Postconcussive Sequelae in Contact Sport: Rugby versus Non-contact Sport Controls

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ABSTRACT

The effects of repeated mild concussive head injury on professional rugby players were examined. Data were collected for rugby players (n=26) and cricket player controls (n=21) using a comprehensive neuropsychological test battery comprising five modalities (Verbal Memory, Visual Memory, Verbal Fluency, Visuoperceptual Tracking and Hand Motor Dexterity) and a self-report Postconcussive Symptomology Questionnaire. Group statistical comparisons of the percentage of individuals with deficit were carried out for (i) rugby versus cricket; (ii) rugby forwards versus rugby backs; and (iii) rugby forwards versus cricket. Rugby players performed significantly poorer than controls on SA WAIS Digit Symbol Substitution subtest and on the Trail Making Test. On Digits Forward and Digit Symbol Incidental Recall, the results approached significance with the rugby players showing a tendency toward impairment on these tests. Rugby players exhibited impairment in areas of visuoperceptual tracking, speed of information processing and attention, and there are tendencies of impairment in verbal and/or visual memory. Results obtained on the self-report questionnaire strongly reinforced cognitive test results and a significant proportion of rugby players reported difficulties with sustained attention, memory and lowered frustration tolerance as well as symptoms of anxiety and depression. It was consistently noted that players in the more full contact positions (rugby forwards) were most susceptible to impairment, confirming that these players, who are exposed to repeated mild head injuries, are at greater risk of exhibiting postconcussive sequelae.

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CHAPTER 1

INTRODUCTION

1.1. Background to Present Research

The present research represents the *first* stage in a national, long-term prospective study of mild head injury (MHI) in contact sport in South Africa. A large body of data was obtained from the neuropsychological assessments and self-report questionnaires of top league South African rugby and cricket players. Using this data, three broad research questions were devised and written up as three separate MA mini-theses (Dickinson, 1998; Ancer, 1998; and Reid, 1998). The motivation behind this division of data into three separate projects was primarily based on manageability, i.e. the large body of data could be broken down into three smaller and manageable units. More significantly, these separate analyses cover a cross-section of important questions in the study of mild head injury in contact sport, and each project allows for different angles on the same issue to be examined *in depth*.

1.2. Research Question

The overarching question addressed at all levels of the aforementioned large-scale investigation arose out of the findings from the growing, yet still relatively new, body of research in the field of mild head injury in contact sport. It would seem that there is heightened concern about the possible *cumulative* effects of concussive and sub-concussive mild head injuries in contact sports such as soccer, boxing and rugby. Following this concern, the hypothesis which is increasingly under examination in this field of research is whether or not people who play contact sport display cognitive or behavioural deficits and symptoms which are indicative of mild closed head injury.

What is also evident from existing research is that in both the general or clinical populations, there appears to be a dearth of empirical studies on repeated mild head injury and the cumulative effects of such trauma. It seems evident that these populations, relative to contact sport-playing populations, are not at a high risk of sustaining repeated head injuries as they are not exposed to events or activities that may lead to such injury. However, in populations of contact sport players, the risk of repeated head injuries is much greater and therefore, this population makes the study of such injury more accessible. In this vein, Barth et al. (1989) have recommended that sports injuries can be utilised as a "laboratory" model for acceleration/deceleration mild head injury in the general population. These authors report further that in most sports, great efforts have been made to evaluate and eliminate the potential for *significant* or *severe* head trauma, but similar efforts have *not* been made in the case of sports-related *mild* head injuries: "until recently, very little has been written about mild head trauma in sports because it was not seen as a major problem" (p. 258).

Broadly speaking, the present research addresses the following research question: What, if any, is the statistical difference between contact sport players and non-contact sport players on a) neuropsychological testing and b) self-report postconcussive symptomology? More specifically, as indicated by the title of this thesis, this project represents a comparative analysis of the percentage of individuals with cognitive deficit and postconcussive symptomology in rugby and non-contact sport (cricket) controls. A dual focus on cognitive deficits *and* postconcussive symptomology following *repeated mild head injury* will be the framework within which this study is to be presented. The aim is not only to understand the sequelae of mild closed head injury with specificity to contact sport, but also to reflect on the consequences of mild closed head injury in general.

CHAPTER 2

LITERATURE REVIEW

2.1. Mild Head Injury: Definition, Classification and Nomenclature

2.1.1. Introduction to the Debate

The question of mild head injury has raised many controversial issues surrounding its definition, the classification of severity and the use of appropriate nomenclature. "Definitions of concussion and mild and minor head injury have varied considerably in the literature" (Binder, 1986, p. 324). By way of a broad example, the term itself, "mild head injury" has been used interchangeably (sometimes by the same authors) with terms such as "diffuse brain injury" (Gennarelli, 1987), "mild traumatic brain injury" (Lezak, 1995), "cerebral concussive injury" (Gennarelli, 1987; Ommaya and Gennarelli, 1974), "minor head injury" (Dacey, Vollmer and Dikmen, 1993; Levin et al., 1987), "minor closed head injury" (Richardson, 1990), "minor brain injury" (Rutherford, 1989) and "minor traumatic brain injury" (Povlishock and Coburn, 1989; Wood, 1990). As will become clear in the ensuing discussion, these nomenclature problems are inextricably linked with issues of the measures and classification of severity of a head injury: how a head injury is classified will determine it's definition and its label (nomenclature).

These are important issues at a time when research in the area of mild head injury is growing in significance amongst a diverse range of professional fields including neuropsychology, sports medicine, rehabilitation, neurology and neurosurgery. It would seem that in such a culture of multidisciplinary research, a 'common language' would greatly benefit the exchange and dissemination of information. This viewpoint is supported by Levin et al. (1987) who, in their discussion on methodological issues in outcome studies, argue that "the longstanding controversy surrounding research on outcome of minor head injury and the postconcussional syndrome (PCS) reflects *ambiguities in definition* [italics added], inconsistencies in criteria for patient selection, variation in procedures for neurobehavioural assessment and difficulty in obtaining follow-up data" (p. 262). According to these authors, non-uniform criteria for defining mild head injury "have undoubtedly contributed to disparities in reported outcome" (1987, p. 262).

At the outset, it should be noted that by way of a broad distinction, *closed head injuries* (which are the focus of the present study) are conventionally distinguished from *penetrating head injuries* of the sort produced by sharp instruments such as knives, or by explosively propelled missiles such as bullets or fragments of shells. Closed and penetrating head injuries differ not merely in terms of likely external causes, but also in patterns of neurological and neuropsychological deficit to which they tend to give rise. Closed head injuries are more likely to produce disturbances of

consciousness and diffuse cerebral damage and can be defined as "*an injury to the head in which the primary mechanism of damage is one of blunt impact*" (Richardson, 1990, p. 3). This distinction between closed and penetrating head injuries is a clear one. In contrast, within the area of severity of closed mild head injury, the classification thereof and hence its definition, where the distinguishing features are significantly more complicated.

2.1.2. Head Injury Classification and Measures of Severity

According to Lezak, "the need to triage patients both for treatment purposes and for outcome prediction" (1995, p. 172) has led to the development of a generally accepted classification system based on the presence, degree and duration of coma called the *Glasgow Coma Scale* (GCS) (Teasdale and Jennet, 1974)[1]. The severity of a head injury is classified according to scores obtained on the GCS response chart (see Table 2-1, below).

Table 2-1. Severity Classification Criteria for the Glasgow Coma Scale

Classification	GCS		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

Teasdale and Jennet (1974)

Despite its demonstrated usefulness[2], especially as a predictor of outcome, the GCS has some inherent problems (Lezak, 1995). While it is a generally useful guideline to severity of injury, "the times that the GCS was measured and the circumstances surrounding the first few hours and days after the injury must be taken into account in determining how much weight to give it as a predictor in the individual case" (Lezak, 1995, p. 756).

An alternative basis for the classification of closed head injury in terms of severity is the duration of *post-traumatic amnesia* (PTA). PTA refers to the patient's inability to form new memories for a particular period after recovering consciousness. A detailed classification based on the duration of PTA was proposed by Teasdale and Jennet (1981) (see Table 2-2, p. 5). It has been found that the duration of PTA correlates well with GCS ratings (Levin, Grossman and Benton, 1982) such that estimates of severity of injury on the basis of PTA are generally in agreement and parallel the GCS severity range except for some finer scaling at the extremes (Lezak, 1995).

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[1] See Table 18-3 in Lezak (1995, p. 755) for Glasgow Coma Scale Response Chart.

[2] The GCS has widespread support and appears to take preference over other level of consciousness classifications such as those proposed by Becker et al., (1982 in Dacey et al., 1993); Ransohoff et al., (1975 in Dacey et al., 1993) and the Grady Coma Scale (in Dacey et al., 1993, p. 160).

Table 2-2. Estimates of Severity of Head Injury Based on PTA Duration

Duration of PTA	Level of Severity
< 5 minutes	Very mild
5 - 60 minutes	Mild
1 - 24 hours	Moderate
1 - 7 days	Severe
1 - 4 weeks	Very Severe
Longer than 4 weeks	Extremely Severe

Jennet and Teasdale (1981)

Lezak (1995) points out that difficulties in defining and therefore determining the duration of PTA have made its usefulness as a measure of severity questionable in some cases. For example, many patients with relatively minor head injuries 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. Despite these shortcomings, Jennet (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).

Satz et al. (1997), in a particularly thorough and useful review of 40 mild head injury studies spanning 25 years (1970-1995), report that researchers have used six different approaches to classify head injury. These include (1) use of the GCS only, (2) GCS plus other, (3) loss of consciousness (LOC) (not GCS) plus other, (4) post-traumatic amnesia only, (5) type of hospitalisation, and (6) self-report questionnaire. "Other" variables included duration of altered consciousness, linear or depressed skull fracture, CT abnormality, length of hospitalisation, neurological signs or all of the above.

With respect to the classification and measures of severity of head injury, what can be concluded with certainty at this stage, is that across the age span (from children and adolescents to adults), there is *no single, widely used, and commonly accepted system of severity classification*. Satz et. al (1997) make the unequivocal assertion that 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).

2.1.3. Definition of Mild Head Injury

There appears to be greater agreement on definitions of more severe head injuries. The above discussion on measures of severity demonstrated that the symptoms traditionally used to classify the severity of a head injury during the acute period of hospitalisation include alterations in consciousness (GCS), durations of LOC, and changes in orientation and memory (PTA). Satz et al. (1997) point out that "although 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" (p. 107). The lack of appropriate measures and the apparently transient nature of the symptoms in the mild ranges of head injury have resulted in various definitions. For the purposes clarification, it is important to review these definitions.

Rutherford (1989) asserted that the term "minor brain injury" would be a less ambiguous and more suitable term than "minor head injury" since this latter term can include injuries such as facial lacerations, a dislocated jaw or "a bite to the pinna of the ear" (p. 217). This suggestion does not seem to have acquired a major following in that influential writers (e.g. Barth et al., 1989; Binder, 1986; Boll, 1983; Levin et al., 1987; Lezak, 1995; Rimel, Giordani, Barth, Boll and Jane., 1981; Satz et al., 1997) have maintained their use of the term "*head injury*"(although as a sub-heading, Lezak [1995] refers to "Mild Traumatic Brain Injury" [p. 182]).

Following the development of the GCS, Rimel and her co-workers defined "minor head trauma" as: "*a cranial trauma resulting in a loss of consciousness of 20 minutes or less, a Glasgow Coma Scale score of 13 or better, and the need for 48 hours or less of hospitalisation*"[3] [italics added] (1981, p. 222). This range of GCS score corresponds to spontaneous eye opening, ability to obey commands, and a verbal response no worse than confused or incoherent speech. Levin et al. (1987) argue that the Rimel et al. (1981) definition should be adopted by future investigations because of the widespread use of the GCS by neurosurgeons and its high interobserver reliability. The definition *has* been used in subsequent studies (e.g., Barth et al., 1983; Gentillini et al., 1985), but again, there is no consistency in the literature (Satz et al., 1997). For example, Lezak's definition is slightly different; she states that "*with less than 20-30 minutes of loss of consciousness (LOC) if any, and PTA measured in hours rather than days, most cases of head trauma result in mild brain injury* [italics added]" (1995, p. 182).

In an attempt to address the issue of the definition of mild head injury, Asarnow et al. (1995) argue that

> severity of head injury is really a dimension. Assigning labels such as "mild" and "minor" to arbitrary cut points along that dimension so as to establish categories of severity only ends up reifying the arbitrary cut points. The critical need is not to establish a consensus of definition of the terms "mild and moderate closed head injury". What is needed is an operational definition of closed head injury severity along multiple dimensions (e.g. loss of consciousness and post-traumatic amnesia). (p. 119 in Satz et al., 1997, p. 127).

This assertion by Asarnow et al., implies that the definition for mild closed head injury should be *empirically* determined using variables that already show promise based on a priori or empirical methods (such as PTA or LOC). In other words, these authors are arguing that how one chooses to define the extreme tail of the mild head injury spectrum is, at the present time, arbitrary, and

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[3] The specification of a maximum duration of hospitalisation of 48 hours effectively excluded patients with delayed epidural haematomas, severe extracranial injuries, or serious medical complications (Rimel et al., 1981).

therefore "*researchers should seek to determine the threshold along the injury spectrum that produces a measurable effect on outcome morbidity based on the putative risk factors*" (Satz et al., 1997, p. 128). However, because there are major methodological limitations to such a retrospective empirical approach, the minimal threshold for functional morbidity is still unknown, and the suggestion proposed by Asarnow et al. should proceed with caution[4].

Clearly, further investigations are needed if there is to be any consensus on the definition of mild head injury. At present, it seems safe to conclude that the definition of mild head injury does not entail a fixed, clearly circumscribed set of criteria. Instead, it is more useful to speak of the spectrum (or the "dimension", to use Asarnow et al.'s term) of mild head injury along which there are various degrees of severity, as well as various presentations of initial and/or persisting symptoms (in terms of LOC or PTA or GCS score or combinations of these). This means that on one level, mild head injury is an all-encompassing term which may include, for example, LOC for one patient, a longer or shorter duration of LOC for another, and no LOC with PTA for yet another. In recent efforts to define mild head injury for research purposes, Esselman and Uomoto (1995, in Satz et al., 1997) in conjunction with the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitative Medicine[5] have expanded the spectrum of mild head injury to include *different grades of injury severity*. It was recommended the mild head injury be defined by the measure of *at least one* of the following:

> (a) any period of loss of consciousness for less than 30 min, with GCS of 13 to 15 following the loss of consciousness; (b) any loss of memory for events immediately before or after the accident with post-traumatic amnesia for less than 24 hr; (c) any alteration in the mental state at the time of the accident (e.g. dazed, disorientated, or confused); and (d) focal neurological deficit(s) (e.g. double vision, loss of balance, taste or smell) that may or may not be transient (in Satz et al., p. 128).

This definition highlights several important issues:

(1) it represents a useful integration of criteria which are not only most commonly researched, but also, most commonly used in clinical practice;

(2) more specifically, it appears to incorporate the most widely used measures of severity of a head injury (e.g., duration of LOC, duration of PTA, GCS score, and/or orientation);

(3) because of the exclusion of a hospitalisation criteria, it encourages investigation with participants who have not been hospitalised; and

(4) it also incorporates *wider* limits of what is traditionally defined as mild head injury (for example when compared to Rimel et al.'s [1981] definition) and therefore increases the prevalence rate of mild head injury.

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[4] The proposal put forward by Asarnow et al., is currently under investigation by the same authors at the University of California (UCLA).

[5] The present author's opening assertion regarding the interdisciplinary interest in MHI, is borne out here.

Pending further investigation (such as that proposed by Asarnow et al., 1995), it is proposed that for the purposes of this thesis, a working definition of mild head injury would best be served by the criteria outlined in this definition.

2.1.4. Definition of *Concussion* in the Context of Mild Head Injury

The term "concussion" or "cerebral concussion" appears at every turn in discussions on mild head injury. However, like "mild head injury", "concussion" is also a term to which many different meanings have been attributed. In addition, the terms 'mild head injury', 'cerebral concussion' and 'concussive head injury' are *frequently* used interchangeably in the literature. It is thus important to consider the definition of concussion with respect to the definition of mild head injury.

A definition which appears regularly in the literature and is quoted by authors such as Lezak (1995) and Levin et al. (1989) is the definition proposed by Rutherford (1989) who suggests that

> Concussion is 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. (p. 217).

As Lezak (1995) points out, in recommending that concussion be defined as "an acceleration/deceleration injury to the head" which is typically but not necessarily accompanied by amnesia, "Rutherford has attempted to extend this diagnosis to the many cases of minor head injury in which behavioural sequelae are consistent with this type of brain damage but loss of consciousness is questionable" (p. 178).

In a study wherein the researchers correlated experimental and clinical observations on blunt head injuries, Ommaya and Gennarelli (1974) proposed a formal hypothesis which defined cerebral concussion as

> a *graded set of clinical syndromes* following head injury wherein increasing severity of disturbance in level and content of consciousness is caused by mechanically induced strains affecting the brain in a centripetal sequence of disruptive effect on function and structure. The effects of this sequence always begin at the surfaces of the brain in the mild cases and extend inwards to affect the diencephalic-mesencephalic core at the most severe levels of trauma [italics added] (p. 638).

This hypothesis was elaborated into a proposed classification of the possible grades of concussion (see Table 2-3, p. 9). The grades of severity according to this scale are seen to be as a result of the initial impact or impulse which then causes shear strains to the nerve fibres in the brain[6].

In addition to Ommaya and Gennarelli's classification of concussive syndromes, there have been subsequent noteworthy classification systems of concussion (Kulund, 1982, in Dacey et al., 1993;

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[6] This is similar to Rutherford's (1989) notion of concussion being an 'acceleration/deceleration injury'.

Nelson et al., 1984; and Torg, 1982, in Dacey et al., 1993). While these classifications (see Table 2-4, p. 9) are useful, they are problematic in the sense that they are vastly different from each other, and durations of, for example, LOC, PTA and RGA are not always specified. These factors will in all likelihood lead to discrepancies in diagnosis and may then further complicate the already complicated classification/definition scenario.

Table 2-3. Ommaya and Gennarelli's Hypothesis for the Syndromes of Cerebral Concussion

Grade	Description	Outcome
I	confusion	normal consciousness without amnesia
II	confusion + amnesia	normal consciousness with post-traumatic amnesia (PTA) only
III	confusion + amnesia	normal consciousness with PTA plus retrograde amnesia (RGA)
IV	coma (paralytic) ---> confusion and amnesia	normal consciousness with PTA plus RGA
V	coma	persistent vegetative state
VI	coma	death

Ommaya and Gennarelli (1974, pp. 633-654).

Table 2-4. Classifications of Concussion

Torg (1982)	Kulund (1982)	Nelson et al. (1984)
Grade I	*Mild*	*Grade 0*
"Bell rung"	Stunned, dazed	Head struck / moved rapidly
Short-term confusion	No confusion, dizziness	Not stunned / dazed initially
Unsteady gait	No nausea, visual disturbance	Headaches and difficulty concentrating
Dazed appearance	Feels well after 1-2 mins.	*Grade 1*
Loss of Consciousness (LOC)	Coordination	Stunned / dazed initially
PTA	*Moderate*	No LOC or amnesia
Grade 2	LOC	"Bell rung"
PTA	Mental confusion	Sensorium clears quickly (< 1 min)
Vertigo	Retrograde amnesia	*Grade 2*
Grade 3	Tinnitus, dizziness	Headache
PTA	Skill recovery may be rapid	Cloudy sensorium > 1 min.
Retrograde amnesia	*Severe*	No LOC
Vertigo	Longer LOC	Possible tinnitus, amnesia
Grade 4	Headache, confusion	May be irritable, confused, dizzy, hyperexcitable
Immediate, transient LOC	PTA	*Grade 3*
Grade 5	Retrograde amnesia	LOC < 1 min.
Paralytic coma		Not comatose (arousable w/ noxious stimuli)
Cardiorespiratory arrest		Grade 2 symptoms during recovery
Grade 6		*Grade 4*
Death		LOC > 1 min.
		Not comatose
		Grade 2 symptoms during recovery

Reproduced with permission form Nelson et al. (1984) in Dacey et al., (1993, p. 161)

A further critique of these classification systems, is that the gradings are at first confusing in the sense that, for example, a Grade VI on Ommaya and Gennarelli's (1974) classification and a Grade 6 on Torg's (1982) classification both culminate in "death" which would surely be as a result of a fairly severe head injury and not a minor or trivial insult. Such discrepancies have important implications for the definition of concussion and mild head injury because one could infer from the classification that not all concussions are 'mild' (concussions are generally considered to be minor trauma). Therefore, to use the terms "concussion" and "mild head injury" interchangeably may be incorrect. This apparent confusion warrants clarification and is addressed in the ensuing discussion.

The clinical usage of the term "concussion" has been influenced by assumptions about the underlying pathophysiology and the wider effects of closed head injury upon neurological function. To begin with, concussion (or *commotio cerebri*) was traditionally defined as the transient loss of consciousness without permanent damage to the brain (e.g., Denny-Brown and Russell, 1941; Symonds, 1928; Ward, 1966 in Richardson, 1990). However, such a distinction has long been recognised to be problematic (Symonds, 1962). Ommaya and Gennarelli (1974) argue that the modern concept of cerebral concussion includes at least the possibility of permanent brain damage.

Furthermore, it is widely accepted that the axonal damage involved in concussive head injury (due to rotation and/or acceleration/deceleration forces acting on the brain) causes diffuse (as opposed to focal) damage. Gennarelli (1987) points out that considerable debate continues to confuse the nomenclature of such diffuse brain injuries. He argues that because these injuries represent a *continuous spectrum of the same pathophysiology*, the varieties of diffuse brain injury are not fundamentally different from one another. This is unlike focal injuries where the pathophysiology of each lesion is different and thus individual lesion names are more readily agreed upon. "Instead, the diffuse brain injuries represent *differences in the quantity of brain damage, not the type*; thus any subdivision of them is somewhat arbitrary [italics added]" (Gennarelli, 1987, p. 109). Gennarelli goes on to argue that "axonal damage" would be a more apt descriptor for concussion, but, argues Gennarerlli, the term concussion is so well established that it would serve no purpose to discontinue its use.

Gennarelli makes a broad distinction between concussion and Diffuse Axonal Injury (DAI) (*a full account of DAI is provided in section 2.3.1*). Within each of these categories, there are further subdivisions. Firstly there is a distinction between what Gennarelli calls the Mild Cerebral Concussion Syndromes and Classical Cerebral Concussion. The *mild concussion* syndromes are identical to Grades I, II and III in Ommaya and Gennarelli's (1974) classification of cerebral concussion syndromes (see Table 2-3, p.9) where there is confusion and disorientation with or without amnesia. *Classical cerebral concussion*[7] is defined in terms of a reversible coma occurring at the instant of trauma, which may be accompanied by cardiovascular and pulmonary function changes

[7] In her discussion of Gennarelli's definition of Classical Cerebral Concussion, Lezak (1995, p. 178) (perhaps mistakenly or by way of typographical error) incorrectly refers to Classical Cerebral Concussion as "Classic Concussion".

and neurologic abnormalities, including decerebrate posturing (stiffened body position), pupillary changes and seizure-like activity, all of which dissipate within the first 20-30 minutes after the event. This type of concussion corresponds to Grade IV in Ommaya and Gennarelli's (1974) classification of cerebral concussion syndromes (see Table 2-3, p.9).

Secondly, Gennarelli differentiates between three severities of *Diffuse Axonal Injury* (DAI) (see Table 2-5, below).

Table 2-5. Degrees of Severity of Diffuse Axonal Injury (DAI)

Severity	Description
Mild DAI	Coma lasts from 6 to 24 hours.
Moderate DAI	Coma lasts more than 24 hours without prominent, long-lasting brainstem signs.
Severe DAI (previously 'diffuse white matter shearing injury')	Patient is deeply unconscious after injury and remains so for a prolonged period of time; presence and persistence of abnormal brainstem signs.

Gennarelli (1987, p. 114-115).

It is useful to observe that what is classified as 'mild DAI' (coma lasting 6 - 24 hours) is classified as a 'severe head injury' on the GCS scale (coma lasting longer than 6 hours after admission). It is therefore important to note that mild DAI does not constitute a mild head injury. Rather, the severities of DAI are in actual fact descriptions of moderate to severe brain trauma and would correspond to Grades V and VI (Ommaya and Gennarelli, 1974) and Grades 5 and 6 (Torg, 1982) of the classification of concussive syndromes (see Table 2-4, p.10). On the other hand, the *descriptions of the mild concussion syndromes and classical cerebral concussion, can be referred to as mild head injuries*. These categories of diffuse injuries from mild concussion to severe DAI represent the "continuous spectrum" to which Gennarelli refers. Recent work by Blumbergs et al. (1994 in Satz et al., 1997) supports the notion of continuity of pathophysiology from mild head injury to severe head injury using the concept of DAI. Gennarelli points out that because there is no diagnostic procedure that is capable of determining the amount of axonal damage present, the severity of DAI is determined clinically and this would appear to be the case for concussion as well.

2.1.5. Synthesis and Perspectives

With respect to the criteria for mild head injury considered in the definition discussed above (section 2.1.3), it is clear that the definitions of concussion provided by Gennarelli (1987), fulfil the criteria for mild head injury. It can thus be concluded that mild concussion and classical cerebral concussion are classified as mild closed head injuries. However, it is extremely important to bear in mind that what we are referring to are different points (albeit arbitrary) on a continuum of diffuse injury and it is therefore possible that a more severe injury (resulting in a greater degree of axonal damage) would

not be classified as mild, but rather as mild to moderate DAI or even as a Grade 5 concussion (Torg, 1982), depending on which classification system one wishes to use (taking into consideration that mild DAI or a Grade 5 concussion would not constitute a mild head injury, but rather a moderate to severe head injury).

For the purposes of the present research, Gennarelli's definitions of concussion will serve as guidelines and the terms "mild concussion", "cerebral concussion", "concussive head injury" or "mild head injury" will be used interchangeably. There will be instances when the present author is reviewing another researcher's work wherein the terminology will be slightly different. However, in most cases, it is understood that by and large, we are referring to mild head injury as synonymous with concussion, using the following parameters:

(a) any period of loss of consciousness for less than 30 min, with GCS of 13 to 15 following the loss of consciousness;

(b) any loss of memory for events immediately before or after the accident with post-traumatic amnesia for less than 24 hours;

(c) any alteration in the mental state at the time of the accident (e.g. dazed, disorientated, or confused); and

(d) focal neurological deficit(s) (e.g. double vision, loss of balance, taste or smell) that may or may not be transient.

2.2. Epidemiology and Demographic Characteristics of Mild Head Injury

The incidence of impairments resulting from head trauma has primary importance for those concerned with the planning of emergency medical care systems, acute care programs and inpatient rehabilitation programs, as well as for evaluation of the effects of prevention and early intervention initiatives (Willer, Abosch and Dahmer, 1990). According to Willer et al. (1990), the literature on incidence falls into three broad categories: those which report the incidence of head injury; those which report the incidence of head injury leading to hospitalisation; and finally, those which report the incidence of head injury leading to brain injury and disability. Willer et al. (1990) point out that there is a need for greater information at the third level in order to plan effectively for rehabilitation services.

However, the exact incidence of minor head injury is difficult to determine for a number of reasons (Dacey and Dikmen, 1987, p. 126). First, most health surveys concentrate on hospitalisations, and since most mild head injuries (due to their apparent triviality) either go unreported or are treated in a doctor's and/or emergency rooms without hospitalisation, these health surveys are inaccurate. Second, descriptive tools such as the International Classification of Diseases (ICD) include related injuries such as face lacerations in the category for head injuries thus skewing the statistic. In this regard, it is evident from epidemiological studies that many of these reports differ in their definitions of head injury and brain injury. Because there is lack of uniformity of definition, there is also

disparity in the ICD codes used to identify extant cases of head injury or brain injury. Third, patients who have sustained multiple injuries that include a minor head injury may be classified according to their most severe or complex injury, thus ignoring the head injury. In the South African context, there are no comprehensive epidemiological studies on mild head injury. This means that one is left with the option of inference, i.e. one is to infer from epidemiological studies in, for example, the USA and UK. This is highly problematic in that the population demographics are different in each country. It is nevertheless of interest to present a brief review of a number of significant epidemiological studies.

Richardson (1990) reported that "the number of individuals who receive a head injury each year that is sufficiently serious to lead them to seek treatment at a hospital is roughly 600 000 in England, 84 000 in Scotland and 2 200 000 in the USA" (p. 38). In an estimate of US brain injury disability using data from 7 incidence studies (Annegers et al., 1980; Cooper, Tabbador and Houser, 1981; Jagger et al., 1984; Kalsbeck et al., 1980; Klauber, Marshall and Barrett-Connor, 1981; Kraus et al., 1984; and Whitman, Coonley-Hoganson and Desai, 1984 in Kraus, 1990, p. 17) reported the following estimates:

1. Brain injury incidence = 200/100 000
2. US population size, 1984 = 235 million
3. Total new cases in 1984 = 470 000
4. Prehospital brain injury deaths = 70 000
5. Total cases admitted to hospital alive = 400 000
6. US hospital admission by severity:
   mild = 320 000 (80%)
   moderate = 40 000 (10%)
   severe = 40 000 (10%)
7. Discharge rate (alive):
   mild = 100% discharged alive
   moderate = 93% discharged alive
   severe = 42 % discharged alive

What is noteworthy about these estimates is that mild head injuries far outweigh both moderate and severe injuries. Kurtze (1982 in Levin et al., 1987) reported that closed head injury is the most common cause of neurological disability in the USA. These reports make a study of mild head injury a crucial and necessary endeavour. In addition, since there is such a high incidence of mild head injury, the implication is that there is a significant proportion of 'survivor' head injured patients with disability. Kraus (1987) estimates approximately 32 000 cases of disability per year in the mild head injury category, hence the importance of Willer et al.'s (1990) argument for more studies on disability so that rehabilitation services can be improved.

Studies have shown that head injury is higher in males than in females, in young adults than in children or the elderly (Annegers et al., 1980; Cooper et al., 1983; Jennett et al., 1977; Kalsbeek et al., 1980; Kraus et al., 1984; Marshall et al., 1983; Rowbotham, Maciver, Dickson and Bousfield, 1954; Steadman and Graham, 1970, in Richardson, 1990). The incidence of head injury is also higher among the working classes (Kraus, 1978; Whitman et al., 1984, in Richardson, 1990) and it is higher following alcohol consumption (Heilman et al., 1971; Jennet et al., 1977 in Richardson, 1990).

Field (1976, in Richardson, 1990) concluded that road traffic accidents were the major cause of injuries amongst adults, that falls were the major cause of injuries amongst children, and that domestic accidents were the major cause of injuries amongst the elderly. Cases of assault[8] (Richardson, 1990) account for up to 20% of adult injuries, and Kerr et al. (1971 in Richardson, 1990) found that industrial accidents reflected 14% of admissions following head injury.

2.3. The Sequelae of Mild Closed Head Injury

2.3.1. Pathophysiology and Neuropathological Sequelae

As recently as 1993, Dacey et al. wrote that "despite the frequent occurrence of minor head injury, its pathology and pathophysiology are very poorly understood" (p.162). However, as Lezak (1995) argues, an understanding of the underlying neuropathology of a particular neurological disorder is vital in any neuropsychological study. While it is beyond the scope of this review to present a comprehensive dissertation on the so-called "geography" (Lezak, 1995) of the brain, it is important for the purposes of this research, to focus on the current understandings of the neuropathology of closed head injury. It is interesting to note that the present-day descriptions of closed head injury and it's pathophysiology represent a fairly 'modern' evolution of theory and practice which has it's origins in research spanning approximately 50 to 60 years.

By and large, contemporary perspectives on head injury and its pathophysiology include and expand on the research preceding current understandings. In broad terms, the contemporary position is that a closed head injury, which is sufficient to cause even a brief disturbance of consciousness, may produce detectable structural damage, and in some cases this may occur without subjective complaints by the patient. For example, Sekino et al. (1981, in Binder, 1986) reported that routine CT scans conducted within 2 weeks of injury were abnormal in 36 out of 500 cases, yet 13 of the 36 with CT abnormalities had no subjective complaints.

It is generally accepted that in closed head injury, damage typically occurs in two stages (a detailed account of these mechanisms can be found in Lezak, 1995): a) the *primary injury* which occurs at the

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[8] Richardson lists a variety of sources of assault (1990, p. 36).

time of impact and b) the *secondary* injury which constitutes the physiological effects ensuing from the primary injury. Secondary injuries will not be discussed here but can be resourced in various places, most notably Lezak (1995) and Walsh, (1991). In brief, secondary lesions include "ischaemia, anoxia, oedema and brain distortion due to intracranial bleeding" (Walsh, 1991, p. 168). The primary injury will be discussed in some detail. It will become clear that these mechanisms are worthy of attention in a study on mild closed head injury in contact sport.

When a relatively still victim receives a blow to the head, this kind of static injury results in a rapid sequence of events, "beginning with the inward moulding of the skull at point of impact [*coup lesion*] and a compensatory adjacent outbending" (Lezak, 1995, p. 177). *Contrecoup lesions*, or brain contusions (bruises), often occur in the area opposite the blow. Lezak (1995) points out that coup and contrecoup lesions account for the specific and localizable behavioural changes that accompany closed head injuries.

Unlike the static injury, where damage can be relatively focal, when there is a great deal of momentum on impact as in the case of motor vehicle accidents and whiplash injuries, "clearly distinguishable focal deficits are much less likely to be seen. In such cases damage tends to be widespread with patterns of multifocal or bilateral damage and no clearcut evidence of lateralisation, regardless of the site of impact (Levin, et al., 1982).

This form of generalised or *diffuse* injury is closely associated with the mechanism in which there occurs a translatory force and rotational acceleration of the brain within the bony protuberances of the skull, resulting in frontal and temporal lobe lesions. The movement of the brain within the skull places strains on the delicate nerve fibres and blood vessels and stretches them to the point of shearing (Strich, 1961) - generally referred to as the "shear-strain" model of brain injury. These shearing effects result in microscopic lesions (Oppenheimer, 1968) in the brain which tend to be concentrated in the frontal and temporal lobes as well as in the "interfaces between grey and white matter around the basal ganglia, periventricular zones. corpus callosum, and brainstem fibre tracts" (Mendelow and Teasdale, 1984; Pang, 1989 in Lezak, 1995, p. 177).

At the neuronal level, this rapid acceleration/deceleration results in damage to axons in cerebral and brain stem white matter, and in serious injuries, in the cerebellum too. This kind of axonal damage is called *diffuse axonal injury* (DAI), a term used to describe prolonged traumatic coma that is not due to mass lesions or ischaemic insults. The mechanism of rotational acceleration is more likely to cause concussion and diffuse axonal injury and is less likely to cause focal injury. The centripetal forces of rotational injuries do not damage the mesencephalic centres of consciousness as much as the temporal and limbic areas, a finding that has been related to the clinical observation of memory loss and confusion without loss of consciousness (Ommaya and Gennarelli, 1974).

In data obtained using animal models, Povlishock and Coburn (1989) demonstrated that "focal axonal damage is a consistent feature of minor to moderate traumatic brain injury and thus would suggest that a comparable axonal response is occurring in head-injured humans (p.47)[9]. Thus what Strich (1961) in her necropsy study of 75 patients with "apparently uncomplicated head injuries" (p.443) earlier defined as "diffuse severe degeneration of white matter" (p. 448), is now widely recognised as diffuse axonal injury[10]. Gennarelli (1987) describes the pathophysiology of DAI thus:

> Depending on the severity of injury, axonal damage may occur in isolation or in conjunction with actual tissue tears. The latter appear as small areas where the brain is sufficiently damaged so that both axons and small blood vessels are torn; these then appear as small haemorrhages, usually within the central area of the brain. These haemorrhagic tissue tears are the only macroscopic manifestation of DAI. An especially frequent combination of macroscopic tissue tears occurs in the corpus callosum and the dorsolateral quadrant of the rostral brainstem at the superior cerebellar peduncle. (p. 115-116).

According to Lezak (1995), besides the scattered tiny (*petechial*) haemorrhages seen with diffuse axonal damage which occur mostly in frontal and temporal lobe white matter, larger blood vessels may be torn on impact. In closed head injuries, haemorrhages cause sub- or epidural hematomas (swellings filled with blood) within the skull. "Closed head injuries with haemorrhages tend to be more serious than when the damage is due to DAI alone" (Lezak, 1995, p. 179).

As was noted in section 2.1. (above) Gennarelli (1987) draws a distinction between *concussion* and *diffuse axonal injury*, placing concussion at the less severe end of the spectrum of diffuse brain injuries. He distinguishes between two forms of concussion (see Table 2-3, p. 9, for the description of Ommaya and Gennarelli's (1974) hypothesis for the syndromes of cerebral concussion). In the case of *mild cerebral concussion*, Gennarelli states that although consciousness is preserved, it is clear that some degree of cerebral dysfunction has occurred. The fact that memory mechanisms seem to be the most sensitive to trauma, suggests that the cerebral hemispheres rather than the brainstem are the recipient of mild injury forces:

> The degree of cerebral cortical dysfunction, however is not sufficient to disconnect the influence of the cerebral hemispheres from the brainstem-activating system, and therefore consciousness is preserved. No other cortical functions except memory seem at jeopardy, and the only residual deficits that patients with mild concussion syndromes have is brief retrograde or post-traumatic amnesia. (Gennarelli, 1987, p. 112).

In the case of *classical cerebral concussion*, Gennarelli warns that it is important for the clinician to distinguish the symptomology resulting from concussion from that arising as a result of focal injury.

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[9] These authors have elsewhere made the point that animal models can replicate many of the features of human head injury (1989, p. 38).

[10] Graham, Adams and Gennarelli (1987, p. 74) list a variety of synonyms which have been used by various researchers for this form of neuropathology, namely: "shearing injury", "diffuse damage of immediate impact type", diffuse white matter shearing injury", "inner cerebral trauma" and the more recent and commonly used term, "diffuse axonal injury".

According to Gennarelli (1987) the mechanisms that underlie classical cerebral concussion are but an extension of those of the mild concussion syndromes:

> ...not only have the mechanical stresses and strains on the brain caused dysfunction of those cortical functions involving memory, but they have in this instance caused sufficient physiological disturbance to temporarily cause diffuse cerebral hemispheric disconnection from the brainstem reticular activating system. Because this dysfunction is physiological and not structural, when the electro-chemical milieu of the brain returns to normal, the usual interaction between the cerebral hemispheres and brainstem is re-established and consciousness returns. (p. 113).

The neuropathology involved in the spectrum of concussive syndromes (see Table 2-3, p. 9) as proposed by these authors is described as follows: cortical-subcortical disconnection (C.S.D) for grades I and II; C.S.D + diencephalic disconnection (C.S.D.D) for grades II and III; and C.S.D.D + mesencephalic disconnection (C.S.D.M.D) for grades IV to VI (Gennarelli and Ommaya, 1974, p. 633-654).

Mild head injuries such as cerebral concussion have been classically viewed as essentially reversible syndromes without detectable pathology (Denny-Brown et al., in Ommaya and Gennarelli, 1974, p. 633). The most common definition of cerebral concussion suggested that it was a *transient* loss of neurological function without macroscopic or microscopic abnormalities. Studies using magnetic resonance imaging (MRI) have shown that lesions to the grey and white matter of the brain itself may well resolve within three months following a minor closed head injury; any residual lesions are likely to be extraparenchymal abnormalities, such as chronic subdural haematomas (Levin et al., 1987). Furthermore, it is still recognised that permanent damage is not an inevitable consequence of a single concussive blow and that "a single uncomplicated minor head injury produces no permanent disabling neurobehavioural impairment in the great majority of patients who are free of a pre-existing neuropsychiatric disorder and substance abuse" (Levin et al., 1987, p. 234).

However, research is accumulating wherein findings suggest that minor and moderate head injuries are associated with structural change in various foci throughout the brain (Povlishock and Coburn, 1989). In a seminal study on microscopic lesions in the brain following head injury, Oppenheimer (1968) stated that

> the point to be stressed in regard to these cases of 'concussion' is that permanent damage, in the form of microscopic destructive foci, can be inflicted on the brain by what are regarded as trivial head injuries. If such injuries are repeated (as they may be, for instance in boxing), one would anticipate that a *progressive, cumulative loss of tissue, and of nervous function* [italics added], would occur. (p. 306).

The "progressive, *cumulative* loss of tissue and nervous function" as pointed out by Oppenheimer (1968) was supported by the findings of Gronwall and Wrightson (1974) on the cumulative effects of concussion, who stated that

> "[i]f, as seems likely from pathological studies, the mechanism of concussion of all grades of severity includes neuronal damage, the most probable explanation of the

> cumulation of the effects of concussion is that each event destroys neurons, diminishing the reserve available and making the loss evident under the stress of further injury (p. 997).

Gennarelli (1987) reported that although the great majority of patients with classical cerebral concussion have no sequelae other than amnesia for the events of impact, some patients may have more long-lasting, although subtle, neurological deficiencies. He concludes that "further investigation of these sequelae must be done" (1987, p. 113). Such further investigation would seem warranted in an area where there appears to be a fine line between the force necessary to cause a transient functional impairment and that sufficient to produce irreversible structural change (Richardson, 1990).

There has thus been an evolution in the concept of cerebral concussion in that researchers (e.g., Binder; 1986; Hooper, 1969; Levin et al., 1987; Rimel, Giordani, Barth, Boll and Jane, 1981; Symonds, 1962; Walker, 1973 in Ommaya and Gennarelli, 1974) began to suggest that concussion should not be confined to cases in which there is immediate loss of consciousness with rapid and complete recovery,

> "...but should include the many cases in which the initial symptoms are the same but with subsequent long continued disturbances in consciousness, often followed by residual symptoms - concussion in the above sense depends on diffuse injury to nerve cells and fibres sustained at the moment of the accident, the effects of which may or may not be reversible" (Ommaya and Gennarelli, 1974, p. 633).

Jennet and Teasdale (1981) drew the conclusion that "even a brief concussion usually entails some structural damage to the brain" and that "the damage done by and the symptoms subsequently suffered after mild head injuries are frequently underestimated" (in McKinlay, Brooks and Bond, 1983, p. 1084). A similar assertion was made by Rimel et al.: "...our observations tend to support Sir Charles Symonds' statement that 'It is questionable whether the effects of concussion, however light, are ever completely reversible' " (1981, p. 227). Richardson (1990) supports this position when he stated that:

> In considering neuropathological evidence obtained from patients who have sustained severe or even fatal injuries, therefore, it should be born in mind that nowadays the difference between these patients and those who have sustained relatively mild or minor head injuries is conceived of as a quantitative rather than a qualitative one (p. 40).

2.3.2. Neuropsychological Sequelae and the Postconcussion Syndrome (PCS)

A number of patients with minor head injuries complain of symptoms for weeks, months or years after the injury (Rutherford, 1977). These symptoms - which can include both *objectively measured* deficits as well as *subjective* complaints - constitute a constellation of symptoms of what is now commonly know as the Postconcussive Syndrome (PCS). Earlier research (Gronwall and Wrightson, 1974) indicated that:

> If we exclude the patients whose symptoms are due to readily identifiable conditions such as subdural haematoma, post-traumatic hydrocephalus, dizziness, and vertigo due to skeletal or peripheral-nerve involvement, we are left with a group whose complaints are remarkably uniform. They cannot concentrate, their memory is poor, they tire easily and they are irritable. Attempts to work bring on headache. These will be recognised as the symptoms of the postconcussion syndrome (p. 607).

The research into PCS suggests that cognitive deficits in attention and concentration, memory and information processing (established via formal testing), and related subjectively reported symptomology such as irritability, fatigue, headache, difficulty concentrating, memory problems and depression and/or anxiety, often co-exist. A review of this literature is imperative for the present study where there is the stated dual focus on both objectively measured and self-report postconcussive symptomology.

While the present review of studies on the post-concussive sequelae of mild head injury is not exhaustive, a number of studies which are of particular relevance to the present research will be highlighted. Where appropriate, the studies are presented individually so that the idiosyncrasies of each (particularly with respect to neuropsychological batteries, functions measured and outcome) could be highlighted.

2.3.2.1. *PCS: Objective Measures*

Gronwall and Wrightson (1974) made use of the paced auditory serial addition test (PASAT) - a measure for the rate of information processing - to assess patients with PTA < 24 hours. The authors found that concussed patients can process a *limited* number of items as swiftly as normal controls. "However, as the number of items increases, at a critical point the performance of the concussed patient falls off, and diverges further from that of controls as more items are added" (Gronwall and Wrightson, 1974, p. 608). In other words, the finding here suggests that higher levels of information processing, particularly sequential processing were affected 1 month post-injury. Gronwall and Wrightson (1974) were the first researchers to suggest that a reduction in the rate of information processing is an important factor in the genesis of the postconcussion syndrome.

This aspect of information processing deficits appears elsewhere in the literature (e.g. Leininger, Gramling, Farrell, Kreutzer, and Peck, 1990; Levin et al., 1987; Rimel et al., 1981). Information processing capacity can be described as "the number of operations the brain can carry out at the same time" (Gronwall, 1989, p. 154). In a useful discussion on information processing, Gronwall (1989) argues that after mild head injury, patients have difficulties in all areas that require them to analyse more items of information than they can handle simultaneously: They present as *slow* - it takes longer to process the 'bits' of information; they present as *distractible* - irrelevant stimuli are monitored at the same time as attending to relevant stimuli; they present as *forgetful* - while

concentrating on point A, the processing space for simultaneously concentrating on point B is lost; they present as *inattentive* - 'too much' information cannot be taken in and processed. As Gronwall points out, these are all aspects of attention and the relation between attention deficits and impaired information processing skills following mild head injury have been documented (Gronwall and Wrightson, 1974; Levin et al., 1987).

One aspect of attention which has not been researched as widely, is vigilance and reaction time (Brouwer and Wolffelar, 1985; and McCarthy, 1977, in Gronwall, 1989). Vigilance demands the ability to attend to uninteresting stimuli for an extended period of time, i.e. sustained attention. One month post-injury, vigilance performance was found to be unimpaired under normal task conditions, but fell short under task conditions requiring sustained effortful processing (Parasuraman, Mutter and Molloy, 1991). In a separate study, reaction time was slowed immediately after and 6 weeks after trauma, but improved between 6 weeks and 6 months after mild head injury (MacFlynn, Montgomery, Fenton and Rutherford, 1984). Selective attention and reaction time difficulties have been reported by Gentillini, Nichelli and Schoenburger (1987). Visual information processing deficits have been observed on paradigms demanding attention and concentration (Chitra-Mariadas, Rao, Gangadhar and Hedge, 1989).

Rimel et al. (1981) conducted a prospective study at the Virginia Medical Centre to obtain the profile of a large group of patients with minor head injury in terms of neurological status on admission and premorbid factors that might contribute to outcome, and then to assess the overall status of the patients three months later. The research incorporated both psychosocial and neuropsychological assessment. The relatively extensive neuropsychological assessment battery included the Halstead Neuropsychology Battery, Wechsler Scales of Intelligence, Memory tests and the Wide Range Achievement Test. The subject sample was controlled for age (no subject was older than 55) and education (> 6 years). Of the sixty-nine patients who underwent neuropsychological assessment, there was evidence of mild neuropsychological impairment in the vast majority of the Halstead-Reitan Neuropsychological Procedures, including tests of higher level cognitive functioning, new problem-solving skills, attention, concentration and judgement. The most important finding of this and other similar studies (Rutherford, 1977) was the large number of patients with minor head injury who were experiencing difficulties 3 months post-injury.

In a controlled study of mild head injury due to MVA, McLean, Temkin, Dikmen and Wyler (1983) administered a brief battery of neuropsychological tests at 3 days and 1 month post-injury. Four measures were utilised, namely (a) Selective Reminding Test, (b) Galveston Orientation and Amnesia Test (GOAT), (c) Stroop Colour Test and (d) three measures of self-perception - self-perception of overall functioning, discomfort scales and head injury symptom checklist. The results indicated that patients with PTA > 24 hours differed significantly from both the PTA $\leq$ 24 hours and control groups in areas of memory (storage and retrieval), orientation, speed and distractibility 3 days post-injury. By 1 month post-injury, however, the study yielded non-significant results which, according to these

authors, pointed to a "substantial recovery" (p. 372). This finding is discrepant with that of Gronwall and Wrightson, (1974, 1981) and Rimel et al. (1981) who demonstrated significant problems at 1 and 3 months post-injury respectively. As possible explanations for these differences, McLean et al. (1983) cite the following: a) inappropriate use of controls in previous studies; b) failure to screen for pre-existing conditions in prior research, thus confusing the effects of the injury with pre-injury factors; c) possible practice effects in their own study; and d) differences in the neuropsychological measures across different studies.

In an attempt to document the relationship between cognitive, emotional and behavioural sequelae associated with minor head trauma, Barth et al. (1983) evaluated 71 patients (using the same criteria for mild head injury as used by Rimel et al., 1981). The extensive assessment battery included the Wechsler Adult Intelligence Scale (WAIS), Halstead-Reitan neuropsychological Test Battery, Wechsler Memory Scale (WMS) and Minnesota Multiphasic Personality Inventory, and was administered three months post-injury. The results suggested impairment in memory and visuospatial deficits. Cognitive deficits did not seem related to the duration of loss of consciousness (Leininger et al. 1990), post-traumatic amnesia, and the presence of sensory and motor deficits.

In a three-centre study (Levin et al., 1987), *subacute* disturbances in attention, memory (Ruff et al., 1987), and information processing were found during the first few days to one week following minor head trauma and the authors concluded that

> "although the follow-up data indicate that by 1 to 3 months most patients sustaining a minor head injury exhibit cognitive recovery to within the range of matched control subjects, a residue of isolated neurobehavioural defects may occasionally persist for a longer duration" (Levin et al., p. 240).

An interesting and significant study was conducted by Bohnen, Jolles and Twijnstra (1992) who argued that most previous studies have compared head-injured patients with a control group who had not suffered a concussion. Instead, these authors aimed to test the hypothesis that patients with postconcussive symptoms 6 months after mild head injury have cognitive deficits as compared with matched, symptom-free mild head injury patients and healthy control subjects. The psychometric battery comprised of (a) computerised version of the Auditory Verbal Learning Test; (b) the Stroop Colour Word Interference Test; and (c) a computerised divided attention task. The results indicated that the subgroup of mild head injury patients who report postconcussion symptoms at 6 months following an uncomplicated mild head injury may demonstrate deficits on tests of attention and information processing. In contrast, patients who had recovered from an uncomplicated mild head injury did not differ in cognitive functioning from healthy control subjects. The authors concluded that "the less adequate neuropsychological functioning of symptomatic patients warrants further investigation" (p. 694).

The significance of the Bohnen et al. (1992) study (particularly for the present research) is that it highlighted the importance of a focus on the relationship between cognitive deficits and behavioural sequelae. In a commentary article referring to the Bohnen et al. (1992) research, Lehman states that the study is "a welcome addition to the emerging body of evidence which suggests that such 'minor' head injuries may have long-term sequelae" (p. 696).

In contrast to the above studies where there is evidence of adverse outcomes, here have been reports of neuropsychological studies in which there were null outcomes (e.g. Bawden et al, 1985; Levin et al., 1982, 1994). For example, in a controlled study, Gentillini et al. (1985) (again using the same criteria for inclusion as Rimel et al., 1981), administered a neuropsychological battery consisting of six tests tapping attention, memory and intelligence: (1) selective attention test, (2) Digits forward test, (3) Word recognition test, (4) Buschke's test, (5) Working memory test, and (6) Raven test. The researchers concluded that "in spite of a general trend toward lower performances, head injured patients did not have significant impairment when the two experimental groups were compared with the appropriate statistical methods" (p. 139) and that therefore, "it can be reasonably concluded that if there is structural damage after mild head injury, it generally recovers from the neuropsychological standpoint within one month after the trauma" (p. 139).

2.3.2.2. *PCS: Subjective Measures*

In addition to the research purely on objectively measured neuropsychological deficits, there is a growing body of research on the nature and impact of neurobehavioural symptomology which are subjectively reported by patients in the days and weeks following trauma. Rutherford (1977) reported symptoms present 6 weeks after accidental mild head injury, including headache (24.8%), anxiety (19.3%), insomnia (15.2%), dizziness (14.5%), irritability (9.0%), fatigue (9.0%), loss of concentration (8.3%) and loss of memory (8.3%). Rimel et al. (1981) found that out of 424 patients 3 months post-injury, the most frequent subjective complaint was headache (78%) and memory difficulties (59%) and only one sixth of the subject sample were complaint free. Although the most frequently reported symptoms in Levin et al.'s (1987) study were headache (71%), fatigability (55.5%), and dizziness (50.3%), factor analysis of all symptoms revealed a cognitive-depressive factor which included complaints of depression, impaired recent and remote memory, poor concentration and impaired thinking. This constellation of symptoms has been confirmed in similar studies (Dikmen, Temkin and Armsden, 1989). McLean et al. (1983) found symptoms such as headaches, fatigue, dizziness, blurred vision and memory difficulties at 3 days post-injury and at 1 month, fatigue, blurred vision and memory difficulties persisted. Barth et al. (1983) reported mild dysphoria and 'general psychological discomfort' with an accompanying decrease in adaptive functioning following mild head injury. Lidvall et al. (1974, in Szymanski and Linn, 1992), found that by three months post-injury, anxiety and headache were the most prominent symptoms. In a one year follow-up study, headache was found to be the most commonly reported postconcussive symptom (Alves, Macciocchi and Barth, 1993). Several studies have reported that persisting post-concussive

symptoms have adversely affected the patients' return to gainful employment (Barth et al., 1983; Dikmen et al., 1989; Gronwall and Wrightson, 1975; McLean et al., 1983; Rimel et al., 1981).

Rutherford (1989) distinguishes between what may be considered the common early and late symptoms of concussion (see Table 2-6, below). The *early* symptoms are what the patient complains of immediately after regaining full consciousness, and are typical complaints on the following morning. The *late* symptoms are those that are reported at clinical visits a few weeks later.

Table 2-6. Early and Late Concussive Symptoms (Rutherford, 1989)

Early Symptoms	Late Symptoms
Headache	Headache
Dizziness	Dizziness
Vomiting	Irritability
Nausea	Anxiety
Drowsiness	Depression
Blurred Vision	Poor memory
	Poor concentration
	Insomnia
	Fatigue
	Poor hearing
	Poor Vision

The aetiology of persisting symptoms post-injury have been the subject of some debate. Rutherford (1977) outlines various research endeavours which have differed in their opinions regarding the organic versus functional aetiology of these symptoms: the author cites Miller (1961) who believed that the PCS occurred in cases of trivial rather than severe head injury and was related to questions of compensation; Taylor (1967), who argued that the condition may be partly organic; and Lidvall, Linderoth and Norlin (1974) who concluded that the condition is neurotic. Alternatively, Gronwall and Wrightson (1974) maintain that the condition starts as organic damage to the brain causing poor intellectual function, and that the loss of self-confidence arising from this leads to a neurosis. Barth et al. (1983) argue that although the argument for psychogenic determinants of the postconcussive syndrome certainly has merit, "recent histological, neurophysiological and neuropsychological data point to the possibility of a specific neuropathological contribution [shear-strain model] to postconcussive symptoms in cases of minor head trauma" (p. 531). It would seem that the most comprehensive conclusion that can be drawn with respect to aetiology of the PCS is that there is an interplay of both organic and functional elements (Lishman, 1988; Rutherford, 1977, p. 4). Lishman (1988) concluded that from the studies to date, it appears that both pyhsiogenic and psychogenic influences are important. However, Lishman maintains that where mild to moderate injuries are concerned, organic factors are chiefly relevant in the earlier stages, whereas persisting symptoms are perpetuated by secondary neurotic developments, often of a complex nature. Gualtierri (1995) proposed that where the persisting deficits are more problematic, this may be due to the following situations: (a) what appears to be a mild head injury is really a severe injury; (b) PCS has evolved into a post-traumatic depression which goes unrecognised and untreated; (c) a premorbid psychiatric

condition is mobilised around the mild head injury as a focal (but not a causative) event; and (d) the patient shows signs of a "functional" condition, such as conversion disorder, somatoform pain disorder, compensation neurosis, or malingering.

2.3.3. Synthesis and Perspectives

Research on the neuropsychological sequelae of mild head injury established via formal testing, appears to suggest that slowed information processing is the most commonly found cognitive deficit. Impairment in attention and concentration, particularly sustained and/or divided attention, vigilance, reaction time, and memory are also frequently reported. These deficits may or may not co-exist with the subjectively reported sequelae such as irritability, memory problems, difficulty concentrating, depression and anxiety.

Taking into account the findings of various research studies, a useful presentation of the range of possible symptoms associated with the PCS is provided by Anderson (1995) (see Table 2-7, below).

Table 2-7. Range of Symptoms Associated with PCS

Somatic:	headache, dizziness, vertigo, insomnia, vomiting, fatigue and weakness, loss of appetite, drowsiness, blurred vision, strabismus, menstrual irregularities, decreased noise tolerance, sensitivity to medications and alcohol, restlessness, clumsiness, 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, somatisation and hypochondriasis, denial of symptoms, apathy or lack of spontaneity, personality change.

(Anderson, 1996, p. 24)

The Bohnen et al. (1992) study highlighted the issue that studies of patients with mild head injury cover a heterogeneous population, as there are subgroups of patients who recover quickly, within days, whereas others have persistent postconcussive symptoms extending weeks or months post-injury. This point regarding the variability in outcome is addressed in several reviews on the sequelae of mild head injury (e.g. Binder, 1986; Boll, 1983; Evans, 1992; Gualtierri, 1995; Satz, 1997; and Szymanski and Linn, 1992) and is evident in the studies reviewed in the above discussion. It is important to note that there is clear evidence of cognitive deficits in the first few days to one week following a mild head injury (Barth et al., 1983; Dikmen et al., 1989; Fisher, 1982 in Binder, 1986; Gronwall and Wrightson, 1974; MacFlynn et al., 1984; McLean et al., 1983; Yarnell and Lynch, 1973), but as Binder (1986) points out, "there is inconsistent evidence of prolonged cognitive impairment as a sequelae of mild head injuries" (p. 326) and follow-up studies are sparse (Barth et al., 1983; Ruff et al., 1989). In the review by Satz et al. (1997), the authors maintain that as injury

severity increases, more variability in the findings are reported. It would seem that these conflicting results may be due to aspects such as (a) the heterogeneity of the mild head injury population samples; (b) the different time intervals after injury at which assessment takes place; (c) the sensitivity of the selected cognitive tests in detecting post-traumatic brain dysfunction, (d) various methodologies employed; and (e) the appropriateness of control groups.

Despite the variability and apparent inconsistency of findings in the literature, there is no doubt that the PCS is common, and although these symptoms decline with time, the literature indicates that, in a number of cases, these symptoms appear to persist beyond the 1 month post-injury time-frame and go on to present as *chronic* effects of mild head injury. It would seem that the critical nature of such persisting symptomolgy as revealed in various studies, has fuelled the DSM-IV proposal for a Postconcussional Disorder (c.f. section 2.5).

Conflicting data are reported as to what factors predict the persistence of PCS after mild head injury. Age, sex and duration of PTA may of relevance (Rutherford et al., 1977) but other studies report a lack of correlation between these prognostic factors and persistence of PCS (Dikmen et al., 1989; Jakobsen et al., 1997 in Bohnen and Jolles, 1992). It has been shown that multiple concussions may have a cumulative effect in that they delay the cognitive and subjective recovery from a new head injury (Gronwall and Wrightson, 1975). This latter finding has received very little follow-up in the literature mainly, it would seem, due to the fact that there are few people in the general population who are sustaining more than 2 or three mild head injuries. This implies that the present study with its focus on a contact-sport population may be a viable and necessary option if questions regarding the *possible chronic sequelae and cumulative effects of mild head injury* are to be adequately addressed.

In addition, it has become increasingly clear that both a purely psychogenic and a purely organic viewpoint have serious shortcomings in explaining the persistence of postconcussive symptoms. Many years ago, Symonds (1962; 1942, in Bohnen and Jolles, 1992) suggested that the aetiology of PCS was cerebral damage, but that a symptomatic expression of PCS depended on a psychological interaction with the neurological damage. He argued that it was unnatural and impossible to separate the physiogenic and the psychogenic. Thus, what Rutherford (1977) saw as the interplay of organic and functional pathology, is now widely supported in the literature and commonly referred to as the "interaction concept" (Bohnen and Jolles, 1992; Lishman, 1988).

2.4. The Diagnostic and Statistical Manual (DSM)-IV Proposal of a Postconcussional Disorder

The growing body of research on PCS (as reviewed above) has alerted the attention of the Diagnostic and Statistical Manual of Mental Disorders (DSM) in that a proposed "Postconcussional Disorder" has been included in the most recent DSM-IV (1994) (see Table 2-8, below). It appears that the proposal of such a disorder is an attempt to (a) establish a common language for researchers and clinicians who are interested in studying the cognitive and behavioural sequelae of concussive head

injury; and (b) address definition and classification inconsistencies. According the DSM-IV, the essential feature of the proposed Postconcussional Disorder is an acquired impairment in cognitive functioning, accompanied by specific neurobehavioural symptoms, that occurs as a consequence of a closed head injury of sufficient severity to produce a significant cerebral concussion.

Table 2-8. DSM-IV Research Criteria for Postconcussional Disorder

A.	A history of head trauma that has caused significant cerebral concussion. **Note:** The manifestations of concussion include loss of consciousness, post-traumatic amnesia, and less commonly, posttraumatic onset of seizures. The specific method of defining this criterion needs to be established by further research.
B.	Evidence from neuropsychological testing or quantified cognitive assessment of difficulty in attention (concentrating, shifting focus of attention, performing simultaneous cognitive tasks) or memory (learning or recalling information).
C.	Three (or more) of the following occur shortly after the trauma and last at least 3 months: (1) becoming easily fatigued (2) disordered sleep (3) headache (4) vertigo or dizziness (5) irritability or aggression with little or no provocation (6) anxiety, depression, or affective lability (7) changes in personality (e.g., social or sexual inappropriateness) (8) apathy or lack of spontaneity
D.	The symptoms in Criteria B and C have their onset following head trauma or else represent a substantial worsening of pre-existing symptoms.
E.	The disturbance causes sufficient impairment in social or occupational functioning and represent a significant decline from a previous level of functioning. In school-age children, the impairment may be manifested by a significant worsening in school or academic performance dating from the trauma.
F.	The symptoms do not meet the criteria for Dementia due to Head Trauma and are not better accounted for by another mental disorder (e.g., Amnestic Disorder Due to Head Trauma, Personality Change Due to Head Trauma).

(DSM-IV, 1994, p. 705-706).

Currently, individuals who fulfil the research criteria for postconcussional disorder, would be given the DSM-IV diagnosis of Cognitive Disorder Not Otherwise Specified. In terms of a differential diagnosis, if the head trauma results in dementia (e.g., memory impairment and at least one other cognitive impairment), postconcussional disorder would not be considered. Mild neurocognitive disorder (which, like postconcussional disorder, is also included as a criteria set for further study) is differentiated from postconcussional disorder by the specific pattern of cognitive, somatic and behavioural symptoms and the presence of a specific aetiology (i.e., closed head injury). Individuals with Somatisation Disorder and Undifferentiated Somatoform Disorder may manifest similar behavioural or somatic symptoms; however, these disorders do not have the specific closed head injury aetiology or measurable impairment in cognitive functioning. Postconcussional disorder is also distinguished from Factitious Disorder (the need to assume to sick role) and Malingering (in

which the desire for compensation may lead to the production or prolongation of symptoms due to closed head injury).

The criteria for the proposed postconcussional disorder including its possible subtypes, has continued to stimulate debate in the literature (Brown, Fann, and Grant, 1994; Henry, 1994; Hoffman, 1994; Tucker, 1994) and is at present an unresolved issue. Brown et al. (1994) argue that any proposed criteria for the postconcussional disorder, especially in terms of quantitating the length of PTA, will be subject to the criticism that they are arbitrary. Hoffman (1994) contends that Brown et al.'s suggestion to exclude postconcussional disorder from the DSM-IV, ignores instances in which patients suffer significant cognitive dysfunction following relatively minor injuries with no loss of consciousness. According to Hoffman, to exclude the disorder would allow no legitimate place for these patients to be diagnosed, and he proposes instead that certain subtypes of the disorder be considered. These controversies serve to confirm the lack of resolution which exists in the area of PCS, and point to the need for further research.

2.5. Mild Head Injury in Contact Sport

In most sports, there have been substantial efforts to evaluate and eradicate head trauma in the severe range. However, this has not been the case with most sports-related mild head injuries. Barth et al., (1989) maintain that athletes do not typically complain about "minor injuries" because the ramifications of admitting to an injury could include losing one's position on a team, missing an opportunity to impress coaches, national selectors and/or fans, or being seen as 'weak' and a failure. In this sense, "assessing the prevalence of sports-related concussion or mild head injury is problematic for the simple reason that most cases go unreported" (Anderson, 1995, p. 23)

The controversies surrounding the definition and classification of head injuries also exist in the context of mild head injury in sports (Anderson, 1995; Cantu, 1986; Kelly et al., 1991; Nelson, 1984; Torg et al., 1979). The most important implications of the confusion surrounding the definition and classification of mild head injury in contact sport are (a) defining and diagnosing the various severities of *concussion* on and off the field; (b) that the condition, due to its apparent triviality, can potentially be underdiagnosed and (c) *there are no universally accepted criteria for determining when an athlete may safely return to competition after having suffered a concussion* (Cantu, 1986; Kelly et al., 1991; Lindsay, McLatchie and Jennet, 1980). This is a fairly serious issue in the light of the possible cumulative effects of concussive head injuries and the obvious need to prevent long-term and irreversible damage. As will become clear in the review that follows, one of the most fundamental and serious concerns in the inquiry into mild head injury in contact sport, is the athlete who is subject to repeated minor head injury over an extended period of time.

2.5.1. Epidemiological Studies of Head Injuries in Contact Sport

A number of studies and reviews (Drew and Templer, 1992; Templer and Drew, 1992) have focused on the incidence of head injuries in various sports. In one study (Lindsay, McLatchie and Jennet, 1980) soccer- and rugby-players sustained the 3rd and 6th highest number of head injuries respectively in the series. A Cambridge study reported that rugby and soccer were the 2nd and 3rd highest contributors to head injuries. This was the case for all severity variables including length of PTA, intracranial complications, fractures of the skull or face, and permanent deficit (Gleave, 1986). In a New Zealand study, McKenna, Borman, Findlay and de Boer (1986, in Abreau, Templer, Schuyler and Hutchison, 1990), reported that soccer was the winter sport that contributed to the second most head injuries, while *rugby contributed to the most head injuries in this study.* In countries, such as the UK, USA and South Africa, where rugby and soccer are popular and widely played sports, one could assume that these statistics may be very similar.

Several studies have addressed the incidence of injury in American football. Gurdjian and Gurdjian (1978 in Richardson, 1990) pointed to subdural haematomas as an important cause of deaths in American college football, even among players wearing protective helmets. The most common type of injury is helmet-to-helmet collision in the course of tackling or blocking manoeuvres (Barth et al., 1989). The National Football Head and Neck Injury Registry documented 1 129 injuries between 1971 and 1978 that involved hospitalisation for more than 72 hours, surgical intervention, fracture-dislocation, permanent paralysis, or death (Torg et al., 1979). These authors reported an increase in the number of cervical spine injuries due to the recently outlawed tackling technique called *spearing*, which involved using the head and helmet to knock down a player. A retrospective study by Gerberich, Priest, Boen, Straub and Maxwell (1983) researched the concussion incidences and severity in 3 063 secondary school varsity (first team) football players. Postconcussive symptoms were reported by some players up to nine months postseason. The authors note that the rate of 19% of players with at least one possible concussion in one season, and the fact that probable concussions accounted for 24 % of all injuries are conservative figures.

> A finding of this many concussion episodes, together with the way they were handled, suggests problems of defining and understanding concussion and differences in diagnosis and diagnostic criteria by medical personnel or other individuals presumably trained in injury assessment Gerberich et al., 1983, p. 1373).

A prospective study of head and neck injuries in 342 football players at the University of Iowa in the USA, found an incidence of 175 injuries in 100 players during the 8 year period of the study (Albright et al., 1985). Mueller and Blythe (1987) noted that head and cervical spine fatalities accounted for 84.6% of all football fatalities from 1954 through 1984. Maroon et al. (1980, in Barth et al., 1989) reported that most severe football head injuries occur during defensive blocking or tackling manoeuvres.

In South Africa, there have been a number of research endeavours into the incidence and nature of rugby injuries. Such research has developed out of a concern about incidents of death and paralysis following *neck* and *spinal* injuries (Allie, 1991; Roux, Goedeke, Visser, van Zyl and Noakes, 1987). In two studies on the effects of *head* injuries by Nathan, Goedeke and Noakes (1983) and Roux et al. (1987), findings revealed that concussion accounts for the single most common injury in rugby and it is the players in the *top teams* that are at greatest risk of sustaining an injury. Authors in these studies report that these figures are probably underestimates as a high percentage of sub-concussive incidents go unreported.

2.6. Neuropsychological Studies of Mild Head Injury in Contact Sport

2.6.1. Boxing

The earliest account of neurological, cognitive and behavioural impairment resulting from boxing is the now famous "Punch Drunk" article (Martland, 1928). The author highlighted neurologic symptoms such as mild confusion and an unsteady gait early on, and later, speech and motor deficits as well as upper-extremity and head tremors. Martland noted that this syndrome develops into a movement disorder similar to Parkinson's Disease, usually involving unsteady gait and considerable mental decline. Diffuse cerebral atrophy often occurs and has been variously labelled as "dementia pugilistica (Lampert and Hardman, 1984) and "traumatic boxer's encephalopathy" (Mawdsley and Ferguson, 1963 in Barth et al., 1989). Roberts (1969, in Cooper, 1987) describes a syndrome progressing from dysarthria and disturbance of balance to severe ataxia, pyramidal and extrapyramidal signs, and dementia. In this study, incidence among boxers and severity of signs increased with exposure in the ring which highlighted the cumulative nature of repeated damage.

Using the PASAT scores as measures of information processing, Gronwall and Wrightson (1975) found that, after a second concussion, the rate at which subjects (drawn from a non-sports population) were able to process information, was reduced more than in controls who had been concussed once, and they took longer to recover than controls. These authors concluded that "*the effects of concussion seem to be cumulative, and this has important implications for sports where concussion injury is common* [italics added]" (p. 995). Research into moderate and severe head injury in boxing has been well documented and the cumulative effects in both pathological and neurological terms of *repeated* mild head injuries in boxers are widely recognised (Casson, Sham, Campbell, Tarlau and DiDomenico, 1982; Kaste et al., 1982; McCunney and Russo, 1984; McLatchie et al., 1987; Ryan, 1987). Kaste et al. (1982) concluded that the effects of repeated concussions are cumulative, and beyond a yet to be determined number of concussions, the neuropathology is likely to be irreversible. The neurological abnormalities found in the Kaste et al. (1982) study have been confirmed elsewhere (Jordan, 1987; McLatchie et al., 1986; Ross, Casson, Siegel and Cole, 1987; Sironi and Ravagnati, 1983). Casson et al. (1982) conducted detailed neurological examinations, EEG and CT scans on 10 professional boxers and found mild to moderate cerebral atrophy in more than half of the subject

sample. These authors suggested that the abnormalities were not due to knockouts (no boxer had been knocked out more than twice), but rather, due to *multiple subconcussive blows to the head.*

Neuropsychological studies in boxing have found mild impairment on the Trail Making Test (Kaste et al., 1982). Boxers performed significantly poorer than controls on the Inglis Word Learning Test and on the copy and immediate recall of the Rey Complex Figure (McLatchie et al., 1987). In this latter study, there were no differences between boxers and controls on the Wechlser Memory Word Learning Test, digit span, and on story recall. Using tests of verbal and visuospatial memory, attention, information processing, motor function and intellectual abilities on amateur boxers, no significant differences were detected between boxers and controls (Brooks, Kupshik, Wilson, Galbraith and Ward, 1987). The authors considered the relatively short boxing career and the superior intellectual abilities of the boxers (relative to controls) as reasons for the null outcome in their study. Cognitive functions of 23 amateur boxers were assessed immediately before and after a fight using an extensive neuropsychological battery (Heilbronner, Henry and Carson-Brewers, 1991). Impairments were noted in verbal recall (specifically retrieval deficit for newly learned and symbolic material) and incidental memory (on Digit Symbol Incidental Recall) post-fight. The lack of impairment on the WAIS Digit Symbol Substitution subtest, post-fight, was explained as a response to heightened autonomic nervous system activity not to increased central processing speed. A similar study was conducted by Butler, Forsythe, Beverly and Adams (1993) who reported no neuropsychological dysfunction due to boxing. In the present author's view, a major methodological problem in the Butler et al. (1993) study was that the control group, which comprised 31 water polo players and 47 rugby players, was highly inappropriate since there is increasing evidence that rugby players are themselves highly susceptible to cognitive deficits due to mild head injuries. Haglund and Eriksson (1993) reported differences on the Finger Tapping Test between amateur boxers, soccer players and field athletes. Ross et al. (1987) found greater impairment on memory tests than on "nonmemory" tests (incl. Trail Making Test, Digit Symbol Test, and Bender Gestalt Test). These authors concluded that the "development of abnormal neuropsychological test scores on periodic screening of boxers might be the earliest and first sign of subtle chronic brain damage.... It is hoped that such a testing scenario might prevent many of the more florid and clinically obvious cases of chronic brain injury that are often seen in boxers with long careers" (Ross et al., 1987, p. 50). In 1986, Drew, Templer, Schuyler, Newell and Cannon concluded that appreciable brain damage and associated neuropsychological deficits in today's active boxers may be the rule rather than the exception" (p. 525).

The use of head gear in boxing has been suggested by various authors (Casson et al., 1982; McLatchie et al., 1987). However, as was revealed in the discussion on the pathophysiology of mild head injury (see section 2.3.1), the research indicates that it is not primarily the *force* of the blow to the head, but the *rotation* and/or *acceleration/deceleration forces* which result in diffuse neuronal damage. A headguard may well help to prevent focal damage, but by adding weight to the head, it may even increase the degree of rotation (Timperley, 1982). A recent South African newspaper

article reported that the British government is examining proposals to ban punching to the head and face in a bid to eliminate the threat of brain damage that has "blighted" boxing in recent years. Labour MP Paul Flynn told the Sunday Telegraph that action was needed to protect boxers after a year which had seen three bouts resulting in boxers requiring neurosurgery (The Citizen, 1998).

2.6.2. Soccer

In the game of soccer, controlled head contact with the ball, or heading, is as valuable a skill as shooting on goal, but it places the player at risk of injury from the ball as well as from other players simultaneously trying to head the ball. In addition, the minor head injuries soccer players sustain in collisions are probably underdiagnosed (Dailey and Barsan, 1992). In one study, 33% of all soccer injuries were due to heading the ball (McKenna et al., 1986, in Abreau et al., 1990).

As early as 1925, Hey (in Dailey and Barsan, 1992) reported the death of a young soccer player who developed a subdural haematoma after repeatedly heading a wet (and therefore, heavier) ball. Tysvaer, Storli and Bachen (1989) conducted a neurologic and electroencephalographic (EEG) study of soccer injuries due to heading the ball. An increased incidence of EEG abnormalities was found in players compared with non-football playing, non-injured controls. The authors concluded that "the high incidence of EEG changes is probably due to the result of a *cumulative* effect of repeated head traumas" (p. 573). Spear (1995) reviewed recent research regarding the link between playing UK football (soccer) and the risk of developing dementia and examines the possible link between head injury and Alzheimer's Disease (AD). The research review suggests that football players are at much greater risk of recurrent minor head injury than the general population and that amyloid deposition, associated with severe head injury, can cause pathological changes similar to AD.

Surprisingly, neuropsychological studies on soccer players are almost non-existent. Abreau et al. (1990) compared the performance of 31 soccer players and 31 tennis players using a neuropsychological battery which included the Raven Progressive Matrices, Symbol Digit Modalities Test, Perceptual Speed Test and PASAT. There were no significant differences between the two groups on neuropsychological testing. There was however, within the soccer player group, a significant negative correlation between number of games played and performance on the PASAT. Although this research was centred more around neuropsychological testing than subjective symptoms, the latter more strongly differentiated the soccer and tennis players. Soccer players reported experiencing headaches, blurred vision, dizziness, and passing out after a game. The researchers concluded that their results provided only tentative support for detrimental effects to the brain due to soccer, and stated that they could make no comment about the permanency of such effects. In case studies of two soccer goalies reported by Dailey and Barsan (1992), one subject had persistent headache and concentration difficulties 2 days after injury. The headache and concentration problems persisted intermittently 6 weeks post-injury and postconcussive syndrome was

diagnosed. The second subject reported persisting symptoms of confusion, decreased concentration, right-side hearing loss and three months after injury, the subject was unable to return to work.

2.6.3. American Football

Empirical attention has been paid to football-related trauma, but, like other contact sports (such as boxing and soccer), there are few controlled prospective studies in the literature. As pointed out by Barth et al. (1989), epidemiological, descriptive, retrospective and case studies provide most of our understanding of football head injury. The recent surge of interest in mild head injury in the clinical population, has generated interest in high school, university and professional football playing communities in the USA, with particular focus on early identification, recovery, return to practice and athletic competition, and improvement of equipment, rules, and coaching techniques. Some writers (e.g., Wilberger, 1988) maintain that football players may be at greater risk than boxers because the incidence of minor head injury may be four to five times higher.

The research on cognitive and behavioural sequelae of mild head injury in football is sparse. Kelly et al. (1991) report the case of a high school football player who died of diffuse brain swelling after repeated concussions without loss of consciousness. Saunders and Harbaugh (1984) suggested that in the athlete who has suffered a minor head injury, CT scanning should be done before medical clearance for resumption of contact sports if any postconcussive symptoms such as headache, lightheadedness, dizziness, blurred vision, nausea, or lethargy persist. These authors also warned against the deleterious effects of "sequential minor impact injuries" or the "second impact syndrome (SIS)" which is characterised by rapid brain swelling and herniation following a second injury (Saunders and Harbaugh, 1984). Between 1980 and 1993, the National Centre for Catastrophic Sports Injury Research in Chapel Hill, North Carolina, USA, identified 35 probable cases of SIS among football players alone (Cantu, 1995). In 1973, Yarnell and Lynch identified several amnestic syndromes in mildly concussed ("dinged") football players. Marked post-traumatic short-term memory impairment occurred without any apparent alteration in consciousness while others had delayed retrograde amnesia without loss of consciousness.

Until 1989, there were no prospective neuropsychological studies in football (Barth et al., 1989). In the Barth et al. (1989) study, assessment procedures included the Trail Making Test A and B, the Symbol Digit Test and the PASAT. Psychological test scores were gathered at preseason, 24 hours, 5 days and 10 days post-injury, and at postseason. Possible practice effects were noted on the Trail Making Test. However, the authors reported deficits in the area of information processing and a positive correlation between the improvement in concussive symptoms such as headache, memory, dizziness and nausea and improvement on cognitive testing (a similar finding to Bohnen et al., 1992). Recovery following the 10-day period post-injury was also noted. According to Barth et al. (1989), "questions still remain regarding the full extent of recovery and compensation, the short- and

long-term effects of *multiple head trauma* [italics added], and factors predisposing a player to the risk of mild head injury" (1989, p.272.)

2.6.4. Rugby and Australian Rules Football

Boll (1983) suggested that because of the full contact nature of the sport, rugby players sustain several "silent" or "quiet" mild head injuries over the course of their rugby playing careers and for this reason, rugby players represent a valid target population for a preliminary investigation into the cumulative effects of repeated mild head injuries. In rugby, heavy falls are the most common cause of head injury, and tackle collisions, boots or fists make a significant contribution (Gleave, 1986).

Maddocks and Saling (1991) obtained baseline (pre-injury) measures in a sample of 130 Australian Rules Football (rugby) players using a limited neuropsychological test battery comprising the Paced Auditory Serial Addition Test (PASAT), Digit Symbol Substitution Test (DSST) and Four-Choice Reaction Time, involving measures of Decision Time (DT) and Movement Time (MT). Ten players subsequently concussed were re-tested at 5 days post-injury. Relative to the age-matched control group, analyses of covariance showed poorer performances following concussion on the DSST and DT measures. The results suggested that neuropsychological deficits are detectable in the early stages following mild concussive injury. Maddocks, Saling and Dicker (1995) examined whether the existence of a previous concussive head injury leads to poorer performance on the Digit Symbol (DS) subtest of the Wechsler Adult Intelligence Scale-Revised, 6 months after injury, in 198 Australian Rules Footballers (all players were <30 years old.) Although age and greater exposure to the game were found to increase the likelihood of concussive injury, the findings suggested that tests such as DS may only reveal effects within the first weeks after concussion (Barth et al., 1989; Maddocks and Saling 1991), and that DS performance does not differentiate concussed from non-concussed players by the sixth month following trauma.

In a study on the persistent visuospatial attention deficits following mild head injury also in Australian Rules Football players, Cremona-Meteyard and Geffen (1994) found that "a persistent consequence of mild head injury might be an inability to act quickly in response to expected spatial events" (abstract). Shuttleworth-Jordan, Puchert and Balarin (1993) conducted a controlled study on the effects of mild concussive head injury on South African university rugby players using the Denckla Finger Tapping, Purdue Pegboard, Digit Span, Digit Supraspan, and Trail Making Tests. The study found (1) pre- and post season differences between concussed rugby players and controls; and (2) repeated differences between concussed rugby players and controls at 1 week, 2 months and 3 months post-injury. Patterns of deficit, suggestive of closed head injury, were found in areas of hand motor speed, verbal memory and information processing and deficits were present for at least 3 months in concussed players. These authors point out the potentially hazardous significance of such deficits on the academic performance of South African university and high school students. The Shuttleworth-Jordan et al. (1993) study represents a significant improvement on previous rugby

research in that repeated differences between rugby players and non-contact sport controls were utilised and a wider variety of tests was included in the battery. A recent Australian study of mild head injury and speed of information processing in professional rugby league players (Hinton-Bayre, Geffen, McFarland, 1997), showed that measures of speed of information processing were sensitive to impairment in the postacute phase, 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 stated that further studies using multiple baseline measures from individually administered assessments would confirm the consistency of this finding.

2.6.5. Synthesis and Perspectives

The studies of mild head injury in contact sports are of particular importance for the present research in that these studies have highlighted an important factor contributing to the severity of head trauma, namely *the cumulative effects of multiple blows (impact) and/or acceleration/deceleration (nonimpact) injuries to the head, not necessarily resulting in knockouts or loss of consciousness.* The working hypothesis here is that contact sport players receive *successive blows to the head* (e.g., *blunt impacts* from a knee or elbow during a maul in rugby) and/or they sustain *successive subconcussive whiplash-like injuries* (e.g., *rotation and acceleration/deceleration injuries* from a hard tackle in rugby). There is a growing body of research which suggests that these injuries result in pathophysiological, neuropsychological and neurobehavioural sequelae. For these reasons, a study of the possible *cumulative* effects of concussive mild head injuries is not only feasible, but also highly advantageous.

As was the case in studies on the neuropsychological sequelae and postconcussive symptoms of mild head injury in the general or clinical population (section 2.3.2), the findings across studies in contact sport are also inconsistent and variable. This fact warrants further study in the area of mild head injury in contact sport because it is only through constant refining of methodological issues, and re-testing of previous findings, that a uniform and complete picture will begin to emerge. This assertion is substantiated by Barth et al., (1989) who concluded that

> [s]tudying other football teams and sports with differing premorbid characteristics and mechanisms of injury, as well as appropriate control groups, will undoubtedly contribute to our better understanding of what we have now come to view as the spectrum of mild head injury (p. 272).

CHAPTER 3

METHODOLOGY

3.1. Participants

The subjects for this study comprised top league, professional rugby and cricket players who received a neuropsychological assessment as part of the seasonal medical, psychological, kinaesthetic and fitness assessment procedure at the Sport Science Institute in Cape Town. Players who were selected for the national squads of the respective teams were automatically included in the study - the rugby squad consisted of 26 players (15 forwards and 11 backs) and the cricket squad (matched control group) consisted of 21 players. The rationale for selecting top league players for this study was that these players have a longstanding career in their respective sports and thus represent an appropriate "laboratory group" (Barth et al., 1989) for the study of mild head injury in contact sport as well as mild head injury in general. Subjects were excluded from this study if they had ever suffered from a neurological or psychiatric/psychological disorder, if they had ever had the diagnosis of a learning disorder and/or if they had ever failed a standard at school.

(see Table 3-1, below, for a summary of the participant sample demographic data).

Table 3-1. Demographic Data of Participant Sample

Group	No. of Subjects	Age (mean)	Years of Education (mean)	Estimated Premorbid IQ (mean)
Rugby	26	27.5	14.2	119.2
Cricket	21	27.1	13.7	121.1
Forwards	15	27.2	13.9	117.2
Backs	11	27.8	14.6	121.9

The rugby players were assessed, pre-season, in February 1997 and the cricket players were assessed, post-season, in April 1997. The availability of players for assessment was largely contingent upon the national and international competitive commitments of the respective teams. In addition, from the perspective of the Sport Science Institute, there was a substantial amount of logistical preparation and planning involved in coordinating the efforts of the medical, psychological and other professionals in an assessment procedure of such a broad scale. In the case of the cricket team, these factors were particularly influential in ruling out the possibility of assessing the players pre-season as opposed to post-season.

3.2. Data Collection Procedures

3.2.1. Pre-assessment Phase

Prior to the assessment procedure in Cape Town, several planning meetings were held by the core research team of three Intern Clinical Psychologists together with their supervisor. The aim of these meetings was a) to discuss the short- and long-term goals of the research; b) to design the test battery and confer on the rationale behind the tests included and excluded from the battery; and c) to discuss the test administration procedures. The overall objective here was to ensure that each player was assessed according to a *strictly* standardised procedure.

3.2.2. Assessment Phase

Each participant was assessed individually by an assessor from a core team of three highly trained Intern Clinical Psychologists. Due to time constraints, three assistant-assessors were enlisted in Cape Town to aid the team in administration of the assessment battery. The assistant group comprised of two qualified Clinical Psychologists and one Intern Clinical Psychologist. All assessors received their Clinical Psychology Masters degrees at the same university and were well acquainted with the tests included in the battery. The assistants were rigorously briefed as to the standardised procedure that was required in the administration of tests for this particular study. Scoring of the test protocols administered by the assistants was carried out by the original team of three interns, thus ensuring inter-rater reliability.

3.2.2.1. *Consent form and pre-assessment questionnaire*

Prior to administration of the neuropsychological tests, the participant was requested to complete a consent form (see Appendixes A and B). This was followed by a pre-assessment questionnaire (see Appendix D) which tapped a) important biographical information such as age, highest level of education achieved, and current level of functioning; b) sporting history; and c) head injury history (sports-related and non-sports related). This questionnaire was designed by the research team to tap information which would be critical in making decisions on whether or not the subject should be excluded from the study; it was an aid in establishing an estimate of the subject's general intellectual functioning; and the sporting/head injury history provided an important context within which the subjects' test protocols were analysed and interpreted.

3.2.2.2. *Neuropsychological assessment*

A wide-ranging neuropsychological battery was administered to each participant in both groups. This battery comprised of tests in six major modalities, namely (i) General Intellectual Functioning (selected subtests from the South African - Wechsler Adult Intelligence Scale [SA - WAIS]); (ii) Verbal Memory; (iii) Visual Memory; (iv) Verbal Fluency; (v) Visuoperceptual Tracking; and (vi) Hand Motor Dexterity (see Appendixes C and E for the full assessment schedule and administration procedures). The tests administered in each area were as follows:

(i) General Intellectual Functioning
- SA WAIS subtests: Comprehension
  Picture Completion
  Digit Symbol Substitution

(ii) Verbal Memory
- SA WAIS Digit Span
- Wechsler Memory Scale (WMS) Associate Learning (Immediate and Delayed Recall)

(iii) Visual Memory
- SA WAIS Digit Symbol Substitution Incidental Recall
- WMS Visual Reproduction of Designs (Immediate and Delayed Recall)

(iv) Verbal Fluency
- Words-in-a-Minute (Unstructured Verbal Fluency)
- S-Words-in-a-Minute (Structured Verbal Fluency)

(v) Visuoperceptual Tracking
- SA WAIS Digit Symbol Substitution[11]
- Trail Making Test (Part A and B) (from the Halstead Battery)

(vi) Hand Motor Dexterity
- Finger Tapping Test (two trials on both preferred and non-preferred hand)

3.2.2.3. *Postconcussive Symptomolgy Questionnaire (PCSQ)*

In addition to the neuropsychological battery, a 31-item self-report postconcussive symptomology questionnaire (PCSQ) tapping a wide range of possible postconcussive symptoms was administered to each participant (see Appendix F). The 31 questions in the questionnaire were designed around 14 content areas (see Table 3-2, below) and were drawn from a variety of sources (Burbach, 1987; Levin et al., 1987; Lezak, 1995; Lishman, 1978; Walsh, 1985).

TABLE 3-2. Content Areas of Postconcussive Symptomology Questionnaire (PCSQ)

1. Physical/neurological symptoms (incl. headaches, eyesight, fatigue, dizziness, seizures, sensitivity to noise)	8. Frustration tolerance
2. Perceptual disturbances	9. Depression
3. Sexual problems	10. Social withdrawal
4. Speech and language	11. Restlessness
5. Memory	12. Vegetative symptoms
6. Attention and concentration	13. Anxiety
7. Emotional lability	14. Aggression

3.2.3. Post-assessment Phase

A similar process to the initial pre-assessment phase was conducted in the post-assessment phase. Several meetings were held by the core team and their supervisor to discuss scoring and inter-rater

[11] Scores obtained from this subtest were used in the calculation of the estimated premorbid IQs, but the test scores and level of impairment on this test, are considered and presented as part of the Visuoperceptual Tracking modality.

reliability. Following this consultation phase, all relevant data was captured onto *one* comprehensive spreadsheet. This spreadsheet contains all demographic information, test scores and questionnaire responses.

3.3. Data Analysis Procedures

3.3.1. Phase One: Calculation of Deficit on Neuropsychological Assessment

The first phase of data analysis for the present research involved calculating the level of deficit shown by each player in each of the tests administered. The level of severity of deficit was determined according to the degree to which a test score significantly deviated from the best-available normative data at the time of data analysis. Thus, where there was no appropriate norm and standard deviation (SD) available, the level of deficit was not calculated. This was the case for the Wechsler Memory Scale (WMS) Associate Learning Delayed Recall and for the SA WAIS Digit Symbol Substitution Delayed Recall.

Three levels of impairment were devised as follows:

i) None - test score = less than 1 SD from the norm

ii) Mild - test score = equal to or greater than1 SD from the norm

iii) Moderate/Severe - test score = equal to or greater than 2 or 3 SDs from the norm

For the purposes of statistical calculation, each level of severity was encoded as follows:

(i) None = 0 (ii) Mild = 1 (iii) Moderate/Severe = 3

3.3.2. Phase Two: Preparation for Statistical Analysis

The results obtained from the *neuropsychological assessments* were calculated as follows:

Within each group (i.e., rugby and cricket, rugby forwards and rugby backs), the number (n) of subjects who showed deficit in each of the three categories of impairment (namely "none", "mild", "moderate/severe") was calculated. This number was then translated into a percentage (%) which represented the *proportion* of subjects in each group falling into each of the three levels of impairment.

The results obtained from the self-report *PCSQ* were calculated as follows:

Within each group (i.e. rugby and cricket, rugby forwards and rugby backs), the number (n) of subjects who answered "never", "sometimes" or "often" to each question on the questionnaire was calculated. This number was then translated into a percentage (%) which represented the *proportion* of subjects in each group falling into each of the three categories.

3.3.3. Phase Three: Statistical Comparison of Neuropsychological Assessment

Ferguson (1988) argued that "because of a simple relationship for 1 degree of freedom between x^2 and the normal deviate, x^2 provides an [appropriate] procedure for testing the significance of the difference between proportions" (p. 211; Radloff, personal communication, 1998).

In the third phase of data analysis, the overall comparative analysis was carried out on three levels in terms of the groups that were compared, namely: (i) rugby versus cricket; (ii) rugby forwards versus rugby backs; and (iii) forwards versus cricket. The rugby versus cricket comparison addressed the general trends and possible differences across both groups as a whole. The forwards versus backs comparison addressed the hypothesis that given the more full contact nature of the forward positions in the rugby team, there may be differences *within* the rugby group, i.e. between forwards and backs. The forwards versus cricket comparison tested the hypothesis that if there were differences between the forwards and the backs, then significant differences may also exist between forwards and controls. Together, the three levels of analysis addressed the overall hypothesis that full-contact sport players are more susceptible to the effects of cumulative mild head injuries than non-contact sport players.

The data was analysed using the Pearson x^2 statistic to (a) compare the percentage of players in each group who showed deficit on each test in the assessment battery; and (b) to compare the percentage of players in each group who reported a presence of postconcussive symptomology. The results were interpreted in terms of two measures of significance, namely *significant* ($p<0.05$) and *approaching significant* ($0.15>p>0.05$). Because of the relatively small subject sample, using a measure which approached significance was seen to be an appropriate method for noting possible *indicators* of impairment.

3.4. Data Presentation

The data are presented in table format in Chapter 4. Both the neuropsychological assessment and postconcussive symptomology results are presented in three sections according to the groups being compared, namely (a) rugby versus cricket; (b) forwards versus backs; and (c) forwards versus cricket. The neuropsychological tests are classified into five modalities and presented in the following order: a) Verbal Memory, b) Visual Memory, c) Verbal Fluency, d) Visuoperceptual Tracking and e) Hand Motor Dexterity.

3.5. Ethical Considerations

Prior to the assessment process, the participants were requested to sign a consent form (see Appendixes A and B). All participants agreed to take part in the study. Participants were informed that data obtained in the testing would be used for group research and publication purposes only and

individual results would remain *totally confidential and anonymous*. Given the high profile status of the participants, every effort was made to ensure the anonymity of individual players' results. It was explained to all participants that should an assessment yield results which may be cause for concern, no substantive career decisions would be made on the basis of the initial test results prior to further in-depth neuropsychological assessment. Participants were invited to contact the assessment team personally should they wish to discuss any aspect of the assessment procedure and/or results.

CHAPTER 4

RESULTS

The comparative results for the neuropsychological assessment and the Postconcussive Symptomology Questionnaire (PCSQ) are presented according to three levels of group comparison, namely (1) rugby versus cricket; (2) forwards versus backs; and (3) forwards versus cricket.

Neuropsychological assessment results

The cognitive tests are grouped and presented in terms of five modalities, namely (i) Verbal Memory, (ii) Visual Memory, (iii) Verbal Fluency, (iv) Visuoperceptual Tracking, and (v) Hand Motor Dexterity. The results indicating the number (n) and percentage (%) of players with deficit on a specified cognitive test in each modality, together with the x^2 statistic, are presented as follows:

In the hand motor dexterity modality, specifically Tables 4-5, 4-10, and 4-15, there are instances of *no statistic* reported because in these cases *all* subjects have no impairment, thus rendering a statistical comparison null and void.

Postconcussive Symptomology Questionnaire (PCSQ) results

The results indicating the number (n) and percentage (%) of subject responses on the PCSQ, together with the x^2 statistic are presented as follows:

On item 6 (seizures) and item 11 (sexual difficulties) across all three comparisons, *no statistic* is reported because on these items, all subjects gave the same response (i.e., "never") thus rendering a statistical comparison null and void.

NOTE: **In all tables, significance and approaching significance are represented as follows:**

** **indicates *significance* ($p < 0.05$)**

* **indicates *approaching significance*** **($0.15 > p > 0.05$)**

Neuropsychological Assessment: RUGBY versus CRICKET

Table 4-1. VERBAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	RUGBY			CRICKET			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forwards									
n	20	6	0	16	2	3			
%	76.9	23.1	0.0	76.2	9.5	14.3	4.969	2	0.0834*
Digits Backwards									
n	23	2	1	16	5	0			
%	88.5	7.7	3.8	76.2	23.8	0.0	3.045	2	0.2182
Digit Supraspan									
n	22	3	1	15	4	2			
%	84.6	11.5	3.8	71.4	19.0	9.5	1.283	2	0.5265
WMS Associate Learning (Easy) Immed. Recall									
n	25	0	1	19	0	2			
%	96.2	0.0	3.8	90.5	0.0	9.5	0.627	1	0.4286
WMS Associate Learning (Hard) Immed. Recall									
n	21	4	1	17	3	1			
%	80.8	15.4	3.8	81.0	14.3	4.8	0.032	2	0.9839

Table 4-2 . VISUAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	RUGBY			CRICKET			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall									
n	18	3	5	17	3	1			
%	69.2	11.5	19.2	81.0	14.3	4.8	2.188	2	0.3349
WMS Visual Reproduction Immed. Recall									
n	22	2	2	16	2	3			
%	84.6	7.7	7.7	76.2	9.5	14.3	0.622	2	0.7325
WMS Visual Reproduction Delayed Recall									
n	20	6	0	18	3	0			
%	76.9	23.1	0.0	85.7	14.3	0.0	0.580	1	0.4463

Table 4-3. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Deficit.

TEST	RUGBY			CRICKET			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	16	9	1	13	8	0			
%	61.5	34.6	3.8	61.9	38.1	0.0	0.847	2	0.6548
Structured Verbal Fluency									
n	24	1	1	16	4	1			
%	92.3	3.8	3.8	76.2	19.0	4.8	2.901	2	0.2345

Neuropsychological Assessment: RUGBY versus CRICKET (*Continued*)

Table 4-4. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Deficit.

TEST	RUGBY			CRICKET			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution									
n	20	6	0	20	0	1			
%	76.9	23.1	0.0	95.2	0.0	4.8	6.542	2	0.038 **
Trail Making Test A									
n	22	3	1	20	1	0			
%	84.6	11.5	3.8	95.2	4.8	0.0	1.581	2	0.4536
Trail Making Test B									
n	21	2	3	21	0	0			
%	80.8	7.7	11.5	100.0	0.0	0.0	4.519	2	0.1044 *

Table 4-5. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Deficit.

TEST	RUGBY			CRICKET			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Finger Tapping Test I (Preferred Hand)									
n	23	2	0	16	4	0			
%	92.0	8.0	0.0	80.0	20.0	0.0	1.385	1	0.2393
Finger Tapping Test I (Non-preferred Hand)									
n	25	0	0	19	2	0			
%	100.0	0.0	0.0	90.5	9.5	0.0	2.489	1	0.1146 *
Finger Tapping Test II (Preferred Hand)									
n	25	0	0	20	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*[1]		
Finger Tapping Test II (Non-preferred Hand)									
n	26	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*		

[1] Where *no statistic* is reported, all subjects have no impairment thus rendering a statistical comparision null and void.

Neuropsychological Assessment: FORWARDS versus BACKS

Table 4-6. VERBAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forwards									
n	11	4	0	9	2	0			
%	73.3	26.7	0.0	81.8	18.2	0.0	0.257	1	0.6119
Digits Backwards									
n	12	2	1	11	0	0			
%	80.0	13.3	6.7	100.0	0.0	0.0	2.478	2	0.2884
Digit Supraspan									
n	13	2	0	9	1	1			
%	86.7	13.3	0.0	81.8	9.1	9.1	1.48	2	0.4771
WMS Associate Learning (Easy) Immed. Recall									
n	14	0	1	11	0	0			
%	93.3	0.0	6.7	100.0	0.0	0.0	0.763	1	0.3825
WMS Associate Learning (Hard) Immed. Recall									
n	12	2	1	9	2	0			
%	80.0	13.3	6.7	81.8	18.2	0.0	0.833	2	0.6594

Table 4-7. VISUAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall									
n	8	3	4	10	0	1			
%	53.3	20.0	26.7	90.9	0.0	9.1	4.514	2	0.1047 *
WMS Visual Reproduction Immed. Recall									
n	13	1	1	9	1	1			
%	86.7	6.7	6.7	81.8	9.1	9.1	0.115	2	0.9443
WMS Visual Reproduction Delayed Recall									
n	13	2	0	17	4	0			
%	86.7	13.3	0.0	63.6	36.4	0.0	1.896	1	0.1685

Table 4-8. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	7	7	1	9	2	0			
%	46.7	46.7	6.7	81.8	18.2	0.0	3.495	2	0.1742
Structured Verbal Fluency									
n	13	1	1	11	0	0			
%	86.7	6.7	6.7	100.0	0.0	0.0	1.589	2	0.4518

Neuropsychological Assessment: FORWARDS versus BACKS (*Continued*)

Table 4-9. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution							5.720	1	0.0168 **
n	9	6	0	11	0	0			
%	60.0	40.0	0.0	100.0	0.0	0.0			
Trail Making Test A							0.922	2	0.6308
n	12	2	1	10	1	0			
%	80.0	13.3	6.7	90.9	9.1	0.0			
Trail Making Test B							4.540	2	0.1033 *
n	10	2	3	11	0	0			
%	66.7	13.3	20.0	100.0	0.0	0.0			

Table 4-10. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Finger Tapping Test I (Preferred Hand)									
n	13	1	0	10	1	0			
%	92.9	7.1	0.0	90.9	9.1	0.0	0.032	1	0.8586
Finger Tapping Test I (Non-preferred Hand)									
n	14	0	0	11	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*		
Finger Tapping Test II (Preferred Hand)									
n	14	0	0	11	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*		
Finger Tapping Test II (Non-preferred Hand)									
n	15	0	0	11	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*		

Neuropsychological Assessment: FORWARDS versus CRICKET

Table 4-11. VERBAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			CRICKET			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forward									
n	11	4	0	16	2	3			
%	73.3	26.7	0.0	76.2	9.5	14.3	3.695	2	0.1576
Digits Backward									
n	12	2	1	16	5	0			
%	80.0	13.3	6.7	76.2	23.8	0.0	1.910	2	0.3848
Digit Supraspan									
n	13	2	0	15	4	2			
%	86.7	13.3	0.0	71.4	19.0	9.5	1.861	2	0.3943
WMS Associate Learning (Easy) Immed. Recall									
n	14	0	1	19	0	2			
%	93.3	0.0	6.7	90.5	0.0	9.5	0.094	1	0.7598
WMS Associate Learning (Hard) Immed. Recall									
n	12	2	1	17	3	1			
%	80.0	13.3	6.7	81.0	14.3	4.8	0.064	2	0.9686

Table 4-12. VISUAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			CRICKET			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall									
n	8	3	4	17	3	1			
%	53.3	20.0	26.7	81.0	14.3	4.8	4.155	2	0.1252 *
WMS Visual Reproduction Immed. Recall									
n	13	1	1	16	2	3			
%	86.7	6.7	6.7	76.2	9.5	14.3	0.662	2	0.7182
WMS Visual Reproduction Delayed Recall									
n	13	2	0	18	3	0			
%	86.7	13.3	0.0	85.7	14.3	0.0	0.007	1	0.9351

Table 4-13. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			CRICKET			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	7	7	1	13	8	0			
%	46.7	46.7	6.7	61.9	38.1	0.0	1.920	2	0.3829
Structured Verbal Fluency									
n	13	1	1	16	4	1			
%	86.7	6.7	6.7	76.2	19	4.8	1.142	2	0.5649

Neuropsychological Assessment: FORWARDS versus CRICKET (*Continued*)

Table 4-14. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			CRICKET			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution									
n	9	6	0	20	0	1			
%	60.0	40.0	0.0	95.2	0.0	4.8	10.463	2	0.0053 **
Trail Making Test A									
n	12	2	1	20	1	0			
%	80	13.3	6.7	95.2	4.8	0	2.400	2	0.3012
Trail Making Test B									
n	10	2	3	21	0	0			
%	66.7	13.3	20.0	100.0	0.0	0.0	8.129	2	0.0172 **

Table 4-15. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			CRICKET			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Finger Tapping Test I (Preferred Hand)									
n	13	1	0	16	4	0			
%	92.9	7.1	0.0	80.0	20.0	0.0	1.085	1	0.2975
Finger Tapping Test I (Non-preferred Hand)									
n	14	0	0	19	2	0			
%	100.0	0.0	0.0	90.5	9.5	0.0	1.414	1	0.2344
Finger Tapping Test II (Preferred Hand)									
n	14	0	0	20	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*		
Finger Tapping Test II (Non-preferred Hand)									
n	15	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*		

Postconcussive Symptomology: RUGBY versus CRICKET

Table 4-16. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionna

Question	RUGBY			CRICKET			x^2	df	p
	Never	Sometimes	Often	Never	Sometimes	Often			
1. Headaches									
n	17	9	0	4	16	1			
%	65.4	34.6	0.0	19.0	76.2	4.8	10.596	2	0.0050 **
2. Eyesight									
n	25	0	1	17	4	0			
%	96.2	0.0	3.8	81.0	19.0	0.0	6.060	2	0.0483 **
3. Hearing									
n	24	2	0	19	1	1			
%	92.3	7.7	0.0	90.5	4.8	4.8	1.399	2	0.4969
4. Weakness in Limbs									
n	24	2	0	17	3	1			
%	92.3	7.7	0.0	81.0	14.3	4.8	1.885	2	0.3897
5. Clumsiness									
n	24	2	0	19	1	1			
%	92.3	7.7	0.0	90.5	4.8	4.8	1.399	2	0.4969
6. Seizures									
n	26	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*[1]		
7. Dizziness									
n	18	7	1	18	3	0			
%	69.2	26.9	3.8	85.7	14.3	0.0	2.092	2	0.3514
8. Fatigue									
n	22	4	0	14	6	1			
%	84.6	15.4	0.0	66.7	28.6	4.8	2.676	2	0.2624
9. Sensitivity to Noise									
n	19	7	0	16	3	2			
%	73.1	26.9	0.0	76.2	14.3	9.5	3.363	2	0.1861
10. Hallucinations									
n	22	3	1	20	1	0			
%	84.6	11.5	3.8	95.2	4.8	0.0	1.581	2	0.4536
11. Sexual Difficulties									
n	26	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*		
12. Speech Difficulties									
n	24	2	0	20	1	0			
%	92.3	7.7	0.0	95.2	4.8	0.0	0.167	1	0.6828
13. Clumsy Speech									
n	13	13	0	15	6	0			
%	50.0	50.0	0.0	71.4	28.6	0.0	2.215	1	0.1367 *
14. Stutter									
n	23	3	0	20	1	0			
%	88.5	11.5	0.0	95.2	4.8	0.0	0.685	1	0.4078
15. Slurred Speech									
n	24	2	0	20	1	0			
%	92.3	7.7	0.0	95.2	4.8	0.0	0.167	1	0.6828
16. Memory									
n	19	7	0	13	8	0			
%	73.1	26.9	0.0	61.9	38.1	0.0	0.667	1	0.4140

[1]Where *no statistic* is reported, all subjects gave the same response, thus rendering a statistical comparision null and void.

Table 4-16. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionna (*continued*).

Question	RUGBY			CRICKET			x^2	df	*p*
	Never	Sometimes	Often	Never	Sometimes	Often			
17. Attention/Concentration							3.122	2	0.2099
n	18	6	2	10	10	1			
%	69.2	23.1	7.7	47.6	47.6	4.8			
18. Sustained Attention							6.268	2	0.0435 **
n	7	18	1	11	7	3			
%	26.9	69.2	3.8	52.4	33.3	14.3			
19. Impatience							0.136	2	0.9341
n	4	15	7	4	12	5			
%	15.4	57.7	26.9	19.0	57.1	23.8			
20. Irritability							2.431	2	0.2966
n	8	17	1	11	9	1			
%	30.8	65.4	3.8	52.4	42.9	4.8			
21. Easily Angered							1.529	2	0.4656
n	11	12	3	12	6	3			
%	42.3	46.2	11.5	57.1	28.6	14.3			
22. Depressed							2.504	2	0.2859
n	16	10	0	9	11	1			
%	61.5	38.5	0.0	42.9	52.4	4.8			
23. Social Contact							2.833	2	0.2425
n	1	9	16	2	3	16			
%	3.8	34.6	61.5	9.5	14.3	76.2			
24. Restlessness							0.668	2	0.7160
n	16	7	3	11	8	2			
%	61.5	26.9	11.5	52.4	38.1	9.5			
25. Sleep Difficulties							0.937	2	0.6259
n	19	6	1	13	6	2			
%	73.1	23.1	3.8	61.9	28.6	9.5			
26. Appetite Difficulties							0.011	1	0.9162
n	22	4	0	18	3	0			
%	84.6	15.4	0.0	85.7	14.3	0.0			
27. Anxiety							0.399	2	0.8193
n	11	14	1	7	13	1			
%	42.3	53.8	3.8	33.3	61.9	4.8			
28. Worry							0.167	2	0.9199
n	12	12	2	10	10	1			
%	46.2	46.2	7.7	47.6	47.6	4.8			
29. Argumentative							4.327	2	0.1149 *
n	10	12	4	14	4	3			
%	38.5	46.2	15.4	66.7	19.0	14.3			
30. Short-tempered							0.086	1	0.7699
n	15	11	0	13	8	0			
%	57.7	42.3	0.0	61.9	38.1	0.0			
31. Aggression							0.167	1	0.6828
n	24	2	0	20	1	0			
%	92.3	7.7	0.0	95.2	4.8	0.0			

Postconcussive Symptomology: FORWARDS versus BACKS

Table 4-17. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnai

Question	FORWARDS			BACKS			x^2	df	p
	Never	Sometimes	Often	Never	Sometimes	Often			
1. Headaches									
n	9	6	0	8	3	0			
%	60.0	40.0	0.0	72.7	27.3	0.0	0.454	1	0.5004
2. Eyesight									
n	14	0	1	11	0	0			
%	93.3	0.0	6.7	100.0	0.0	0.0	0.763	1	0.3825
3. Hearing									
n	13	2	0	11	0	0			
%	86.7	13.3	0.0	100.0	0.0	0.0	1.589	1	0.2075
4. Weakness in Limbs									
n	14	1	0	10	1	0			
%	93.3	6.7	0.0	90.9	9.1	0.0	0.053	1	0.8187
5. Clumsiness									
n	13	2	0	11	0	0			
%	86.7	13.3	0.0	100.0	0.0	0.0	1.589	1	0.2075
6. Seizures									
n	15	0	0	11	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*		
7. Dizziness									
n	10	4	1	8	3	0			
%	66.7	26.7	6.7	72.7	27.3	0.0	0.768	2	0.6812
8. Fatigue									
n	12	3	0	10	1	0			
%	80.0	20.0	0.0	90.9	9.1	0.0	0.580	1	0.4462
9. Sensitivity to Noise									
n	11	4	0	8	3	0			
%	73.3	26.7	0.0	72.7	27.3	0.0	0.001	1	0.9725
10. Hallucinations									
n	12	3	0	10	0	1			
%	80.0	20.0	0.0	90.9	0.0	9.1	3.653	2	0.1610
11. Sexual Difficulties									
n	15	0	0	11	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*		
12. Speech Difficulties									
n	13	2	0	11	0	0			
%	86.7	13.3	0.0	100.0	0.0	0.0	1.589	1	0.2075
13. Clumsy Speech									
n	6	9	0	7	4	0			
%	40.0	60.0	0.0	63.7	36.4	0.0	1.418	1	0.2337
14. Stutter									
n	13	2	0	10	1	0			
%	86.7	13.3	0.0	90.9	9.1	0.0	0.112	1	0.7380
15. Slurred Speech									
n	14	1	0	10	1	0			
%	93.3	6.7	0.0	90.9	9.1	0.0	0.053	1	0.8187
16. Memory									
n	8	7	0	11	0	0			
%	53.3	46.7	0.0	100.0	0.0	0.0	7.025	1	0.0080 **

Postconcussive Symptomology: FORWARDS versus BACKS (*Continued*)

Table 4-17. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnai (*continued*).

Question	FORWARDS			BACKS			x^2	df	*p*
	Never	Sometimes	Often	Never	Sometimes	Often			
17. Attention/Concentration									
n	9	4	2	9	2	0			
%	60.0	26.7	13.3	81.8	18.2	0.0	2.101	2	0.3498
18. Sustained Attention									
n	3	11	1	4	7	0			
%	20.0	73.3	6.7	36.4	63.6	0.0	1.451	2	0.4842
19. Impatience									
n	2	10	3	2	5	4			
%	13.3	66.7	20.0	18.2	45.5	36.4	1.223	2	0.5425
20. Irritability									
n	3	11	1	5	6	0			
%	20.0	73.3	6.7	45.5	54.5	0.0	2.412	2	0.2993
21. Easily Angered									
n	5	8	2	6	4	1			
%	33.3	53.3	13.3	54.5	36.4	9.1	1.17	2	0.5571
22. Depressed									
n	7	8	0	9	2	0			
%	46.7	53.3	0.0	81.8	18.2	0.0	3.313	1	0.0687 *
23. Social Contact									
n	1	3	11	0	6	5			
%	6.7	20.0	73.3	0.0	54.5	45.5	3.723	2	0.1555
24. Restlessness									
n	7	5	3	9	2	0			
%	46.7	33.3	20.0	81.8	18.2	0.0	4.015	2	0.1343 *
25. Sleep Difficulties									
n	10	5	0	9	1	1			
%	66.7	33.3	0.0	81.8	9.1	9.1	3.179	2	0.2040
26. Appetite Difficulties									
n	11	4	0	11	0	0			
%	73.3	26.7	0.0	100.0	0.0	0.0	3.467	1	0.0626 *
27. Anxiety									
n	3	11	1	8	3	0			
%	20.0	73.3	6.7	72.7	27.3	0.0	7.404	2	0.0247 **
28. Worry									
n	5	8	2	7	4	0			
%	33.3	53.3	13.3	63.6	36.4	0.0	3.125	2	0.2096
29. Argumentative									
n	3	10	2	7	2	2			
%	20.0	66.7	13.3	63.6	18.2	18.2	6.471	2	0.0393 **
30. Short-tempered									
n	7	8	0	8	3	0			
%	46.7	53.3	0.0	72.7	27.3	0.0	1.766	1	0.1839
31. Aggression									
n	13	2	0	11	0	0			
%	86.7	13.3	0.0	100.0	0.0	0.0	1.589	1	0.2075

Postconcussive Symptomology: FORWARDS versus CRICKET

Table 4-18. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire

Question	FORWARDS			CRICKET			x^2	df	p
	Never	Sometimes	Often	Never	Sometimes	Often			
1. Headaches									
n	9	6	0	4	16	1			
%	60.0	40.0	0.0	19.0	76.2	4.8	6.653	2	0.0359 **
2. Eyesight									
n	14	0	1	17	4	0			
%	93.3	0.0	6.7	81.0	19.0	0.0	4.413	2	0.1101 *
3. Hearing									
n	13	2	0	19	1	1			
%	86.7	13.3	0.0	90.5	4.8	4.8	1.500	2	0.4724
4. Weakness in Limbs									
n	14	1	0	17	3	1			
%	93.3	6.7	0.0	81.0	14.3	4.8	1.327	2	0.5150
5. Clumsiness									
n	13	2	0	19	1	1			
%	86.7	13.3	0.0	90.5	4.8	4.8	1.500	2	0.4724
6. Seizures									
n	15	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*		
7. Dizziness									
n	10	4	1	18	3	0			
%	66.7	26.7	6.7	85.7	14.3	0.0	2.498	2	0.2868
8. Fatigue									
n	12	3	0	14	6	1			
%	80.0	20.0	0.0	66.7	28.6	4.8	1.187	2	0.5524
9. Sensitivity to Noise									
n	11	4	0	16	3	2			
%	73.3	26.7	0.0	76.2	14.3	9.5	2.128	2	0.3451
10. Hallucinations									
n	12	3	0	20	1	0			
%	80.0	20.0	0.0	95.2	4.8	0.0	2.057	1	0.1515
11. Sexual Difficulties									
n	15	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No statistic*		
12. Speech Difficulties									
n	13	2	0	20	1	0			
%	86.7	13.3	0.0	95.2	4.8	0.0	0.842	1	0.3590
13. Clumsy Speech									
n	6	9	0	15	6	0			
%	40.0	60.0	0.0	71.4	28.6	0.0	3.556	1	0.0593 *
14. Stutter									
n	13	2	0	20	1	0			
%	86.7	13.3	0.0	95.2	4.8	0.0	0.842	1	0.3590
15. Slurred Speech									
n	14	1	0	20	1	0			
%	93.3	6.7	0.0	95.2	4.8	0.0	0.061	1	0.8057
16. Memory									
n	8	7	0	13	8	0			
%	53.3	46.7	0.0	61.9	38.1	0.0	0.264	1	0.6071

Table 4-18. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire (*continued*).

Question	FORWARDS			CRICKET			x^2	df	*p*
	Never	Sometimes	Often	Never	Sometimes	Often			
17. Attention/Concentration									
n	9	4	2	10	10	1			
%	60.0	26.7	13.3	47.6	47.6	4.8	2.013	2	0.3654
18. Sustained Attention									
n	3	11	1	11	7	3			
%	20.0	73.3	6.7	52.4	33.3	14.3	5.616	2	0.0603 *
19. Impatience									
n	2	10	3	4	12	5			
%	13.3	66.7	20.0	19.0	57.1	23.8	0.358	2	0.8359
20. Irritability									
n	3	11	1	11	9	1			
%	20.0	73.3	6.7	52.4	42.9	4.8	3.879	2	0.1438 *
21. Easily Angered									
n	5	8	2	12	6	3			
%	33.3	53.3	13.3	57.1	28.6	14.3	2.436	2	0.2959
22. Depressed									
n	7	8	0	9	11	1			
%	46.7	53.3	0.0	42.9	52.4	4.8	0.744	2	0.6892
23. Social Contact									
n	1	3	11	2	3	16			
%	6.7	20.0	73.3	9.5	14.3	76.2	0.267	2	0.8752
24. Restlessness									
n	7	5	3	11	8	2			
%	46.7	33.3	20.0	52.4	38.1	9.5	0.804	2	0.6691
25. Sleep Difficulties									
n	10	5	0	13	6	2			
%	66.7	33.3	0.0	61.9	28.6	9.5	1.525	2	0.4666
26. Appetite Difficulties									
n	11	4	0	18	3	0			
%	73.3	26.7	0.0	85.7	14.3	0.0	0.856	1	0.3548
27. Anxiety									
n	3	11	1	7	13	1			
%	20.0	73.3	6.7	33.3	61.9	4.8	0.789	2	0.6742
28. Worry									
n	5	8	2	10	10	1			
%	33.3	53.3	13.3	47.6	47.6	4.8	1.257	2	0.5334
29. Argumentative									
n	3	10	2	14	4	3			
%	20.0	66.7	13.3	66.7	19.0	14.3	9.143	2	0.0103 **
30. Short-tempered									
n	7	8	0	13	8	0			
%	46.7	53.3	0.0	61.9	38.1	0.0	0.823	1	0.3643
31. Aggression									
n	13	2	0	20	1	0			
%	86.7	13.3	0.0	95.2	4.8	0.0	0.842	1	0.3590

4.1. Summary of Significant Results

With regard to mean estimated premorbid IQ, there was no significant difference between the rugby and cricket groups ($p = 0.57$) and between the rugby forwards and backs ($p = 0.33$). In terms of mean years of education, there was no significant difference between rugby and cricket (p=0.24) and between the forwards and backs (p=0.18). There was also no significant difference between the rugby and cricket groups (p=0.77) and between the forwards and backs (p=0.58) in terms of mean age.

4.1.1. Neuropsychological Assessment

In the presentation of this summary, the significant differences and differences which approach significance will be isolated with respect to particular tests. This will be followed by a description of the nature and direction of these differences for the affected tests.

Significant Results **($p < 0.05$):**

There was a significant difference in the level of deficit on two neuropsychological tests: (1) the *SA WAIS Digit Symbol Substitution (DSS)* subtest yielded significant differences across all three comparisons, namely rugby versus cricket (p=0.03), forwards versus backs (p=0.01), and forwards versus cricket (p=0.005); and (2) the *Trail Making Test B (TMT-B)* yielded significant differences in the forwards versus cricket comparison (p=0.02).

(1) *SA WAIS Digit Symbol Substitution (DSS)*:
In the rugby versus cricket comparison, the largest difference appeared in the mild level of deficit (>1SD below the norm) where there were 23.1% (n=6) of the rugby players as opposed to 0.0% (n=0) of the controls with mild deficit (see Table 4-4). In the forwards versus backs comparison, the largest difference was once again in the mild category of deficit where there were 40% (n=6) of the forwards as opposed to 0% (n=0) of the backs with mild deficit (see Table 4-9). All back player scores on the DSS subtest fell within the norm, thus yielding a result of 100% of the backs with no impairment. In other words, all players in the rugby group with mild impairment on the DSS test, were in fact forward players. Because of this, there is a significant difference between the rugby forward and cricket groups on the DSS test (see Table 4-14) with the forwards performing significantly poorer than controls.

(2) *Trail Making Test B (TMT-B)*:
In the forwards versus cricket comparison, there were 13.3% (n=2) of the forwards as opposed to 0.0% (n=0) of the cricket players with mild impairment (see Table 4-14); and, there were 20% (n=3) of the forwards as opposed to 0% of the cricket players with moderate to severe impairment (>2 or 3 SDs below the norm). Thus, all of the controls had no impairment on the *TMT-B*, while the

proportion of rugby players who had impairment on this test (both mild and moderate to severe), were all forward players (see Table 4-14).

***Results Approaching Significance* (0.15 > *p* > 0.05):**

The results indicate that there were four neuropsychological tests in which the comparisons of the level of deficit *approached* significance and are thus possible indicators of a tendency toward impairment: (1) the *SA WAIS Digits Forward (DF)* subtest yielded results approaching significance in the rugby versus cricket comparison (p=0.08); (2) *Digit Symbol Incidental Recall (DSIR)* yielded a result approaching significance in the forwards versus backs comparison (p=0.1); (3) the *TMT-B* yielded a result approaching significance in the rugby versus cricket comparison (p=0.1) and in the forwards versus backs comparison (p=0.1); and (4) *Finger Tapping* Test 1 (Non-preferred Hand) (p=0.11).

(1) *SA WAIS Digits Forward (DF)*:
In the rugby versus cricket comparison, there were 23.1% (n=6) of the rugby players in contrast with 9.5% (n=2) of the controls who had mild impairment on the *DF* test (see Table 4-1). In the cricket group, 14.3% (n=3) displayed moderate to severe impairment, while no players in the rugby group had impairment in the moderate to severe category (see Table 4-1).

(2) *Digit Symbol Incidental Recall (DSIR)*:
In the forwards versus backs comparison, 20% (n=3) of the forwards group had mild impairment and 26.7% (n=4) had moderate to severe impairment on the DSIR test. This is in contrast to the backs group where 0% have mild impairment and 9.1% (n=1) have moderate to severe impairment (see Table 4-7). These statistics reveal that the majority of rugby players who have any level of deficit on this test, are forwards (7 out of 8). All back players, except for one, have no impairment on the DSIR test.

(3) *Trail Making Test-B (TMT-B)*:
In the rugby versus cricket comparison, a similar scenario to the profile on the DSIR test is revealed on the TMT-B. In the rugby group, 7.7% (n=2) were mildly impaired and 11.5% (n=3) were moderately to severely impaired on the TMT-B (see Table 4-4). All subjects in the control group had scores which fell within the norm, thus showing no deficit on this test.

In the forwards group, there are 13.3% (n=2) of players with mild impairment, and 20% (n=3) with moderate to severe impairment. This is in contrast to the backs, where no player was impaired on the TMT-B. This implies that all 5 of the rugby players who have impairment on this test, are forwards, hence the indicative difference in the rugby versus cricket comparison.

(4) *Finger Tapping Test I* (Non-preferred Hand):

In the rugby versus cricket comparison, there was a small proportion of cricket players (9.5%, n=2) with mild impairment on this test. This is in contrast to the rugby group who had no impairment (see Table 4-5).

All other cognitive tests yielded non-significant results.

4.1.2. Self-report Postconcussive Symptomology

In the presentation of this summary, the significant differences and differences which approach significance will be isolated with respect to particular symptoms. This will be followed by a description of the nature and direction of these differences for the affected symptoms.

Significant Results **($p < 0.05$):**

In the rugby versus cricket comparison (see Table 4-16), there was a significant difference between the groups on the following three symptoms: (1) *headache* (item 1) (p=0.005); (2) *eyesight difficulties* (item 2) (p=0.05); and (3) *sustained attention* (item 18) (p=0.04).

In the forwards versus backs comparison (see Table 4-17), there was a significant difference between the groups on the following three symptoms: (1) *memory difficulties* (item 16) (p=0.008); *anxiety* (item 27) (p=0.02); and (3) *argumentativeness* (item 29) (p=0.04).

In the forwards versus cricket comparison (see Table 4-18), there was a significant difference between the groups on the following two symptoms: (1) *headache* (item 1) (p=0.04); and (2) *argumentativeness* (item 29) (p=0.01).

(1) *Headache* (item 1):

In the rugby versus cricket comparison, more cricket players than rugby players reported headaches in that 76.2% (n=16) of the cricket group as opposed to 34.6% (n=9) of the rugby group reported 'sometimes' experiencing this symptom. In the forwards versus cricket comparison, more cricket players (76.2%, n=16) than forwards (40%, n=6) report 'sometimes' having headaches. However, it is useful to note that the two-thirds of rugby players who report experiencing occasional headaches, were forwards (6 out of 9).

(2) *Eyesight* (item 2):

In the rugby versus cricket comparison, virtually all of the rugby group (96.2%, n=25) reported 'never' having eyesight difficulties, whereas 19% (n=4) of the cricket players reported 'sometimes' experiencing eyesight difficulties.

(3) *Sustained attention* (item 18) :

In the rugby versus cricket comparison, there were significantly more rugby than cricket players who reported 'sometimes' encountering difficulties with sustained attention. Sixty-nine percent (n=18) of the rugby group, in contrast to 33.3% (n=7) of the cricket group reported problems in sustained attention. The forwards versus backs comparison revealed that the majority of rugby players who reported this symptom were forwards (11 out of 18).

(4) *Memory* (item 16):

In the forwards versus backs comparison, significantly more forwards than backs reported memory difficulties. All backs (100%) reported 'never', while 47% (n=7) of the forwards reported 'sometimes' experiencing difficulties with memory. This comparison reveals that all rugby players who reported problems with memory, are forwards. This is in contrast to the majority of controls (61.9%, n=13) who reported 'never' having memory difficulties.

(5) *Anxiety* (item 27):

The forwards versus backs comparison showed that significantly more forwards than backs experience anxiety. This comparison also revealed that the majority of rugby players who reported anxiety symptoms, were forwards (14 out of 15). In the forwards group, 73.3% (n=11) as opposed to 27.3% (n=3) of the backs reported 'sometimes' experiencing anxiety.

(6) *Argumentativeness* (item 29):

In the forwards versus backs comparison, there were significantly more forwards (66.7%, n=10) than backs (18.2%, n=2) who reported 'sometimes' being argumentative. In the forwards versus cricket comparison, only 19% (n=4) of the cricket group reported being occasionally argumentative thus revealing that the forwards reported feeling argumentative significantly more than controls. This comparison also revealed that the majority of rugby players who reported this symptom, are forwards (12 out of 16).

Results Approaching Significance **($0.15 > p > 0.05$):**

In the rugby versus cricket comparison, there were results approaching significance on the following two symptoms: (1) *clumsy speech* (p=0.14); and (2) *argumentativeness* (p=0.12).

In the forwards versus backs comparison, results which approach significance were on the following two symptoms: (1) *depression* (p=0.07); and (2) *appetite difficulties* (p=0.06).

In the forwards versus cricket comparison, there were results which approached significance on the following four symptoms: (1) *eyesight* (p=0.11); (2) *clumsy speech* (item 13) (p=0.06); (3) *sustained attention* (p=0.06); and (4) *irritability* (p=0.14).

(1) *Clumsy speech* (item 13):

In the rugby versus cricket comparison, more rugby players (50%, n=13) than controls (28.6%, n=6) reported 'sometimes' finding their speech to be clumsy. In the forwards versus cricket comparison a similar results which approaches significance is revealed in that 60% (n=9) of the forwards versus 28.6% (n=6) of the controls reported the symptom of clumsy speech to occur 'sometimes'. This comparison also reveals that the majority of rugby players who reported clumsy speech, are forwards (9 out of 13).

(2) *Argumentativeness* (item 29):

As pointed out above, the forwards versus backs comparison yielded a significant result on this symptom. In the rugby versus cricket comparison the result approached significance in that 46.2% (n=12) of the rugby players in contrast with 19% (n=4) of the controls reported 'sometimes' being argumentative.

(3) *Depression* (item 22):

In the forwards versus backs comparison, depression was reported more often by forwards than by backs. This result approached significance in that 53% (n=8) of the forwards as opposed to 18.2% (n=2) of the backs reported 'sometimes' feeling depressed.

(4) *Appetite* (item 26):

In the forwards versus backs comparison, all back players reported 'never' experiencing appetite difficulties, whereas 26.7% (n=4) of the forwards reported 'sometimes' having appetite difficulties. All the rugby players who reported this symptoms were forwards (4 out of 4).

(5) *Eyesight* (item 2):

As noted above, this symptom yielded a significant result in the rugby versus cricket comparison. The forwards versus cricket comparison yielded a result which approaches significance in that all, except for 1, of the forwards reported 'never' having eyesight problems. This is contrast to 19% (n=4) of the controls who reported 'sometimes' experiencing problems with eyesight. Clearly, the majority of both forwards and backs (i.e. the whole rugby group) reported no problems with eyesight while a small number of controls reported difficulty in this area.

(6) *Sustained attention* (item 18):

As noted above, this symptom yielded a significant result in the rugby versus cricket comparison where significantly more rugby players reported difficulties in sustained attention when compared to controls. In the forwards versus cricket comparison, 73.3% (n=11) of the forwards as opposed to 33.3% (n=7) of the controls reported 'sometimes' having problems in sustained attention. This comparison reveals that the majority of rugby players who reported this symptom, are forwards (12 out of 19).

(7) *Irritability* (item 20):

In the forwards versus cricket comparison, there were 73.3% (n=11) of the forwards in contrast with 42.9% (n=9) of the controls who reported 'sometimes' feeling symptoms of irritability. The majority of rugby players who reported irritability, were forwards (12 out of 18).

All other items on the PCSQ yielded non-significant results.

CHAPTER 5

DISCUSSION

In order to provide a context within which to discuss and present the findings of this thesis, it was important to consider the definition of mild head injury *per se*, as well as to define concussion with respect to mild head injury. Given the variation in definitions and the inconsistency of severity classifications in previous research, this was a complex task. Nevertheless, with regard to the definition of these terms, it was finally concluded that concussion, even in its mildest form, can be classified as a mild head injury and that, as is the case in several previous studies, the terms may be used interchangeably. Since it was not the aim of this study to classify and isolate concussed from non-concussed subjects (as was the case in Shuttleworth-Jordan et al., 1993), the controversies in definition did not directly have relevance to outcome in the present research. However, the definition was vital in order to establish correct and appropriate parameters within which to study and refer to 'concussion' and 'mild head injury'.

The overarching objective of this study was to consider the cumulative effects of successive mild head injuries. The aim was to compare the results obtained by contact sport players and non-contact sport controls on a) formal neuropsychological testing, and b) a self-report postconcussive symptomology questionnaire. In this process, the following working hypotheses were applied: (1) contact sport players receive *successive blows to the head* (e.g., *blunt impacts* from a knee or elbow during a maul in rugby) and/or they sustain *successive concussive and subconcussive (whiplash-like) head injuries* (e.g., *rotation and acceleration/deceleration injuries* from a hard tackle in rugby); and (2) because of these injuries, there will be persisting patterns of deficit commensurate with deficits in closed mild head injury.

With this aim in mind, and in order to facilitate the comparison between the rugby and control groups, the research needed to control for certain key variables which typically influence neuropsychological test results, namely age, education, and estimated premorbid IQ. On all three variables, there were no significant differences between the groups and therefore, these factors could not account for the comparative differences between the groups.

One variable that was potentially problematic for the comparison of results, was that unlike the rugby group who were assessed pre-season, the cricket group (controls) was assessed post-season. It was evident from clinical observation and from reports by the cricketers themselves, that as a group, there was a certain level of fatigue present. To a large extent, this was due to the fact that a long, highly competitive and stressful season, in which the players had spent many months away from home, had culminated in a five-day test match which ended the day prior to the assessment. It is also noteworthy that the cricketers lost this test match series which affected the overall mood of players and contributed to an observable despondency. This was in contrast to the rugby team who were assessed

pre-season; the rugby players were clearly motivated and responded with much enthusiasm to the entire assessment procedure. It is thus important to consider the possibility that the level of fatigue in the control group produced depressed test results, thus obscuring both (a) the optimal control group level of functioning, and hence (b) the detection of possible deficits between the rugby group and control group. However, this means that any differences that *were* found, could represent the bare minimum of what might have been expected; had the control group been functioning at an optimal level, more differences may have been revealed. Thus, whilst being sub-optimal, in that the full impact of negative effects in the rugby group might have been lost, the present comparison produced a more stringent analysis in that where there are significant findings, these can be considered to be relatively robust.

5.1. Neuropsychological Assessment

5.1.1. Significant Results

SA WAIS Digit Symbol Substitution (DSS)

The most notable finding on neuropsychological testing was the significant difference across all comparisons on the *SA WAIS Digit Symbol Substitution (DSS)* subtest. Results on this test revealed that while no deficit was found in the control group, a significant proportion (23%, n=6) of the rugby group were found to be mildly impaired on this test. In addition, upon finer analysis, it was found that of the proportion of rugby players who were found to be impaired on this test, *all* were forwards. Hence, there were no backs with impairment on this test. This latter result contributed to the significant difference on the forwards versus controls comparison. This implies that it is players who play in the more full-contact positions (e.g., scrum positions) who are more likely to be impaired on DSS.

According to Lezak (1995), motor persistence, sustained attention, response speed and visuomotor coordination play important roles in a subject's performance on this test. Maddocks, Saling and Dicker (1995) point out that DSS is the one of most practical tests of speed of information processing. In addition,

> this test is consistently more sensitive to brain damage than other WIS [Wechsler Intelligence Scale] battery tests in that its score is most likely to be depressed even when damage is minimal, and to be among the most depressed when the other tests are affected as well (Lezak, 1995, p. 378).

Because DSS is not sensitive to the exact locus of a lesion, it is generally of little use in predicting the laterality of a lesion. This aspect of DSS's non-specific sensitivity to cerebral dysfunction means that it is affected by a wide range of performance components and importantly, is more useful where diffuse damage is suspected. Clearly, in the present study where there is a focus on the degrees of diffuse axonal damage associated with mild concussive head injury, as well as the cumulative effects

thereof, a finding of significant differences between rugby and controls (and more particularly, between forwards and controls) on DSS is a highly potent result.

This finding is consistent with studies wherein neuropsychological tests of speed of information processing such as DSS, have been shown to be sensitive to the effects of concussive head injury in American Football (Barth et al., 1989) and Australian Rules Football (Maddocks and Dicker, 1989; Maddocks and Saling, 1991).

However, the present finding of significant differences on test performance in DSS, is inconsistent with that of Maddocks, Saling and Dicker (1995) who reported normal levels of performance on this test six months or longer after injury in Australian Rules Football. These authors suggested that despite previous findings (Barth et al., 1989), there were no residual effects from earlier concussive injuries on function as measured by DSS and therefore found no support for the notion of cumulative effects from repeated concussive injury found by Gronwall and Wrightson (1975). Maddocks et al. argue further that the Gronwall and Wrightson study included subjects who had suffered mild head injuries due to motor vehicle accidents (MVAs) and that therefore, significant acceleration/ deceleration forces could have affected their subjects. According to Maddocks et al., this is in contrast with concussive head injury in sport where it is likely that smaller deceleration forces are operating thus resulting in milder forms of injury. This is a plausible argument, but it is based on a theoretical, as opposed to empirical, distinction. It is also unclear what variables might have affected the difference in outcome in the Maddocks et al. and the present study. It may be that the differing nature of the sports in these studies (i.e. Australian Rules Football and Rugby), influenced the results. However, it would seem more plausible that the reason for the difference in findings here is methodological in that Maddocks et al. used the traditional method of comparing means, whereas the present study looked for individual deficit and *proportions* of deficit within the groups. It appears that this latter method may have been a more powerful mechanism for detecting effects. Moreover, the most potent methodological aspect to the present study proved to be the isolation of positional differences (within the rugby group) which Maddocks et al. did not do. As was discussed in Chapter 2, (section 2.1, above) mild head injuries occur along a spectrum of increasing severity, and to discount the cumulative effects of "milder forms of injury" (such as may be sustained by contact sport players) would seem premature at this stage. Certainly, the finding of impairment on DSS in the present research supports this conclusion and it is imperative that further investigations be conducted.

Trail Making Test B (TMT-B)

The second significant result obtained on neuropsychological testing in this study, was found in the forwards versus cricket comparison on the Trail Making Test, Part B (TMT-B). Relative to controls who showed no impairment on this test, there was a proportion of rugby players who had mild (13.3%) and moderate (20%) impairment on the TMT-B. Both the rugby versus cricket comparison, and the forwards versus backs comparison on this test yielded a result which approached significance

indicating a tendency towards a significant difference between rugby and controls. Upon finer analysis, it was found that of the proportion (33.3%) of rugby players with deficit on this test, *all* were forwards, resulting in a significant difference between the forwards and controls. Thus, no rugby back players had impairment on this test, whereas 2 forwards had mild impairment and 3 had moderate impairment (a total of 5 out of 15 forwards with impairment). As was the case for DSS, this finding suggests that it is players who play in the more full contact positions in the rugby team (i.e., forwards) who are most susceptible to impairment on TMT-B. This difference between forwards and backs is compatible with the findings of Shuttleworth-Jordan (1983) who found that the backs showed a significant practice effect on TMT-B which was not present for the forwards.

Like DSS, the Trail Making Test is a test of visuoperceptual and visuomotor tracking. The test thus involves the dual components of motor speed and attention and because of this, it is highly sensitive to the effects of brain injury. In particular, Part B on this test involves more complex conceptual tracking and a greater degree of mental flexibility, in that the subject must attend to more than one stimulus at a time (divided attention). The finding of deficits in the rugby group (more specifically, in the forwards group) on this test, thus suggests that contact-sport such as rugby contributes to deficits in mental flexibility, divided attention and visuoperceptual tracking. This finding is consistent with previous studies wherein it was found that TMT performances by patients with mild head trauma were slower than those of controls (Leininger et al., 1990; Shuttleworth-Jordan et al., 1993). Kaste et al. (1992) found significant differences between boxers and controls on both parts of TMT.

5.1.2. Synthesis of Significant Neuropsychological Assessment Results

On both DSS and TMT, it is unlikely that deficits in hand motor dexterity were contributing to lowered scores on these tests. There was a negligible proportion of cricket players (9.5%, n=2) who had mild impairment on the first trial of the Finger Tapping test (Non-preferred Hand). The exact reason for this instance of lowered scores in the control group relative to rugby is peculiar, but seems to be related to the fact that all rugby and cricket players reported having sustained hand or finger injuries in the course of their sporting careers. Thus any cerebral attributions to lowered scores on hand motor dexterity tests would be problematic.

Taken together, the results on DSS and TMT-B strongly suggest that relative to controls, rugby players exhibit at least mild impairment in the visuoperceptual tracking function. This is similar to previous research findings on mild head injury, where deficits were detected in the essential features of visuoperceptual tracking such as speed of information processing (Abreau et al., 1990; Barth et al., 1989; Gronwall and Wrightson, 1974; Hinton-Bayre et al., 1997; Leininger et al., 1990; Levin et al., 1987; Rimel et al., 1981; Shuttleworth-Jordan et al., 1993), sustained effortful attention (Parasuraman et al., 1991), complex attention and mental flexibility (Levin et al., 1987; Gentillini et al., 1987). The present finding also supports previous studies in the literature where it was evident that the most

common and consistent deficit due to mild head injury, was in the area of speed of information processing.

More specifically, the findings suggest that the forwards are showing more impairment than the backs. This implies that it is *players who play in the more full-contact positions* (e.g., scrum positions) *who are more likely to be impaired in the functions measured by DSS and TMT-B.* To clarify, it is the forwards in the rugby team, more so than the backs, who are involved in a number of scrums, mauls and hard tackles which result in repeated head to head, head to torso, or head to ground collisions during a game. This is similar to Barth et al.'s (1989) report that the most common type of injury in American football is helmet-to-helmet collision, by players in the offensive line, in the course of tackling or blocking manoeuvres. The present finding of a significant difference between the rugby forwards and the backs (and hence between rugby and controls) in the functions of visuoperceptual tracking, may thus be evidence for the cumulative effects of repeated subconcussive and concussive mild head injury. Given the evidence of some variability across findings as well as an instance of a null outcome with respect to the cumulative effects of repeated trauma (Maddocks et al., 1995), further investigations are required.

5.1.3. Results Approaching Significance

SA WAIS Digits Forward (DF)

The rugby versus cricket comparison yielded a result in which the percentage of rugby players (23.1%) with mild impairment on DF was almost significantly greater than the percentage of controls (9.5%) with mild impairment on DF. However, while no players in the rugby group showed moderate to severe impairment on this test, 14.3% of cricket players had impairment in that range.

Lezak (1995) points out that what DF measures is more closely related to efficiency of attention (freedom from distractibility) than to what is commonly thought of as memory. This aspect is corroborated elsewhere (Gronwall, 1987; Ruff et al., 1989). The finding, which is simply indicative of a possible trend, that 23.1% of the rugby players exhibit mild impairment on this test, may be associated with mild attention deficit showing up in the rugby group which then impacted on other tests also sensitive to deficits in attention, most notably, DSS and TMT-B. However, the fact that three cricketers showed moderate impairment on this test, raises the speculation that (a) mean test scores on tests involving attention and freedom from distractibility might have been affected in the control group, thus further obscuring comparisons; and (b) the fatigue observed and reported in the cricket group had a detrimental effect on performance in this test. On the whole, the proportion of players with deficit on DF are small, thus conclusions and observations with respect to this test should be conservative.

Digit Symbol Substitution Incidental Recall (DSIR)

The analysis of the percentages of players with deficit on DSIR revealed that relative to controls, there is greater tendency for the rugby group to show both mild (11.5%) and moderate to severe (19.2%) impairment on DSIR. The most notable difference in the proportion of players with deficit in the rugby versus cricket comparison, appears to fall within the moderate to severe range of deficit. In addition, the forwards versus backs comparison indicated that of the percentage of rugby players with impairment on this test, the vast majority (88%) are forwards, hence the comparative result that approaches significance on the forwards versus controls comparison. This implies that it is the players in the more full contact positions who are susceptible to deficits on this test.

This pattern appears to be closely linked to that observed on the DSS test. It would appear that the significantly poorer performance of the rugby (specifically forwards) group on DSS when compared to controls, tended to affect the recall of newly learned material or memory function required for DSIR. This may suggest that the attentional deficits implied in the poor performance on DSS, may have impacted on the incidental recall trial of the same test. The results here are indicative of possible verbal memory deficits in the rugby group. This is particularly the case for players in the forward positions.

5.1.4. Synthesis of Neuropsychological Assessment Results Approaching Significance

While the results obtained on the DF and DSIR tests should be interpreted with caution, they are nevertheless indicative of a possible trend towards attentional and/or verbal and visual memory deficits in the rugby group, particularly among forward players. Deficits in attention (Gronwall, 1989; Levin, 1987; Rimel, 1981) and in memory and distractibility (Barth et al., 1983; McLean et al., 1983; Ruff et al., 1987) following mild head injury have been documented and seem to substantiate the tendencies noted on the DF and DSIR tests. In a boxing study, Heilbronner et al. (1991) reported impairments in verbal recall (specifically retrieval deficit for newly learned and symbolic material) and incidental memory (on DSIR) post-fight. Hart, Kwentus, Wade and Hamer (1987 in Shuttleworth-Jordan and Bode, 1995) found that DSIR is clinically useful in distinguishing mild dementia of the Alzheimer's type from depressive pseudodementia in that DSIR is less affected in depression than in organic dementia. According to Hart et l., DSIR is also a better discriminator between depression and dementia than tests of psychomotor speed, verbal recall and design reproduction. These findings may have significant import into the present study where diffuse (dementia-like) damage is suspected among full contact sport players. Deficits on the DF test appear to be less associated with an actual memory component and more associated with an attentional component (Ruff et al., 1989) than what would be the case on the DSIR test. However, the aspect of speed of information processing and what Lezak (1995) calls efficiency of attention (freedom from distractibility) may have compromised performance on DSIR (as was the case on DSS).

5.2 Self-report Postconcussive Symptomology

5.2.1. Significant Results

Headache and Eyesight

There was a significant difference noted in the rugby versus cricket comparison and in the forwards versus cricket comparison in terms of the percentage of players reporting the headache symptom. The percentage of cricket players who reported 'sometimes' experiencing these symptoms, was significantly greater than (a) the percentage of rugby players and (b) the percentage of forwards. There was no difference between the forwards and backs groups on this symptom.

In the rugby versus cricket comparison, significantly more controls reported 'sometimes' experiencing problems with eyesight than rugby players. The difference between forwards and controls on this symptom approached significance where the percentage of controls reporting this symptom was somewhat greater than the percentage of forwards who reported this symptom. There was no difference between the forwards and backs on this symptom.

Although headaches were reported more frequently than eyesight difficulties, these two symptoms appear to be related, particularly in light of the fact that it is commonly known that eyesight difficulties and headaches often co-exist. Given the nature of the questions asked in the questionnaire, the exact nature and extent of these difficulties is unknown. Poor vision and/or headache have been consistently reported in the literature as symptoms which occur particularly in the acute phase following mild head injury (Alves et al., 1993; Levin et al., 1987; McLean et al, 1983; Rimel et al., 1981; Rutherford et al., 1977). However, the present research findings diverge from previous research in that it is the non-head injury controls and not the hypothetically head inured rugby player group who reported these symptoms. This anomaly warrants consideration. As was noted above, the cricket players were assessed at the end of a long and stressful season, and immediately following a five-day test match. In addition, the fatigue level of the players has been highlighted. It is possible that these factors were contributing to the player's susceptibility to headache and vision problems, particularly in the light of the fact that cricket involves standing for many hours in a hot sun. The possibility that these symptoms may be common amongst cricketers may be related to the fact that in recent years, the incidence of players who wear protective hats and specialised sunglasses has increased dramatically. On the other hand, this may be conjecture and it is possible that there is a proportion of cricketers who are exhibiting commonly reported postconcussive symptoms. This is an outside possibility since a number of cricketers have played both rugby and soccer in their junior and high school years. However, it is evident that these symptoms are the *only* instance (both on self-report measures and testing) where the control group are impaired relative to the rugby group, and there is no other support for head injury sequelae in the

control group at all. This strongly suggests that the cluster of headache and eyesight difficulties is due to post-season fatigue and stress rather than postconcussive symptomology.

Sustained Attention

Difficulty in sustained attention (or freedom from distractibility) was reported by a significantly greater proportion of rugby players (69.2%) than cricket players (33.3%) who 'sometimes' experience this symptom. The frequency of this symptom in the forwards and backs seems similar at first (there is no significant difference on this comparison), but the forwards versus cricket comparison approaches significance which indicates that there is a tendency for the forwards to report this symptom more than backs.

Difficulties in concentration and attention have been reported in previous research (Dikmen et al, 1989; Gronwall, 1989; Levin et al., 1987; Rutherford et al., 1977) thus corroborating the present result. This finding is extremely pertinent in the light of the findings on neuropsychological testing where tests requiring a degree of sustained attention and freedom from distractibility, most notably DSS and TMT-B, but also DF and DSIR proved to be the most affected. In addition, the indication that more rugby forwards than backs reported difficulty with sustained attention, supplements the finding that significantly more forwards than backs were compromised on the tests of DSS, DSIR, and TMT-B.

Memory

With respect to memory, there was no significant difference between rugby players and controls. However, the forwards versus backs comparison yielded a significant difference in the frequency of reported memory problems. This comparison revealed that of the proportion (26.9%, n=7) of rugby players who reported memory difficulties, *all* were forwards. While no backs reported memory problems, almost half the forwards reported this symptom. The subjective reports of memory problems corroborates what was found on neuropsychological testing where there is an indicator, approaching significance, of verbal and/or visual memory deficits which may have influenced performance on DSIR.

It is unclear, due to the open-ended nature of the question (item 16 on the Postconcussive Symptomology Questionnaire), whether the memory difficulties reported here were associated with recent, intermediate or long-term memory. In addition, as Lezak (1995) points out,

> people with mental efficiency problems associated with diffuse damage frequently interpret their experiences of slowed information processing and attentional deficits as memory problems, even when learning is affected only mildly.... Thus they complain of 'poor memory', but analysis of their performance on memory and attention tests typically implicates reduced auditory span, difficulty doing (or processing) more than one stimulus at a time, and verbal retrieval problems" (p. 181).

While the results in this study, both on cognitive testing (particularly DSS, TMT-B, DF and DSIR) and self-report symptomology, seem to corroborate Lezak's argument, it would seem that further investigations are needed to clarify whether or not these complaints of memory difficulties are directly associated with memory *per se*, or if they are related to an attentional deficit such as what Lezak discussed.

In any event, this result is extremely important in that, again, it is the forwards more so than the backs who are impaired on key tests and self-report symptoms. The subjective report of memory difficulties has occurred frequently in the literature (Barth et al., 1989; Levin et al., 1987; Rimel et al., 1981; Rutherford, 1977). However, there is no previous research which suggests that it is exclusively players in the more full contact positions who have memory deficits. The highly significant finding of subjective complaints of memory difficulties *by rugby forward players* represents a more finely differentiated analysis and warrants further investigation.

Argumentativeness

The analysis of the percentage of players who reported this symptom revealed a pattern in which the difference between the rugby and control groups as a whole approached significance, but the differences between the forwards and backs and between the forwards and controls was highly significant. The now familiar pattern of forwards being more affected on certain symptoms and tests, is repeated here. Of the 16 players who reported this symptom, 12 were forwards and 4 were backs, indicating that the vast majority of the percentage of rugby players who reported feeling argumentative, were forwards. This implies that players who occupy the more full contact positions in the rugby team are exhibiting symptoms associated with disinhibition and low levels of frustration tolerance.

It may be argued that the nature of a forward position in rugby would attract players who are naturally more aggressive and who are drawn to full contact competition. The nature of the sport may also socialise the player into more aggressive behaviour - such behaviour is apparently condoned and rewarded by fellow team members. This may mean that a symptom such as argumentativeness, which is essentially an aggressive symptom, would be expected from these players. This may raise the issue of whether or not this symptom is a postconcussive symptom or what we are seeing is a personality variable. It is possible that a combination of the two variables (postconcussive and personality) could explain the occurrence of this symptom. As this finding has not been addressed in previous research, it represents a novel area worthy of further consideration.

Anxiety

As was the case for the memory symptom, a similar scenario was found in the report of anxiety. There was no significant difference between rugby and controls on this symptom. However within

the rugby group, there is a significant difference between the forwards and backs, where the forwards reported 'sometimes' feeling anxious more frequently than backs. This analysis revealed that of the proportion of rugby players who reported this symptom, the vast majority (80%) comprised forward players. Of the 15 rugby players who reported this symptom, 12 were forwards. This implies that it is the players who are involved in the more full contact positions of the rugby team, are more susceptible to experiencing anxiety symptoms. Self-report anxiety symptoms have been found in the relevant literature (Rutherford, 1977; Lidvall et al., 1974, in Szymanski and Linn, 1992).

5.2.2. Synthesis of Significant Postconcussive Symptomology Results

There is evidence of a cluster of symptoms occurring in the rugby group which distinguishes that group from controls. This cluster comprises the symptoms of sustained attention, memory, argumentativeness and anxiety. In all of these symptoms, it was *consistently* found that it was the forwards, more so than the backs, who were most affected. This is a highly significant finding given it's consistent occurrence. Hence, there is a strong indication that the forwards, i.e. players in the more full contact positions who are subjected to repeated mild head injuries more often than the backs, are reporting the most postconcussive symptoms. This means that to view the rugby group as a whole without focusing on the differences found in position played, would significantly obscure the findings. Two of the four symptoms, namely sustained attention and argumentativeness, significantly distinguished rugby from controls. The symptoms of sustained attention and memory appear to have significant correlates in the findings on neuropsychological testing, particularly in DSS, TMT-B, DSIR and DF. The cluster of symptoms reported here is suggestive of deficits that would be associated with diffuse cerebral damage due to the cumulative effects of closed mild head injury.

The anomalous findings of headache and eyesight problems being more frequently reported by controls than by rugby appears to be most satisfactorily explained as (a) an idiosyncratic consequence of the nature of the game of cricket itself, and (b) associated with the post-seasonal stage of the assessment.

5.2.3. Results Approaching Significance

Clumsy Speech

A result which approached significance on this symptom was noted in the rugby versus cricket comparison. The difference between forwards and backs was non-significant, but the difference between forwards and controls approached significance which suggests that the forwards have a tendency towards clumsy speech. An analysis of the verbal fluency test reveals that, particularly on unstructured verbal fluency (a test more sensitive to diffuse damage than structured verbal fluency), there were more forwards than backs with mild impairment. While the differences on performance in verbal fluency as a modality were non-significant, the pattern noted here may be an indicator for

further research. Despite the testing component, the percentage of players who reported clumsy speech points to possible deficits in verbal fluency in rugby players, particularly forwards. Speech difficulties have not been reported in mild head injury studies reviewed in this thesis. However, in the present study, the report of speech difficulties does tie in with the general cluster of postconcussive symptoms evident in particularly in the forwards group. However, the fact that the finding with respect to speech difficulties only approaches significance, means that conclusions regarding this symptom should be made with caution at this stage.

Irritability, Depression and Appetite

In the case of the irritability symptom, there was a difference which approached significance in forwards versus cricket comparison. In the case of depression and appetite difficulties, there was a difference which approached significance in the forwards versus backs comparison. In all three cases, there are possible indications that the forwards are more susceptible to this cluster of postconcussive symptoms which are associated with the sequelae of mild head injury.

5.2.4. Synthesis of Postconcussive Symptomology Results Approaching Significance

Aside from the symptom of clumsy speech which may be associated with a neurocognitive deficit, there are indications of a possibility that the rugby players, more particularly the forwards, are possibly exhibiting neuropsychiatric symptoms associated with a mood disturbance. This constellation of symptoms is similar to Levin et al.'s (1987) study where the authors noted a cognitive-depressive factor present in their subject sample. In addition, Rutherford (1977) found irritability and depression to be significant factors and Barth et al. (1983) reported symptoms which amounted to "dysphoria" and a "general psychological discomfort" (p. 532).

5.3. Conclusions

The present research set out to compare the percentage of rugby players with the percentage of cricket players (non-contact sport controls) who exhibit (a) objectively measured cognitive deficit, and (b) self-report postconcussive symptomology. The most consistent and significant result obtained in this study was that in both cognitive tests and self-report postconcussive symptomology, those players who occupy the more full contact positions in the rugby team, namely forwards, are most susceptible to impairment. It is clear that the relative lack of impairment shown by the backs compared to the forwards, has the effect of raising the mean scores in the rugby group as a whole, and therefore, the differentiation of forwards from backs proved to be a powerful methodological device. In addition, the *detection of individual deficit* and the *comparison of proportions of deficit* within each group, proved to be highly effective in detecting the presence or absence of deficit.

In terms of the sequelae found in the present study, the most significant neurocognitive deficits were found in areas of speed of information processing, reduced mental flexibility, attention and concentration, sustained attention, verbal and/or visual memory and new learning. The most significant neuropsychiatric complaints were found in areas of anxiety, depression, irritability and lowered frustration tolerance (argumentativeness). The only somatic complaints were those of headache and eyesight difficulties reported by the cricketers. No somatic complaints were reported by the rugby players.

In the present study, the most sensitive cognitive tests were Digit Symbol Substitution and the Trail Making Test. These key indicators of impairment not only distinguished the rugby group from controls, but also produced a conceptually coherent argument supporting the presence of effects due to mild head injury. It is also evident from the results in this study that the self-report items strongly reinforce the objective test results. In conjunction, the test results and self-reported symptoms reveal clear patterns of postconcussive sequelae which can be associated with the effects of mild closed head injury.

5.4. Evaluation and Recommendations for Future Research

The post-season fatigue of the cricket players and the implications thereof has been addressed in the above discussion. In order to obtain optimal results in both groups and, therefore, to eradicate the interference of post-season circumstances, it is recommended that both the rugby group and controls be tested at the same stage, preferably pre-season.

Reports obtained from the cricket players indicated that many cricketers have also played rugby or soccer in their sporting careers (particularly at school where participation in those sports is compulsory at most South African high schools). It is recommended that a non-contact sport control group comprising, for example, hockey players, who typically do not have a history of playing contact sport at school, be included in future research. Since hockey and rugby are both winter sports, individuals have to select one or the other, and therefore, hockey players typically have not played rugby as well. It may also be beneficial to include a non-sport playing *matched* control group as a third control group. Since the results in the present study indicate that the rugby player group comprises individuals who are high functioning, control groups should be matched according to estimated premorbid IQ, age and education.

In terms of the size of the subject sample in the present study, it should be borne in mind that the subject sample is relatively small and that a larger sample would be useful in detecting patterns of impairment.

The neuropsychological test battery for the present study was seen to be adequate and in fact highlighted certain key indicators of impairment associated with mild head injury, namely Digit

Symbol Substitution and the Trail Making Test. This may be an indication that a smaller test battery than the one used in the present study is warranted, particularly in cases where time and manpower are lacking. On the other hand, it is recommended that a comprehensive test battery be utilised if there are questions about the full extent of deficits present. A more comprehensive battery would also allow for the estimation of premorbid IQ which is essential in ensuring the equivalence of comparative groups.

The test battery used in this study could be improved by including a verbal memory test which does not have a ceiling effect. In other words, because subjects in the present research were high functioning, memory tests of greater difficulty would be more discriminating. This could be in the form of a list learning task, such as the Auditory Verbal Learning Test (AVLT) and should include a recognition trial. It should be noted that the AVLT also has a ceiling effect with high functioning individuals, but its advantage is that the examiner has the opportunity to examine the *learning curve* obtained over six trials (including the recognition trial). A longer digit learning task could also be included. In both instances, i.e. list learning and digit learning, the inclusion of these tasks would assist in clarifying whether or not impairment on certain tests are related to memory or attentional deficits.

The questionnaire used in this study appears to cover an appropriate and relevant cross-section of postconcussive symptoms. However, the distinction between "sometimes", "never", and "often" is somewhat arbitrary. The questionnaire could be refined to use a wider range of possible answers by using a Likert scale in combination with a qualitative component such as a follow-up interview. This would ensure that in cases where there is ambiguity, clarification can be sought. It would also ensure that information such as the aetiology and onset of symptoms could be obtained.

5.5. Final Synthesis

Mild head injury in contact sport continues to stimulate debate and controversy, particularly in areas of definition, classification and effects. By comparing the proportion of individuals with deficit on formal neuropsychological testing and self-report measures, the findings of the present study strongly suggest that rugby players are showing signs of impairment which are commensurate with the effects of closed mild head injury. More specifically, the findings in this research indicate that players in the more full contact positions, namely forwards, are most susceptible to deficit. The differentiation of rugby forward and back players was a powerful methodological tool in the present analysis in that it allowed for a refinement in the detection of effects. The fact that such refinement yielded highly significant results suggests that a differentiation (based on the position played) within the rugby group should be utilised in future research.

The overall results reported in this study are seen to make a valuable contribution to the growing body of research in the area of mild head injury in a contact sport such as rugby. An extremely

important consideration is the possibility of the permanence of the deficits detected in this study. The literature reviewed in Chapter 2 strongly suggests that even the mildest concussive head injury may result in persisting and irreversible neuropathological and neuropsychological sequelae. If, as the present study indicates, rugby forward players are most susceptible to impairment, the cumulative, and possibly irreversible effects of repeated mild head injury in this group, is cause for grave concern and certainly warrants further in-depth investigation.

CHAPTER 6

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Appendix A

Consent Form: Rugby

NEUROPSYCHOLOGICAL ASSESSMENT

CONSENT FORM

I hereby agree to undergo a neuropsychological assessment of my cognitive functioning on the following understanding:

1. This testing will provide the means to identify impairments in the areas of language fluency, attention and memory, visuoperceptual and fine hand motor skills, which may or may not be due to head injuries. The data from this testing will be used for group research and publication purposes in which the individual results will remain totally confidential and anonymous.

2. Specific findings for individuals will be made available in the form of a brief report to the sports physicians of the Sports Science Institute of South Africa, and will form part of a comprehensive report for the South African Rugby Football Union. These individual results will be released to the two above-mentioned bodies on the understanding that they are based on a preliminary research assessment, do not constitute a full clinical assessment, and hence in themselves should not be used to make substantive career decisions. It is understood, however, that the assessment may reveal important indicators of cognitive difficulties which would be in the best interests of an individual to follow up. Should such a follow-up neuropsychological assessment be indicated, this can be arranged on request. It would involve supplementary testing and personalised counselling about the risks involved in playing contact sport considering that individual's particular life circumstances.

Name:

Signed: Date:

Appendix B

Consent Form: Cricket

NEUROPSYCHOLOGICAL ASSESSMENT

CONSENT FORM

I hereby agree to undergo a neuropsychological assessment of my cognitive functioning on the following understanding:

1. The assessments will take around 2 hours per person, and involve a series of questions and a variety of intellectual tests which are usually quite enjoyable for the testee.

2. The results will serve as a *normal control group* data base for research into mild head injury sustained in contact sports such as football and rugby.

3. The data from this research will be used exclusively for research and publication purposes in which individual results will remain confidential and anonymous.

4. However, if any cricketer would like feed-back on the outcome of their assessment, this can be arranged. Furthermore, on request individual results will be kept on confidential file at the Psychology Clinic in the event that they might be useful for subsequent professional purposes at some later date - for example, should any player suffer a head injury in a motor vehicle accident (MVA).

Appendix C

Assessment Schedule

NEUROPSYCHOLOGICAL TESTING: NATIONAL RUGBY TEAM

ASSESSMENT SCHEDULE

1. Pre-assessment Questionnaire
2. Postconcussive Symptomology Questionnaire
3. SA WAIS Digit Symbol Substitution *including* INCIDENTAL RECALL
4. Trail Making Test A and B
5. Words-in-a-Minute
6. "S" Words-in-a-Minute
7. Finger Tapping Test I
8. Digit Symbol DELAYED RECALL (20 mins)
9. WMS - Memory for Designs - IMMEDIATE RECALL
10. SA WAIS Picture Completion
11. SA WAIS Comprehension
12. WMS - Memory for Designs - DELAYED RECALL (20 mins)
13. WMS - Associate Learning - IMMEDIATE RECALL
14. SA WAIS Digit Span
15. Digit Supraspan A and B
16. Finger Tapping Test II
17. WMS - Associate Learning - DELAYED RECALL (20 mins)

Appendix D

Pre-assessment Questionnaire

RHODES UNIVERSITY PSYCHOLOGY DEPARTMENT

Pre-assessment Questionnaire

NAME:_____ **DATE OF BIRTH:**_____

ADDRESS:_____

PHONE:_____ **HIGHEST QUALIFICATION:**_____

FIRST LANGUAGE:_____

- **GENERAL HISTORY**

Question 1

Did you ever fail a year at school? **[] Yes** **[] No**

If **Yes**, when?_____ For what reason?_____

Question 2

What symbol did you achieve for your Senior Certificate (matric)?_____

If qualification lower than matric, please state average mark attained_____

Question 3

What was your final result at University?

Undergraduate:_____

Postgraduate:_____

Question 4

Have you had any other occupations aside from professional rugby? **[] Yes** **[] No**

If **Yes**, please specify_____

_____

Question 5

Have you ever been diagnosed with a learning disorder? **[] Yes** **[] No**

If **Yes**, what disorder was diagnosed?_____

Question 6

Have you ever suffered from a neurological disorder? **[] Yes** **[] No**

If **Yes**, what disorder was diagnosed?_____

Question 7

Have you ever been diagnosed with a psychiatric disorder? **[] Yes** **[] No**

If **Yes**, what disorder was diagnosed?_____

Question 8

Are you currently taking any form of medication? **[] Yes** **[] No**

If **Yes**, please specify_____

Question 9

Do you smoke? **[] Yes** **[] No**

If **Yes**, how much?_____

Question 10

Do you consider yourself to be a normal drinker? (By 'normal' we mean drinking *less than* or *as much as* most other people). **[] Yes** **[] No**

Question 11

Have you ever felt that you should cut down on your drinking? **[] Yes** **[] No**

Question 12

What other forms of substances do you take?_____

How often?_____

Question 13

Have you ever sustained a head injury or concussion that was *not* related to sport (e.g. motor vehicle accident). ***Note to examiner: DO NOT INCLUDE SPORTS-RELATED INJURIES HERE.***

[] Yes **[] No**

If **yes**, date/s? **Injury 1**_____ **Injury 2**_____

Injury 1

- What caused the injury/concussion?_____

_____

_____

- Did you lose consciousness? **[] Yes** **[] No**

  If **Yes**, for how long?_____
- Did you lose your memory? **[] Yes** **[] No**

  If **Yes**, for how long?_____
- Were you hospitalised? **[] Yes** **[] No**

  If **Yes**, for how long?_____

Injury 2

- What caused the injury/concussion?_____

_____

_____

- Did you lose consciousness? **[] Yes** **[] No**

  If **Yes**, for how long?_____
- Did you lose your memory? **[] Yes** **[] No**

  If **Yes**, for how long?_____
- Were you hospitalised? **[] Yes** **[] No**

  If **Yes**, for how long?_____

- **SPORTS HISTORY**

Question 14

a) At what age did you first start playing rugby?_____

b) What team/s did you play for in high school?_____

c) What was the position you played most often?_____

d) How long have you been playing provincial/national rugby?_____

_____

e) In which position do you play now?_____

Question 15

a) Have you ever sustained a head injury or concussion during a game of rugby?

[] Yes **[] No**

If **Yes**, date/s? **Injury 1**_____**Injury 2**_____

Injury 3_____**Injury 4**_____**Injury 5**_____

Injury 1

- What caused the injury/concussion?_____

_____

_____

- Were you dazed or confused? **[] Yes** **[] No**

  If **Yes**, for how long?_____

- Did you lose consciousness? **[] Yes** **[] No**

  If **Yes**, for how long?_____

- Did you lose your memory? **[] Yes** **[] No**

  If **Yes**, for how long?_____

- Were you hospitalised? **[] Yes** **[] No**

  If **Yes**, for how long?_____

- Did you have any other symptoms or difficulties? **[] Yes** **[] No**

If **Yes**, please specify_____

_____

Injury 2

- What caused the injury/concussion?_____

_____

_____

- Were you dazed or confused? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose consciousness? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose your memory? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Were you hospitalised? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you have any other symptoms or difficulties? [] **Yes** [] **No**

  If **Yes**, please specify_____

_____

Injury 3

- What caused the injury/concussion?_____

_____

_____

- Were you dazed or confused? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose consciousness? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose your memory? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Were you hospitalised? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you have any other symptoms or difficulties? [] **Yes** [] **No**

  If **Yes**, please specify_____

_____

Injury 4

- What caused the injury/concussion?_____

_____

- Were you dazed or confused? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose consciousness? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose your memory? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Were you hospitalised? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you have any other symptoms or difficulties? [] **Yes** [] **No**

  If **Yes**, please specify_____

_____

Injury 5

- What caused the injury/concussion?_____

_____

_____

- Were you dazed or confused? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose consciousness? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose your memory? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Were you hospitalised? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you have any other symptoms or difficulties? [] **Yes** [] **No**

  If **Yes**, please specify_____

_____

b) What other injuries have you sustained while playing rugby?_____

_____

_____

Question 16

a) What other sports do you/have you play/ed? **(QUERY BOXING)**_____

_____

b) Have you ever sustained a head injury or concussion while playing a sport *other than rugby*?

[] **Yes** [] **No**

If **Yes**, date/s? **Injury 1**_____**Injury 2**_____**Injury 3**_____

Injury 1

- What caused the injury/concussion?_____

  _____

  _____

- Were you dazed or confused? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose consciousness? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose your memory? [] **Yes** [] **No**

If **Yes**, for how long?_____

- Were you hospitalised? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you have any other symptoms or difficulties? [] **Yes** [] **No**

  If **Yes**, please specify_____

_____

Injury 2

- What caused the injury/concussion?_____

_____

_____

- Were you dazed or confused? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose consciousness? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose your memory? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Were you hospitalised? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you have any other symptoms or difficulties? [] **Yes** [] **No**

  If **Yes**, please specify_____

_____

Injury 3

- What caused the injury/concussion?_____

_____

_____

- Were you dazed or confused? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose consciousness? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you lose your memory? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Were you hospitalised? [] **Yes** [] **No**

  If **Yes**, for how long?_____

- Did you have any other symptoms or difficulties? [] **Yes** [] **No**

  If **Yes**, please specify_____

_____

Appendix E

Neuropsychological Test Battery

Test 10

DIGIT-SYMBOL SUBSTITUTION

Directions

This test was originally taken from the Army Performance Test, but altered as to the directions and time allowed. Wechsler set a time limit of 1½ minutes.

Place the Digit-Symbol sheet before the subject and indicate the key at the top.

Say: "Look at these little boxes or squares. You will notice that each has a number in the upper part and a sign or mark in the lower part. Every number has a different sign" (indicate). "Now, down here" (point to the sample) "there are some more of the boxes, but this time they have only the numbers at the top and the spaces underneath are empty. You have to put into each of the spaces the mark that belongs (corresponds) to the number at the top. The first number is 2, so we have to put in this mark" (pointing to the key – examiner then fills in the 2-sign). "The next is a 3, so we put in this mark" (indicating the sign and filling it in).

The examiner then fills in the rest of the examples personally, asking the subject in each case to point out the appropriate symbol. Do not permit the subject to do the examples, as he must be shown the correct substitutions in the examples.

When all the examples have been filled in, say:

"Now I want you to go on from here yourself and put into each space the sign that belongs to the number at the top. Take each in order as it comes and do not leave any out. Work as quickly as you can and see how many you can do in 1½ minutes."

If the subject begins erasing or correcting an incorrect solution tell him to leave it and go on with the next.

Record on the score sheet the time taken for each five symbols, as an indication of variability in speed of performance. Also make a note of the subject's method of work, etc.

- MAKE A NOTE OF HOW MANY TIMES SHE LOOKS IN 90s. BUT ALLOW TIME S TO FINISH UP TO THE END OF THE SECOND LAST, THICK, VERTICAL LINE.

Test 10

DIGIT-SYMBOL SUBSTITUTION

Scoring

The score is the total number of symbols correctly entered. Precision and neatness are disregarded, but recorded symbols must be identifiable.

For the symbol ⋀ , subjects sometimes use the letter "N". Grant ½-point for each such reversal.

On the other hand

L for L

V or U for U

U for ⋀

3, > or ⊃ for ⊐

to maximum score

Maximum Score: 67

X. SYFERS VERVANG DEUR SIMBOLE.
X. DIGIT SYMBOL SUBSTITUTION.

NAAM
NAME .....

Datum
Date .....

SLEUTEL
KEY

1	2	3	4	5	6	7	8	9
–	⋀	⊐	L	∪	O	∧	X	=

VOORBEELD SAMPLE								TOETS BEGIN TEST BEGINS																	
2	1	3	1	2	4	3	5	3	1	2	1	3	2	1	4	2	3	5	2	3	1	4	6	3	
1	5	4	2	7	6	3	5	7	2	8	5	4	6	3	7	2	8	1	9	5	8	4	7	3	
6	2	5	1	9	2	8	3	7	4	6	5	9	4	8	3	7	2	6	1	5	4	6	3	7	

Aantal korrek 120" Number correct 90"	Aantal half korrek 120" Number half correct 90"	TOTAAL 120" TOTAL 90"

RGN 170-485

X. SYFERS VERVANG DEUR SIMBOLE.
X. DIGIT SYMBOL SUBSTITUTION. — IMMEDIATE

NAAM
NAME .....

Datum
Date .....

SLEUTEL
KEY

1	2	3	4	5	6	7	8	9

TRAIL MAKING TEST - METHOD

TRAIL A

The subject's task is to connect the circles in numerical sequence as quickly as possible with a penciled line. The subject should be cautioned to be careful to avoid making mistakes. However, if the subject makes a mistake the examiner calls it to his attention immediately, indicates the last circle correctly reached, and asks the subject to proceed from that point. Thus, in practice, mistakes contribute to the time required for completion of the task.

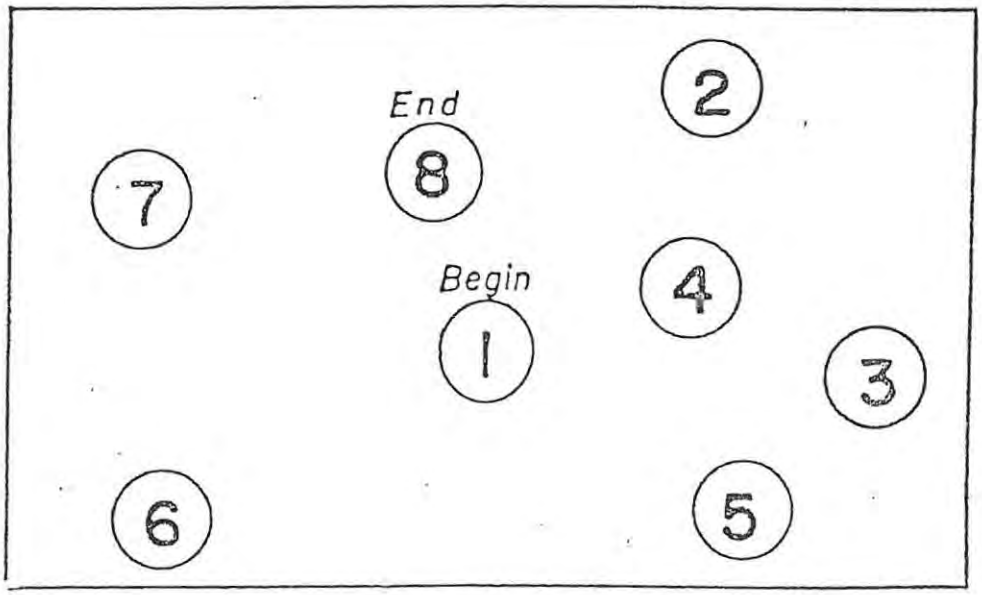
TRAIL B

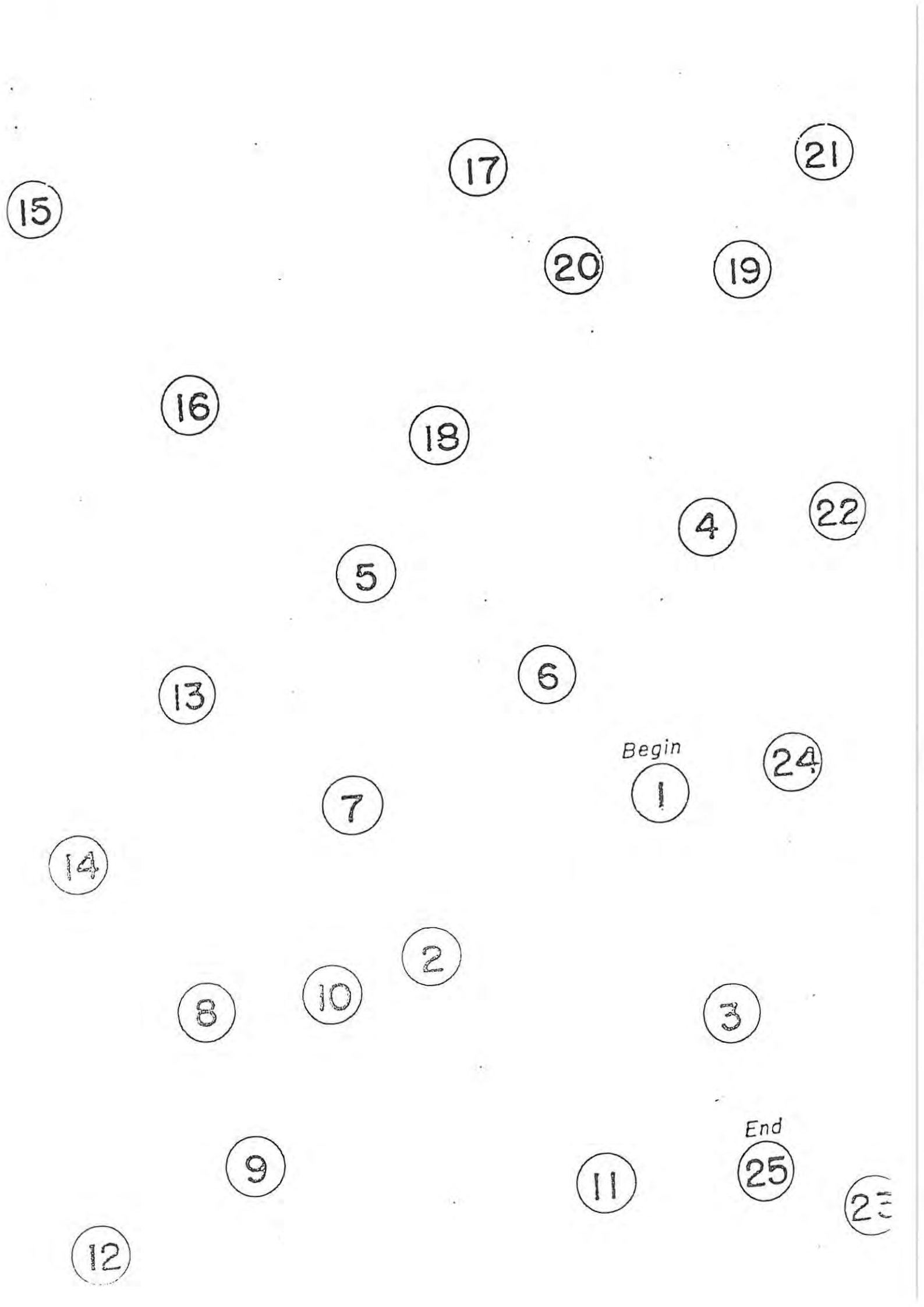
As for Trail A, except the subject is directed to begin at number 1, to draw a line to A, then to 2, next to B, and so on, alternating between numbers and letters until he reaches the end.

TRAIL MAKING

Part A

SAMPLE




17
21
15
20
19
16
18
4
22
5
6
13
Begin
24
1
7
14
2
10
8
3
End
9
25
11
12

WORDS-IN-A-MINUTE

Testee's name:

Instruction: "I would like you to say as many different words as you can think of. You must say the words as fast as you can and I will count them. You can say any words *except* proper nouns (like your name or the name of a city) and words with a different suffix (like sit and sitting). Counting or sentences are also not allowed. Do you understand? Just keep going, I will tell you to stop after one minute".

Instructions to be repeated if the subject does not understand what is required.

Score:

Notes or Observations:

"S" WORDS-IN-A-MINUTE

Testee's name:

Instruction: "Now I would like you to say as many words as you can think of that begin with the letter "S". You must say the words as fast as you can and I will count them. You can say any words *except* proper nouns (like your name or the name of a city) and words with a different suffix (like sit and sitting). Counting or sentences are also not allowed. Do you understand? Just keep going; I will tell you to stop after one minute".

Instruction to be repeated if the subject does not understand what is required.

Score:

Notes or Observations:

FINGER TAPPING TEST A

Testee's name:

Instruction: "Place both your elbows on the table (examiner models what is required) and touch each finger to your thumb in turn starting with your index finger (examiner can again model what is required). Practice that. When I say go, I would like you to do this as fast as you can until I tell you to stop. Be sure to touch each finger and do not go backwards. Are you ready? Go".

Score:

Preferred Hand:

Non-preferred Hand:

Notes or Observations:

X. SYFERS VERVANG DEUR SIMBOLE.
X. DIGIT SYMBOL SUBSTITUTION. – DELAYED

NAAM
NAME .....

Datum
Date .....

SLEUTEL
KEY

1	2	3	4	5	6	7	8	9

WMS Form II - Memory for Designs - IMMEDIATE RECALL

Testee's name:

Instruction: *Cards 1 and 2:* "I am going to show you a drawing. You will have just 10 seconds to look at it. Then, I shall take it away and let you draw it from memory. Don't begin to draw until I say go. Ready? Go".

Card 3: "Here is one that is a little harder. This card has two designs on it. I want you to look at both of them carefully - again you will have only 10 seconds to look at the card, then I shall take it away and let you draw both designs; the one on the left side - here (pointing to space in which subject is to make drawing) and the right one - here (pointing). Ready?"

Score

Card 1:

Card 2:

Card 3:

Notes or Observations:

Test 7

PICTURE COMPLETION

Directions

The test consists of 15 drawings, each of which has a part missing. The cards are presented in numerical order and the subject has to name or indicate the missing part in each.

Say: "I am going to show you some pictures, in each of which there is something missing. Look at each picture carefully and tell me the most important thing missing. Now, look at this picture" (presenting No. 1). "What important part is missing?"

If the correct answer is given, proceed with the test, saying in each case: "Now what is missing in this one?"

If the subject fails to detect the omission in No. 1,

Say: "You see, the nose is missing".

If he fails the second also, he is again helped, thus:

"You see, the pig's tail is missing here"

From the third picture onwards no further help is given. The examiner simply presents each card, asking what is missing.

Sometimes the subject mentions an inessential missing part. The first time this occurs, the examiner says:

"Yes, but what is the most important thing missing?"

A correct answer given within the time limit will be scored as correct. If this comment is repeated for any of the remaining presentations, the subject will not score except in the case of No. 13 (Mirror). Here, if the subject says that the hand is missing, say:

"Yes, and what else?"

"Hand" alone, or "Powderpuff" alone does not score.

If the subject mentions more than one missing part, ask which is the most important and score accordingly.

The time limit is 20 seconds for each picture. If the correct answer is not given within this time, score as a failure and pass on to the next picture.

N.B.: All times and responses are to be recorded.

Present all 15 cards. Use the timer in such a way that the subject realises that he is being timed, but do not make any remark to this effect. If the subject quickly gives an incorrect answer, wait in silence until the end of the 20 seconds; a spontaneous correction made within this period may be credited.

Test 7

PICTURE COMPLETION

Scoring

1 point for each picture for which a correct response is given within the time limit. No half-marks.

Maximum Score: 15

PICTURE COMPLETION
VOLTOOIING VAN PRENTE

RESPONSE/ANTWOORD

1		
2		
3		
4		
5		
6		
7		
8		
9		
10		
11		
12		
13		
14		
15		

SCORE
TELLING .....

GENERAL COMPREHENSION
ALGEMENE BEGRIP

RESPONSE/ANTWOORD

1		
2		
3		
4		
5		
6		
7		
8		
9		
10		

REMARKS
OPMERKINGS .....

.....

.....

.....

.....

Test 2

GENERAL COMPREHENSION

Questions

1. What is the thing to do if you find an envelope in the street that is sealed and addressed and has a new stamp on it?
2. What should you do if, while sitting in the cinema (bioscope, theatre) you are the first person to discover a fire (see smoke and fire)?
3. Why should we keep away from bad company?
4. Why should people pay taxes?
5. Why are shoes made of leather?
6. Why does land in a city cost more than land in the country?
7. Why must a motor vehicle be licensed before it may be used?
8. Why are laws necessary?
9. Why must a person who wishes to travel outside his own country obtain a passport?
10. Why are people who are born deaf usually unable to talk?

Test 2

GENERAL COMPREHENSION

Directions

Be sure that the subject is attending when you give the question. Young subjects and clinical patients sometimes find it difficult to remember the entire question from a single statement of it. It is therefore advisable to **repeat the question** if no response is obtained after 10 to 15 seconds, but **do not abbreviate or alter the wording.**

Say: "Now I am going to ask you some questions and I want you to tell me what you think in each case. There is no fixed answer. Just tell me what you think. Here is the first one ....."

Record the subject's responses verbatim. If the answer is very long-winded and he speaks rapidly, so that the whole of his statement cannot be noted, record the salient points, trying to preserve as much of the answer as possible.

It is sometimes necessary to encourage the subject. This may be done by means of such remarks as "Yes?". "Go ahead", etc. If a response is not clear, add "Please explain further" or "Can you explain to me a little more clearly?". Ask no questions which may indicate the type of answer required.

N.B.: Never pass on to the next question before making certain that the meaning of each answer is clear. Examiners are advised to keep the Guide to Marking before them while administering the test, particularly as specific answers requiring amplification are noted there.

e.g., Q.2 "Report it", "Report it to the manager".

Here the examiner must find out what object the subject has in mind and should grant full marks only if it is made clear that the management may be expected to take charge in order to prevent panic and see that the fire is dealt with.

It is important to **note down such explanations.** Do not merely state "Explained".

N.B.: If more than one answer is given, ask the subject which he considers most important and score on that basis.

Ask all the questions, except for subjects with very low intelligence.

Test 2

GENERAL COMPREHENSION

Scoring

In scoring this test 2, 1 or 0 marks are given, according to the generalisation and quality of the response. It is therefore re-emphasised that the examiner must **persevere in order to discover exactly what is meant where responses are not clear. This is particularly important in the case of simpler persons who express themselves badly,** or of those who answer obliquely, but who seem to have the correct principle in mind. Unless doubtful responses are investigated, difficulty will be experienced in allotting marks.

The accompanying guide to scoring gives the criteria for acceptable 2 and 1 scores, in addition to examples of which responses clearly fall into one or the other category and of those of a type which may leave the examiner in doubt as to where they fall.

Total Score: The sum of marks on the 10 questions

Maximum : 20

WMS Form II - Memory for Designs - DELAYED RECALL

Testee's name:

Instruction: "Earlier you memorised designs off cards presented to you for 10 seconds. I would like to see how many of those designs you can remember and draw now."

Score:

Notes or Observations:

WMS Form II - Associate Learning - IMMEDIATE RECALL

Testee's name:

Instruction: "I am going to read to you a list of words, two at a time. Listen carefully because when I am finished reading I shall expect you to remember the words that go together. For example, if the words were EAST-WEST; GOLD-SILVER, when I say the word EAST, you will answer (pause) WEST. And when I say the word GOLD, you would, of course, answer (pause) SILVER. Do you understand?

When subject is clear as to directions, continue as follows: "Now listen carefully to the list as I read it." Read first presentation at the rate of 1 pair every two seconds.

After reading the first presentation, allow 5 seconds and test by presenting first recall list. Give first word of pair and allow a maximum of 5 seconds for response. If subject gives correct reply, say, "That's right," and proceed with the next pair. If subject gives incorrect reply, say, "No," supply the correct association, and proceed with the following words.

After the first recall has been completed, allow a 10-second interval and give second presentation list proceeding as before. Repeat a second time, making THREE presentations and recall tests in all.

VII. ASSOCIATE LEARNING

First Presentation	Second Presentation	Third Presentation
Come - Go	Knife - Sharp	Country - France
Lead - Pencil	Jury - Eagle	Necktie - Cracker
In - Although	Country - France	Murder - Crime
Country - France	Lead - Pencil	Dig - Guilty
Dig - Guilty	Necktie - Cracker	Come - Go
Lock - Door	Murder - Crime	In - Although
Jury - Eagle	Lock - Door	Lock - Door
Murder - Crime	Come - Go	Jury - Eagle
Knife - Sharp	Dig - Guilty	Lead - Pencil
Necktie - Cracker	In - Although	Knife - Sharp

First Recall	Easy	Hard	Second Recall	Easy	Hard	Third Recall	Easy	Hard
Knife	____		Lock	____		Lead	____	
Lead	____		Dig		____	Lock	____	
Jury		____	Come	____		Necktie		____
Country	____		Jury		____	Come	____	
In		____	Knife	____		Dig		____
Murder	____		Country	____		Country	____	
Necktie		____	In		____	Jury		____
Lock	____		Murder	____		Knife	____	
Come	____		Necktie		____	In		____
Dig		____	Lead	____		Murder	____	
TOTAL	____	____	TOTAL	____	____	TOTAL	____	____

Easy 1)____
2)____
3)____
(A)Total____
A + 2____
Hard 1)____
2)____
3)____
(B)Total____

SCORE

$\frac{A}{2} + B =$ ____

DIGIT SPAN AND SUPRASPAN

1.DIGITS FORWARDS

" I am going to say some numbers. Listen carefully and when I have finished say them right after me."

They fail the test after the incorrect repitition of both trials of a span.
At this point the Digits Forwards test is complete and the score is the best span number achieved.
Thus if they fail both sets of 5 but passed one set of 4, their score is 4. If they get 10 digits forwards correct - then improvise until you have established their span- i.e. if they pass 10 digits forward- try 11 forward etc until they fail twice in a row.

2. DIGITS SUPRSPAN A:

After the second consecutive failure of a span on Digits Forward, the Digit Supraspan test follows.
"I will say that one again and see if you can get it this time".

The first repition of the previously failed span counts as learning trial 1 on this test. Continue to repeat this span until it's learnt correctly or has not been learnt by 9 trials. In other words, the lowest score they can get on the supraspan test is 1 and that's if they get it correct the very first time the span is repeated.

3. DIGIT SUPRASPAN B

After they have a Supraspan A score you get a Supraspan B score. This is the score for the amount of times it takes for them to get the supraspan correct TWICE IN A ROW.
"Let's see if you can get that right again"

If they have a supraspan A score of 4 trials and they are able to repeat the span on the 5th trial- they receive a supraspan B score of 5. If they get the 5th trial wrong- they would need to get the 6th and 7th trials correct to get a supraspan B score of 7. Continue until the 10th trial if necessary. If they are still unable to get the span correct twice in a row they receive a score of 10+.

4. DIGITS BACKWARDS

"I am going to say some more numbers. This time I want you to say them to me backwards. For example if I say 6-2-9, you must say.... (wait for them to say 9-2-6).
The test is failed after 2 consecutive failures of a span on DiGits forwards, and the score is the highest backwards span achieved.

DIGITS FORWARD	Score	DIGITS BACKWARDS	Score
6-4-3-9	4	2-8-3	3
7-2-8-6	4	4-1-5	3
4-2-7-3-1	5	3-2-7-9	4
7-5-8-3-6	5	4-9-6-8	4
6-1-9-4-7-3	6	1-5-2-8-6	5
3-9-2-4-8-7	6	6-1-8-4-3	5
5-9-1-7-4-2-3	7	5-2-9-4-1-8	6
4-1-7-9-3-8-6	7	7-2-4-8-5-6	6
5-8-1-9-2-6-4-7	8	8-1-2-9-3-6-5	7
3-8-2-9-5-1-7-4	8	4-7-3-9-1-2-8	7
7-5-8-3-6-3-2-7-9	9	4-7-2-6-9-1-5-8	8
4-2-7-3-1-8-1-2-6	9	7-4-5-1-2-9-8-3	8
6-1-9-4-7-3-5-2-9-4	10	2-8-4-1-7-9-5-4-6	9
4-7-3-9-1-2-8-3-2-7	10	8-6-9-3-5-7-1-4-2	9

DIGITSUPRASPAN LEARNING TRIAL 1 2 3 4 5 6 7 8 9
(Mark x when wrong, tick when correct)

1st Tick = SupraspanA

2nd Tick = SupraspanB

FINGER TAPPING TEST B

Testee's name:

Instruction: "I would now like to repeat the finger tapping test that we did earlier. To refresh your memory, place both your elbows on the table (examiner models what is required) and touch each finger to your thumb in turn starting with your index finger (examiner can again model what is required). When I say go, I would like you to do this as fast as you can until I tell you to stop. Be sure to touch each finger and do not go backwards. Are you ready? Go".

Score:

Preferred Hand:

Non-preferred Hand:

Notes or Observations:

WMS Form II - Associate Learning - DELAYED RECALL

Testee's name:

Instruction: "I read a list of pairs of words to you earlier. I would like to read the first words to you once more and see whether you can remember the word that goes together with the word I read out. For example, if I say EAST, you would answer (pause) WEST. Ready?"

VII. ASSOCIATE LEARNING

First Presentation	Second Presentation	Third Presentation
Come - Go	Knife - Sharp	Country - France
Lead - Pencil	Jury - Eagle	Necktie - Cracker
In - Although	Country - France	Murder - Crime
Country - France	Lead - Pencil	Dig - Guilty
Dig - Guilty	Necktie - Cracker	Come - Go
Lock - Door	Murder - Crime	In - Although
Jury - Eagle	Lock - Door	Lock - Door
Murder - Crime	Come - Go	Jury - Eagle
Knife - Sharp	Dig - Guilty	Lead - Pencil
Necktie - Cracker	In - Although	Knife - Sharp

First Recall	Easy	Hard	Second Recall	Easy	Hard	Third Recall	Easy	Hard
Knife	____		Lock	____		Lead	____	
Lead	____		Dig		____	Lock	____	
Jury		____	Come	____		Necktie		____
Country	____		Jury		____	Come	____	
In		____	Knife	____		Dig		____
Murder	____		Country	____		Country	____	
Necktie		____	In		____	Jury		____
Lock	____		Murder	____		Knife	____	
Come	____		Necktie		____	In		____
Dig		____	Lead	____		Murder	____	
TOTAL	____	____	TOTAL	____	____	TOTAL	____	____

Easy 1)____
2)____
3)____
(A)Total____
A + 2____
Hard 1)____
2)____
3)____
(B)Total____

SCORE
$\frac{A}{2}$ + B = ____

Score:

Notes or Observations:

Appendix F

Postconcussive Symptomology Questionnaire

RHODES UNIVERSITY PSYCHOLOGY DEPARTMENT

Symptom Check List

PLEASE ANSWER EACH OF THE FOLLOWING QUESTIONS BY INDICATING THE DEGREE TO WHICH THE QUESTION APPLIES TO YOU NOW.

NAME:_____

1. Do you suffer from headaches?	0 Never	0 Sometimes	0 Often
2. Do you have poor eyesight?	0 Never	0 Sometimes	0 Often
3. Do have difficulty hearing?	0 Never	0 Sometimes	0 Often
4. Do you experience weakness in your limbs?	0 Never	0 Sometimes	0 Often
5. Are you clumsy?	0 Never	0 Sometimes	0 Often
6. Do you have fits or seizures?	0 Never	0 Sometimes	0 Often
7. Do you become dizzy?	0 Never	0 Sometimes	0 Often
8. Do you become tired easily?	0 Never	0 Sometimes	0 Often
9. Are you very sensitive to noise?	0 Never	0 Sometimes	0 Often
10. Have you ever felt that you were seeing, hearing, or feeling unusual things?	0 Never	0 Sometimes	0 Often
11. Are you experiencing any sexual problems?	0 Never	0 Sometimes	0 Often
12. Do you having any problems with your speech?	0 Never	0 Sometimes	0 Often
13. Do you stumble over your words when you speak?	0 Never	0 Sometimes	0 Often
14. Do you stutter or stammer?	0 Never	0 Sometimes	0 Often

15. Do you slur your words? 0 Never 0 Sometimes 0 Often

16. Do you have memory difficulties? 0 Never 0 Sometimes 0 Often

17. Do you have problems with attention and concentration? 0 Never 0 Sometimes 0 Often

18. Does your attention wander while following a conversation

    or when you are watching TV or reading? 0 Never 0 Sometimes 0 Often

19. Are you impatient? 0 Never 0 Sometimes 0 Often

20. Are you irritable? 0 Never 0 Sometimes 0 Often

21. Do you become easily angry or hurt? 0 Never 0 Sometimes 0 Often

22. Do you feel sad or 'down in the dumps' or depressed? 0 Never 0 Sometimes 0 Often

23. Do you enjoy seeing your friends and having social contact?

    0 Never 0 Sometimes 0 Often

24. Do you suffer from restlessness? 0 Never 0 Sometimes 0 Often

25. Do you have problems sleeping? 0 Never 0 Sometimes 0 Often

26. Is there a problem with your appetite? 0 Never 0 Sometimes 0 Often

27. Do you feel nervous or anxious? 0 Never 0 Sometimes 0 Often

28. Do you feel worried or on edge? 0 Never 0 Sometimes 0 Often

29. Are you argumentative? 0 Never 0 Sometimes 0 Often

30. Do you feel short-tempered? 0 Never 0 Sometimes 0 Often

31. Do you become aggressive for no apparent reason? 0 Never 0 Sometimes 0 Often