin, Mattis, et al. (1987), significant difficulties were detected at one week, but not at three months following injury.

The findings of early, but not late, neuropsychological problems following MHI in representative samples are also in line with the earlier work of Gronwall and associates (1974; 1975; 1981).
In the above mentioned three centre corroborative study, Ruff et al. (1989) focused on the assessment of visual and verbal memory and digit span, following MHI within seven days, one month and three months post-injury. On both the verbal and visual memory measures the MHI groups scored significantly lower than control groups at baseline, although significant improvements were noted at both the one month and three month follow up on both verbal and visual memory. In contrast, performance on the Digit Span test remained at about the same level on all three examinations. The authors account for this by drawing on evidence in the literature to suggest that performance on the Digit Span test often remains relatively intact in amnestic patients.

The above mentioned studies failed to find evidence of neuropsychological compromise much beyond one to three months post-injury. However, an inherent difficulty which affects these studies is the lack of an objective measure of pre and post-injury effects, which may obscure the relatively subtle nature of the deficits in a MHI group. This inability to make direct comparisons with premorbid data prevents the researchers from pronouncing unequivocally that patients have in fact returned to their previous levels of functioning.

Evidence of Persisting Deficits three or more months post Mild Head Injury

Studies which found significant deficit at three months post-injury include Rimel et al. (1981) and Barth et al. (1983). Rimel et al. found convincing evidence that considerable disability occurs three months after MHI including difficulties with attention, concentration, memory and judgement. Barth et al. (1983) found that a significant proportion of patients showed cognitive impairment at three months post-injury in the areas of memory and visuospatial skills. Whilst the test batteries used in the Barth et al. and Rimel et al. studies were comprehensive, the studies were limited due to the absence of premorbid data and by their failure to utilize matched control groups or a repeated measures control group to account for practice effects.

A more carefully controlled study conducted by Leininger et al. (1990) using matched, uninjured controls, found evidence of deficits in information processing, verbal learning and reasoning at 22 months post-injury. These findings are supported by Raskin, Mateer and Tweeten (1988) who found patients with MHI demonstrated significant impairment on measures of complex attention, working memory and verbal learning, particularly on time dependent tasks 21 months post-injury. A recent study by Klonoff and Lamb (1998) found that patients with a history of MHI presented with chronic and unusually severe deficits almost three years post-injury. The authors acknowledge the limitation of the restricted sample size used, making generalizability of findings problematic.

The study by Ewing et al. (1980) mentioned above provides evidence that patients who appear to have made a full functional recovery from a MHI show persistent cognitive 'fragility' to central nervous system stressors. They compared performance under mild hypoxia in a group of university students who had made a full 'recovery' from MHI between one and three years before the study was conducted with a matched group of control students who had never had a head injury. They found that on memory and vigilance tasks the MHI group performed significantly poorer than controls under hypoxic stress. Thus, although all the students who had MHI more than a year before the study was carried out had returned to full time university work and were performing at the same level as before their accidents, the MHI had left a residual effect which impaired the ability to withstand another central nervous system stressor. The authors concluded that in all probability each concussive event destroys neurons, diminishing the reserve availability and making the loss evident under the stress of further injury.

A later study which found impairment under alternative test conditions was conducted by Parasuraman, Mutter and Malloy (1991). These authors compared the sustained attention performance of mild head injured patients and uninjured control subjects during the first month of injury using a high-event-rate visual vigilance task, combined with stimulus degradation to induce the need for effortful or controlled processing. They found that vigilance performance after MHI is comparatively normal under conditions that encourage automatic processing but deficient under conditions requiring substantial effort to process stimuli.

While the studies reported in this section have demonstrated evidence of continued impaired cognitive functioning up to 22 months post-injury, recent comprehensive reviews conducted by Binder et al. (1997) and Satz et al. (1997) do not provide support for this notion. Binder et al. conducted a meta-analytic review of available prospective and quasi prospective studies on MHI in adults. The review examined 11 independent samples from eight publications which met the following inclusion criteria: patients studied at least three months after MHI; patients selected because of a history of MHI rather than because they were symptomatic; an attrition rate of less than 50% for longitudinal studies; and studies of adults only. The authors concluded that results suggest only a weak association between MHI and persistent cognitive deficits. They advocate that small effect sizes force a clinician to exercise considerable caution before diagnosing brain injury after MHI. While Binder (1997) acknowledges that the neurological basis for sustained neuropsychological problems cannot be completely dismissed, he claims that at present there is little evidence of a neurological aetiology for the most persisting complaints. Similarly Satz et al. (1997) following a comprehensive review of research into MHI in children and adolescents, found that in general many of the studies failed to show adverse affects on neuropsychological functioning following MHI. Satz et al. caution, however, that these findings should not be treated as definitive and recommends "cautious acceptance of the null hypothesis...until more definitive studies are conducted" (p.107).

With regard to the permanency of deficit, there is a lack of consistency in terms of research findings regarding outcome following MHI. Research has generally indicated that MHI results in measurable acute cognitive deficit, and in most cases, deficit following single uncomplicated MHI resolves within three months. However, emerging research indicates that some patients exhibit residual cognitive deficit in the presence of additional stressors or as a result of cumulative effects.

2.2.3.3 Etiology of Persisting Symptoms

The etiology of persisting symptoms following single MHI is a source of ongoing debate. Studies generally have failed to control for the effects of previous injury. This represents a major omission in light of data indicating that recovery on a complex concentration task is slower in patients with a previous MHI (Gronwall & Wrightson, 1975) and the relationship between the presence of cerebral atrophy and the number of previous fights in boxers (Casson, Sham, Campbell, Tarlau & DiDomenico, 1982). It is clear that the older patient, like a person with a previous head injury, is at greater risk for prolonged disability (Rutherford, 1989; Gronwall & Wrightson, 1981). Socioeconomic status is also related to disability after concussive injuries. In the study by Rimel et al., 1981, 100% of managerial level patients had returned to work three months post MHI whereas only 57% of the unskilled labourers had resumed work at that time. While socioeconomic status may be related to the period of disability it is not related to symptomatology. Miller (1961) has implied that the lower classes have insufficient motivation to work because their jobs are undesirable, but professionals and managers may return to work more rapidly as they have more control over their work. The validity of the length of PTA as a predictor of outcome after MHI is problematic when short; is difficult to assess; is unreliably reported by patients after three months and is not a consistently valid predictor of symptomatology and disability or of EEG abnormalities (Gronwall & Wrightson, 1981). Other factors cited include premorbid constitutional and personality problems, pre-injury stressful events and posttraumatic stress (Evans 1992).

There is also substantial literature emerging on the diagnosis of symptoms magnification and financial incentives contributing to reported cognitive deficits in MHI (Alexander, 1995; Dacey et al., 1993; Klonoff & Lamb, 1998; Lishman, 1987; Raskin et al., 1998). McMordie, 1988, asserted that patients recover from their symptoms after settlement in litigation occurs. Persisting symptoms are thus viewed as psychogenic in origin and represent a 'compensation neurosis'. This simplistic characterization of the symptoms of mild head injured patients is questioned by Binder (1986) and Dikmen, Temkin and Armsden (1989) on the grounds that there is evidence of neuropathological and neurophysiological changes post-injury and evidence that changes in symptom rates were not only linked to time of litigation settlement. For example, in longitudinal studies, Rutherford (1989) observed that many patients showed decreased symptom rates prior to litigation settlement and about one third of the patients followed continued to exhibit

symptoms one year post settlement (almost three years post-accident). Other studies have revealed symptoms and dysfunction in patients not engaged in litigation (Leininger et al., 1990). Morse and Montgomery (1992) point out that although some patients may report fewer symptoms after a lengthy, stressful litigation process, changes in symptom reporting in these patients do not necessarily demonstrate that their cognitive and emotional performances have returned to premorbid levels at this time. Both Lishman (1987) and Rutherford (1989) believe that there is an interplay of both organic and functional elements and that both physiogenic and psychogenic influences are important.

In sum, the etiology of persisting symptomatology remains controversial and evidence tends to point towards a mixed psychogenic and organic basis. Particular subgroups of patients appear to be at risk for continued significant deficits on testing beyond one to six months post-injury. In general, the literature suggests that patients at greatest risk for chronic sequelae following a MHI are older (>40 years of age), have had a previous head injury, are high achievers or in demanding occupations, have lower socioeconomic status and/or have family or social stressors.

2.3 CUMULATIVE MILD HEAD INJURY

Macciocchi et al. (1998) emphasize the inadequacy of only examining short term outcomes in regard to the resolution of impairments following MHI. They warn about the possibility of long term deleterious effects of MHI, in particular repeated MHI such as sustained in contact sport. In fact according to De Villiers (1987) one of the most disturbing features of mild concussive or subconcussive head injury is that the effects are cumulative. Binder (1997) expresses surprise that, considering how frequently a relationship between repeated head injuries and slower recovery has been cited, the major supporting evidence that multiple injuries are worse than one injury is so sparse.

Outside of research within the field of contact sport, there appears to be limited formal research in the area of cumulative MHI. A notable exception is the work of Gronwall and Wrightson. In 1975 these authors were able to show that even a single concussion renders the brain slower to recover from a subsequent episode. Using the PASAT they demonstrated that rates of information processing were slower after a second MHI than after a single injury of equivalent severity, despite a mean of four and a half years between the two. The time taken to recover to

normal levels of functioning was also significantly delayed. They thus concluded that multiple concussions have a cumulative effect in that they delay the cognitive and subjective recovery from a new head injury. In a population study with a large sample Carlsson, Svardsdudd, and Welin (1987) found a significant difference on some cognitive measures between men with multiple injuries as compared to those with single injuries, but the effect size was negligible. Binder (1997) points out that it is possible that patients may recover clinically from a single MHI while continuing to suffer from a reduction in brain reserve capacity. As mentioned earlier, Satz (1993) hypothesized that people with reduced brain reserve capacity have lower thresholds for the expression of impaired test performance after any cognitive stressor such as neurological disease or perhaps MHI. This assertion is supported by studies such as that conducted by Ewing et al., 1980, (see paragraph 2.2.3.1) when a small sample of asymptomatic, young patients with MHI exhibited cognitive deficits during the stress of mild hypoxia.

Additional support for the deleterious effects of cumulative MHI emanates from the rapidly growing body of well controlled studies of contact sport players. As noted in the introduction, Binder (1997) asserts that contact sports players (he mentions professional football players in particular) represent a special risk due to the extremely large number of blows they are exposed to. Thus, in the same vein that Barth et al. (1989) recommended that sports injuries be utilised as a 'laboratory' model for acceleration/deceleration MHI, *contact* sports can be viewed as a convenient 'laboratory' for identifying the cognitive effects of *cumulative* MHI in particular.

2.3.1 MILD HEAD INJURIES IN CONTACT SPORTS

The growing awareness that MHI is an often overlooked condition that may result in neuropsychological dysfunction, together with the emerging evidence that the risk of permanent residual cognitive impairment increases consequent on cumulative MHI, has resulted in an increasing focus on MHI in contact sports. MHI is frequently observed in sports participants. These injuries are generally of a milder degree and usually are not associated with other traumatic injuries. Cantu (1995) points out that there appear to be few sports that do not carry some risk of a MHI. In fact some sports have blows to the head as an expected or even intended 'sporting activity'. Included in this category are boxing, soccer, Rugby League and Rugby Union, Australian Rules Football and American Football (Drew & Templer, 1992).

2.3.1.1 Incidence of Head Injuries in Contact Sport

Assessing the prevalence of sports-related MHI is problematic as in most cases it goes unreported (Anderson, 1996). Authors such as Barth et al. (1989), Cantu (1997) and Sturmi, Smith and Lombardo (1998) point to the collusion that frequently takes place between players, coaches and other interested parties in terms of minimizing symptoms for the purpose of continued athletic participation. They highlight possible ramifications of athletes acknowledging what they consider 'minor injuries'. These include losing one's position on a team, missing an opportunity to impress coaches, national selectors and fans, or being seen as a failure. In light of the fact that sports ethos demands that its participants ignore their injuries, Cantu (1997) maintains that most incidence figures are likely to be an underestimate, particularly those concerning the more mild head injuries. Sports related injuries account for 5 to 10% of all head injuries treated in hospitals (Tysvaer, 1992). These figures probably underestimate the frequency of head injury as sportsmen who have sustained a MHI do not always report to the hospital (Warren & Bailes, 1998).

A number of studies have examined the incidence of head injury in American Football specifically. It is difficult to compare these studies with each other directly because of inconsistencies in injury definition. According to Cantu (1997) more than 250 000 concussions occur annually in football. Twenty percent of high school football players suffer concussion during a single football season, some more than once (Warren & Bailes, 1998). Based on the history of head injuries and years of experience of a large group of athletes, Barnes et al. (1998) calculated that there was a 50% probability of a player sustaining a head injury within a 10 year playing period. Commensurate with these estimations are alarming calculations by Binder (1997) who estimates that professional football probably results in thousands of player to player collisions with many of the blows either directly to the head or causing the head to shake. He describes how each National Football League season in America involves at least six months and 20 games. Assuming two full contact practices and one game per week a player might receive up to 75 blows per week, or up to 6000 in four years. The National Collegiate Athletic Association (NCAA) in America has conducted an extensive injury surveillance of all sports and is able to determine a true incidence by reporting injuries as expressed by the number per 1000 athlete exposures (Green & Jordan, 1998). Based on data collected by the NCAA covering the years 1991-1996 the overall concussion rate in football is 0.31 concussions per 1000 athlete

exposures. The rates of concussion in football can be directly compared with those of soccer as the surveillance system (during the same time period) found an identical rate of concussion (0.31 per 1000 athlete exposures) for soccer as for football. Green and Jordan (1998) explain how a concussion rate of 0.31 per 1000 athlete exposures represents a significant risk for this injury when measured over the course of a player's entire career. They cite a retrospective study they conducted of 235 participants at the 1995 Major League Soccer Combine which revealed that the average player had been actively involved in soccer for 18 years. Over that period they calculate that a player can easily participate in at least 4000 practices and games (i.e. 4000 exposures) throughout his playing career. There have been several studies performed in other countries that have revealed that concussions make up from 7% to 24% of all soccer injuries. Green and Jordan (1998) point out that these studies differ from the NCAA data in that there is no actual rate (i.e. per athlete exposures) at which injuries occur.

With respect to studies in South Africa, Nathan, Goedeke and Noakes (1983) reported an epidemiological study of all serious rugby injuries during one playing season in a well defined population of schoolboys. A total of 79 injuries was recorded during the season. The overall incidence, expressed as the number of injuries per boy-hours of rugby (15 boys playing for one hour equals 15 boy hours of rugby) was 1/395 boy-hours of rugby. If only matches are considered the overall incidence of injury was 1/119 boy-hours of match play. Of the 79 injuries head, neck and facial injuries accounted for 38% of all injuries. In terms of type of injury, concussion was the most common, accounting for 21.5% of injuries.

In a later study conducted by Roux, Goedeke, Visser, Van Zyl and Noakes (1987) similar techniques were used to study rugby injuries in a much larger number of high schools. Of the 26 high schools examined, 320 teams were fielded, playing 3 350 matches. Of the 495 injuries recorded 71.3% occurred during matches and 28.7% during practices. This corresponded closely to the Nathan et al. (1983) study cited above in which 62.5% of all injuries occurred during matches and 37.5% during practices. Considering matches separately the overall injury incidence was one injury for every 142 boy-hours of match play. Of the 495 injuries head and neck injuries accounted for 29% of the injuries. Concussion accounted for only 12% of all injuries which was considered an unusual finding since the majority of schoolboy studies show that concussion is the most common injury. The authors found that there was marked

underreporting of concussion by schools monitored by correspondence. Of the schools closely monitored concussion accounted for 18.4% of injuries.

Studies have also revealed that players' positions affect the frequency and the nature of injuries. Stephenson, Gissane and Jennings (1996) investigated the incidence of injury in English professional Rugby League over a period of four playing seasons. Table 4 below reflects the incidence of injury for forwards and backs.

Table 4. Distribution of Injury between Forward and Backline Players

Position	Number	Rate per 1000 hours	95% Confidence Interval
Forwards	277	139	124-155
Backs	215	93	81-105
Total	492	114	105-124

(In Stephenson et al., 1996, p. 333).

From this it can be seen that forwards are injured more frequently than backs in absolute terms. When the rate is standardized for the number of players (six forwards and seven backs), the injury rate differences are even larger (forwards 139 v backs 93 per 1000 hours; $P< 0.05$). In line with these findings, Gibbs (1993) and Seward, Orchard, Hazard and Collinson (1993) have also reported that forwards received a larger than expected number of injuries based on the number of player positions. The greater rates of injury seen in forwards is probably due to their being involved in more repetitive body contact than backline players. For example the forwards are the players who participate in the scrum which results in repeated head to head and/or head to torso pressure and collisions which predispose these players in particular to cumulative brain damage effects (Shuttleworth-Jordan et al., 1993).

Similarly research into the concussion history of elite soccer players conducted by Barnes et al. (1998) revealed that 76% of the concussions reported were sustained by athletes playing forward or midfield. An examination of the studies and incidence figures mentioned above reveal that figures replicate each other between studies and even across countries.

2.3.1.2 Boxing

The 'knockout' and at times a technical knockout in boxing are examples of concussion. Boxing is the only sport in which the rules allow a contestant to inflect cerebral injury of such a magnitude that the opponent will be rendered unconscious. Injury to the cranium and its contents is a direct product of boxing and not an accident as in all other forms of sports. Structural damage to the brain may be produced in two ways:

1. Acute damage may result from one or more severe blows during a single fight resulting in haemorrhage/contusion, unconsciousness, focal paralysis and occasionally death.
2. Cumulative injury may occur over a much longer period of time and is associated with a chronic neurological disorder (post traumatic encephalopathy) most frequently encountered in professional boxers (De Villiers, 1987).

Martland (1928) introduced the term 'punch drunk' to the medical profession when he described the symptoms of several aging retired professional boxers. The medical profession has since coined 'traumatic encephalopathy', 'dementia pugilistica' and 'chronic progressive traumatic encephalopthay of boxers' (Lezak, 1995) to indicate the same syndrome. These terms refer to the chronic, progressive consequences of brain tissue damage resulting from the repeated head trauma of boxing.

Martland's often quoted classic description of the severe punch drunk syndrome has been little changed over the years. The picture of 'punch-drunkenness' in retired boxers is widely recognised, but the supporters of boxing have sought to explain it on the grounds of coincidentally occurring neurological disease or have attributed it to alcoholism rather than to the patient's boxing career (Lishman, 1987). Several series of cases have combined to suggest a syndrome with highly characteristic features, but have been open to criticism on the grounds of special selection (Butler, Forsythe, Beverly, Adams, 1993). Roberts' 1969 extensive survey, was therefore particularly important in establishing the syndrome as a valid entity, and in providing clear indications that the boxing career had been responsible (in Gronwall & Wrightson, 1975). In its fully developed form, the syndrome consists of cerebellar, pyramidal and extrapyramidal features, along with a varying degree of intellectual deterioration. The unusual combination of neurological features provides a characteristic picture and suggests that

a distinctive pathological process is responsible. Severe examples date mostly from boxing careers pursued before the Second World War when medical control over boxing was less rigorous than at present.

There are indications, especially from recent CT scan studies, that present safeguards are still inadequate to prevent brain injury. Kaste et al. (1982) suggest that modern medical control creates a dangerous illusion of safety. Roberts traced a random sample of professional boxers who had held a professional licence for at least three years (between 1929 and 1955) and found that 17% showed evidence of the characteristic syndrome. Approximately one third were judged to be affected severely enough to be recognised by a layperson as 'punch drunk'. The prevalence of the syndrome and the severity of the symptoms increased with exposure to boxing which highlighted the cumulative nature of repeated damage (in Grownwall & Wrightson, 1975). By the later 1960s many researchers felt that major brain damage in boxers was being adequately prevented. In 1966 Blonstein (cited in Drew & Templer, 1992) reported to the Royal Society of Medicine that "with shorter bouts and modern medical control, the punch drunk syndrome has not been seen during the last twenty years in active professional boxers " (p. 649). A 1974 study by Harvey and Davis is the last reported incidence in the research literature of the classical punch drunk syndrome with full range of behavioural symptoms (Drew & Templer, 1992).

Many experts on boxing injuries had contended that by 1980 major neurological impairment in boxers had been an occurrence of the past. Since 1982 however, a number of research articles have reported continued evidence of brain damage in modern fighters in spite of the lack of gross neurological symptoms. Casson et al. (1984) studied 18 boxers using a combination of neurological, CT, EEG and neuropsychological exams. The authors found a significant correlation between number of professional bouts and the percentage of impaired or abnormal test results. They concluded that this relationship indicates the cumulative effect of multiple subconcussive blows to the head and that these blows are a likely etiology. Studies which use a combination of techniques reveal that brain damage still occurs in modern day boxers. These abnormalities appear in CT scans (atrophy), EEG findings (abnormalities) and a few in functional neurological exams (confusion, disorientation, and memory loss). Generally, the deficits were noted in retired boxers and in some cases they tended to correlate with increased number of bouts. With these findings there is increasing concern that brain damage may be occurring from early on in a boxer's career when he first experiences subconcussive blows and that these blows may be resulting in cumulative damage (Butler et al., 1993; Casson et al., 1982; Haglund & Eriksson , 1993; Ross et al., 1983; Ryan, 1987).

A number of studies have been undertaken in order to examine cognitive impairment in professional boxers (Casson et al., 1982; Casson et al., 1984; Drew & Templer, 1992; Jordan, 1987; Lampert & Hardman, 1984; McCunney & Russo, 1984; Ross et al., 1983). These studies have generally found impaired performances on neuropsychological tests and have correlated test results with abnormal CT scans and EEGs. Abnormal performances have been demonstrated on tests measuring attention, concentration, immediate and delayed memory, new learning, sequencing abilities, speed of information processing and on overall impairment indices. On tests of motor speed, nonverbal auditory perception and abstraction abilities, professional boxers have not shown major differences compared to controls. These studies however are plagued by methodological flaws including a failure to report mean scores, a lack of matched control groups and a lack of stated criteria for test abnormality.

Subsequent research seems to indicate that brain damage may also occur in amateur boxers (Haglund & Eriksson, 1993; Heilbronner, Henry and Carson-Brewer, 1991; McLatchie et al., 1987). In general, where results have suggested minor neuropsychological changes in amateur boxers, changes include: impairments in verbal learning and memory; in poor copy and recall of a complex geometric figure; slower complex reaction times, but faster movement and simple reaction times. No differences were demonstrated on tasks of attention and concentration, verbal memory, visuomotor functions, associate learning and information processing speed. Other studies conducted failed to find evidence that well controlled amateur boxing produced cognitive impairment (Brooks, Kupshik, Wilson, Galbraith and Ward, 1987; Butler et al., 1993). Methodological weaknesses of these studies include self selection (some of the subjects examined were volunteers and thus may not have been representative of boxers in general), the lack of estimates of premorbid ability and the lack of carefully matched control groups. Brooks et al. recognize the need for a longitudinal study of amateur boxers, or studies of amateur boxers with long boxing histories.

Haglund and Eriksson (1993) suggest that a possible reason that more signs of chronic brain damage were not found in amateur boxers is due to a natural selection, i.e. boxers who fight well and do not get hit as often continue boxing, while less successful boxers give up and therefore do not sustain chronic brain damage. Authors such as Brooks et al. (1987) and Heilbronner et al. (1991) also point to the strict regulations governing amateur boxing compared to professional boxing in which referees stop fights extremely quickly at the first sign of distress on the part of one of the boxers. This may explain failures in some studies to find any significant differences between amateur boxers and controls on neuropsychological measures.

Drew and Templer (1992), Casson et al. (1982) and McLatchie et al. (1987) discuss the use of helmets or adding padding in the gloves of boxers. However as the present objective of boxing is to render an opponent unconscious through blows to the head, any attempt to reduce the effectiveness of those blows will be controversial. Drew and Templer (1992) also point out that added padding to the hands and head may actually increase the amount of damage sustained through repeated acceleration/deceleration of the brain inside the skull. Thus the protection may result in more blows being sustained with greater force before the opponent is rendered unconscious. Short of a ban on boxing the most effective method of preventing severe cognitive deficits to boxers may be to limit their careers when early signs of brain impairment can be detected such as is currently being attempted in California (Drew & Templer).

2.3.1.3 Soccer

Soccer is the most widely played team sport in the world with an estimated 200 million registered participants (Matser, Kessels, Jordan, Lezak & Troost, 1998). Head injury in soccer can occur from head to head contact, falls with the head hitting the ground, and the head being struck by the ball. In fact the deliberate use of the head to propel an object is relatively unique to soccer. It has been estimated that if a soccer player plays 300 games during his soccer career, he will receive about 2000 blows to the head from heading (Tysvaer & Storli, 1989). Bruce, Schut and Sutton found that 5% of professional soccer players receive a concussion in any given season (1984).

Abreau, Templer, Schuyler and Hutchison (1990) compared the performances of 31 collegiate soccer players and 31 collegiate tennis players on the Raven Progressive Matrices, Symbol Digit Modalities, Perceptual Speed and PASAT Tests with respect to self-reported postconcussional symptoms. They found no significant differences between the groups on the cognitive tests, but did find a significant negative correlation between the number of games played and performance on the PASAT. Also a significantly greater number of soccer players than tennis players retrospectively reported symptoms of headaches, blurred vision and dizziness and passing out after a game. Due to the small sample size, the lack of premorbid data and the retrospective nature of this study, the authors concede that their results only provide tentative support for the detrimental effects to the brain. In addition no comment could be made about the permanency of such effects.

Stronger evidence of the presence of cognitive impairment was provided by Tysvaer and Lochen (1991) who reported on 37 former football players of the Norwegian national team who underwent neurological and EEG examination to investigate the incidence of head injuries due to heading the ball. An increased incidence of EEG abnormalities was found in players compared with non-football playing, non-injured controls of similar age. The high incidence of EEG changes in these players were attributed to the result of a cumulative effect due to repeated head traumas. The neuropsychological test battery consisted of measures of general intelligence (the Wechsler Adult Intelligence Scale- Revised [WAIS-R]); sustained attention, concentration and mental flexibility (Trail-Making Test); aphasia, sensory skills, several motor functions and visual memory (Benton Visual Retention Test). On these tests soccer players showed consistently higher proportions of impaired functioning. In respect to findings on the WAIS-R the soccer players exhibited evidence of cognitive impairment and were considerably more variable than controls on the Verbal Scale and showed greater difficulty with Digit Span (a measure of attention and short term memory). In regard to the Trail Making Test, the soccer players' scores were significantly poorer on both Part A and Part B of the test. The distinction between the two groups was greater on Part B, the more complex of the two parts. Amongst the former soccer players, the authors found a higher degree of neuropsychological impairment in headers than in non headers. The results of the study led to the authors' conclusion that "blows to the head by heading show convincing evidence of brain damage similar to that found in patients who have sustained minor head injuries" (Tysvaer & Lochen, 1991, p. 59). The finding

of greater cognitive impairment in headers compared with non headers was supported by research conducted by Witol and Webbe (1993) and Matser et al. (1998). Witol and Webbe conducted a study specifically focussing on the effects of heading in soccer players. Their findings reveal impairments in attention, concentration, cognitive flexibility and general intellectual functioning in soccer players who were frequent headers and who also had a history of frequent heading. Matser et al. (1998) compared the cognitive performance of Dutch professional soccer players with a control group of professional non-contact sport athletes. The soccer players demonstrated impairments in memory, planning and visuoperceptual processing. The authors found that forward and defensive players were more vulnerable to cognitive impairment as they are more likely to head the ball and thus experience a higher frequency of soccer related concussions.

In contrast to the above studies which tend to support the notion that repetitive heading of the ball leads to chronic brain injury, other studies have failed to show any correlation between career heading exposure and the development of a chronic encephalopathy (Boden, Kirkendall & Garret, 1998; Jordan, Green, Galanty, Mandelbaum & Jabour, 1996). Boden et al. conducted a prospective study of concussion incidence in elite soccer players and concluded that long term encephalopathic changes in soccer players are more likely a result of multiple concussions sustained playing the game than repetitively heading the soccer ball. This is supported by Jordan et al. who evaluated elite, active soccer players and matched them with a control group of elite track athletes. They estimated career exposure to heading and found no evidence that this exposure correlated with symptoms that might be attributed to chronic encephalopathy. In addition increased heading exposure did not correlate with abnormalities on brain MRIs. They conclude that any evidence of encephalopathy in soccer players relates more to acute head injuries received playing soccer (for example, collisions) than to repetitive heading. This led to speculation that repetitive heading may exacerbate the effects of an acute head injury. The researchers concluded that soccer exposes the player to a substantial risk of acute head injury, although not necessarily as a result of heading. Baroff (1998) points out however, that the absence of apparent heading effects in the Jordan et al. study may be related to the fact that this was a much younger population in comparison with studies by Tysvaer and Lochen (1991). Cumulative subacute effects of heading may not be apparent until a later age.

In sum, the above studies provide evidence that participation in professional soccer may be related to impairments in cognitive functioning typically associated with diffuse cerebral damage. The question of whether these deficits result from heading the ball per se or prior episodes of MHI or possibly a combination of the two is not yet resolved. It is likely that the soccer player with a history of acute head injury may be at greater risk from heading than a counterpart without such a history (Baroff, 1998).

2.3.1.4 Rugby/Football

While the focus of the present research is on MHI in players of Rugby Union (the only type of rugby played in South Africa), there are strong parallels with sports such as Rugby League, Australian Rules Football and American Football. For clarification purposes a brief comparison of these four sports is provided.

All four sports mentioned above involve physical collisions between players that cause acceleration-deceleration impacts to the head. Tackling, scrumming and other collisions between players can result in this type of injury (Shuttleworth-Jordan et al., 1993). An important aspect of all four sports is tackling which occurs when a player is held by one or more opponents and in the process he is brought to the ground. The player may often be running at speed when tackled or blocked (physically prevented from moving forward). This may result in the sudden deceleration causing MHI. There are some differentiating features between the four sports which include for example, differing numbers of players in the scrum and the fact that only in American Football is the use of protective clothing mandatory. However, the strong parallels which clearly exist make research findings from Rugby League, Australian Rules Football and American Football relevant to the present study on players of Rugby Union.

American Football

Head and neck injuries have been perennial problems in American Football. Since the formation of the National Collegiate Athletic Association in response to fatalities and paraplegia resulting from the sport, public concern for the safety of the athlete has continued to be of primary importance in the evolution of American Football (Cantu, 1997; Albright, Mcauley, Martin, Crowley & Foster, 1985). The enormous popularity of the sport has made it the prototype wherein adequate injury statistics have been kept and several major injury prevention steps have

been legislated at the national level. However statistics of catastrophic injury are not reflective of the more common MHI termed 'stingers'. Bruce, Schut and Sutton (1984) reported that football accounts for 40% of all concussive MHI in school sports. In a survey of 3 064 football players in 103 Minnesota high schools following the 1977 season, 19% of the players suffered possible concussive MHI. Albright et al., 1985, conducted a study of 342 college football players at the University of Iowa over a 8 year period and found a total of 115 head injuries. They also found that players who received a second head injury in college football were apparently more affected by the second injury. Players who reported a high school head injury resulting in non football participation for a day or more, were more likely to have lost time for a college head injury, a phenomenon the authors interpreted as sensitization caused by earlier injury.

In 1989 Barth et al. conducted a prospective neuropsychological study of American college football players in which they utilized premorbid baseline data and controlled for test practice effects with two groups (college students and mild orthopaedic injury patients) who received repeated testing. The neuropsychological test battery which was administered to head injured players at 24 hours, 5 days, 10 days post trauma and at post-season included the Trial Making, The Symbol Digit and PASAT tests. In addition a self-report questionnaire was used to assess psychosocial consequences. They found impaired information processing abilities among players with reported MHI with a pattern of rapid but possibly incomplete recovery up to 10 days. There was no difference between the recovery curve of the head injured subjects and the controls on the Trail Making Test which may have resulted from possible practice effects. Symptoms of headache, memory, nausea, dizziness and weakness reported by the concussed subjects showed the same recovery pattern as the cognitive tests.

Australian Rules Football

Australian Rules Football is one of the most popular sports in Australia, with over 15 000 teams registered with the National Australian Football Council (Maddocks, Saling & Dicker, 1995). At a professional level it has been found that nearly 25% of injuries are to the head and neck region, and that 5% of total injuries are concussive in nature (Dicker, McColl & Sali, 1986).

Research on concussive head injuries amongst Australian Rules Footballers has been conducted by two main groups of researchers consisting of Maddocks, Saling and colleagues (1991 & 1995) and Geffen and colleagues (1995 & 1997). Maddocks and Saling (1991) conducted a study which involved using baseline premorbid data and a matched control group. The neuropsychological battery included the Digit Symbol Substitution Test, the PASAT, and Four-Choice Reaction Time measure involving measures of Decision Time and Movement Time. Relative to controls poorer performances following concussion were noted on the Digit Symbol, Decision Time and Movement Time measures. Methodological strengths of this study include the fact that it was a prospective study which made use of pre-injury data and incorporated repeated testing of matched control groups to control for practice effects. However, methodological weaknesses include a limited test battery; failure to deal with the permanent effects of concussive head injury and failure to consider the possible effects of unreported mild concussive and subconcussive head injury. A later study by Maddocks et al. (1995) utilised baseline data obtained from Australian Rules footballers. Over a period of eight years, 200 players were assessed prior to the commencement of the season in which they joined the club. An estimate of premorbid IQ was obtained from demographic variables such as age, occupation and years of education. A retrospective concussive history was obtained from each subject. The Digit Symbol subtest was then administered individually to each subject. Results revealed normal levels of performance six months or longer after injury. A limitation of the study however, was that it was retrospective in nature and thus concussive history had to be derived from the players' accounts without medical confirmation. Other limitations included the use of the Digit Symbol test in isolation and the fact that no control group was used.

A recent study conducted by Hinton-Bayre, Geffen, and McFarland (1997) attempted to determine sensitivity to cognitive impairment immediately following concussive head injury in professional rugby league players. They found that speed of information processing and speed of comprehension were impaired in the postacute phase of MHI, whereas the untimed task of word recognition was not. Speed of comprehension was more sensitive to post-injury impairment than either Digit Symbol Substitution or Symbol Digit Modalities tests. The authors suggest that further studies using multiple baseline measures from individually administered assessments would confirm the consistency of this finding. Cremona-Meteyard and Geffen (1995) also examined more permanent, persisting effects of cumulative MHI. The researchers

looked at persistent visuospatial attention deficits following MHI in Australian Rules football players. They compared two groups of Australian Rules football players with MHI to non-injured sportsmen using a cued reaction time task which measured the reaction time benefit of valid directional cueing and the reaction time cost of miscueing. In the first experiment, nine footballers tested within two weeks of sustaining their injury showed the same cost as controls in speed of response to targets in the unexpected visual field. However, their responses to targets in the expected location showed only a minor benefit compared to controls. On retesting a year later their pattern of performance had not altered although overall reaction time had improved. Cremona-Meteyard and Geffen conclude that a persistent consequence of MHI may be an inability to act rapidly in response to expected spatial events.

Rugby Union

The only research on Rugby Union (albeit played in countries including the UK, New Zealand, Australia and South Africa) appears to have been conducted in South Africa, steered by Shuttleworth-Jordan et al. (1993). There were two aspects to their research: (i) an analysis of pre- and post-season differences between reportedly non-head injured rugby players and controls, and (ii) an analysis of repeated test differences between rugby players with a MHI and matched controls. The test battery included the Denckla Finger Tapping, Purdue Pegboard, Digit Span, Digit Supraspan and Trail Making Tests. The pre-season comparison between the rugby players and controls suggested the presence of impairments in working memory, verbal new learning ability and hand motor dexterity in the rugby players - a pattern of deficits typically associated with closed head injury due to diffuse brain damage effects. One finding inconsistent with the general trend of these results was a significantly faster Finger Tapping test score in the rugby group compared to the controls. The authors explain that the differences amount to points of a second and it was considered that this test could not be scored rigorously enough to ensure reliable differences with respect to points of a second. They thus conclude that the direction of this Finger Tapping test result in isolation had little interpretive validity (Shuttleworth-Jordan et al., 1993).

The post-season comparison revealed that the rugby players demonstrated significantly less capacity to benefit from practice in contrast with the control groups. These findings were ascribed to the permanent effects of a head injury sustained in a previous rugby season, or the recent effects of subconcussive or unreported concussive effects during that rugby season, or a

combination of both, in the rugby playing group. Also of significance, when the rugby playing group was compared with respect to position of play, the forwards showed no significant improvement on any of the tests in contrast to the backline who were able to benefit from practice. The authors propose that these findings suggest that the forwards were showing greater impairment than the backline.

In regard to the section of the study comprising repeated test measures on concussed players, there were indications of impairments in working memory, verbal new learning and hand motor dexterity in the mild head injured rugby playing groups. These results are highly comparable with the pattern of impairment noted in the 'non head injured' rugby playing group on the pre- and post-season comparisons. Considered together, the authors suggest that the results across the two sections of the study consistently show the rugby playing groups to be compromised on functions which are known to be sensitive to the diffuse brain damage which classically accompanies a closed head injury. The authors state that these results provide compelling indications that the consequences of mild concussive and subconcussive head injuries in rugby result in working memory, verbal memory and hand motor dexterity impairments which take at least three months to recover and in many instances may not recover entirely.

The study was able to overcome some of the limitations of previous studies due to the use of baseline premorbid data; the repeat testing of control groups to control for practice effects; the examination of the recovery curve up to three months; and the examination of the effects of previous concussive or unreported subconcussive head injury.

2.3.2 SYNOPSIS: MILD HEAD INJURY IN CONTACT SPORTS

Studies of MHI in contact sports have generally suffered from methodological flaws (such as an absence of carefully matched control groups, lack of estimates of premorbid ability, limited test batteries and failure to deal with permanent effects) resulting in inconsistent findings. Nevertheless most of these studies have brought to the fore the potential dangers of the cumulative effects of multiple MHI. Generally research into the cognitive effects of MHI in contact sports (such as boxing, soccer, and football/rugby) reveal patterns of deficits amongst players that are typically associated with diffuse brain damage. It is assumed that these deficits are due to the fact that players are consistently subjected to concussive and subconcussive injuries.

2.3.3 LOCATING CUMULATIVE MILD HEAD INJURY WITHIN A THEORETICAL CONTEXT

A limitation of almost all studies in the field of MHI is that research has been conducted in an atheoretical context. This problem is emphasized by Shuttleworth-Jordan (1999) who raises the concern that the recent reviews of Satz et al. (1997) and Binder et al. (1997) tend to rely primarily on an array of limited empirical findings, and that such debates are premised upon the seeming absence of relatively short term group MHI effects, rather than on broader and more complex theoretical issues.

Shuttleworth-Jordan's key contention is that almost without exception, literature in the field of MHI is empirically based rather than theoretically articulated. In order to comprehend research results in this area research requires a theoretical context, which she provides. She uses Satz's (1993) Brain Reserve Capacity (BRC) Threshold Theory and Jordan's (1997) model of inter-individual variability to explain how the use of theoretical concepts may allow for the identification of patterns that might otherwise not be apparent.

Satz's BRC theory postulates a threshold factor which exists prior to the manifestation of symptoms caused by disease in the central nervous system. This threshold represents the critical amount of brain tissue at which normal functioning can be sustained. This model holds that brain reserve capacity thresholds vary between individuals. General intelligence and educational levels represent indirect measures of an individual's BRC threshold. In terms of Satz's theory, a higher BRC is likely to act as a protective factor, decreasing the risk of functional impairment. Therefore the greater the BRC, the less likelihood there is of an individual exhibiting symptoms of neurological impairment. Correspondingly, individuals with lower BRC are more likely to exhibit neuropsychological deficit because lower BRC acts as a vulnerability factor creating greater risk of functional impairment. In terms of this theory any reduction in BRC due to neurological pathology is likely to increase an individual's vulnerability to functional impairment. Satz points to a number of factors which serve as threshold lowering factors and enhance vulnerability to symptom onset. These factors include ageing, low education and IQ, high task challenge, gender effects and additional cerebral insult.

BRC theory thus holds that there is a functional impairment cut off point which varies between individuals depending on the presence of differing vulnerability and protective factors. Pre-existing differential vulnerability factors will therefore lead to a variability in symptom presentation following MHI. Accordingly, Jordan's (1997) 'Shuttle' model of variability, developed within the context of BRC theory, postulates that owing to different levels of pre-existing cerebral reserve in association with the onset of neural attrition due to normal ageing, the presentation of symptomatology in brain damaged groups will occur differentially between individuals. This will be reflected in an increased variability of cognitive scores associated with the ageing process and declining raw scores. Specifically, due to protective factors which raise the threshold of symptom onset, a notable proportion of individuals will not present with much cognitive fall-off. Alternatively, due to vulnerability factors which lower the threshold of symptom onset, a notable proportion of individuals will show particularly marked fall-off. This then results in a significantly widened distribution of scores. Later, as neural attrition advances in later old age, protective factors resulting in raised BRC thresholds for some individuals become less effective and previously good scorers will perform closer to the norm. This results in a reduction in markedly variable symptom presentation between individuals. The distribution of scores will narrow again and be reflected in reduced variability of cognitive test scores in association with continued lowering of mean scores (Jordan, 1997).

Jordan (1997) extrapolates this model from the slow neural attrition that occurs with ageing and suggests that this pattern of inter-individual variability is likely to occur for other groups of individuals with accumulating degrees of mild brain insults. She asserts that cumulative mild traumatic head injury in particular, is likely to provide a good example of this occurrence. Combining indications from the 'Shuttle' model of variability and BRC theory, with evidence for cognitive impairments following participation in contact sports, Jordan proposes the following hypothetical indications for a MHI study in rugby.

She hypothesises that the curvilinear pattern of inter-individual variability in cognitive test performance associated with ageing will be replicated, in that rugby playing groups (exposed to long term multiple MHI) are likely to demonstrate increased variability on scores of cognitive tasks, in comparison with non-contact sport controls. This variability is expected because of the interaction of cumulative cerebral impacts superimposed on pre-existing differences in BRC

thresholds. These result in varying symptom onset for different individuals within a rugby playing group. Moreover different positions of play will contribute to significantly increased score variability in that a significant proportion of forward players within a rugby group (who risk more frequent exposure to concussive and subconcussive head injuries than backline players) are likely to reveal early symptom onset. In contrast to this, a significant proportion of the less exposed backline players are less likely to show evidence of cognitive deficit. Thus within a rugby group a significant proportion of the more exposed forward players are likely to present with early symptom onset while the more protected backline players are likely to exhibit symptoms only at a later stage, consequently leading to a widened distribution of scores (and thus increased variability).

Jordan (1997) further ventures that much like the extensive neural attrition associated with very advanced ageing, extended exposure to cumulative MHI experienced by the forwards may eventually cause the majority of forward players to perform poorly across tests. This would lead to scores falling close to the mean and thus result in a narrowing of score variability within the forwards subgroup.

Finally Jordan (1997) hypothesised that these variability effects would be manifest on tasks known to be sensitive to the effects of diffuse cerebral damage commensurate with the process of normal ageing as well as in cumulative cerebral insults.

2.3.4 Synopsis

Shuttleworth-Jordan proposes that Satz' BRC threshold concept, together with the 'Shuttle' model of variability provide a theoretical lens which allows for identification of patterns that might not otherwise be apparent and allow for the advancement of research in this field beyond the level of mere description. Thus, this study is theoretically premised upon Satz's BRC theory and the primary aspect of Jordan's 'Shuttle' variability model which proposes an initial increase of variability in the cognitive scores of a group of adults with cumulative MHI.

2.4 AIMS OF THIS RESEARCH

This research study was designed to take into account both empirical research findings suggesting the presence of chronic neuropsychological deficits in contact sports players, and indications from the 'Shuttle' variability model (Jordan, 1997) and Satz's Brain Reserve Capacity Theory (1993).

To further existing South African research into the cumulative effects of MHI in rugby players (conducted on university rugby players), it was decided to compare the performance of professional rugby players with that of professional players of a non-contact sport (cricket) who arguably are not exposed to long term cumulative concussive and subconcussive MHI. It was decided to utilize a more comprehensive battery of tests than has been used in previous research. This test battery aimed to incorporate an extensive spectrum of key cognitive modalities typically compromised in closed head injury. These modalities, as defined by Lezak (1995), include attention and concentration, memory/new learning, language fluency, visuoperceptual tracking and fine hand motor dexterity. Tests assessing premorbid intelligence for the most part, seem to have been excluded in previous research (with the exception of Maddocks & Saling, 1991). In order to address this limitation, tests which are typically not sensitive to impairments associated with MHI were also included providing an indication of premorbid cognitive functioning. Additionally, previous studies do not appear to have examined the effects of positional differences in contact sports. The present research accounted for positional differences within the rugby group by comparing the test performances of the rugby forward and the rugby backline subgroups. This was motivated by the fact that forward players (in contrast to backline players) are more susceptible to multiple subconcussive head injuries from collisions, tackling and scrumming.

2.5 RESEARCH HYPOTHESES

In terms of the indications from the empirical research reviewed above and the theoretical notions from Satz's BRC theory and Jordan's 'Shuttle' model of variability, the following hypotheses were posed:

1. It was hypothesised that on measures sensitive to diffuse brain damage, the overall performance of the rugby players (as reflected in *mean* scores) would be significantly poorer than that of the cricket players due to the long term cumulative mild concussive and subconcussive head injuries that the rugby playing group is exposed to (whereas the cricket players are not) which would serve to reduce BRC to a greater extent within the rugby playing group. Further it was hypothesized that within the rugby playing group reductions in BRC would occur differentially for players depending on whether they are in forward or backline positions, due to their differential exposure to cumulative concussive and subconcussive MHI which would increase *variability* of scores in the rugby group relative to the cricket group.

2. More specifically it was hypothesized that on measures sensitive to diffuse brain damage, the overall performance of the forward players (as reflected in *mean* scores) would be significantly poorer than that of the backline players due to the greater exposure of the forward players (relative to the backline players) to long term cumulative mild concussive and subconcussive head injuries, which would serve to reduce BRC to a greater extent within the forward playing group.

CHAPTER THREE
METHODOLOGY

3.1 PARTICIPANTS

This study forms part of a larger study into the effects of mild closed head injury in rugby which is being conducted by the Rhodes Psychology Clinic, the South African Rugby Football Union (SARFU) and the Sports Science Institute (Cape Town). The participants were drawn from professional rugby and cricket players. The sample included all designated players selected to undergo a series of physical and psychological tests at the Sports Science Institute. The rationale for using professional rugby players is that due to the nature of the sport and the length of playing careers, players are vulnerable to sustaining cumulative MHI. The use of professional cricket players as a control group was motivated by the fact that cricket is a non-contact sport and thus players were less likely to have suffered cumulative head injuries during their professional sport playing careers. Although the cricket players may have played rugby at school, the nature of sport at a professional level would have demanded a choice be made, presumably limiting the cricketers' long term exposure to cumulative head injuries at a professional rugby playing level. It was considered that the professional cricket players would compare closely to the rugby players in terms of demographic data such as age, education level and premorbid level of functioning.

Players with a reported history of substance abuse, any neurological disorder known to effect cognitive functioning negatively, and any reported previous moderate or severe head injuries, were excluded from the study in order to minimize confounding variables.

Table 5: Summary of Number and Percentage of Players with at least one Reported Prior Mild Head and Hand/Finger Injury

Team	No. of Players with MHI	No. of Players with Hand/Finger Injuries
Rugby n = 25	n = 19 (76%)	n = 21 (84%)
Cricket n = 21	n = 18 (85,7%)	n = 16 (76,2%)

Note: For a breakdown of the frequency and cause of head injuries for rugby and cricket players see Appendix A.

Of the 25 rugby players, 19 reported at least one MHI due to any cause and of these 76% occurred during rugby. Of the 21 cricket players, 18 reported at least one MHI due to any cause and of these 57% were due to playing schoolboy rugby. Of the 25 rugby players, 21 (84%) reported at least one prior hand injury while 16 of 21 cricket players (76%) reported at least one prior hand/finger injury.

Participants who had suffered MHI were not excluded from the sample in either group for two reasons. Firstly, the focus of the study was the effects of cumulative MHI over a long term period from school playing rugby days into their professional playing careers. Secondly, while 18 of the 21 cricket players reported histories of isolated MHI, they were not excluded as the study was looking at the more prolonged exposure to long term cumulative MHI which was anticipated in the rugby group. Similarly, 19 of the 25 rugby players reported histories of at least one MHI. Nonetheless, it is assumed that all players would have sustained continued concussive and subconcussive head injuries, many of which would not have been reported. Significantly, all but one of the cricket players played rugby at school and of the 37 reported head injuries, 21 occurred while playing school level rugby.

As the prior hand injuries reported by players at the time of testing did not preclude their participation in their professional rugby or cricket teams, these injuries did not significantly interfere with their gross hand motor dexterity and thus these injuries were not considered grounds for exclusion from this study.

None of the rugby or cricket players interviewed met any of the exclusion criteria which as noted above were: players with a reported history of substance abuse or any neurological disorder known to negatively effect cognitive functioning and players with a history of moderate or severe head injuries.

Table 6: Demographic Data of Participants

Group	n	Age	P value	Years Education	P value	Estimated Premorbid IQ	P value
Rugby	26	27.5	0.77	14.2	0.24	119.2	0.57
Cricket	21	27.1	0.77	13.7	0.24	121.1	0.57
Rugby Forwards	15	27.2	0.58	13.9	0.18	117.2	0.33
Rugby Backline	11	27.8	0.58	14.6	0.18	121.8	0.33

Analyses conducted on the demographic data (see Table 7) revealed that there were no significant differences between the rugby and cricket players with respect to age (P = 0.77), years of education (0.24) or estimated premorbid IQ levels (P = 0.57) and no significant differences between the rugby forward and backline players with respect to age (P = 0.58), years of education (P= 0.18) or estimated premorbid IQ levels (P = 0.33).

The final sample thus included 26 rugby players and 21 cricket players with highly equivalent demographic data with respect to age, years of education and estimated premorbid IQ.

3.2 PROCEDURE

Players were tested at the Sports Science Institute in Cape Town. The rugby players were assessed between the 2nd and the 5th of February 1997 during their pre-season medical and psychological evaluation. The cricket players were tested between the 14th and 16th of April 1997 during their post-season medical and psychological evaluation. The tight schedules of the national sporting teams meant that assessment opportunities were restricted and thus it was not possible to assess both teams pre-season.

The research team consisted of a research co-ordinator and three researchers. Due to the time constraints associated with attempting to assess professional teams, the services of research assistants were engaged. One research assistant was employed to aid with the assessment of the rugby players over a period of three days, and three research assistants were employed to expedite the assessment of the cricket players over two days. All researchers and research assistants were psychology graduates trained in the administration of neuropsychological testing. Inter-rater reliability was maintained by the training of both researchers and research assistants in standardized test administration procedures. Standardised written instructions were attached to the test material to further ensure uniform administration.

In an introductory group meeting the nature and purpose of the assessment was explained to the participants. Prior to testing, the procedures were reiterated to each individual. Participants were then required to sign written consent forms (see Appendix B). Testing took approximately two hours. Noting Lezak's description of a "sterile" environment, characterised by an absence of contaminants which could impact on the veracity of tests (1995, p. 137), participants were tested separately in a quiet environment free of distractions. Whilst all participants were fluent in English, several were Afrikaans first language speakers and one participant was Xhosa speaking (see Table 7). As an Afrikaans translation of the WMS Paired Associated learning test was available Afrikaans speaking players were given the option of being tested in Afrikaans for the verbal fluency and verbal memory tests. Of a total of 21 Afrikaans speaking participants, 16 elected to do so. The Xhosa speaking participant was tested in English as there was no Xhosa translation of the verbal memory test available at the time of testing. Justification for this is gained from Shuttleworth-Jordan (1996) who analysed comparative research data on English versus Xhosa first language university students on the following tests: SAWAIS Digit Span and Digit Symbol subtests including Digits Forwards, Backwards, Difference, Supraspan and Digit Symbol Incidental Recall; three subtests from the Wechsler Memory Scale including Paired Associates, Logical Memory and Visual Reproduction, immediate and 30 minute delayed recall; Verbal Fluency - Words in one minute; The Trail Making Test and the Finger Tapping Tests. In spite of a general trend towards lowered scores in the Xhosa language group, across all the tests the mean scores of both language groups fell within one standard deviation of each other. Shuttleworth-Jordan considers this lowering to be of trivial clinical significance. Based on these findings she supports the use of standardized cognitive tests with urbanized black populations,

particularly more educationally advantaged students who are studying in the medium of English. In consideration of the fact that the Xhosa speaking participant in this study attended an English medium school and spoke English fluently it was considered that a marginal lowering would not significantly effect his overall test performance, or influence the group analyses.

TABLE 7. First Language of Participants

Sport	n	English	Afrikaans	Xhosa
Cricket	21	18	2	1
Rugby	25	6	19	0

3.2.1 QUESTIONNAIRES (See Appendix C)

A questionnaire was designed for the purposes of this research which drew information on educational qualifications, occupational history, sport playing history, previous head injuries as well as exclusion type criteria. In order to assess the frequency of residual postconcussive symptoms suffered by players, a symptom checklist was administered.

3.2.2 NEUROPSYCHOLOGICAL TEST BATTERY (See Appendix D)

The neuropsychological test battery utilized was designed to provide information on the players' estimated premorbid level of functioning as well as current functioning across a comprehensive spectrum of key cognitive modalities typically compromised in closed head injury, including: attention and concentration, memory/new learning, language fluency, visuoperceptual tracking and fine hand motor functioning.

The following tests were administered: South African Wechsler Adult Intelligence Scale (SAWAIS) Digit Symbol Substitution subtest; Digit Symbol Substitution subtest - Incidental Recall; Trail Making Test (TMT), Part A and Part B; Words-in-One-Minute Unstructured Verbal Fluency Test; "S" Words Verbal Fluency Test; Sequential Finger Tapping Test; Wechsler Memory Scale (WMS) Memory for Designs subtest; Picture Completion subtest; SAWAIS Comprehension subtest; WMS Memory for Designs Delayed Recall; WMS Paired Associated

Learning subtest; SAWAIS Digit Span subtest; Digit Supraspan; Sequential Finger Tapping - repeat trial; WMS Paired Associate Learning Delayed Recall. These are all tests regularly used in neuropsychological assessment. Unless otherwise specified, standardized procedures as described below were used for test administration.

3.2.2.1 Tests of General Intellectual Functioning

Estimates of levels of premorbid cognitive functioning are important in that they provide a yardstick or comparison standard for each individual player allowing for the detection and quantification of cognitive deficit. Lezak supports the validity of using a single test score or, more desirably, a cluster of scores to estimate premorbid ability of intellectual functioning, utilizing the best performance method. This assumption flows from the phenomena of "transituational consistency" of cognitive behaviour (1995, p. 106). Thus to estimate premorbid levels of functioning two subtests of general intellectual functioning drawn from the SAWAIS were administered (Comprehension and Picture Completion). For all instances of normal scores (8.5+) on these two subtests, a pro-rated IQ was calculated. This method was utilized for all participants except for participant no. 22 where a defective score on Picture Completion was recorded. This score contradicted his performance across other tests as well as his educational level, a variable usually correlating well with IQ. In this instance the score obtained on Comprehension only was used to estimate the premorbid IQ. This is in accordance with Lezak (1995) who condones the use of a single score as an indicator of premorbid IQ. (For estimated premorbid IQ and education levels, see Appendix E).

SAWAIS Comprehension

This SAWAIS subtest is designed to test verbal reasoning and social judgement and is likely to be one of the better indicators of premorbid ability as verbal reasoning holds up well in the presence of diffuse damage (Lezak, 1995). The test consists of 10 questions which invoke understandings of given situations and entail the participant providing answers to specific tasks or problems. Participants are instructed that there is no one correct answer to each question, rather, they have to say what they think in each case. Participants are questioned further should their responses require elaboration. Instructions were taken from the SAWAIS Manual (1969).

SAWAIS Picture Completion

Picture Completion is a test of visual reasoning which involves both visuoperceptual and verbal abilities. It is potentially a good indicator of premorbid ability as it consistently demonstrates resilience to the effects of diffuse cerebral damage (Lezak, 1995). The Picture Completion subtest consists of 15 drawings each of which lacks a single important element or item within the picture. The pictures, arranged in order of increasing difficulty, are shown individually and the participants are required to identify the missing element within a 20 second time limit. Instructions were taken from the SAWAIS Manual (1969).

3.2.2.2 Tests of Verbal Memory

SAWAIS Digit Span

Digit Span is used to measure span of immediate verbal recall. The test consists of Digits Forward and Digits Backward which both involve auditory attention and depend on a short term retention capacity. While some studies have reported only a composite score combining Digits Forward and Digits Backward (Ruff et al., 1989) it was decided to report and analyse Digit Span Forward and Digit Span Backward as separate tests as they involve different mental processes and are thus affected differently by brain damage (Lezak, 1995). The SAWAIS Digit Span was used and instructions were taken from the SAWAIS Manual (1969).

Digits Forward Span: Digits Forwards measures what Lezak (1995) terms 'freedom from distractibility'. This is more closely related to the efficiency of attention than to what is commonly thought of as memory. While Digits Forwards span of some patients is likely to fall below normal limits in the first few months following head injury, it is likely to return to normal levels during subsequent years (Lezak 1995). It is also less vulnerable to diffuse damage than Digits Backward. The test consists of a number of random number sequences that the researcher reads aloud at a rate of one number per second. Participants are required to repeat, in the correct order, each number sequence immediately after the researcher reads it to them. Each trial consists of a pair of sequences consisting of the same number of digits. If participants are able to repeat a sequence, the researcher progresses onto the next trial which consists of a sequence with an extra digit. The test is discontinued after two sequences of a trial are failed. The score is the longest sequence of numbers correctly repeated.

Digits Supraspan: This is a test of verbal new learning ability and is thus sensitive to diffuse brain damage (Shuttleworth-Jordan, 1992). Digits Supraspan is an extended version of the Digits Forward Test and the method proposed by McFie (1975) was used. This test involves learning the span above the normal digit span forwards. Thus after participants fail two consecutive sequences of the same length, the researcher repeats the last failed sequence until participants recall it correctly. The score is the number of trials taken to learn the span (Supraspan A).

Digits Supraspan Sustained Learning: A second supraspan score was yielded on this test (Supraspan B). Participants were required to repeat the supraspan consecutively in order to determine, more sensitively, the number of trials taken for real learning to take place.

Digits Backwards*:* In relation to Digits Forwards, Digits Backwards requires considerably greater transformation of the stimulus input prior to recall (Sattler, 1992). The mental image of the numerical sequence must be held longer and must also be manipulated before it is restated. This taps working memory and is particularly sensitive to diffuse damage as may be expected in closed head injury (Lezak, 1995). Participants are instructed to repeat in reverse order the sequence of numbers read out by the researcher.

WMS Paired Associate Learning

The version used was taken from Form 1 of the WMS manual (Wechsler, 1945). The Afrikaans translation by Burbach (1987) was used for Afrikaans speaking participants. While the Afrikaans version is not standardized all the various comparison groups were administered the same test. The test consists of a series of ten paired words, made up of five easy pairs and five hard pairs. The easy pairs consist of words normally associated with one another while the hard pairs consist of words not normally associated with one another and which are thus more difficult to learn. The researcher reads out the sequence of pairs and then reads out only the first word of each pair and the participant is instructed to recall the associated word. The test consists of three presentations. Old associate learning ability is required to remember the easy pairs while new learning ability is required to remember the hard pairs. Consequently the hard pairs are more susceptible to the effects of brain damage (Lezak, 1995). As a composite score would have lacked sensitivity in the detection of cerebral impairment, scores for the easy and hard pairs were analysed and reported separately.

WMS Paired Associate Learning Delayed Recall

As delayed memory is typically more sensitive to the effects of diffuse brain damage (Lezak, 1995), a 20 minute delayed version of this test was administered to the participants. In the delayed version of this test the first word of the pair is read out and the participant is required to recall the associated word.

3.2.2.3 Tests of Visual Memory

Digit Symbol Incidental Recall (Immediate)

This particular test elicits recent memory function which is sensitive to the effects of diffuse brain damage. The short form method of the incidental recall test (Shuttleworth-Jordan & Bode, 1995) was used. The Digit Symbol test (see tests of visuoperceptual tracking) was completed in the normal way. The participant filled in the squares to the end of the second last row, although the researcher noted which square the participant filled in after 90 seconds had lapsed. The researcher then gave the participant a sheet of paper marked with the numbers on which the participant had to fill in as many of the matching symbols as could be recalled.

Digit Symbol Incidental Recall (Delayed)

A delayed recall version of the incidental recall test was administered. After a 20 minute delay participants are again given a sheet of paper marked with the numbers on which they are asked to fill in as many matching symbols as they can.

WMS Visual Reproduction

The test involves immediate recall and is particularly sensitive to right hemisphere head trauma (Lezak, 1995). The Form 1 version from the WMS manual (Wechsler, 1945) was used. The test consists of three cards: Card I and Card II consist of one design each and Card III consist of two designs. Each card is shown to the participant for 10 seconds. Following each exposure participants are instructed to draw what they remembered of the design.

WMS Visual Reproduction Delayed Recall

A delayed version of this test was administered. After a 20 minute delay the participants are given a clean sheet of paper on which they are instructed to draw the designs from memory.

3.2.2.4 Tests of Verbal Fluency

Words-In-One-Minute (Terman & Merrin 1973)

This is an unstructured test of verbal fluency. Administration of this test follows Lezak's description of the test (1995). The researcher instructs the participants to say as many words as they can think of as quickly as they can. Participants were instructed to desist from the following: using proper nouns, using the same word whilst changing only the suffix, constructing sentences or counting. The participant was stopped after one minute.

S-Words-In-One-Minute

This variation of the Words-In-One-Minute test, as described by Lezak (1995), involves the participants having to say as many words as they can think of, as quickly as they can, beginning with the letter S. Instructions were the same as those for the unstructured verbal fluency test described above.

3.2.2.5 Tests of Visuoperceptual Tracking

SAWAIS Digit Symbol

This test involves complex visuoperceptual tracking and is consistently sensitive to brain damage. Lezak (1995) notes that its score is most likely to be depressed even when damage is minimal and to be among the most depressed when other tests are affected as well. The SAWAIS version of the Digit Symbol test is made up of three rows of 67 blank squares each paired with a randomly allocated number from one to nine. Above these rows is a printed key which pairs each number with a different symbol. The participant is required to fill in the blank spaces with the symbol paired with the number in the test's key, as systematically and as rapidly as possible. Instructions were as per the SAWAIS Manual (1969). Following a demonstration of the procedure by the researcher of the first 8 blocks, the participant is instructed 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. Participants are told to fill in the symbols in the order presented without leaving any out. The researcher notes the number of blocks completed in 90 seconds.

Trail Making Test (TMT)

The Trail Making Test is a measure of sustained attention and concentration, which requires sequential problem solving and ability to keep two things in mind simultaneously (Barth et al., 1989). Again this test is a particularly effective means of detecting the effects of mild head trauma (Lezak, 1995). The test is administered in two parts (A and B).

Part A consists of a series of numbers within circles on a work sheet. Participants are instructed to draw lines connecting the circles in a consecutive sequence as quickly as they can without lifting the pencil from the paper. If participants make a mistake it is brought to their attention at that point and they are required to correct it immediately. The score is the number of seconds taken to complete the trial. Prior to proceeding with the test participants complete a practice sample.

The format and administration of Part B is similar to Part A. Part B consists of a series of numbers and letters within circles on a worksheet. Participants are instructed to connect alternating numbers and letters in the correct sequence. As Part B involves working memory, complex visuoperceptual tracking and the ability to shift a response set, in the presence of diffuse brain damage scores on Part B are likely to be discernibly depressed relative to those on Part A.

3.2.2.6 Tests of Hand-Motor Dexterity

Finger Tapping Test (Denckla, 1973)

As brain damage tends to have a slowing effect on finger tapping rate, bilateral slowing would indicate diffuse brain damage in the absence of any physical impairment. The participant is instructed to place both elbows on the table (the researcher demonstrates what is required) and with one hand at a time, to touch each finger to the thumb beginning with the index finger as quickly as they can. The score is the number of seconds taken by the participant to do five sets of sequential taps. Two trials of the test were administered in order to elicit the participant's best performance.

3.3 DATA PROCESSING

In order to ensure reliability, test protocols were marked only by the three researchers who met at various intervals to ensure a consistent standard of scoring. The data were then analysed in different ways to form three separate research projects as follows:

1) A direct comparison of group scores of the rugby versus the cricket players and the rugby forwards versus the rugby backline players across all tests administered.
2) A comparison of the cognitive profiles of rugby versus the cricket players and the rugby forwards versus the rugby backline players with available normative data.
3) A comparison of the proportion of cognitive deficit present in the rugby versus the cricket players and the rugby forwards versus the rugby backline players in each functional modality, as well as a comparison of the frequency of cognitive deficit and postconcussive symptomology.

The present research comprised the first level of analysis, that is a direct comparison of test scores of the rugby versus the cricket players and the rugby forwards versus the rugby backline.

Statistical Procedure

For every test the means and standard deviations were calculated for each of the rugby, cricket, rugby forward and rugby backline groups. For every test comparisons were made between the means and standard deviations of the rugby and cricket players and the means and standard deviations of the rugby forward and rugby backline players using independent t - tests. A non parametric Mann Whitney U Test was used to compare the medians of the rugby and cricket players' scores and the medians of the rugby forwards and rugby backline players.

CHAPTER FOUR
RESULTS

4.1 INTRODUCTION

A comparison of the neuropsychological test results for (i) the rugby and cricket players and (ii) rugby forwards and rugby backline players will be presented separately.

4.2 COMPARISON OF RUGBY AND CRICKET PLAYERS' TEST RESULTS

4.2.1 COMPARISON OF MEANS

In comparison with the rugby players, the cricket players showed significantly poorer performance on Finger Tapping 1 preferred ($P < 0.01$), Finger Tapping 1 non-preferred ($P < 0.01$), Finger Tapping 2 preferred ($P < 0.05$), and on Finger Tapping 2 non-preferred ($P < 0.05$).

4.2.2 COMPARISON OF MEDIANS

In comparison with the rugby players, the cricket players showed significantly poorer performance on Finger Tapping 1 preferred ($P < 0.05$), Finger Tapping 1 non-preferred ($P < 0.01$) and on Finger Tapping 2 preferred ($P < 0.05$).

4.2.3 COMPARISON OF VARIABILITY

In comparison with the cricket players, the rugby players showed significantly increased variability on the Digit Symbol Substitution subtest ($P < 0.05$), on the Trail Making Test - Part B ($P < 0.05$) and on Finger Tapping (second trial) non-preferred ($P < 0.05$).

4.3 COMPARISON OF RUGBY FORWARDS AND RUGBY BACKLINE TEST RESULTS

4.3.1 COMPARISON OF MEANS

In comparison with the backline players, the forwards showed a significantly poorer performance on the Digits Backwards Test ($P < 0.01$), Digit Symbol Incidental Recall (Delayed) ($P < 0.01$), Digit Symbol Substitution subtest ($P < 0.01$), Trail Making Test- Part A ($P < 0.05$), Trail Making Test- Part B ($P < 0.01$), Finger Tapping 1 non-preferred ($P < 0.01$) and on Finger Tapping 2 non-preferred ($P < 0.05$).

4.3.2 COMPARISON OF MEDIANS

In comparison with the backline players, the forwards showed a significantly poorer performance on the Digits Backwards Test ($P < 0.01$), Digit Symbol Incidental Recall- Immediate ($P < 0.05$), Digit Symbol Incidental Recall (Delayed) ($P < 0.05$), Digit Symbol Substitution subtest ($P < 0.01$), Trail Making Test - Part A ($P < 0.05$), Trail Making Test - Part B ($P < 0.01$), Finger Tapping 1 non-preferred ($P < 0.05$) and on Finger Tapping 2 preferred ($P < 0.05$).

4.3.3 COMPARISON OF VARIABILITY

In comparison with the backline players, the forwards showed significantly increased variability on Digit Symbol Incidental Recall (Delayed) ($P < 0.01$).

Table 8 : Comparison of Means (t Statistic), Medians (Non Parametric Statistic), and Standard Deviations (F Statistic) of Rugby and Cricket Players.

Test	Rugby			Cricket			t-Statistic	Non-Parametric	F-Statistic
	n	Mean	SD	n	Mean	SD			
Tests of Verbal Memory									
Digits Forward	26	7.42	0.99	21	7.33	1.35	0.26	280.0	1.53
Digits Backward	26	6.15	1.38	21	5.95	1.50	0.48	292.5	0.82
Digit Supraspan	26	2.30	1.32	21	2.76	2.21	0.87	256.0	2.45
Digit Supraspan Sustained Learning	26	3.38	1.58	21	3.67	2.11	-0.53	260.5	1.23
WMS Pairs (easy) - Immed	26	8.81	0.43	21	8.50	0.82	1.66	338.5	3.01
WMS Pairs (hard) - Immed	26	8.08	2.80	21	8.36	3.13	-0.32	250.5	0.23
WMS Pairs (easy) - Delay	26	5.92	0.27	21	5.95	0.21	-0.40	265.0	0.65
WMS Pairs (hard) - Delay	26	3.26	1.07	21	3.42	0.81	-0.56	262.0	1.80
Tests of Visual Memory									
Digit Symbol Incidental Recall - Immed	26	6.73	2.35	21	6.86	1.71	-0.21	285.5	2.80
Digit Symbol Incidental Recall - Delay	26	6.90	2.26	21	6.95	1.99	0.08	279.5	1.07
Visual Reproduction - Immed	26	11.96	1.40	21	11.29	1.65	1.52	340.5	0.22
Visual Reproduction - Delay	26	11.30	1.89	21	10.95	1.43	0.71	315.0	3.80
Tests of Verbal Fluency									
Words-in-One-Minute	26	38.92	7.78	21	39.48	7.14	-0.25	271.5	0.16
S-Words-in-One-Minute	26	17.50	4.58	21	17.10	5.10	0.29	285.0	0.13
Tests of Visuoperceptual Tracking									
Digit Symbol Substitution	26	52.60	9.06	21	50.44	6.90	0.94	309.0	4.25 *
Trail Making A	26	27.66	9.12	21	25.76	6.14	0.82	301.0	2.64
Trail Making B	26	58.64	18.60	21	53.95	11.00	1.07	306.0	4.57 *
Tests of Hand Motor Dexterity									
Finger Tapping 1 preferred	26	5.14	0.95	21	5.86	0.77	-2.73 **	145.0 *	0.93
Finger Tapping 1 non-preferred	26	4.90	0.85	21	5.75	0.68	-3.67 **	119.5 **	0.30
Finger Tapping 2 preferred	26	4.73	0.71	21	5.25	0.58	-2.64 *	159.0 *	0.42
Finger Tapping 2 non-preferred	26	4.70	0.85	21	5.20	0.60	-2.31 *	189.5	6.03 *

Significant Difference (* $p < 0,05$; ** $p < 0,01$)

Table 9:Comparison of Means (t Statistic), Medians (Non Parametric Statistic) and Standard Deviations (F Statistic) of Rugby Forward and Backline Players.

Test	Forwards			Backline			t-Statistic	Non-Parametric	F-Statistic
	n	Mean	SD	n	Mean	SD			
Tests of Verbal Memory									
Digits Forward	15	7.40	1.05	11	7.45	0.93	-0.14	80.5	0.48
Digits Backward	15	5.47	1.19	11	7.09	1.04	-3.62 **	24.5 **	1.12
Digit Supraspan	15	2.27	1.16	11	2.36	1.57	-0.18	84.5	0.27
Digit Supraspan Sustained Learning	15	3.40	1.64	11	3.36	1.57	0.06	83.5	0.01
WMS Pairs (easy) - Immed	15	8.73	0.53	11	8.91	0.20	-1.04	69.0	2.96
WMS Pairs (hard) - Immed	15	7.87	2.88	11	8.36	2.80	-0.44	75.5	0.04
WMS Pairs (easy) - Delay	15	5.93	0.25	11	5.90	0.30	0.22	84.5	0.19
WMS Pairs (hard) - Delay	15	3.33	1.11	11	3.18	1.07	0.35	91.0	0.00
Tests of Visual Memory									
Digit Symbol Incidental Recall - Immed	15	6.00	2.30	11	7.72	2.10	-1.96	44.5 *	1.89
Digit Symbol Incidental Recall - Delay	15	5.97	2.40	11	8,18	1.25	-3.05 **	35.0 *	7.96 **
Visual Reproduction - Immed	15	11.73	1.33	11	12.27	1.49	-0.97	59.5	0.01
Visual Reproduction - Delay	15	11.53	1.73	11	11.00	2.14	0.70	95.0	1.14
Tests of Verbal Fluency									
Words-in-One-Minute	15	37.00	8.58	11	41.55	5.92	-1.51	56.0	0.94
S-Words-in-One-Minute	15	16.67	4.84	11	18.64	4.15	-1.09	70.0	0.17
Tests of Visuoperceptual Tracking									
Digit Symbol Substitution	15	47.97	7.86	11	58.91	6.56	-3.75 **	26.5 **	1.60
Trail Making A	15	30.69	9.24	11	23.54	7.50	2.11 *	120.5 *	0.03
Trail Making B	15	67.42	16.69	11	46.66	14.19	3.33 **	137.5 **	0.56
Tests of Hand Motor Dexterity									
Finger Tapping 1 preferred	15	5.40	0.74	11	4.79	1.12	1.65	106.5	2.07
Finger Tapping 1 non-preferred	15	5.30	0.65	11	4.41	0.84	2.98 **	122.0 *	0.76
Finger Tapping 2 preferred	15	4.94	0.65	11	4.45	0.72	1.82	122.0 *	0.11
Finger Tapping 2 non-preferred	15	4.99	0.73	11	4.30	0.87	2.19 *	118.0	1.25

Significant Difference (* $p < 0,05$; ** $p < 0,01$)

CHAPTER FIVE
DISCUSSION

5.1 INTRODUCTION

The objective of this research was to investigate the effect of cumulative concussive and sub-concussive MHI amongst professional rugby players. The aim was to compare the cognitive performance of professional rugby players to a control group of professional players of a non-contact sport (cricket).

With regard to cognitive performance it was hypothesised that on measures known to be sensitive to the effects of diffuse neuronal damage, the overall performance of the rugby players (as reflected in *mean* scores) would be significantly poorer than that of the cricket players, due to the long term cumulative mild concussive and subconcussive head injuries that the rugby playing group is exposed to (whereas the cricket players are not), which would serve to reduce BRC to a greater extent within the rugby playing group. Further it was hypothesized that within the rugby playing group reductions in BRC would occur differentially for players depending on whether they are in forward or backline positions, due to their differential exposure to cumulative concussive and subconcussive MHI thereby increasing *variability* of scores in the rugby group relative to the cricket group. More specifically it was hypothesized that on measures sensitive to diffuse brain damage, the overall performance of the forward players (as reflected in *mean* scores) would be significantly poorer than that of the backline players, due to the forward players greater exposure (relative to backline players) to long term cumulative mild concussive and subconcussive head injuries, which would serve to reduce BRC to a greater extent within the forward playing group.

In order to limit the influence of confounding variables it was vital that there was a consistency in terms of key subject variables which may account for differences between comparative groups. Thus it was necessary to control factors known to influence neuropsychological tests results such as age, education level and estimated premorbid IQ. Analyses conducted on the demographic data revealed that there were no significant differences between the rugby and cricket players with respect to age ($P = 0.77$), years of education ($P = 0.24$) or estimated premorbid IQ levels ($P = 0.57$) and no significant differences between the rugby forward and

backline players with respect to age (P = 0.58), years of education (P= 0.18) or estimated premorbid IQ levels (P = 0.33). These variables suggest that the groups had similar abilities and thus differences in performances on the neuropsychological battery could be ascribed to cognitive impairment caused by closed head injury.

A confounding variable that was not controlled for, was the timing of the assessments. Under ideal circumstances the cricket team should have been assessed during their pre-season evaluation (as opposed to their post-season evaluation) in order to match the testing conditions of the rugby team more accurately. However, as explained previously, national and international commitments of the respective teams limited assessment opportunities. As a result the cricket team, who had lost their test series the day before the assessments began, appeared tired and demotivated compared to the rugby players. This may have adversely influenced the cricketers' test performances. Whilst this may have masked the effects of cognitive fall off on the part of rugby players compared to cricket players, it also limits the possibility of exaggerating test performance differences, thereby producing a more stringent evaluation.

5.2 NEUROPSYCHOLOGICAL TEST RESULTS

5.2.1 COMPARISON OF RUGBY PLAYERS AND CRICKET PLAYERS

With regard to both *mean* and *median* scores, the only instances of significant differences between the rugby players versus the cricket players were for tests of Finger Tapping. With regard to *variability* of scores the most notable differences between the two groups were the significant differences in variability found on the Digit Symbol subtest, the Trail Making Test - Part B, and on the Finger Tapping (second trial) non-preferred Test. Tests on which significant differences were found will be discussed separately.

5.2.1.1 Finger Tapping

In comparison with the rugby players the cricket players showed significantly poorer *mean* score performance on Finger Tapping 1 preferred, Finger Tapping 1 non-preferred and on Finger Tapping 2 preferred. These findings may be related to the fact that 76% of rugby players and 84% of cricket players reported having sustained hand or finger injuries in the course of their sporting careers (see Table 5). These injuries possibly confound the use of the Finger Tapping test as an uncomplicated indicator of brain damage effects since variable hand injuries may

affect tests specifically related to hand motor dexterity. Of note however, Shuttleworth-Jordan et al. (1993) also found that rugby players, relative to controls, demonstrated superior performance on Finger Tapping tests. It is thus possible that rugby players as a group have particularly well developed hand motor functioning. As coarse hand finger dexterity is required for the game of rugby, it is likely that rugby players tend to possess superior hand motor functioning and/or these skills develop during the playing of the game. It is thus possible that these skills in the rugby players are so highly developed that MHI effects may not be detected on this test.

5.2.1.2 Digit Symbol subtest

While there was no significant *mean* score difference between the rugby players relative to the cricket players on the Digit Symbol subtest, in comparison with the cricket players the rugby players showed significantly increased *variability* of scores. Digit Symbol is a test of psychomotor performance that is relatively unaffected by intellectual prowess, memory or learning (Lezak, 1995). Maddocks et al. (1995) consider this task to be one of the most practical tests of speed of information processing. The test involves complex visuoperceptual tracking, motor persistence, sustained attention and response speed (Lezak 1995). Russell (1986) notes that Digit Symbol is the WAIS-R subtest most sensitive to central nervous system dysfunction. Similarly, Lezak notes that the Digit Symbol subtest score is most likely to be depressed even when damage is minimal and to be among the most depressed when other tests are affected as well. Commensurate with this, Russell (1986) reports a study on the scatter patterns of the WAIS in which patients with diffuse brain damage, right hemisphere damage and left hemisphere damage, all scored worst on the Digit Symbol. Thus, considering the extreme sensitivity of the Digit Symbol subtest to brain damage, the increased variability of scores suggests that a significant proportion of the rugby players have reached the critical BRC threshold and are thus exhibiting deficits strongly indicative of diffuse cerebral damage.

The increased variability of scores within the rugby group compared with the cricketers within the present study can be explained in terms of the curvilinear pattern of inter-individual variability associated with brain damaged groups, conceptualized in Jordan's 'Shuttle' model of variability described above (Jordan, 1997). In terms of this model, the increased variability of scores suggests that within the rugby group a significant proportion of players have reached the critical threshold where they are no longer able to adjust to deficit and are thus beginning to exhibit cognitive fall-off while a notable proportion of more preserved players are not presenting with cognitive deficit. Taking into account Jordan's model of variability, the significantly greater variability of scores within the rugby playing group on the Digit Symbol subtest invalidates the 'null' indications of average effects.

The finding that a significant proportion of rugby players are showing impairments on this test links with indications from other research into MHI in rugby. Barth et al. (1989) and Hinton-Bayre et al. (1997) found that in the sub-acute phase of single MHI, Digit Symbol was found to be a sensitive indicator of brain damage. While Maddocks and Saling (1991) found that the Digit Symbol subtest performance did not differentiate concussed and nonconcussed players by the 6th month post trauma, it is important to note that these authors compared means only. This is a limitation of a great deal of research which has typically concerned itself with mean scores without taking variability of scores into account. Because the present study considered differences in variability (rather than mean differences only), the sensitivity of the Digit Symbol subtest was revealed.

5.2.1.3 Trail Making Test - Part B (TMT-B)

While there was no significant *mean* score differences between the rugby players relative to the cricket players on the TMT-B, in comparison with the cricket players the rugby players showed significantly increased *variability* of test scores. TMT, like Digit Symbol, is a test of visuoperceptual and visuomotor tracking with motor speed and agility making a strong contribution to success on this task (Lezak, 1995). Like most other tests involving the dual components of motor speed and attention functions, the TMT is highly vulnerable to the effects of brain injury. In particular, Part B tests complex visuoperceptual tracking, mental flexibility, alertness, concentration and working memory and is likely to be more markedly lower than on Part A in the presence of diffuse brain damage (Lezak, 1995). As argued for Digit Symbol above, as TMT-B is a test sensitive to brain damage, the finding of greater variability of scores within the rugby playing group on this task, relative to the cricket players, suggests that a significant proportion of individuals within the rugby group demonstrate deficits in mental flexibility, divided attention and visuoperceptual tracking. As discussed with respect to Digit Symbol, the greater variability of scores within the rugby group is commensurate with Jordan's 'Shuttle' variability model (1997), suggesting that within the rugby group, a significant

proportion of players have reached the critical BRC threshold level and, as they are no longer able to adjust to deficit, are beginning to demonstrate cognitive impairment while a notable proportion of more preserved players are not yet displaying evidence of cognitive fall off.

The finding that a significant proportion of rugby players are demonstrating deficits on TMT-B in this study, links with previous studies in the sports arena (albeit for studies of *mean* score comparisons only) where it was found that TMT performances by patients with MHI are slower than those of control subjects (Shuttleworth Jordan, 1993). Barth et al. (1989) also recorded impaired performances on the TMT amongst American Football players, although, as mentioned above, this was confined to the sub acute phase of MHI. Kaste et al. (1982) found that boxers (not restricted to those with MHI) performed significantly slower than controls on both parts of the TMT. In addition they found that slowing increases with severity of injury.

5.2.1.4 Synopsis: Comparison of Rugby Players' and Cricket Players' Results

In sum, an analysis of the significant *mean* score differences between the rugby and cricket groups did not provide support for indications commensurate with the presence of diffuse brain damage in the rugby group. The only significant differences found were significantly poorer performances on the part of the cricket players relative to the rugby players on tests of Finger Tapping. Evidence of superior finger tapping abilities in rugby players has been noted in previous studies (Shuttleworth-Jordan et al., 1993) suggesting that this test has not been a good discriminator of diffuse brain damage for a professional rugby playing group.

The significantly increased *variability* of scores for the rugby players compared with the cricketers on the Digit Symbol subtest and on the TMT-B invalidates the null indications of average effects. Together, Digit Symbol and TMT-B are frequently cited in the neuropsychological literature as the most sensitive indicators of diffuse damage consequent on mild closed head injury. Both tests have shown up deficits in prior sports related head injury studies. Digit Symbol and TMT-B are both tests of complex visuoperceptual and visuomotor tracking and as they involve the dual components of motor speed and attention, they are highly sensitive to the effects of brain injury. Thus, although the mean scores of the rugby players are not significantly different to those of the cricketers, the significantly greater variability of scores within the rugby group compared to the cricket group on both these tests indicate that a

significant proportion of rugby players are demonstrating impairments typically associated with diffuse cerebral damage resulting from MHI. This notable finding would have been missed had only mean score differences between the groups been examined.

5.2.2 COMPARISON OF RUGBY FORWARDS AND RUGBY BACKLINE PLAYERS

The only test on which significant differences were found across all three levels of comparison (mean scores, median scores, and variability) was the Digit Symbol Incidental Recall (Delayed) on which the forwards' mean and median scores were significantly poorer than the backline players and the forwards showed increased variability of scores relative to the backline. With regard to both mean and median scores, relative to the backline, the forward players showed a significantly poorer performance on the Digits Backwards Test, Digit Symbol, TMT- A and TMT-B, Finger Tapping 1 non-preferred and Finger Tapping 2 non-preferred. With regards to median scores only, in comparison with the backline, the forward players showed a significantly poorer performance on Digit Symbol Incidental Recall (Immediate). Tests on which significant differences were found will be discussed separately.

5.2.2.1 Digit Symbol Substitution subtest

In comparison with the backline, the forwards showed a significantly poorer performance on Digit Symbol Substitution reflected in significant differences on both a comparison of mean and median scores. As discussed in paragraph 5.2.1.2 the rugby group as a whole showed a significantly increased variability of scores relative to the cricket group on this test, implying that a significant proportion of the players' performances on this test were dropping off relative to the rest of the group. The significantly poorer mean performance of the forwards relative to the backline explains the increased variability of scores recorded amongst the rugby group as a whole and confirms specifically that it was the group of forward players whose performances were dropping off relative to the rest of the group. As noted above, this test involves complex visuoperceptual tracking and is highly sensitive to the effects of diffuse damage (Lezak, 1995). The impaired performance of the forwards compared to the backline players is thus indicative of impaired information processing abilities on the part of the forwards. As stated earlier these deficits are associated with diffuse damage consequent on mild closed head injury.

5.2.2.2 Trail Making Test, Part A and Part B

The forward players recorded a significantly poorer performance relative to the backline players on TMT-A and TMT-B on both comparisons of means and medians. As discussed in paragraph 5.2.1.3 the TMT taps cognitive functioning related to performance skills, attention, serial retention, integration and problem solving. Lezak (1995) asserts that slow performances on both Parts A and B point to the likelihood of brain damage. As discussed in paragraph 5.2.1.2 in regard to Digit Symbol, the rugby group as a whole showed a significantly increased variability of scores relative to the cricket group on TMT-B suggesting that a significant proportion of the players' performances on TMT-B were dropping off relative to the rest of the group. The significantly poorer mean performance of the forwards relative to the backline explicates the increased variability of scores recorded amongst the rugby group as a whole. As with Digit Symbol this result establishes that it was the forwards whose performances were dropping off relative to the rest of the rugby group. These results suggest that the forwards in particular are showing deficits in mental flexibility, divided attention and visuoperceptual tracking.

5.2.2.3 Digits Backward

A comparison of *mean* scores revealed that the rugby forward players recorded a significantly poorer performance relative to the rugby backline on Digits Backwards. Digits Backwards is an effortful double-tracking activity calling upon working memory in that the subject is required to perform a set of alternative cognitive tasks (Lezak, 1995). Since Digits Backwards is a test of working memory, it is particularly sensitive to diffuse brain damage (Shuttleworth-Jordan, 1992). This is in contrast to Digits Forwards which tends to remain relatively intact relative to Digits Backwards in the presence of diffuse brain damage (Lezak, 1995). The fact that a significant difference between the forward and backline players was observed on Digits Backwards and not Digit Forwards lends further weight to the suggestion of cerebral diffuse damage being observed amongst the forward players. In light of the fact that Digits Forwards tends to hold in the face of MHI, the lack of significant differences amongst the forward and the backline players on this test is an additional indicator that premorbidly the two subgroups functioned at a similar level.

5.2.2.4 Digit Symbol Incidental Recall (Delayed)

In comparison with the backline, the forwards showed a significantly poorer performance on Digit Symbol Incidental Recall (Delayed) reflected in significant differences on both a comparison of *mean* and *median* scores. In addition, relative to the backline, the forwards showed increased *variability* of scores on this task. This test taps delayed memory function and has been shown to be particularly sensitive to the effects of diffuse brain pathology (Lezak, 1995).

In terms of *mean* and *median* comparisons, the impaired performance of the forwards, relative to the backline on this task, is thus reflective of deficits in delayed visual memory, indicating the presence of diffuse brain injury amongst the rugby forwards. This finding is similar to previous research into MHI which has demonstrated that patients with MHI demonstrate a disproportionate impairment of delayed, as compared with immediate, recall (Dikmen et al., 1989; Stuss et al., 1985). Baddeley, Harris and Sunderland (1987) point out that patients with MHI may perform reasonably well on immediate recall tasks; however, recall shows an exaggerated decline following a delay filled with distraction. This vulnerability to interference was demonstrated by Stuss et al. (1985) who found that patients with MHI who were employed and did not have neurologic deficits were selectively impaired on a task requiring them to recall three consonants after interference delays of varying durations.

In terms of comparisons of *variability*, the significantly increased variability of scores amongst the forward players, relative to the backline players, can be understood in light of the 'Shuttle' model of variability (Jordan, 1997) discussed earlier. The increased variability of scores seen amongst the forwards players on this task is typical of brain damaged groups who, according to the 'Shuttle' model, exhibit an initial increased variability as a significant proportion of the group fall beyond the critical BRC threshold and exhibit functional symptomology, while a substantial proportion have not yet fallen beyond the critical threshold and are still able to adjust to cognitive deficit. The increased variability thus indicates that, at this point, not all forward players have succumbed to cognitive impairment resulting in a wide distribution of scores.

5.2.2.5 Digit Symbol Incidental Recall (Immediate)

While the poorer performance on Digit Symbol Incidental Recall (Immediate) of the forward players relative to the backline players was not significant in terms of *mean* comparisons, a significant difference was revealed when their *medians* were compared. The median, like the mean, is also a measure of central tendency. The median however, is based on where the midpoint of the distribution falls and therefore an equal number of scores fall above and below it. This means that in terms of the results of this test, the midpoint of the distribution of the scores for the forwards was significantly lower than that of the backline players. This indicates that the forwards are demonstrating poorer performances on recent visual memory. The fact that this difference was reflected only on a comparison of group median scores is an indication that although immediate recall is sensitive to the effects of diffuse brain damage (Lezak, 1995), it is not as vulnerable as delayed recall on which significant differences were noted across all levels of comparison.

5.2.2.6 Synopsis: Comparison of Rugby Forward and Rugby Backline Results

The forward players demonstrated deficit relative to the backline players in the modalities of working memory (Digits Backwards, TMT-B), visuoperceptual tracking (Digit Symbol, TMT-A and TMT-B), Verbal Memory (Digits Backwards) and Visual Memory (Digit Symbol Incidental Recall - Immediate and Delayed). On the overall comparison between the rugby and cricket groups, the significantly greater variability within the rugby group indicated that some of the rugby group were falling off relative to the rest of the group on Digit Symbol and TMT-B. The significantly poorer performance of the forward players relative to the backline players on these tests confirms specifically that it was the forward players whose performances were dropping off relative to the rest of the group. As commented upon earlier, the forward players are those most likely to be exposed to the risk of cumulative MHI. Taken together, the deficits recorded amongst the forward players across these modalities is highly consistent with the effects of diffuse cerebral damage associated with cumulative MHI.

5.3 IMPLICATIONS OF THE STUDY

Overall the results of the present research support previous findings of cognitive deficit associated with MHI in rugby (Barth et al., 1989; Hinton Bayre et al., 1997; Maddocks & Saling, 1991; Shuttleworth-Jordan et al., 1993). This research however, differs from previous studies in that, apart from Shuttleworth-Jordan et al., 1993, the studies mentioned above were limited to the acute and subacute phase of single MHI and are therefore unable to comment on the permanency of deficit. This research reflected on relatively chronic, rather than merely acute, sequelae as the rugby players were assessed pre-season and had not sustained a rugby related head injury for at least four months. The positive findings in this research thus support those of previous research into cumulative MHI in general (Gronwall and Wrightson, 1975; Gronwall, 1989) and are consistent with these researchers' findings that cumulative MHI increases the risk of permanent residual cognitive deficit.

This research differs from previous studies in that rather than only examining significant *mean* differences between groups, cognisance was taken of the differences of *variability* of scores between groups. The most salient aspect of this research is the finding that the rugby forwards are performing at a significantly poorer level than the rugby backline on tasks sensitive to the effects of diffuse cerebral damage. The lack of significantly different mean scores between the rugby group as a whole compared to the cricket group may be understood in light of the significantly better performances of the rugby backline relative to the rugby forwards which has the effect of raising the mean scores in the rugby group as a whole. The finding that forward players show greater evidence of impairment than the backline players is commensurate with previous research by Shuttleworth Jordan et al. (1993) who found indications that while rugby backline players and controls were able to benefit from practice on neuropsychological tests, rugby forward players failed to demonstrate improvements in performance despite practice. While the indications of impairment in the 1993 study were subtle and tentative, this research replicates these findings and demonstrates more emphatically that forward players exhibit more marked cognitive impairment compared to backline players.

Findings appear to support the 'Shuttle' model of variability (Jordan, 1997) and the BRC Theory (Satz, 1993) within which Jordan's model is constituted. As described previously (paragraph 2.3.3) the BRC concept is linked to the notion of a threshold factor which exists prior to the

presentation of symptoms due to disease in the central nervous system. Thresholds for the clinical presentation of symptomology vary depending on factors such as gender, education, intelligence and age. The gender factor was automatically controlled in that all participants were male. An analysis of the other demographic data revealed no significant differences between the rugby backline and forward players in regard to education, intelligence and age, suggesting that their respective levels of functioning and premorbid BRC are equivalent.

BRC levels can change in the face of vulnerability factors, including the cumulative neurological insults of MHI. As the neural attrition resulting from these insults advances, protective factors become less effective lowering the threshold of symptom presentation and thus increasing vulnerability to cognitive impairments. This is evident in this study's finding that rugby forward players (because of their more frequent exposure to concussive and subconcussive head injury than the backline players) are falling below the BRC threshold and exhibiting symptoms suggestive of diffuse cerebral damage typically associated with MHI. This phenomenon also contributes to the significantly increased variability in scores observed within the rugby group as a whole compared with the cricket group, since a significant proportion of the more exposed forward players reveal early symptom onset whereas a significant proportion of the less exposed back players do not.

While the backline players do not presently exhibit symptoms of functional cognitive impairment, this does not necessarily indicate a lack of brain injury. Their past and ongoing exposure to further head insults makes them vulnerable to neuronal attrition which in time may result in a lowering of BRC leading to more overt cognitive deficit. There is also evidence that secondary stressors may temporarily lower the critical threshold and reveal evidence of functional impairment. This was seen in studies which reversed null findings following MHI by testing subjects under alternative test conditions. Underlying residual symptomology were exposed by Parasuraman et al., 1991, (paragraph 2.2.3.2) who tested patients with MHI under task conditions requiring sustained effortful processing and by Ewing et al., 1980, (paragraph 2.2.3.1) who tested subjects under hypoxic stress. In addition, the full extent of symptom presentation following neuronal attrition may be delayed until additional neuronal loss occurs. This attrition may occur in the course of any neuropathological process, including the process of normal ageing (Satz, 1993). Moreover, Shuttleworth-Jordan (1999) asserts that the onset of

older age superimposed on top of a prior 'silent' head injury will serve to further reduce BRC, increasing the risk of functional impairment which would not have been present only on the basis of older age. Thus while the backline players may not be showing signs of symptomology at present, their exposure to ongoing cumulative MHI may lead to clinical presentations at a later stage.

5.4 CONCLUSION

The overall results of this study indicate that a significant proportion of the rugby players' performances were falling off relative to the rest of the group on tests vulnerable to the cognitive effects of diffuse brain damage. Comparisons of the performances of the rugby forward and backline players indicated that it was the subgroup of forward players in particular, whose test performances revealed deficits suggestive of diffuse cerebral damage. Specifically deficits were found in working memory, visuoperceptual tracking, verbal memory and visual memory. Thus the findings of the present research indicate that rugby players, in particular rugby forward players, are vulnerable to sustaining permanent cognitive deficit commensurate with diffuse cerebral damage due to the cumulative effects of closed MHI**.**

The implications of this study are particularly alarming when one considers the prominence of rugby in South Africa today which leads to tremendous overt and subtle pressure on schoolboys to participate in the sport. Considering that the incidence of MHI in rugby is conservatively estimated at about 10% of players per season, and given that the majority of players are likely to sustain multiple impacts to the head during the course of long term participation in the sport, it is vital that the hazardous potential of sustaining repeated MHI in rugby is taken seriously. It is thus imperative that sport and school authorities, parents and aspiring rugby players are alerted to the potential risks of exposure to multiple concussive and subconcussive injuries. This awareness will facilitate more informed decisions with regard to matters of rugby policy and participation in the sport.

5.5 EVALUATION AND RECOMMENDATIONS FOR FUTURE RESEARCH

5.5.1 METHODOLOGICAL STRENGTHS OF PRESENT RESEARCH

Compared to earlier research into mild head injuries in contact sports, the present study benefits from the following strengths:

1. The researchers were granted access to professional rugby players. This appears to be the first time that SARFU has granted permission for neuropsychological assessments to be conducted in order to examine the effects of cumulative MHI. Professional rugby players are most likely to have intensive, ongoing exposure to concussive and subconcussive MHI making them a highly appropriate group for the purposes of this research.

2. Positional differences were accounted for within the rugby group which allowed for a refinement in regards to detecting impairments. The differentiation between forward and backline players elicited vital information that may have been missed had this level of comparison been ignored.

3. While research has typically concerned itself with mean scores only, this study also took the variability of scores into account. The interpretation of variability is likely to enhance the possibility of detecting subtle indications of brain damage which could otherwise be overlooked.

4. To control for extraneous variables that may affect cognitive performance, both the rugby and the control group were drawn from a relatively homogeneous group of high functioning, professional sports players. The consequent equivalence of both groups in regard to key demographic variables such as age, education and estimated premorbid IQ levels means that findings can be attributed to cognitive impairment caused by closed head injury rather than differing premorbid abilities.

5. The comprehensive neuropsychological test battery allowed for the emergence of patterns of impairment across a range of key cognitive modalities typically associated with deficits associated with MHI and included tests to estimate premorbid IQ. Previous research in this field has typically been based on significantly more limited test batteries.

5.5.2 METHODOLOGICAL LIMITATIONS OF PRESENT RESEARCH

A strict critical evaluation of this study reveals certain limitations which, arguably, do not significantly detract from the robustness of the findings. These limitations and moderating factors are described below.

1. Whilst the control group used in this study was an improvement over many past studies in that they were well matched professional players of a non-contact sport, it is possible that these players were not as high functioning as they could have been as most of them had a history of playing rugby. Players of a non-contact sport who typically do not have a history of playing contact sport at school would thus have been a more appropriate control group. In addition, as noted previously (paragraph 3.2), national and international commitments of the respective teams limited assessment opportunities. Consequently the cricket team was assessed during its post-season evaluation in contrast to the rugby players who were tested pre-season. The post-season fatigue and seeming despondency on the part of the cricket team may have obscured their optimal level of functioning and thus may have masked the effects of cognitive fall off on the part of rugby players compared to cricket players. Ideally both groups should have been tested pre-season in order for testing conditions to have been more accurately matched. However, while these factors may have under-detected the amount of impairment in the rugby players, they also served as a more stringent measure of comparison in that where there are significant findings these can be considered to be relatively robust.

2. Time constraints meant that premorbid IQ estimates were based on only two tests. However this was still a legitimate method for estimating IQ in that these tests are considered good estimates of general intellectual functioning (Lezak, 1995) and furthermore, Lezak condones the use of even a *single* subtest score in isolation as an adequate estimate of premorbid IQ. Moreover a major omission in most prior studies has been the lack of an attempt to build 'hold' tests into the battery which provide an estimate of premorbid IQ.

3. The sample size used in this research is relatively small. A larger sample would confirm the patterns detected in the present research. However the present research already appears to replicate indications of earlier studies, particularly in regard to positional findings from both rugby and soccer studies (Matser et al., 1998; Shuttleworth-Jordan et al., 1993). Moreover in light of Satz et al.'s (1997) stipulation that 20 or more participants is sufficient for MHI research, the sample size can be considered adequate.

4. Because of the cross sectional nature of this research, the possibility that differences found do not represent premorbid differences cannot be excluded. However the impairments observed in this research are highly consistent with expectations of patterns of deficit typical of diffuse brain injury, thereby increasing the probability that observed differences result from the effects of ongoing cumulative MHI.

5.5.3 RECOMMENDATIONS FOR FUTURE RESEARCH

In light of these limitations the following recommendations for future research are proposed:

1. A long term prospective study involving the assessment of professional rugby players with periodic follow up of players in order to assess delayed future cognitive effects resulting from cumulative MHI.

2. The inclusion of a matched control group of non-contact sportsmen who have not had a history of playing rugby. Hockey players appear to fit this requirement as both hockey and rugby are winter sports, limiting players' participation to one or the other.

3. Assessment procedures of comparative groups to be conducted at the same stage (ie., both groups to be tested pre-season).

4. An increased number of participants in order to confirm indications of this research.

5. The use of an additional assessment of premorbid functioning such as the inclusion of the NART (New Adult Reading Test) or a vocabulary test, on the proviso that they are appropriately standardized for the South African population.

6. It may be useful to incorporate tasks involving increased challenge in order to increase vulnerability to the demonstration of cognitive impairment (for example the use of a stress test). Under these conditions underlying deficits may be revealed.

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