

**RUGBY:
MORE THAN JUST A GAME**

**A STUDY OF THE CUMULATIVE EFFECTS
OF MILD HEAD INJURIES ON HIGH SCHOOL
RUGBY PLAYERS**

A thesis submitted in partial fulfilment of the
requirements for the degree of

MASTER OF ARTS

of

RHODES UNIVERSITY

by

CRISTINA GIAI-COLETTI

June 2002

ABSTRACT

The present study comprises part of an ongoing research study investigating the effects of cumulative mild head injuries on Rugby Union players. The aim of the study was to ascertain whether there are neuropsychological effects of cumulative mild head injuries sustained during the rugby-playing careers of senior schoolboy rugby players. Participants were top-level rugby players from high schools in Grahamstown and Cape Town ($n = 79$) and non-contact sport controls of top-level field hockey players from the same schools ($n = 58$). Group mean comparisons across a battery of neuropsychological tests were carried out between the Total Rugby versus the Total Field Hockey group, and the Rugby Forwards versus the Rugby Backs group. Comparisons between Total Rugby versus Total Field Hockey revealed impaired performance by the rugby players on two tests of visuoperceptual tracking, namely Digit Symbol Substitution and Trail Making Test (Part A). For Rugby Forwards versus Rugby Backs, there were no consistent differences to support the expectation that forwards would perform worse than backs. Forwards performed more poorly than backs on WMS Associate Learning Subtest – Hard (Delayed Recall), whereas backs performed more poorly than forwards on Digits Backwards. This suggests that some individuals in the cohort were starting to exhibit verbal memory deficit, albeit not clearly in association with forward positional play. Overall, results of the present study provide tentative support for the hypothesis that school level rugby players are more susceptible to the effects of cumulative concussive and sub-concussive head injuries than are non-contact sport controls.

ACKNOWLEDGEMENTS

To my supervisor, Professor Ann Edwards, for all your time and continued effort during the process of writing this thesis. Your supervision and constant guidance were greatly appreciated. Thankyou for assisting me with this final phase of my degree.

To Iain Reid for supervising me during the preliminary stages of my thesis. Thankyou for helping me embark on what initially appeared an enormous task, and for assisting me in maintaining perspective.

To Professor Sarah Radloff for the seemingly endless hours of assistance that you willingly poured into the statistical analyses of this research. Thankyou.

To Michael Border – a huge thankyou for being there for me right from the beginning and for never failing to support me when times were rough. You are wonderfully appreciated!

To Kathy and Kirsten who helped with the data collection for this research. A special thankyou for all the long hours spent while we collated and processed the data – you were superb!

To my family for their unceasing support and love, not only during the writing of this thesis, but throughout the entire span of my university career. A special thankyou to my mother, Sandra, who helped me persevere when things seemed unbearable – thankyou for helping to make this possible.

Brendon – your patience, love, support and continual presence were invaluable. Thankyou for helping me to *remember* my purpose amidst the work, for this gave me strength.

TABLE OF CONTENTS

Abstract	ii
Acknowledgements	iii
Table of Contents	iv
CHAPTER 1: INTRODUCTION	1
1.1 Research Context	1
1.2 Research Question	2
CHAPTER 2: LITERATURE REVIEW	4
2.1 Classification of Terms	4
2.1.1 Dominant Types of Head Trauma	4
2.1.2 Mild Head Injury Classification	5
2.1.3 Epidemiology of Mild Head Injury	7
2.1.4 Pathophysiology of Mild Head Injury	8
2.1.5 Mild Head Injury Across Various Age Groups	9
2.1.6 Section Summary	10
2.2 Neuropsychological Sequelae of Mild Head Injury	10
2.2.1 The Post-Concussive Syndrome (PCS)	11
2.2.2 Objective Neuropsychological Sequelae of Mild Head Injury	11
2.2.3 Neuropsychological Recovery Following Mild Head Injury	12
2.2.3.1 Recovery of function within three months post-injury	12
2.2.3.2 Persisting neuropsychological dysfunction	13
2.2.3.3 Neuropsychological sequelae of cumulative mild head injury	16
2.2.3.4 Commentary on neuropsychological dysfunction subsequent to mild head injury	18
2.2.3.5 Brain Reserve Capacity theory	20
2.2.4 Section Summary	21

2.3 Mild Head Injury in Contact Sport	22
2.3.1 Epidemiology	22
2.3.2 Boxing	24
2.3.3 Soccer	26
2.3.4 American Football	29
2.3.5 Australian Rules Football	30
2.3.6 Rugby League	31
2.3.7 Rugby Union	32
2.3.8 Section Summary	36
2.4 Research Hypotheses	36
CHAPTER 3: METHODOLOGY	38
3.1 Participants	38
3.1.1 Selection Criteria	38
3.1.2 Exclusion Criteria	39
3.1.3 Demographic Data	39
3.1.4 Mild Head Injury History	41
3.2 Procedure	42
3.3 Consent and Questionnaires	43
3.4 Neuropsychological Test Battery	43
3.4.1 General Intellectual Functioning	44
3.4.2 Visuoperceptual Tracking	45
3.4.3 Verbal Memory	46
3.4.4 Visual Memory	47
3.5 Data Processing	48
3.6 Statistical Analysis	48

CHAPTER 4: RESULTS	50
4.1 Comparison of Group Means Across all Neuropsychological Tests	50
4.1.1 Total Rugby Versus Total Field Hockey	50
4.1.2 Rugby Forwards Versus Rugby Backs	50
CHAPTER 5: DISCUSSION	53
5.1 Context of the Study	53
5.2 Revisiting the Aims, Methods and Hypotheses of the Study	54
5.3 Discussion of Results	57
5.3.1 Group Mean Comparisons for Total Rugby Versus Total Field Hockey	57
5.3.2 Group Mean Comparisons for Rugby Forwards Versus Rugby Backs	59
5.4 Conclusions and Theoretical Implications of the Research Findings	61
5.5 Evaluation of the Research Study	64
5.6 Recommendations for Future Research	66
REFERENCES	68
APPENDICES	
Appendix I: Letter of Consent	
Appendix II: Demographic Questionnaire	
Appendix III: Assessment Schedule and Neuropsychological Test Battery	

LIST OF TABLES

Table 2.1: Definition of Mild Traumatic Brain Injury	6
Table 3.1: Comparison of Demographic Data for Rugby and Field Hockey Players	40
Table 3.2: Comparison of Demographic Data for Rugby Forwards and Rugby Backs	41
Table 3.3: Comparison of the Incidence of Previous Reported Mild Head Injuries (both sport and non-sport related) in Rugby and Field Hockey Players	42
Table 3.4: Comparison of the Incidence of Previous Reported Mild Head Injuries (both sport and non-sport related) in Rugby Forwards and Rugby Backs	42
Table 4.1: Comparison of Means across Total Rugby and Total Field Hockey	51
Table 4.2: Comparison of Means across Rugby Forwards and Rugby Backs	52

CHAPTER ONE: INTRODUCTION

1.1 RESEARCH CONTEXT

The present study forms part of a larger and ongoing research study investigating the effects of cumulative mild head injuries on Rugby Union players. The study was initiated in 1996 by Rhodes University, the South African Rugby Football Union (SARFU) and the Sports Science Institute in Cape Town. Initially, *professional* rugby players were targeted, and the Springboks versus a comparison group of national cricket players were assessed using a wide neuropsychological test battery and a self-report measure of post-concussive symptoms. Results were analysed via direct comparisons of rugby versus cricket group mean scores (Ancer, 1999). However, it was decided that cricket controls were not an ideal non-contact sport group in that many of these individuals had played rugby as their winter sport. Therefore, a national field hockey group was assessed via the same procedures and was used as an alternative control group, and Springbok rugby versus field hockey group mean scores were compared (Finkelstein, 1999). In addition, the national Under 21 rugby squad was assessed via the same procedures and the test results of the two rugby cohorts (Springbok and Under 21) were combined to enlarge the sample size. This enlarged sample was then compared to the same group of national field hockey players (non-contact sport controls), also via direct comparisons of group mean scores (Finkelstein, 1999). Results of the above research consistently revealed poorer cognitive test performance by the rugby group compared to the control group on tests sensitive to the effects of diffuse brain damage, as well as impaired cognitive performance by the rugby forwards relative to the rugby backs on the same tests.

Next, an analysis at the *school level* was conducted using the same basic assessment procedure to compare the cognitive test performance and post-concussive symptoms of top-level schoolboy rugby players from rugby-playing schools in Cape Town to top-level schoolboy field hockey players (non-contact sport control group) from the same schools. In this study, as with the analyses for the professional players, rugby versus field hockey group mean scores were compared (Ackermann, 2000). Results provided much less support for the presence of cognitive deficit in a high school rugby-playing population than was evidenced for professional players (Ackermann, 2000). A review of all the Rhodes University studies on Rugby Union at both the

professional and school levels suggests that the effects of mild head injury are additive such that older players at the professional level show greater deficit than the younger players at school level (Shuttleworth-Edwards, Border, Reid & Radloff, in press). However, the apparent absence of effects at the school level requires further investigation to confirm this observation.

Thus, it was decided to initiate *a second phase at the school level* in order to enlarge the school sample, which is the aim of the present study. To maintain continuity within the ongoing research study, the present project utilised the same basic methodology as was used by Finkelstein (1999) and Ackermann (2000) to assess a cohort of schoolboy rugby players from a top rugby-playing school in Grahamstown, and a comparison group of field hockey players from the same school. Grahamstown rugby and field hockey samples were then combined with the Cape Town samples to create enlarged samples that produced the basis for the present study: rugby players were compared to field hockey players and rugby forwards were compared to backline players. Data collection and data analysis for this second phase were identical to those of the first phase, except that one-tailed as opposed to two-tailed t-tests were used to compare the group means in an effort to conduct more powerful statistical analyses.

1.2 RESEARCH QUESTION

The necessity of research into the effects of sports-related mild head injury becomes apparent in light of the high incidence of such injury at both the professional and school levels (Bennett & Raymond, 1997; Garraway, Lee, Hutton, Russell & Macleod, 2000). Additionally, mild head injury is often accompanied by residual cognitive symptomatology that may interfere with the cognitive skills necessary for academic success at school (Rimel, Giordani, Barth, Boll & Jane, 1981; Bernhardt, 2000). Furthermore, although much research has investigated the sequelae of single, uncomplicated mild head injury, there is a lack of consensus regarding the chronic effects of *cumulative* mild head injuries (Binder, Rohling & Larrabee, 1997; Satz, Zaucha, McCleary, Asarnow & Becker, 1997).

Players of contact sports are particularly exposed to mild head injuries (Wojtys et al., 1999). Research into the cumulative effects of mild head trauma in the contact sports

suggests that diffuse brain damage may be present amongst these players. This includes research on boxers, soccer players, American footballers, Australian Rules footballers, and Rugby League players (for example, Jordan, Matser, Zimmerman & Zazula, 1996; Matser, Kessels, Lezak & Troost, 2001; Barth, Alves, Ryan, Macciocchi, Rimel, Jane & Nelson, 1989; Maddocks & Saling, 1996; Hinton-Bayre, Geffen & McFarland, 1997). Research into the neuropsychological effects of cumulative mild head injuries in Rugby Union is sparse, with the only apparent research being that conducted at Rhodes University (Shuttleworth-Jordan, Balarin & Puchert, 1993; Shuttleworth-Edwards et al., in press). Furthermore, as most of the research on contact sport has focused on adult players, there is a particular paucity of research examining the effects of multiple concussive and sub-concussive blows at the school level, which may have implications for rugby-playing scholars attempting to write examinations. The present research study aims to address the lack of research on schoolboy players of Rugby Union.

Thus the basic question that is posed by this study is whether there are neuropsychological effects of cumulative mild head injuries sustained during the rugby-playing careers of top-level schoolboy Rugby Union players. As indicated above (see section 1.1), this question is addressed via direct group comparisons of the cognitive performances of top-level schoolboy rugby players and non-contact sport controls across a battery of neuropsychological tests shown to be sensitive to the effects of mild closed head injury. It is hypothesised that rugby players relative to field hockey players, and rugby forwards relative to rugby backs, will perform more poorly on certain neuropsychological tests that are particularly sensitive to the effects of diffuse brain damage. To elucidate the findings, results of the present research study are theoretically contextualised within Satz's (1993) theory of Brain Reserve Capacity.

CHAPTER TWO: LITERATURE REVIEW

2.1 CLASSIFICATION OF TERMS

This section will contextualise mild traumatic head injury within the greater range of head trauma in general. A definition of mild head injury will be provided, followed by a discussion of the epidemiology and pathophysiology of mild head injury, as well as of the typical symptoms with which it is associated.

2.1.1 Dominant Types of Head Trauma

Traumatic head injuries can be classified into two types: open and closed. An *open* head injury is commonly caused by gunshot or exploding shell fragments, and it results in tearing of the scalp, penetration or fracture of the skull, and focal laceration of brain tissue. Damage from an open head injury is usually localised along the path of the intruding object, resulting in relatively circumscribed and predictable cognitive losses, the severity of which depends upon the depth of penetration of the intruding object, as well as the amount of brain tissue damaged (Levin, Benton & Grossman, 1982; Lezak, 1995).

In contrast, a *closed* head injury is the result of blunt trauma to the head, causing linear or rotational acceleration of the brain (when a moving object collides with a stationary or slower moving head), or deceleration (when a moving head and body are impacted upon by a stationary or slower moving object). The damage caused by a closed head injury is primarily diffuse, where tearing or shearing of axons occurs. This is the primary mechanism of injury in closed head trauma, and it occurs at the moment of impact. However, localised effects are sometimes evidenced in closed head injuries, which are the result of coup (the point of impact) and contrecoup (the area opposite the point of impact) lesions, the latter resulting from the brain bouncing back after striking the opposite side of the skull. The severity of closed head injury ranges from mild to moderate to severe, depending on the amount of diffuse damage sustained (Levin et al., 1982; Selby, 2000). The focus of the present research study is on mild traumatic closed head injury.

2.1.2 Mild Head Injury Classification

The classification of mild head injury is a controversial and unresolved issue. According to Satz et al. (1997, p. 126):

This determination represents one of the most fundamental measurement and definition problems confronting researchers of head injury across the age span.

Uzzell (1999) maintains that the difficulties and scepticism in defining and classifying mild head injury have arisen out of initial definitions that were unclear and incomprehensible. Classification criteria are usually based on alterations in consciousness (Glasgow Coma Scale), duration of unconsciousness, changes in orientation and memory (length of posttraumatic amnesia), length of hospitalisation, and neurological status (Satz et al., 1997; Uzzell, 1999). Recently, any alteration in cerebral function, caused by rotational forces exerted onto the head, is referred to as mild traumatic brain injury or concussion (Baker & Patel, 2000; Wojtys et al., 1999). While loss of consciousness usually indicates more severe head injury, loss of consciousness is no longer seen as a prerequisite for classifying mild head injury (Lezak, 1995). Flanagan (1999) describes the definition for mild traumatic brain injury (MTBI) recently developed by the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine, asserting that it has the most specific criteria thus far (see table 2.1). Like prior definitions, this too includes an assessment of consciousness, alterations in mental status, neurological status, a GCS score, and the presence of posttraumatic amnesia. However, it only includes those individuals who sustain either a direct blow to the head or acceleration-deceleration brain injuries (excluding those who meet the criteria due to anoxia, stroke, infection, tumour or seizures), which differentiates this definition from other mild head injury definitions. Additionally, Flanagan (1999) asserts that this definition better distinguishes mild from more severe head trauma than do other definitions of its kind.

Table 2.1: Definition of Mild Traumatic Brain Injury as Developed by the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine

Definition A traumatically induced physiological disruption of brain function, as manifested by at least one of the following: 1. any period of loss of consciousness; 2. any loss of memory of events immediately before or after the accident; 3. any alteration in mental state at the time of the accident (e.g. feeling dazed, disorientated, or confused); and 4. focal neurological deficit(s) that may or may not be transient. but where the severity of the injury does not exceed the following: 1. loss of consciousness of 30 minutes; 2. after 30 minutes, a Glasgow Coma Score of 13-15; and 3. posttraumatic amnesia not greater than 24 hours. This definition includes: 1. the head being struck; 2. the head striking an object; and 3. the brain undergoing an acceleration-deceleration movement without external trauma to the head. This definition excludes stroke, anoxia, tumor, encephalitis, etc.
--

Source: Flanagan (1999)

The lack of consensus in defining mild head injury has proven problematic for research into this area. Evans (1992) postulates that strict criteria are essential to classify mild head injury and to eliminate the effect of confounding variables. He defines mild head injury as a closed head injury where the duration of loss of consciousness is 30 minutes or less, or a sense of being dazed without loss of consciousness; an initial GCS score of 13 to 15, without subsequent deterioration; an absence of focal neurologic deficits; and no evidence of depressed skull fracture, intracranial haematomas, or other neurosurgical pathology. Evan's defining criteria were adopted in the prior rugby head injury studies conducted with professional and school level players (Finkelstein, 1999; Ackermann, 2000), of which this study is a continuation. When comparing the above two definitions, it is evident that Evans' (1992) definition and that described more recently by Flanagan (1999) are identical with regard to the core features that comprise a definition of mild head injury, namely alterations in consciousness should yield a GCS score of 13 to 15, and duration of unconsciousness should be 30 minutes or less. Further, Evans' definition specifically

stipulates criteria for a milder range (i.e. a sense of being dazed with no loss of consciousness and an absence of neurologic deficits), and this form of mild injury is also indicated by the definition posed by Flanagan (1999). Hence, the critical features of Evans' definition are commensurate with current definitions of mild head injury as detailed by Flanagan (1999), and therefore the continued use of Evans' (1992) definition in the current rugby research study is considered appropriate. The important point for the current research purposes is that, according to these criteria, any individual with a GCS score of less than 13 to 15 and with loss of consciousness for longer than 30 minutes would be considered to have sustained a head injury of greater severity than mild, that is, falling outside the mild head injury category.

2.1.3 Epidemiology of Mild Head Injury

Studies on the incidence of mild head injury reveal that it constitutes a serious health concern. According to Kraus and Nourjah (1989) who conducted an epidemiological investigation on victims in California, USA, probably more than 70% of individuals who sustain traumatic head injuries have what would be classified as mild brain injury. However, according to the National Centre of Health Statistics, approximately 85% of all brain injuries are classified as mild (Bennett & Raymond, 1997). Other authors estimate that 2 million persons in the USA experience closed head injuries annually (Brown, Fann & Grant, 1994). In a more recent epidemiological examination of mild and moderate head injury in the USA (Sosin, Snizek & Thurman, 1996), it was discovered that an estimated 1.5 million non-institutionalised civilians sustain a non-fatal brain injury annually, a rate of 618 per 100 000 person-years. Furthermore, motor vehicles were involved in 28% of the brain injuries, and sports and physical activity were responsible for 20%. While motor vehicle related brain injuries tended to be the most severe, most of the mild head injuries were caused by sports and physical activity. In 1989, a study in Auckland, New Zealand revealed an annual incidence of mild head injury of 511 per 100 000 population (Wrightson, 1989). More recently, Wrightson and Gronwall (1998) carried out a study on the incidence of mild head injury in New Zealand, estimating the incidence to be 1769 per 1000 000 population per year for people aged 15 and over. For those under 15 years of age the incidence was put at 2920 per 1000 000 population per year. A prospective cohort study conducted in the United Kingdom revealed that an estimated 1400 young people and adults were still disabled one year after head injury, 90% of

whom had mild brain injuries. This corresponds to a rate of 154 per 100 000 population (Thornhill et al., 2000).

2.1.4 Pathophysiology of Mild Head Injury

Research suggests that a continuity of brain damage exists in closed head injuries, ranging from mild to severe, based on the idea of a spectrum of diffuse axonal injury (DAI) (Gennarelli, 1996; Satz et al., 1997). DAI, the proposed primary mechanism of damage and subsequent cognitive and behavioural difficulties in mild head injury, arises from rotational acceleration forces that produce changes in brain pathophysiology. Rotational acceleration/deceleration forces produce swirling movements of the brain within the rigid skull, resulting in rotational stresses that cause diffuse shearing, stretching or tearing of axons and small vessels, mainly within the central white matter of the hemispheres, the corpus callosum and the long tracts in the brain stem (the latter associated with loss of consciousness). Linear acceleration/deceleration forces usually result in minor focal injuries (coup and contrecoup), and no DAI, as these forces are not accompanied by rotation of the skull relative to the brain. Although loss of consciousness may accompany linear acceleration/deceleration, this is due to profound circulatory disturbances associated with this type of force, and not to high levels of inertial loading that produces DAI and the accompanying disturbances in consciousness (as in rotational acceleration). Both linear acceleration (translational impact) and rotational acceleration (arced impact) are usually present, either simultaneously or successively, in closed head injury, and it is difficult to disentangle the linear and rotary components after the event (Levin et al., 1982; Richardson, 1990). However, DAI can occur in situations involving a freely moving head, such as in whiplash injuries, and does not necessarily involve direct impact to the head (Richardson, 1990).

The presence of DAI after mild head injury has been supported by research. Strich (1956, 1969, cited in Binder, 1986) first described neuronal changes in humans who had died subsequent to sustaining mild head injuries. In 1968, Oppenheimer performed postmortem examinations of five patients who had sustained mild head injuries, but whom had died from other injuries. He discovered microscopic lesions in what appeared, in all other respects, to be normal brains. Blumbergs et al. (1994) documented the topography of axonal injury in five mild head injured patients whom

had died from other causes. By conducting immunostaining for the amyloid precursor protein (APP), which undergoes fast axonal transport, they were able to show multifocal axonal injury in all five patients.

2.1.5 Mild Head Injury Across Various Age Groups

According to Satz et al. (1997), the etiology and subsequent pathophysiology of closed head injury in children differs from that of adults. Traumatic cerebral insults in children are more likely the result of falls, whereas in adolescents and adults vehicular accidents are often a major cause of head injuries (with greater acceleration/deceleration forces to the head). The resulting trauma from falls (as in children) involves less rotational sheering than trauma incurred through vehicular accidents (as in adolescents and adults). Consequently, the effects of closed head injury may be different in children compared to adults. Additionally, diffuse brain swelling, which is more common in pediatric head injury (mild or severe, regardless of coma), may lead to different patterns and levels of neuropsychological or academic-psychosocial outcomes from that expected by adults (Snoek, Minderhoud & Wilmink, 1984). Where children do sustain closed head injuries that are qualitatively similar to those sustained by adolescents and adults (e.g. vehicular accidents), children may be more vulnerable to the subsequent effects of the injury, as children's brains are still in the process of development (Donders & Strom, 2000; Satz et al., 1997). As different cognitive skills have different developmental rates and ages of acquisition, it is possible that those skills that are undergoing rapid cognitive change at the time of the head injury are particularly vulnerable to negative effects due to a lowered critical threshold for impairment in the future (Satz, 2001). For these reasons, it is imperative to be aware of the developmental stage of participants in a target group, as the implications of findings on neuropsychological tests may differ according to this developmental stage. Additionally, test results may be different between adults and children of differing developmental ages (Oddy, 1993). Such an awareness is therefore important when considering and comparing research outcomes during the process of interpretation of research results.

A difficulty arises, however, in defining the dividing point between the age categories: children; adolescents; adults. In a recent study conducted by Donders and Strom (2000), the sample consisted of individuals aged 9-16, whom the authors

referred to as “children and adolescents” (p. 796). A study by Boll and Barth (1983) labelled the ages ranging from 16 upwards as “adults” (p.266). In 1999, Daniel et al. conducted a study on “adolescents” (p. 167) aged between 13 and 18, to examine improvement in cognitive functioning due to natural developmental processes (in the absence of head injury). The authors stated that a period of rapid cognitive change occurs at approximately 14-16 years of age. Their findings supported this phenomenon, revealing how the older subjects (17-18 years old) consistently performed better on all tests, possibly indicating that maturation had already occurred in this age group. On the basis of the afore-mentioned information, the age group of the participants in the present study (aged 15-19) is a bridging group. Incomplete cognitive maturation may be a phenomenon of the younger participants, whereas full cognitive development should have occurred for the older participants.

2.1.6 Section Summary

Mild head injury refers to a closed head trauma wherein there is brief or no loss of consciousness, where the posttraumatic amnesia is relatively short, and where there is an absence of focal neurologic deficit and skull fracture. The epidemiology of mild head injury is high, with the primary mechanism of injury involving diffuse axonal damage. The etiology and pathophysiology of mild head trauma differs between children, adolescents and adults. It is therefore important that the developmental stages of participants in a target group is known prior to interpretation of research results, as the implications of findings on neuropsychological tests may differ according to these developmental stages.

2.2 NEUROPSYCHOLOGICAL SEQUELAE OF MILD HEAD INJURY

That mild head injury may result in neuropsychological and behavioural dysfunction has been recognised for decades. Sir Charles Symmonds, in 1962, made the following remark, “It is, I think, questionable whether the effects of concussion, however slight, are ever completely reversible” (p. 4). It is not uncommon for mildly head injured patients to complain of a variety of cognitive, emotional, somatic, motor and sensory difficulties for a while after sustaining their head injury (Anderson, 1996; Cullum & Thompson, 1997). While the numerous signs and symptoms that are *subjectively* reported following mild head injury are referred to as the Post-Concussive Syndrome (PCS), various *objective* neuropsychological measures have

been devised to assess the cognitive functioning of individuals subsequent to mild head trauma (Bohnen & Jolles, 1992). The neuropsychological sequelae of *cumulative* mild head injury have also been assessed (Erlanger, Kutner, Barth & Barnes, 1999; Maroon et al., 2000), the effects of which can be conceptualised within Satz's (1993) theory of Brain Reserve Capacity.

2.2.1 The Post-Concussive Syndrome (PCS)

Although there is no consensus as to the definition of PCS (Binder, 1997), the most regularly occurring symptoms that constitute PCS are headaches, dizziness, irritability, anxiety, depression, blurred vision, insomnia, fatigue, noise sensitivity, attention and concentration difficulties, and memory problems (Binder, 1986; Evans, 1992; Lezak, 1995). While some PCS symptoms may disappear within a few days of the injury, the cognitive symptoms in particular may persist for weeks, months or years in a small number of individuals (Anderson, 1996).

PCS was initially viewed as psychological in origin as there was insufficient evidence to suggest an organic basis (Uzzell, 1999). The idea of secondary gain cast further doubt upon the existence of PCS (Cullum & Thompson, 1997). Bernhardt (2000) postulates a multifactorial etiology, involving both psychological as well as organic components. Some authors believe that it is inadequate to deny the existence of PCS on the basis of insufficient evidence and lack of instruments sensitive enough to detect it, as has been done in the past (Gronwall & Wrightson, 1975; Uzzell, 1999). A recent study of patients with persisting PCS symptoms utilised a functional imaging technique (evoked and event-related potentials) to determine changes in cognitive functioning subsequent to mild head injury (Gaetz & Weinberg, 2000). Results revealed evidence of electrophysiological changes in brain function, which the authors concluded indicates that PCS "has a substantial biological, as opposed to psychological, basis" (Gaetz & Weinberg, 2000, p. 815).

2.2.2 Objective Neuropsychological Sequelae of Mild Head Injury

Although the deleterious effects of mild head injury have been acknowledged since the 1800's (Erichsen, 1882; Walton, 1883), it was not until the Rimel et al. (1981) study that the morbidity associated with mild head injury was truly recognised. In spite of the numerous studies that have subsequently been conducted on mildly head

injured patients, the outcome following mild brain injury is not well established and the presence of cognitive-behavioural sequelae due to mild head trauma remains controversial (Barth et al., 1983; Bohnen & Jolles, 1992; Raskin, Mateer & Tweeten, 1998).

Research investigating the neuropsychological sequelae of mild head injury has shown that mild head injury results in measurable cognitive deficit. In *adults*, the following cognitive domains have shown to be predominantly affected subsequent to mild head injury: speed of information processing, memory, concentration, reaction time (Binder, 1986; Evans, 1992) and attention (Binder et al., 1997; Evans, 1992). A review of the literature on children and adolescents yields varied results. In *adolescents*, objective cognitive deficit has been identified in the areas of abstraction, reasoning, verbal memory (Spear Bassett & Slater, 1990) and learning (Leathem & Body, 1997; Spear Bassett & Slater, 1990), as well as in language, motor speed, somatosensation and visuospatial functioning (Levin & Eisenberg, 1979). In *children and adolescents*, memory and attention have often been found to be impaired in the mildly head injured (Anderson et al., 1997; Anderson, Catroppa, Morse & Haritou, 1999; Catroppa & Anderson, 1999; Ewing-Cobbs et al., 1998; Satz, 2001). Numerous researchers investigating mildly head injured children and adolescents have also revealed language impairments and frontal lobe dysfunction (Lord-Maes & Obrzut, 1996).

2.2.3 Neuropsychological Recovery Following Mild Head Injury

The process of recovery following mild head injury is controversial, particularly with regard to the presence and etiology of persisting neuropsychological dysfunction (Binder, 1986). It appears that the majority of mildly head injured patients may not suffer persisting symptomatology (Binder et al., 1997). However, evidence exists to support the fact that a minority of individuals are certainly affected (Anderson, 1996; Leininger, Gramling, Farrell, Kreutzer & Peck, 1990).

2.2.3.1 Recovery of function within three months post-injury: Neuropsychological studies of mildly head injured patients have demonstrated that recovery of cognitive functioning, although perhaps incomplete, occurs within a 5-10 day period immediately post-injury (Barth et al., 1989; Macciocchi, Barth, Alves, Rimel & Jane,

1996). Evidence of full recovery within one month also exists (McLean, Temkin, Dikmen & Wyler, 1983; Newcombe, Rabbitt & Briggs, 1994). The majority of studies, however, indicate that recovery of neuropsychological functioning can take as long as up to three months (Dikmen, McLean & Temkin, 1986; Gronwall & Wrightson, 1974; Hugenholtz, Stuss, Stethem & Richard, 1988; Levin et al., 1987).

The University of Colorado conducted an ongoing study (cited in Cullum & Thompson, 1997) exploring the presence of residual symptomatology subsequent to mild head injury, which compared patients with mild head trauma (with and without a previous mild head injury) and control patients who had also been involved in an accident but who had no injury to the head, neck or back. The patients were administered a brief neuropsychological battery of standard measures within one week of their accidents and then again two months later. Preliminary findings revealed no differences in performance at both intervals post-injury. However, this study is weakened by the small sample size, making it difficult to generalise the findings to the greater mild head injury population.

2.2.3.2 Persisting neuropsychological dysfunction: With regard to *duration* of cognitive deficit following mild head injury, the seminal study by Rimel et al. (1981) found persisting morbidity in 66% of 70 patients tested three months after sustaining a single mild head injury. This study was furthered by Barth et al. (1983) who observed measurable cognitive impairment three months after injury in a significant portion of the same cohort of patients, primarily involving attention-concentration, complex information processing, and memory. Although these studies suggest that neuropsychological deficit may persist beyond three months post-injury, they were poorly controlled for age and there was no inclusion of matched control groups.

More carefully controlled studies illustrate that some mildly head injured patients do in fact suffer from persisting symptomatology. Leininger and colleagues (1990) examined mildly head injured patients between one and 22 months post-injury. The patients were divided into two sub-groups (those who had lost consciousness and those who were only disorientated or confused), and both patients and uninjured controls were administered a neuropsychological test battery. Results revealed measurable cognitive deficits involving reasoning, information processing and verbal

learning, with no significant differences in neuropsychological functioning between patients tested within three months post-injury and those tested beyond three months, indicating the presence of residual cognitive dysfunction in this population. No differences were found between the two mild head injury sub-groups. In a later study, Bohnen, Jolles and Twijnstra (1992) compared symptomatic mild head injury patients to non-symptomatic mild head injury patients and to uninjured controls, six months post-injury. Findings demonstrated that symptomatic mild head trauma patients performed less well than non-symptomatic patients on tests of divided and selective attention, and information processing, again indicative of enduring neuropsychological debility consequent on mild head injury.

In a more recent study on the differentiation of long-term differences on testing, two groups of participants, 36 with a history of mild head injury and 36 without, were administered a neuropsychological battery incorporating tests known to be sensitive to the effects of mild head injury (Pinkston, Gouvier & Santa Maria, 2000). Overall, the results failed to show significant differences between groups for the differentiation of long-term mild head injury status. Although revealing, this study is limited by its lack of baseline measurement of functioning. A recent study aimed at determining the incidence of mild head injury in New Zealand, found that referrals made by general practitioners because of persisting symptoms occurred up to one year post-injury in 6% of the mild head injured population (Wrightson & Gronwall, 1998).

The controversy surrounding the duration of residual cognitive symptomatology is further fuelled by studies such as those conducted by Gronwall and Sampson (1974) and Ewing, McCarthy, Gronwall and Wrightson (1980). In the former study mildly head injured patients who had returned to normal functioning approximately six weeks post-injury, were tested again 3-4 years later. At ground level their cognitive performance was essentially normal, but when placed under an hypoxic state (3800 feet in a hyperbaric chamber) they displayed deficits in memory and mental vigilance. The same was evidenced in the Ewing et al. (1980) study where, 1-3 years post-injury, mildly head injured participants displayed deficit on vigilance and memory tests at a simulated altitude of 3800 metres. These deficits were not apparent in their test performance at ground level, nor were they evidenced by the control group. The

authors asserted that such decrement resembles the cognitive dysfunction seen immediately after concussion and in old people.

With regard to children, Gulbrandsen (1984) revealed cognitive deficit in schoolchildren (aged 9-13) tested at an average of six months post-injury. Other researchers observed that at one year post-injury, 6-18% of 51 children and adolescents who had sustained a mild closed head injury showed impairment on a range of neuropsychological tests (Winogron, Knights & Bawden, 1984). The study by Anderson et al. (1999) investigated functional memory skills in 44 pre-school children with varying degrees of injury severity (mild, moderate and severe) using the Rivermead Behavioural Memory Test for Children. Participants were evaluated acutely and at 12 months post-injury. Results demonstrated that during the acute phase of recovery (i.e. within three months post-injury) there was no clear dose-response relationship between injury severity and memory function. However, this relationship developed over time with mildly head injured children displaying functional memory problems at 12 months post-injury, although to a lesser extent than that displayed by the more severely injured children. Although utilising a fairly small sample size, this study was strengthened by its inclusion of healthy controls that were well matched with the participants with regard to age and socio-economic status.

Recovery of neuropsychological functioning following mild head injury is mediated by a number of factors (Binder, 1986). Prognostic variables that affect vulnerability to persisting morbidity following mild head injury include age, premorbid intellectual level, premorbid personality, gender (females tend to recover better), the effects of previous injuries, multiple mild head injuries, socio-economic status, alcohol abuse and the presence of the apolipoprotein E (APOE) genotype (Binder, 1986; Kutner, Erlanger, Tsai, Jordan & Relkin, 2000; Lishman, 1988; Selby, 2000; Wojtys et al., 1999). Often there is no correlation between severity of injury and persistence of symptoms, which is suggestive of a possible psychogenic etiology (Dikmen, Temkin, & Armsden, 1989). According to Cullum & Thompson (1997), psychological variables, secondary gain issues, or both, may affect prognosis following mild head trauma. These authors state, "the typical expectation following mild uncomplicated [traumatic brain injury] is recovery" (Cullum & Thompson, 1997, p. 12), attributing any subsequent deterioration of functioning in the absence of neurological

complications to a psychogenic basis. Such a tenacious view is just one amongst many in the ongoing controversy surrounding residual cognitive deficit consequent on mild head injury. However, studies such as the one conducted by Tellier et al. (1999) demonstrate the possible organic basis for persisting symptoms. These authors found evidence of intracranial abnormalities (via computed tomography scans) in 31% of 48 mildly head injured patients one year post-injury.

2.2.3.3 Neuropsychological sequelae of cumulative mild head injury: Outside of the sports arena, research investigating the residual cognitive deficit associated with *multiple* mild head injuries is sparse. Gronwall and Wrightson (1975) tested 20 young adults after a second concussion to determine the rate at which they processed information. These authors observed that the rate was reduced compared to controls who had been concussed only once, and that they took longer to recover than the controls, concluding that the effects of concussion seemed to be cumulative. The hypothesised explanation that they provided for their findings related to permanent neuronal damage that leaves the patient more vulnerable to further head injuries and that, with repeated mild head injury, eventually dips below threshold capacity, permanently affecting rate of information processing to a measurable extent. These results were corroborated by a study conducted by Gronwall (1989) who utilised the PASAT to assess speed of information processing in a cohort of mildly head injured patients. The author found that older patients (aged 40-49) and patients with a previous head injury were impaired in this regard and that they took longer to recover than those patients with a single episode of mild head injury.

A number of researchers within the sports arena have specifically focused on the neuropsychological effects of cumulative mild head injury. In a recent study by Collins et al. (1999), 393 male college football players were examined in an effort to assess the relationship between history of mild head injury and learning disabilities, and the association between these variables and neuropsychological performance. Of the sample, 129 players (34%) had a history of one previous mild head injury and 79 players (20%) had experienced two or more mild head injuries. The results of the study suggested that history of mild head injury and learning disability are independently related to lower baseline neuropsychological functioning. With particular reference to multiple mild head injuries, the authors observed that those

players with two or more prior mild brain injuries displayed significant long-term deficits in the domains of executive functioning and speed of information processing. These players also showed a tendency to report PCS symptoms. In order to understand their findings within a theoretical framework, the authors employed Satz's (1993) theory of Brain Reserve Capacity (to be described below) as it relates to learning disability and multiple mild head injuries affecting neuronal functioning and therefore cerebral threshold capacity.

In contrast to the above neuropsychological studies, all of which support deleterious effects of cumulative mild head injury, a study by Maddocks, Saling & Dicker (1995) demonstrated no residual cognitive deficit associated with repeated mild head injury in a cohort of mildly head injured Australian Rules footballers. However, this study was characterised by a number of methodological limitations. Firstly, only one neuropsychological test was employed (i.e. the Digit Symbol Substitution Subtest from the WAIS-R). Secondly, the control group comprised supposedly non-head injured footballers, which is problematic due to the high prevalence with which players of contact sport sustain sub-concussive head injuries that often go unnoticed (Baker & Patel, 2000; Bernhardt, 2000). Thirdly, the researchers relied on players accurately reporting their mild head injury histories when it is known that sports players often underreport mild head injuries (Anderson, 1996).

Finally, it is relevant to report on a neurological study conducted by Gaetz, Goodman and Weinberg (2000) on 271 junior hockey players. These authors investigated the cumulative effects of concussion via electrophysiological indices. The players were divided into four groups on the basis of prior concussion history: no previous concussion, one concussion, two concussions, and three or more concussions. Thereafter they were presented with visual stimuli in a variety of forms to which they had to respond verbally. All responses were recorded and event-related potential recordings were noted. The primary finding was that significantly longer P3 latencies (P3 is a cognitive event-related potential) were observed in players who had three or more concussions. This study is one of the first experimental demonstrations in humans that cumulative cognitive damage can result from multiple mild head injuries. The evidence therefore suggests that although some recovery of neuropsychological function may occur subsequent to sustaining a mild head insult (as is evidenced in

patients who return to work and/or school), some permanent residual neuronal damage remains, with the effects of repeated mild head injuries being additive.

2.2.3.4 Commentary on neuropsychological dysfunction subsequent to mild head injury:

Although much research supports the existence of neuropsychological sequelae following mild head injury, recent meta-analytic reviews have led to opposing opinions. In 1997, Binder et al. conducted a meta-analytic review of available prospective and quasi-prospective neuropsychological studies on residual deficits in mildly head injured *adults* (which was narrowed down to eight studies). Their data suggest that measures of attention may be the most sensitive indicators of debility associated with mild head trauma. With regard to persisting neuropsychological effects of mild brain injury, the meta-analysis provided support for a reportedly weak association between cognitive deficits and mild head injury, with no definite causation being revealed. On the basis of their estimates of persistent neuropsychological dysfunction, the authors assert that neuropsychological assessment is likely to have a negative predictive value of over 50%. They conclude that in cases of chronic debility after mild head trauma, clinicians are more likely to be correct when diagnosing no brain injury than when diagnosing brain injury. In his follow-up article, Binder (1997) provides alternative explanations for residual complaints after mild head trauma (including medical and psychiatric illness, malingering, and somatoform disorder), stating that, "Clearly, there is no robust evidence of permanent traumatic brain dysfunction, and alternative explanations must be considered" (Binder, 1997, p. 443).

Satz et al. (1997) conducted a comprehensive review of the literature on mild head injury in *children* (comprising 40 studies from 1970 to 1995). Their results revealed 13 adverse, 18 null, and 9 indeterminate findings related to neuropsychological, academic, or psychosocial outcome. With regard to the adverse outcomes, the authors stated that the effects were merely alterations in neuropsychological performance (at the more extreme tail of the mild injury distribution), and that they were subthreshold, transitory and small (across the entire spectrum of mild head injury). They proceeded to mention that such adverse outcomes should therefore be viewed with "cautious scepticism" (Satz et al., 1997, p. 121). The authors found more variability in neuropsychological outcome with increasing severity in the mild range. Satz et al.

(1997) concluded that the null hypothesis should be accepted as it relates to neuropsychological outcome in mild head injury in children.

In response to the Binder et al. (1997) and Satz et al. (1997) reviews, Shuttleworth-Jordan (1999) comments that the reviews focus heavily on empirical findings of the presence or absence of neuropsychological deficit, while paying little attention to the meaning of such results within a theoretical framework. While Binder et al. (1997) and Satz et al. (1997) fully acknowledge the lowering of brain reserve capacity (BRC) due to other factors, Shuttleworth-Jordan (1999) draws attention to the fact that these authors did not acknowledge that a mild head injury may constitute a new risk factor in itself. Shuttleworth-Jordan (1999) utilises Brain Reserve Capacity theory (as formulated by Satz, 1993, and described later under section 2.2.3.5) to explicate how a subclinical mild brain insult may destroy neurons, thereby diminishing cerebral reserve. It is therefore misleading to take null effects as a sign that full recovery has occurred. Furthermore, Shuttleworth-Jordan (1999) introduces the concept of variability of results as an indication of the presence of cognitive dysfunction, as variability invalidates the 'null' indications of average effects. She asserts that where there are *no* differences between group mean scores, significant differences in variability is an indication that some members of the cohort are displaying cognitive deficit. This was observed in research conducted on professional rugby players, in which significant variability was found between the rugby group and the control group (on tasks sensitive to diffuse brain damage) in the *absence* of significant differences in mean scores (Reid et al., 1999). This variability was taken as an indication of neuropsychological dysfunction amongst some of the rugby players that was being overlooked by the group mean comparisons. The research studies included in the Binder et al. (1997) and Satz et al. (1997) reviews concentrate on group mean comparisons, while ignoring significant variability between groups and the implications thereof for the future functioning of mildly head injured individuals. In this regard, Shuttleworth-Jordan (1999, p.25) concludes:

“... it is fallacious to suggest that null functional outcome at a particular point in time following mild head injury is equivalent to null brain injury, in that different test conditions, other superimposed neurological stressors, and merely the passage of time with respect to such asymptomatic findings might ultimately transfigure into quite the reverse.”

2.2.3.5 Brain Reserve Capacity theory: As indicated above by Shuttleworth-Jordan (1999) and Collins et al. (1999), the theory of Brain Reserve Capacity (BRC), as formally formulated by Satz (1993), is useful in conceptualising the neuropsychological dysfunction associated with cumulative mild head injury. According to Satz (1993), BRC is a hypothetical construct that refers to various protective and vulnerability factors that may modify the clinical expression of brain lesions, and which are therefore related to adaptive behaviour. BRC theory postulates that there is a threshold of brain damage that must be sustained before an individual will display functional impairment, with threshold being affected by factors such as aggregate lesions, disease progression, and challenge. Satz (1993) hypothesized that higher levels of BRC are associated with higher thresholds for exhibiting functional impairment, and that individual differences in brain reserve probably exist that may further alter symptom threshold. Therefore, brain insults of similar severity will more readily become apparent in individuals with lower cognitive reserve than those with higher BRC. Additionally, with cumulative brain insults over time, the risk of worsening functional impairment will be greater in those people with lower BRC than those with higher cognitive reserve. An assumption associated with the theory of BRC is that general intelligence and educational level are indirect, although imprecise, indexes of cognitive reserve. Other factors, namely neuronal attrition associated with aging, (Coffey, Saxton, Ratcliff, Bryan & Lucke, 1999; Mortimer & Pirozolo, 1985), gender (Zhang et al., 1990), brain size (Di Sclafani et al., 1998), history of prior head trauma (Rothweiler, Temkin & Dikmen, 1998), or a combination of these factors (Coffey et al., 1999; Fields, 1991) also affect brain reserve.

The value of BRC theory is saliently evidenced via the conceptualisation of individual variability in patterns of cerebral dysfunction often observed in Alzheimer's dementia (Alexander et al., 1997; Schmand, Smit, Geerlings & Lindeboom, 1997; Unverzagt, Hui, Farlow, Hall & Hendrie, 1998) and in persons infected with human immunodeficiency virus (HIV) (Basso & Bornstein, 2000). Such research provides evidence for BRC theory, showing how premorbid intellectual function and/or level of education may serve as protective or vulnerability factors in the development of functional morbidity associated with Alzheimer's dementia or with HIV infection, the principle of which can be extended to apply to the morbidity associated with cumulative mild head injury. Early mild head trauma may lead to loss of neurons that

is insufficient to manifest as functional impairment. However, with the accumulation of additional mild head injuries and gradual neuronal attrition associated with aging, a prior head injured person may dip below critical threshold, resulting in clinically diagnosable dementia. Satz (1993) illustrates this point by citing research on professional boxers showing that, "the aging process probably accelerated by the cumulative effects of relatively mild to severe head trauma over many years, would summate and lower the temporal threshold of dementia" (p. 286). Similarly, evidence exists that head injury functions as a risk factor in earlier onset Parkinson's disease (Quinn, Rossor & Marsden, 1986), as well as in symptom presentation associated with Alzheimer's Dementia (Mortimer & Pirozzolo, 1985) and multi-infarct dementia (Roth, 1986). The theory of BRC is therefore highly pertinent for the interpretation and conceptualisation of results obtained in mild head injury research, and especially sports head injury that is characterised by multiple blows to the head.

2.2.4 Section Summary

Mild closed head injury is often accompanied by subjectively reported signs and symptoms that are referred to as the Post-Concussive Syndrome (PCS), as well as deficits that are objectively measurable using neuropsychological procedures (Binder, 1986). Although these deficits tend to resolve within three months post-injury in the majority of mild head trauma patients, some patients (children and adults) tend to present with residual cognitive debility. Some research has shown that such disability is objectively observable up to three years post-injury in adults and one-year post-injury amongst children (Gronwall and Sampson, 1974; Winogron et al., 1984). Controversy exists, however, regarding the etiology of persisting symptomatology. The available studies suggest that there is a mixed psychogenic and organic etiology, with factors such as premorbid personality and intellectual level, age, effect of multiple or previous injuries, and APOE genotype affecting prognosis subsequent to mild closed head trauma (Binder, 1986; Kutner et al., 2000; Lishman, 1988; Selby, 2000; Wojtys et al., 1999). Researchers investigating permanent debility consequent on cumulative mild head injury have revealed objective cognitive deficit in the domains of information processing speed and executive functioning, and that residual deficit is measurable using electrophysiological indices (Collins et al., 1999; Gaetz et al., 2000; Gronwall, 1989; Gronwall & Wrightson, 1975). In the face of research that supports the existence of residual cognitive symptomatology post-mild head injury,

recent literature reviews of research conducted on children and adults maintain that public health concerns regarding the long-term effects of mild head trauma are unfounded as there is “no robust evidence of permanent traumatic brain dysfunction” associated with mild head injury (Binder, 1997, p. 443; Binder et al., 1997; Satz et al., 1997). However, the counterargument asserts that variability in the test results of mildly head injured patients must be considered within Satz’s (1993) theoretical framework of Brain Reserve Capacity, so as to avoid dismissing the potentially hazardous effects of such injury on cerebral reserve capacity (Shuttleworth-Jordan, 1999).

2.3 MILD HEAD INJURY IN CONTACT SPORT

Although few sports do not carry some risk of mild brain injury, the greatest risk is in contact sports due to the very nature of these games, where blows to the head may be expected or even intentional (Anderson, 1996). Although previously considered benign, recent studies suggest that sports-related mild head injury is a serious public health problem (Collins et al., 1999; Matser, Kessels, Lezak, Jordan & Troost, 1999). The following section will examine the incidence of mild head trauma in contact sports. Neuropsychological studies of mild head injury in boxing, soccer, Australian Rules Football, and rugby, the latter being the focus of this research, will then be presented.

2.3.1 Epidemiology

A relatively recent statement by the Centers for Disease Control and Prevention (1997) proclaimed that athletic mild head injuries in the United States have reached epidemic proportions. Nevertheless, researchers still have difficulty assessing the prevalence of such injuries as most cases go unreported (Anderson, 1996). Besides many mild head injuries being unnoticed due to their supposedly mild nature, athletes often underreport mild head trauma for fear of letting down the team, being seen as weak, risking losing playing time during a game due to medical disqualification, and/or reducing their probability of playing in the next game (Baker & Patel, 2000; Bernhardt, 2000; Ruchinskas, Francis & Barth, 1997).

Epidemiological research that has been conducted on sports-related mild head injuries reveals that mild head injury occurs in all of the major sports, including American

football, Australian Rules football, boxing, soccer and rugby, amongst others (Baker & Patel, 2000; Erlanger et al., 1999; Maddocks et al., 1995), with an estimated incidence of 300 000 mild head injury cases per year in the United States (Centers for Disease Control and Prevention, 1997). Additionally, mild closed head injury is one of the most frequently reported sporting injuries. It has been found that the sport associated with the highest incidence of such injury in the United States is American football (Bailes & Cantu, 2001). With 1.5 million annual participants, various estimates suggest that 4-20% of these players sustain a mild head insult each season (Collins et al., 1999). A study conducted at the high school level indicated that 40 000 per 1.1 million high school football players sustain a mild head injury per season in the United States (Powell & Barber-Foss, 1999). Research on professional Australian Rules football players found that 25% of all injuries were to the head and neck, and that 5% of all injuries were concussions (Maddocks et al., 1995). According to Ruchinskas et al. (1997), the rate of mild head trauma in boxing ranges from 1-70%, in soccer from 4-22%, and in rugby from 2-25%. In soccer, the basic incidence is 0.96 concussions per team per season, with the overall incidence being 0.6 per 1 000 athlete exposures for men, and 0.4 for women (Boden, Kirkendall & Garrett, 1998). Of significance is that concussion rates per 1000 athlete exposures in American football and men's soccer are equal (Kelly & Rosenberg, 1997).

An epidemiological study on Rugby Union revealed that in rugby, the injury rate varies according to gender and position (Bird, et al., 1998). While females exhibited a rate of 6.1 injuries per 100 player games, males had a higher rate at 10.9 injuries per 100 player games. Positional variation primarily highlighted the differences between forward and backline players, with forwards sustaining more injuries to the head/face and neck, and backs suffering more lower body injuries. Furthermore, results showed that injury rates for games were higher than that for practices. These findings are similar to those of an earlier study that found the wing forward to be the player with the highest rate of injuries, and that head injuries accounted for almost one quarter of all injuries sustained (24%) (Ryan & McQuillan, 1992). According to Bird et al. (1998), the tackle was the phase of play in which most of the concussions occurred (64%). Rucks and mauls were responsible for the remainder of all injury types. A recent epidemiological study found that the introduction of professionalism into Rugby Union has coincided with a marked increase in injuries (27% to 47%) to both

professional and amateur players, with the rise being particularly high in teenage players (20% to 41%) (Garraway et al., 2000). The authors attribute this increase to recent changes in the laws of Rugby Union that encourage more open play, which has probably resulted in more tackles involving a higher degree of momentum or the use of greater force. At the schoolboy level, the incidence of concussion in Rugby Union is reportedly 20% (Roux, Goedecke, Visser, van Zyl & Noakes, 1987), with mild head injuries being reported as the single most common injury sustained (Nathan, Goedecke & Noakes, 1983).

2.3.2 Boxing

Boxing has been a focus of research into sports-related head injury since 1928, as the aim in boxing is to render one's opponent unconscious or helpless through successive blows to the head (Barth et al., 1989; Martland, 1928). Phrases such as "punch drunk", "chronic boxers' encephalopathy" and "Dementia Pugilistica" (DP) have been coined to refer to the diffuse cerebral atrophy that often occurs in professional boxers after retirement (Erlanger et al., 1999; Jordan, 2000). Such chronic traumatic brain injury represents the additive effect of multiple concussive and sub-concussive blows to the head, rendering boxing suitable for research into the cumulative effects of mild head injury (Bailes & Cantu, 2001).

Neuropsychological testing of *professional* boxers has revealed the presence of cognitive deficit associated with head injury in general (Haglund & Eriksson, 1993). Cognitive deficiency has been detected in the areas of attention, concentration, immediate and delayed memory, new learning, sequencing abilities, and speed of information processing (Heilbronner, Henry & Carson-Brewer, 1991).

Other studies conducted on professional boxers have suggested a strong relationship between length of career/number of bouts and measurable cognitive dysfunction, a finding that indicates how neuropsychological functioning is detrimentally affected with successive blows to the head (Bailes & Cantu, 2001). Casson, Sham, Campbell, Tarlau and Didomenico (1982) studied 15 former and active professionals, and three amateurs via neurological examination, EEG, CT, and neuropsychological testing. Of the 15 professionals, 13 had abnormal assessments in at least two measures, and all the amateurs were normal except for mild impairments on one pair of

neuropsychological tests – immediate and delayed verbal memory. Of particular significance was the finding of a direct relationship between length of professional boxing career and the presence and severity of neuropsychological damage. Similar findings were replicated by Jordan et al. (1996) who discovered, via neuropsychological testing of professional boxers, that deficit in attention, concentration and memory was related to various indices of increased sparring exposure, during which most traumatic head injuries occur (not restricted to mild head injuries). Although research suggests that less cognitive damage occurs in amateur boxers (Jordan, 2000), the most extensive study on mild head injury in amateur boxing, which took place over four years, revealed a trend between memory, visuoconstructional ability, and perceptual-motor functions and number of bouts sustained pre-baseline testing (Stewart et al., 1994). The collection of these findings provides evidence for the cumulative effect of multiple head injuries, as the trends evident in research on amateur boxers develop into statistically significant findings at the professional level.

In contrast, results on *amateur* boxers is in dispute (Lezak, 1995). In 1994, Butler reviewed 10 studies that assessed 289 amateur boxers, finding that amateur boxers did not exhibit any significant signs of neuropsychological dysfunction. However, the suggestion was made that a long amateur career may degrade fine motor movements. This finding is consistent with that observed in the Heilbronner et al. (1991) study, which found that boxers with more extensive fight histories had slower dominant and non-dominant hand tapping speed. In the study by Brooks, Kupshik, Galbraith & Ward (1987), 29 active amateur boxers were administered neuropsychological tests, the results of which were compared to those of 19 controls matched for age, ethnicity and educational level. Findings revealed no consistent pattern of deficit (although three players did show significance at the $p < 0.05$ level), and no relationship between cognitive functioning and duration of amateur career. The authors concluded that their study provided no evidence that cognitive abnormalities result from well-controlled amateur boxing. However, the effectiveness of this study was weakened by the lack of baseline measurement, which, according to Jordan (2000), is methodologically essential in research into boxing. Furthermore, no theoretical context was utilised to understand the findings, which resulted in the three significant scores being dismissed on an empirical basis. Butler, Forsythe, Beverly and Adams

(1993) included baseline testing, yet found no significant cognitive deficit at post-fight testing, nor after six-month to two-year follow-up, and no correlation to number of bouts. Besides utilising a relatively small sample size ($n = 86$), the follow-up period may not have been long enough.

Cognitive deficits that have been elucidated by research into amateur boxing have been found in the areas of verbal memory and verbal new learning (Heilbronner et al., 1991; McLatchie et al., 1987), visual memory, visuo-spatial ability, complex reaction time (McLatchie et al., 1987), and perceptual and motor ability (Stewart et al., 1994). A recent study (Ramirez, 1998) examined the effects on attention of repeated mild closed head injuries in amateur boxing. The participants were administered the Test of Variable Attention (TOVA) one hour after the completion of the boxing tournament and again eight weeks later. Results were compared with those of amateur basketball players who were assessed while their tournament was in progress, and matched for age, grade point average, and socio-economic status. Results showed that variability of attention was the only significant mean result in the boxing group, and that the boxers' reaction time was significantly slower than that of the control group, both at the end of the tournament and eight weeks later.

2.3.3 Soccer

Soccer, the most frequently played sport in the world (Statistics on Fifa, 1982), is generally regarded as safe by the general public. However, soccer has been formally classified as a contact sport by the American Academy of Pediatrics (1988). Research into the chronic neuropsychological deficits associated with mild head injury in soccer generally distinguishes between professional and amateur players.

The first research study to have found significant cognitive deficit in *professional* soccer players utilised an extensive neuropsychological test battery, and compared 37 retired players (mean age 48.6 years) to matched (for age and education) hospitalised patients with no history of head or neck injuries, or evidence of brain damage (Tysvaer & Lochen, 1991). The findings revealed that 81% of soccer players had some degree of cognitive deficit, compared with 40% of the controls. Furthermore, 49% of soccer players had moderate to gross impairment in attention, concentration, memory and judgement, while none of the controls were more than mildly impaired.

These findings were supported by the Witol and Webbe (1993) study that found soccer players (predominantly the older age group, and those who headed the ball frequently or who had a history of frequent heading) to exhibit deficits in attention, concentration, cognitive flexibility and general intellectual functioning. In 1998, Matser, Kessels, Jordan, Lezak and Troost conducted a study to determine the presence of chronic traumatic brain injury in professional soccer players, finding these players to exhibit impairments in memory, planning and visuoperceptual tracking, when compared with non-contact sport controls. Moreover, their findings revealed the effect of a dose-response, where cognitive impairment was inversely related to the number of concussions in soccer and the frequency of heading the ball. Furthermore, neuropsychological test performance also varied according to field position, with greater impairment being exhibited by forward and defensive players, who were more likely to experience soccer-related concussions and to head the ball.

In a more recent study, a group of these authors conducted further research on 84 Dutch premier league professional soccer players to identify the specific contribution of soccer-related concussions and headers to neurocognitive dysfunction in this population (Matser et al., 2001). Participants underwent extensive neuropsychological evaluation, and the dose-response relation between number of headers in one professional season and the number of soccer-related concussions on cognitive functioning was investigated. The test battery included measures of abstract reasoning, executive functioning, attention and speed of information processing, memory, visuoperceptual functioning, language, and fine motor behaviour. Results revealed an inverse relationship between number of headers and verbal and visual memory and focused attention, as well as an inverse relationship between number of concussions and visuoperceptual processing and sustained attention – as the number of headers and concussions increased, so cognitive functioning worsened. The authors concluded that, amongst professional soccer players, a dose-response relation exists between neuropsychological impairment and number of headers and concussions, with headers and concussions separately contributing to cognitive dysfunction. Although the study lacked a matched control group, the validity of results was strengthened by the big sample size and by the manner in which the study was well controlled for a number of variables (e.g. level of education, substance abuse, number of non-sports related concussions, and medical and psychiatric

conditions). The indication from the above research is that the effects of chronic mild head injury are additive, with measurable deficit becoming apparent with increased exposure to concussion. However, contradictory findings were revealed by a neurological examination that found no differences between soccer players and track athletes on MRI (Jordan, Green, Galanty, Mandelbaum & Jabour, 1996). The reason for this discrepancy may be due to methodologic issues, as this study did not utilise neuropsychological techniques known to be sensitive to mild head injury (Lezak, 1995).

In a retrospective neuropsychological study aimed at assessing the attention and concentration of *amateur* soccer players (Abreau, Templer, Schuyler & Hutchison, 1990), the authors found a significant negative correlation between number of games played and performance on the PASAT, yet no measurable cognitive deficit on neuropsychological tests in general. The researchers concluded that their findings neither warrant a clean bill of neuropsychological health for soccer players nor indicate any serious harm to the brain. According to the authors, their findings suggest that soccer may in fact cause minor brain damage or dysfunction. In a more recent, well-controlled cross-sectional study (Matser et al., 1999), 33 amateur soccer players were compared to non-contact sport controls (swimmers and track athletes) on the basis of neuropsychological test performance. Compared with controls, amateur soccer players demonstrated mild neurocognitive impairment on tests of planning (39% vs 13%) and memory (27% vs 7%). Additionally, cognitive performance on six of the neuropsychological tests was inversely related to the number of concussions incurred in soccer. From this study it is evident how, at the level of amateur soccer, the dose-response relation between mild head injury and cognitive impairment that is clearly evident in professional players, is already evident to a measurable degree. The indication is that mild neuropsychological impairment that is evident in amateur soccer players may be the forerunner to more pronounced impairment at the professional level, as a result of the cumulative effect of successive mild head injuries sustained during their careers.

2.3.4 American Football

Kutner and colleagues (2000) conducted a study on *professional* American football players to determine whether the cognitive status of these players varies as a function of age and apolipoprotein E (APOE) genotype (with age being an indication of the extent of exposure to football related head injuries). Players completed a computerised neuropsychological test battery that assessed attention, memory, spatial processing, reasoning, and reaction time. Results revealed how older players who possessed APOE genotype exhibited lower than expected cognitive performance relative to all other players studied, including comparably experienced players without APOE genotype and younger APOE genotype carriers. Memory was the cognitive domain most severely affected. While the authors admitted that the findings of their exploratory study revealed only modest support for their hypothesis, and that more research is needed on APOE genotype-exposure interactions amongst this population, they concluded that their study provided preliminary evidence that the cognitive functioning of professional American footballers with repeated exposure to head injury may be affected by age, by the presence of APOE genotype, and by cumulative exposure to contact.

In 1989, Barth et al. carried out a four-year prospective study that determined the neuropsychological deficits of injured *college* American footballers. The neuropsychological test battery was comprised of the Trail Making Test (Parts A and B), the PASAT, and the Symbol Digit Test, which were administered to 2350 players, as well as to a control group of 59 orthopaedic patients and 48 male college students. Testing was conducted at 24 hours post-injury, at five days and ten days post-injury, and at post-season, thereby allowing the researchers to establish a recovery curve. Results revealed that measurable deficits were predominant at 24 hours post-injury in the domains of attention, concentration and information processing, and that recovery took place over the next five to ten days. The authors concluded that a *single* uncomplicated mild head injury shows rapid resolution of symptoms.

The Collins et al. (1999) study (described in section 2.2.3.3) that researched the effects of *multiple* mild head injuries on college football players demonstrated how those players with two or more prior mild head injuries displayed significant long-term deficits in the domains of executive functioning and speed of information

processing. This research on mild head trauma amongst American footballers demonstrates that while recovery from one uncomplicated mild brain injury may be rapid, the effects of multiple mild head injuries are additive, with the presence of the APOE genotype creating increased vulnerability to permanent deficit.

2.3.5 Australian Rules Football

The players of Australian Rules football run a high risk of sustaining mild head trauma, as they are often involved in interceptions and tackles while the oval ball is either kicked or punched around the field, frequently causing collisions between players (Cremona-Meteyard & Geffen, 1994). A neuropsychological study investigating whether the effects of such trauma are detectable utilised the PASAT, the Digit Symbol Substitution Test and Four-Choice Reaction time to obtain baseline measures (Maddocks & Saling, 1996). The sample consisted of 130 *professional* players, 10 of whom sustained a mild head injury and were subsequently re-assessed at five days post-injury (along with members of the control group). Results suggested that persisting neuropsychological changes in information processing and decision time are detectable at five days post-injury. Although the study focused solely on the effects of a single mild head injury, which precludes any comment on the neuropsychological sequelae of multiple mild head injuries, the results are still valuable in demonstrating the deleterious effects of a mild head injury sustained as a result of contact sport.

In an earlier research project investigating the acute and long-term effects of concussion on professional Australian Rules footballers, players who had sustained a mild head injury were assessed using cued reaction time tasks, at two weeks and one year post-injury (Cremona-Meteyard and Geffen, 1994). A control group of elite sportsmen with no history of concussion were used as the control group, and potentially confounding variables were addressed and managed. Although there were no test differences between players and controls during the acute phase, at one-year post-injury the mild head injury group showed a reduced benefit of valid cueing relative to controls. The authors concluded that their research provides convincing evidence of persistent impairment in the ability to direct visuospatial attention following mild head trauma in Australian Rules footballers. The implication is a reduced ability to act quickly in response to expected spatial events (such as a ball

looming towards a player), which puts players at risk of further (and hence multiple) head injury.

2.3.6 Rugby League

The game of Rugby League is extremely physical where players are required to have speed, stamina, strength and agility. Since the ball can only be carried or kicked down the field, players often collide into each other or tackle each other (the latter being an intrinsic part of the game), which result in players being repeatedly exposed to head injury (Gibbs, 1993).

In 1999, Hinton-Bayre, Geffen, Geffen, McFarland and Friis conducted a follow-up study on their 1997 report on the sensitivity of tests of speed of information processing to impairment following mild head injury in *professional* Rugby League players. They aimed at taking practice effects into account and at assessing individual variations following concussion. To achieve the latter, they employed a Reliable Change Index (RCI), which was an impairment criterion of decline. The following measures formed their neuropsychological test battery: Speed of Comprehension, Digit Symbol Substitution, and Symbol Digit Modalities Test. Players were assessed pre-season, and up to three times post-injury: 1-3 days, 1-2 weeks, and 3-5 weeks. Matched controls were also re-assessed at similar intervals towards the end of the season. Group analyses of the data revealed significant decline in players' performance on each test 1-3 days post-injury, but that functioning had returned to baseline within 1-2 weeks post-injury. None of the controls were impaired. After conducting individual analyses, which incorporated use of the RCI, it became apparent that using only one test to measure impairment was insufficient, as it failed to detect up to more than half of the concussed players at 1-3 days post-injury. Furthermore, using the RCI on any one of the three tests improved the test's sensitivity to concussion. Results of the supposedly most sensitive test (Speed of Comprehension) revealed impairment in 11/20 concussed players at 1-3 days post-injury. However, when all three tests were considered, 16/20 players showed deficit on at least one of the three tests when re-assessed 1-3 days post-injury. When the RCI was employed, 7/20 players were still found to be impaired 1-2 weeks post-injury, particularly on Speed of Comprehension, but returned to baseline levels at 3-5 weeks post-injury. This study was limited by a small sample size, by varying amounts of

baseline and post-injury data across players, and by an impairment criterion also based on a small sample. However, it reveals how, when utilising tests sensitive to the effects of mild head trauma, residual symptoms become apparent, especially in those individuals who are exposed to multiple mild head injuries, such as in the game of Rugby League.

2.3.7 Rugby Union

Rugby Union is a full-body contact sport with many injuries resulting from extrinsic forces, including impact and collision at speed. Mild closed head injury, accompanied by joint dislocations and fractures, are the more serious injuries sustained in rugby (Bird et al., 1998). Nonetheless, research into the neuropsychological sequelae of cumulative mild head injury in Rugby Union is sparse.

The first study to examine the effects of mild head injury in Rugby Union appears to have been that conducted by Shuttleworth-Jordan et al. (1993) in South Africa. Although restricted by a small number of concussed players, this study managed to overcome some of the methodological weaknesses of other studies by employing a more extensive test battery, by recording a recovery curve up to three months, as well as by exploring the effects of previous concussive or unreported sub-concussive head injuries. Shuttleworth-Jordan et al. (1993) compared 60 *university* rugby players to non-contact sport controls on measures of hand-motor dexterity (Denckla Finger Tapping and Purdue Pegboard), short-term verbal memory (Digit Span Forwards), verbal new learning (Digit Supraspan Test) and working memory (Trail Making Test Parts A & B and Digit Span Backwards). Baseline premorbid data was collected at pre-season to allow for two levels of analysis: (i) pre- and post-season differences between non-head injured players and matched controls, and (ii) test differences between players with a reported mild head injury sustained during the season and matched controls at pre-season, five days, one month, two months, and three months post-season.

The pre-season comparison between non-concussed players and controls revealed that the non-head injured rugby players revealed a pattern of deficit similar to that seen in diffuse brain damage, with deficits in verbal new learning, working memory, and

hand-motor dexterity. The authors attributed these results to probable permanent deficits in a rugby-playing population. Further indicative of diffuse brain damage, the rugby players showed less capacity than the controls for practice effects between pre- and post-season testing. Positional variation was also noted, with forward players exhibiting greater impairment than backline players. This finding was associated with the nature of play in the scrum that is likely to expose forward players to a high potential for cumulative mild head injury.

Five of the 60 rugby players reported sustaining a mild head insult during the rugby season. Relative to controls, these players displayed significant dysfunction in the areas of verbal new learning, working memory, attention, and hand-motor dexterity at three days post-injury. Although considerable recovery was evident at one month and further recovery at two months post-injury, at three months post-injury the mild head injury group was still not demonstrating a practice effect to the same degree as the controls on Digits Backwards, Digit Difference, Digit Supraspan, and Finger Tapping. The indication was that full recovery had not yet been reached. Furthermore, the pattern of deficit among the head injured group was highly comparable to that recorded among the non-head injured group.

The Shuttleworth-Jordan et al. (1993) research provided the thrust that initiated an ongoing study of the neuropsychological effects of cumulative mild head injuries on Rugby Union players. Initially, *professional* rugby players (the Springboks) and a comparison group of national cricket players were individually assessed using a wide neuropsychological test battery and a self-report measure of post-concussive symptoms. Results were analysed via direct comparisons of rugby versus cricket group mean scores (Ancer, 1999). National field hockey players were then assessed via the same assessment procedures and used as an alternative control group to the cricket players, and Springbok rugby versus field hockey group mean scores were compared (Finkelstein, 1999). Additionally, the national rugby sample was enlarged to incorporate the national Under 21 rugby squad, and results of a wide neuropsychological test battery of the combined rugby sample (Springboks and Under 21 players) were compared to those of the national field hockey sample via direct comparisons of rugby versus field hockey group mean scores (Finkelstein, 1999).

At the professional level, results of visuoperceptual tracking tasks were consistently low, with the rugby group demonstrating worse cognitive test performance than the field hockey group. The unstructured verbal fluency task also revealed significant deficit amongst the rugby players relative to the field hockey players, whereas the hand-motor dexterity task showed better performance by the rugby players relative to the field hockey players.

With regard to rugby forwards versus rugby backs at the professional level, results of visuoperceptual tracking tasks were also consistently low in the direction of poorer cognitive test performance by the rugby forwards relative to the rugby backs. Rugby forwards also displayed worse test performance than the rugby backs on the hand-motor dexterity task, as well as on an attention and concentration task and a verbal memory task. These results revealed the presence of significant cognitive deficit associated with mild closed head injury amongst professional rugby players, with impairment being particularly pronounced amongst the rugby forwards. Although this research was considered methodologically rigorous due to the large sample size, the inclusion of a matched non-contact sport control group, and the use of neuropsychological tests known to be sensitive to the effects of diffuse brain damage, the results only applied to top-level *professional* rugby players and consequently were not representative of the total rugby-playing population.

As a result, an analysis at the *school level* was conducted using the same basic assessment procedure to compare the cognitive test performance and post-concussive symptoms of top-level schoolboy rugby players (from rugby-playing schools in Cape Town) to top-level schoolboy field hockey players from the same schools (non-contact sport controls). The neuropsychological test battery was a refined version of that employed at the professional level, and included measures of general intellectual functioning (Picture Completion, Vocabulary, National Adult Reading Test), attention and concentration (Digit Span, Letter-number Sequencing, STROOP screening test), visuoperceptual tracking (Digit Symbol Substitution, Trail Making Test Parts A & B), memory (WMS Associate Learning, Digit Symbol Incidental recall – Immediate & Delayed, WMS Visual Reproduction), verbal fluency (Words-in-One-Minute, “S” Words-in-One-Minute) and fine hand-motor dexterity (Sequential Finger Tapping).

In this study, as with the analyses for the professional players, rugby versus field hockey group mean scores were compared (Ackermann, 2000).

Results of this school level research revealed only one significant finding on a verbal memory task in the direction of poorer test performance by the rugby group relative to the field hockey group. Unlike findings on professional rugby players, research at the school level did not reveal statistically significant results on visuo-perceptual tracking tasks.

With regard to school level rugby forwards versus school level rugby backs, no significant differences were noted on any of the neuropsychological tests. Such a finding is counter to epidemiological research that indicates a higher incidence of head injuries in rugby forwards compared to rugby backs (Bird et al., 1998). However, this epidemiological research is based on adults, and Ackermann (2000) suggests that her contrasting findings may have been influenced by the fluidity of school level positions, which are possibly not as entrenched as they are at the professional level. Yet, due to the small number of participants in Ackermann's (2000) school sample, and the lack of school level research to corroborate her findings on rugby forwards versus rugby backs, it would seem too premature to suggest that school level forwards will not display more deficit than school level backs.

Ackermann (2000) concluded that the results of the group mean comparisons provided only tentative support for the presence of cognitive deficits in a high school rugby-playing population. While the methodology of this research was an improvement on research conducted at the professional level in that it utilised a more sensitive method for calculating IQ and a more refined and updated version of the test battery, it was limited by the small sample size, making it difficult to generalise the findings to other school level rugby players.

It was therefore decided to initiate a second phase at the school level in order to enlarge the school sample, which is the aim of the present study.

2.3.8 Section Summary

The incidence of sports-related mild head injury is very high, particularly in contact sports such as boxing, soccer, Australian Rules football, American football, Rugby League and Rugby Union. Since the introduction of professionalism into Rugby Union, injury rates have markedly risen, particularly amongst teenage players (Garraway et al., 2000). In boxing, the trend between length of amateur boxing career and presence and severity of cognitive dysfunction is visible as a direct relationship at the professional level (Jordan et al., 1996; Stewart et al., 1994). Professional soccer players have been found to exhibit neuropsychological deficit predominantly in the areas of attention, concentration and memory (Tysvaer & Lochen, 1991). Amateur soccer players also exhibit the dose-response relation between number of concussions and cognitive performance, albeit to a lesser degree than in professional players, with deficit being apparent in planning and memory (Matser et al., 1999). The few studies conducted on American Footballers, Australian Rules Footballers, and Rugby League players have consistently revealed patterns of cognitive deficit among players that are typically associated with diffuse brain damage (Barth et al., 1989; Hinton-Bayre et al., 1999; Maddocks & Saling, 1996). The Shuttleworth-Jordan et al. (1993) study of Rugby Union players suggests that such deficits are likely to be permanent. Results of ongoing research into the effects of cumulative mild head injury in Rugby Union indicates that, at the professional level, neurocognitive deficits suggestive of diffuse brain damage are marked and measurable (Finkelstein, 1999). At the school level, research reveals no overall cognitive dysfunction amongst top-level rugby players, although subtle indications point to incipient neuropsychological deficit in the area of verbal memory function (Ackermann, 2000).

2.4 RESEARCH HYPOTHESES

From the empirical research presented above and the theoretical postulates of Satz (1993), as explicated by Shuttleworth-Edwards et al. (in press), the following hypotheses were posed for the purposes of this study:

1. Top-level schoolboy rugby players, when compared with non-contact sport controls, will exhibit greater neuropsychological impairment on tests sensitive to the effects of diffuse brain damage, as evidenced by a significant difference of group mean scores in the direction of poorer performance by the rugby

players relative to controls. The rationale for this hypothesis is that rugby players are more exposed to the potential for sustaining cumulative mild head injuries due to the nature of the game, which in turn, would serve to lower their brain reserve capacity, rendering them more vulnerable to neuropsychological impairment, particularly on tests sensitive to subtle neurocognitive dysfunction.

2. Top-level schoolboy rugby forwards, when compared with rugby backs, will display greater neuropsychological impairment on tests sensitive to the effects of diffuse brain damage, as evidenced by a significant difference of group mean scores in the direction of poorer performance by the forwards relative to the backs. The rationale is that the forwards' positioning on the field, where they are more exposed to play that involves physical collisions and impacts (due to scrumming and tackling), increases their exposure to cumulative mild head injuries (compared to the positioning of the backs). Such injuries serve to lower cognitive threshold capacity, thereby rendering forwards more vulnerable than backs to neuropsychological dysfunction on tests susceptible to the effects of mild cognitive impairment.

CHAPTER THREE: METHODOLOGY

The present study forms part of a larger and ongoing research study exploring the effects of cumulative mild head injuries on rugby players. Initially, *professional* rugby players were assessed using a wide neuropsychological test battery and a self-report measure of post-concussive symptoms (Finkelstein, 1999). Next, an analysis at the *school level* was conducted using the same basic assessment procedure to compare the cognitive test performance and post-concussive symptoms of top-level schoolboy rugby players (from rugby-playing schools in Cape Town) to top-level schoolboy field hockey players from the same schools (non-contact sport controls) (Ackermann, 2000). Subsequently it was decided to initiate *a second phase at the school level* in order to enlarge the school sample, which is the aim of the present study. To maintain continuity within the ongoing research study, the present project utilised the same basic methodology as was used for the prior studies (Finkelstein, 1999 and Ackermann, 2000) to assess a cohort of schoolboy rugby players from a rugby-playing school in Grahamstown, and a comparison group of field hockey players from the same school. Specifically, the methodology was as follows.

3.1 PARTICIPANTS

3.1.1 Selection Criteria

Participants were selected from top-level senior schoolboy rugby players (chosen due to their lengthy rugby careers and a more intensive and competitive level of play) and top-level senior schoolboy field hockey players from a top rugby-playing school in Grahamstown. The field hockey players were equivalent to the rugby players in terms of level of play (i.e. top teams), quality of school, educational level and age.

The participants included only currently active rugby players aged between 15 and 19 years of age. Field hockey players were used as the non-contact sport control group as both field hockey and rugby are winter sports, which minimised the possibility of athletes being involved in both sports and thereby confounding the research findings. Field hockey players are not exposed to cumulative mild head injury to the same extent as are rugby players. The incidence of mild head injury in field hockey has been reported as 0.46 per 100 player-sessions, which is significantly lower than both

football and Rugby Union (Powell & Barber-Foss, 1999; see earlier review, pp. 22-23).

3.1.2 Exclusion Criteria

In order to control for potentially confounding variables, as well as to maintain continuity between phases, the same exclusion criteria as for the first school phase were applied. These included a reported history of substance abuse, any neurological or psychiatric/psychological disorders, previous diagnosis of a learning disorder, and/or any previous moderate to severe head injury sustained for any reason (such head injuries were considered any injuries greater than mild as defined by Evans (1992) and the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine) (see earlier discussion, pp. 5-7). On the basis of these exclusion criteria, two rugby players were excluded (learning disorder: $n = 2$) and one field hockey player was excluded (neurological disorder: $n = 1$). The final Grahamstown sample included in the data analysis comprised 32 rugby players and 24 field hockey players. When Cape Town samples were combined with the Grahamstown samples, the following groups and sub-groups were established: Total Rugby ($n = 79$); Total Field Hockey ($n = 58$); Rugby Forwards ($n = 45$); and Rugby Backs ($n = 34$). Participants were not excluded on the basis of previous mild head injuries as doing so would have excluded the majority of the rugby players, given the nature of the game. Furthermore, the aim of the study was to examine the effects of *cumulative* mild head injuries.

3.1.3 Demographic Data

As performance on cognitive tests is known to be affected by variables such as age, education level, and IQ (Lezak, 1995), group mean comparisons between the Total Rugby group and the Total Field Hockey group, and within the Total Rugby group (Rugby Forwards versus Rugby Backs), were calculated for these variables using a pooled independent two-sample t -test. Estimated premorbid IQs were individually established and utilised for the aforementioned comparisons. According to Lezak, "The best performance method" for calculating estimated premorbid IQ entails the use of the single highest score of tests that are known to be good indicators of premorbid IQ and that tend to be generally unaffected by brain damage (see discussion in Lezak, 1995, pp. 106-108). Two recognised tests for this purpose

(Lezak, 1995) are the Vocabulary Subtest and Picture Completion Subtest from the Wechsler Adult Intelligence Scale – Third Edition (WAIS-III).

The estimated premorbid IQ of each rugby and field hockey player was calculated using both these tests. As is evident from tables 3.1 and 3.2 below, groups (Total Rugby versus Total Field Hockey; Rugby Forwards versus Rugby Backs) were equivalent in terms of estimated premorbid IQ ($p = 0.0823$ and $p = 0.213$ respectively) as well as level of education ($p = 0.986$ and $p = 0.425$ respectively). From tables 3.1 and 3.2 it is apparent that the Total Rugby group was well matched with the Total Field Hockey group in terms of age ($p = 0.069$), although a significant difference for age was evident between the Rugby Forwards and the Rugby Backs ($p = 0.045$) with the Forwards group being marginally higher (mean = 17.38) compared to the Backs (mean = 17.03). Specifically, the ages ranged from 15-19 amongst the Forwards and from 16-18 amongst the Backs. Clearly, these mean ages appear highly equivalent and the statistical significance appears to be reflecting the wider spread of scores within the Forwards group compared to the Backs. Substantial effects on cognition would not be expected by virtue of this discrepancy in age ranges between the Rugby Forwards and the Rugby Backs in that they are well within a five-year span, which is less than is typically provided for the establishment of “normative” data on cognition tests (Spreen & Strauss, 1991). Furthermore, both groups fall within the same developmental stage of late adolescence as delineated by Santrock (2002) (see earlier discussion, pp. 9-10).

Table 3.1: Comparison of Demographic Data for Rugby and Field Hockey Players

	Sport	n	Mean	SD	t-statistic	p-value
Age	Rugby	79	17.23	0.77	-1.831	0.069
	Hockey	58	16.98	0.78		
Education	Rugby	79	11.10	0.69	0.017	0.986
	Hockey	58	11.10	0.81		
Premorbid IQ	Rugby	79	114.22	17.13	-0.224	0.823
	Hockey	58	113.57	16.09		

* Significant Difference ($p < 0.05$)

Table 3.2: Comparison of Demographic Data for Rugby Forwards and Rugby Backs

	Position	n	Mean	SD	t-statistic	p-value
Age	Forwards	45	17.38	0.81	-2.039	0.045*
	Backs	34	17.03	0.67		
Education	Forwards	45	11.16	0.74	-0.802	0.425
	Backs	34	11.03	0.63		
Premorbid IQ	Forwards	45	116.31	17.13	-1.256	0.213
	Backs	34	111.44	16.99		

* Significant Difference ($p < 0.05$)

3.1.4 Mild Head Injury History

Group mean comparisons of the reported incidences of *total sport and non-sport related* mild head injuries were conducted between the Total Rugby group and the Total Field Hockey group, as well as between Rugby Forwards and Rugby Backs (see tables 3.3 and 3.4).

With regard to Total Rugby versus Total Field Hockey, a significant difference was evident ($p = 0.000$) in the direction of rugby players sustaining more mild head injuries (both sport and non-sport related) than the hockey players. Rugby players reported an average incidence of 2.29 mild head injuries, while hockey players reported a mean incidence of 0.35. Furthermore, the Total Rugby group exhibited a higher standard deviation ($SD = 1.68$) and a much wider range of incidence (Range = 0-7) than did the Total Field Hockey group ($SD = 0.48$; Range = 0-1), suggesting greater variability in the incidence of mild head trauma amongst the rugby players.

When comparing Rugby Forwards versus Rugby Backs, the analysis revealed no significant differences between the groups with regard to mean incidence ($p = 0.49$) and variability ($p = 0.94$). Therefore, there was no difference between the two groups with regard to range of incidence (Range = 0-7 for both Rugby Forwards and Rugby Backs).

**Table 3.3: Comparison of the Incidence of Previous Reported
Mild Head Injuries (both sport and non-sport related)
in Rugby and Field Hockey Players**

Sport	n	Mean	SD	Range	t- statistic	p- value
Rugby	79	2.29	1.68	0-7	-9.766	0.000*
Hockey	58	0.35	0.48	0-1		

* Significant Difference ($p < 0.05$)

**Table 3.4: Comparison of the Incidence of Previous Reported
Mild Head Injuries (both sport and non-sport related)
in Rugby Forwards and Rugby Backs**

Position	n	Mean	SD	Range	t- statistic	p- value
Forwards	45	2.18	1.67	0-7	0.687	0.494
Backs	34	2.44	1.71	0-7		

* Significant Difference ($p < 0.05$)

3.2 PROCEDURE

All the participants were assessed between May and June 2001 at the educational institution from which they were selected. As in the first phase of the school study, assessment took place during the rugby season, as opposed to pre-season, as this allowed for the detection of permanent effects as well as any acute effects of cumulative mild head injury in the rugby players. The core research team (who conducted the assessment) included the current researcher and two post-graduate students in psychology, under the supervision of a trained clinical psychologist involved in earlier research aspects of the ongoing study. All received their training from Rhodes University, and were trained in the administration and scoring of the tests included in the neuropsychological test battery. To maintain continuity within research phases, the test protocols and the questionnaires used were identical to those

employed in the first phase. Furthermore, standardised test instructions, taken either from the original manuals or from Lezak (1995), appeared on each test protocol.

Each participant was tested individually, in a private room, for a approximately one hour. All participants were English speaking and all interviewing and testing took place in English. Prior to testing, each participant was provided with an explanation of the nature and purpose of the assessment, and any concerns were clarified. Furthermore, confidentiality was emphasised by informing the participants that individual results would remain confidential and that only group analyses would be conducted.

3.3 CONSENT AND QUESTIONNAIRES

As all the players in the sample were minors, consent for the research was provided by the school principal acting in his role as *loco parentis* (see Appendix I). The school principal subsequently informed the participants' parents of the research project. Before testing began, each participant was required to complete a demographic questionnaire (see Appendix II) that provided information on biographical details (age, level of education, current level of functioning), sporting history, previous head injuries (both sport and non-sport related), and history of substance abuse. This information was employed for the purpose of matching the groups and of making any necessary exclusions. A self-report post-concussive symptomatology questionnaire was then administered to the participant to elicit any residual post-concussive symptoms. As this questionnaire does not form part of the database for this particular study, it was not included in the appendices.

3.4 NEUROPSYCHOLOGICAL TEST BATTERY

The neuropsychological test battery used in the present study was a reduced version of that utilised in the earlier phase of the school research. In the interests of time economy and more focused research, it was decided only to include tests across cognitive modalities that consistently showed deficit in the prior professional and school level research, namely visuoperceptual tracking and memory. Tests used for the estimation of premorbid IQ were also included. Specifically, the neuropsychological test battery consisted of the following tests: WAS-III Vocabulary Subtest; WAIS-III Picture Completion Subtest; Wechsler Memory Scale (WMS)

Associate Learning Subtest (Immediate and Delayed Recall); South African Wechsler Adult Intelligence Scale (SAWAIS) Digit Symbol Substitution Subtest (Incidental and Delayed Recall); SAWAIS Digit Span Subtest; and Trail Making Test (Parts A and B). These tests measured functioning across major cognitive modalities, including general intelligence, visuo-perceptual tracking, verbal memory, and visual memory. Each test will be discussed in more detail under the respective cognitive modality (see Appendix III for the assessment schedule as well as for the aforementioned neuropsychological tests in order of administration).

3.4.1 General Intellectual Functioning

As a consistent relationship has been found between premorbid intellectual ability and level of impairment subsequent to brain injury (Lezak, 1995), it was necessary to establish the participants' premorbid IQs. This allowed for matching of the groups so that accurate group comparisons could be conducted on the basis of neuropsychological test results. As mentioned in section 3.1.3, two WAIS-III subtests, namely Vocabulary and Picture Completion, were utilised for the purpose of determining estimated premorbid IQs.

WAIS-III Vocabulary Subtest – this test consists of a list of 33 words arranged in order of increasing difficulty. As per the WAIS-III manual (1997) instructions, the participant was required to state the meaning of the word. Vocabulary assesses an individual's established semantic knowledge, language development and general verbal ability, and it correlates highly with general intelligence (Groth-Marnat, Gallagher, Hale & Kaplan, 2000). According to Zillmer, Waechter, Harris and Kahn (1992) (in Lezak, 1995), Vocabulary tends to be among the least affected tests subsequent to diffuse brain injury, and is therefore a good indicator of premorbid intellectual functioning.

WAIS-III Picture Completion Subtest – this test comprises 15 incomplete pictures arranged in order of increasing difficulty. As per the WAIS-III manual (1997) instructions, the participant was to identify an important missing visual component in each picture, within a time limit of 20 seconds per card. Picture Completion assesses visual alertness, visual recognition (long-term visual memory), visual organisation and reasoning abilities, as well as the ability to differentiate essential from irrelevant

or inessential stimuli (Groth-Marnat et al., 2000). Similar to Vocabulary, Picture Completion is a good indicator of premorbid ability as it is minimally affected by brain damage (Crosson, Greene, et al., 1990, in Lezak, 1995).

3.4.2 Visuoperceptual Tracking

SAWAIS Digit Symbol Substitution Subtest – this is a timed task involving perceptually pairing symbols with corresponding digits, followed by rapidly recording the symbols with the corresponding digits. As per the SAWAIS manual (1969) instructions, the examiner first demonstrated by filling in the first eight blank spaces with the symbol corresponding the digit printed above the blank space. The participant was then instructed to do the same by filling in as many of the remaining 67 blank spaces as possible, as quickly and as accurately as possible, without omitting any blocks. The number of blocks completed after a 90 second period was recorded for scoring purposes. Good performance on this test requires a complex interaction of cognitive functions, namely visuoperceptual tracking, sustained attention, psychomotor speed, short-term visual memory, sequencing ability, response speed, visuomotor co-ordination, and mental flexibility (Groth-Marnat et al., 2000; Lezak, 1995). As a result, Digit Symbol Substitution is very sensitive to even subtle cognitive impairment and is therefore a good indicator of diffuse brain damage associated with mild closed head injury (Erlanger et al., 1999; Lezak, 1995). The 90 second SAWAIS version of this test was used rather than the more recent WAIS-III 120 second version, as it is shorter and therefore recommended for economical use in research settings (Shuttleworth-Edwards, in press).

Trail Making Test (Reitan, 1956) – this is a test of complex visual scanning, motor speed, and sustained attention and concentration that requires sequential problem solving (Barth et al., 1989; Spreen & Strauss, 1991). The test consists of two parts (A and B) that will be presented separately. According to Spreen and Strauss (1991), this test is sensitive to the effects of brain damage, with Part B being particularly sensitive to even mild cognitive impairment.

Trail Making Test (Part A): This test comprises a sheet of paper with 25 encircled numbers randomly arranged on the page. The participant was required to draw pencil lines connecting the numbers in numerical order, and to do so as quickly as possible

without lifting the pencil off the paper. The score was the time taken to complete the test. Prior to commencement of the task, the participant was provided with a mini version of the test to complete for practice purposes.

Trail Making Test (Part B): The administration of Part B was similar to that of Part A, with the exception that the participant was required to connect encircled numbers and letters in alternating order. Part B requires complex conceptual tracking, working memory, more mental flexibility and more information processing than Part A. It is therefore more difficult than Part A as well as more susceptible to the effects of diffuse brain damage (Lezak, 1995; Spreen & Strauss, 1991).

3.4.3 Verbal Memory

SAWAIS Digit Span Subtest – this test consists of two separate tests, namely Digits Forwards and Digits Backwards, which involve different cognitive abilities and are differentially affected by brain damage (Lezak, 1995). For these reasons, they are examined individually.

Digits Forwards: As per the SAWAIS manual (1969) instructions, the participant was to repeat a fixed random series of numbers of increasing length, verbalised by the tester. For each digit span, there is another span of equal length (different numbers). When the participant was unable to recall both trials of a span, the test was discontinued. The score was the highest span number achieved. This test assesses immediate auditory and verbal memory, but it is primarily a test of attention or freedom from distraction (Kaufman, McLean & Reynolds, 1991). Digits Backwards is a far more difficult task than Digits Forwards, rendering it more likely to impairment following diffuse brain damage. Hence, Digits Forwards is more likely to hold in such instances (Lezak, 1995).

Digits Backwards: As with Digits Forwards, the participant was to repeat a fixed random series of numbers of increasing length. Although also similar in instruction and scoring to Digits Forwards, this test required the participant to recall the numbers in reverse order. Digits Backwards assesses auditory and verbal memory, and it further requires that the participant store the information in memory while actively

manipulating it. The latter task taps working memory function, which is highly sensitive to diffuse brain damage (Lezak, 1995).

WMS Associate Learning Subtest (Immediate Recall) – the original Wechsler version (Form 1) of the WMS manual (Wechsler, 1945), including the delayed recall task, were used. The participant was read a list of 10-paired words, consisting of six “easy” pairs that are readily associated and four “hard” pairs that are less readily associated. The participant was then cued with the first word of each pair (one pair at a time), and required to recall the corresponding word. This procedure was repeated three times. The “easy” pairs assess the recall of old verbal associations, while the “hard” pairs rely on new verbal learning ability. As a result, the “hard” pairs are more susceptible to the effects of diffuse brain damage (Lezak, 1995).

WMS Associate Learning Subtest (Delayed Recall) – As delayed memory is more vulnerable than immediate memory to the effects of diffuse brain damage (Lezak, 1995), the participant was required to recall the “easy” and “hard” pairs presented earlier, after a 20 minute delay. Again the participant was cued with the first word of each pair (one pair at a time), and expected to recall the association.

3.4.4 Visual Memory

SAWAIS Digit Symbol Incidental Recall (Immediate) – the short form method of this test was used (Shuttleworth-Jordan & Bode, 1995) as originally described by Kaplan et al. (1991, in Lezak, 1995, p. 463). The tester allowed the participant to continue to the second last row, but noted how many blocks the participant had managed to fill in after 90 seconds. The participant was then provided with a sheet of paper on which the digits were printed and was required to draw as many of the corresponding symbols as could be recalled. This is a test of recent memory that has been shown to be susceptible to the effects of diffuse brain damage. Studies point to the diagnostic potential of Digit Symbol incidental recall in the evaluation of cumulative mild head injury in contact sport (Heilbronner et al., 1991; Pettersen & Skelton, 2000; Shuttleworth-Edwards, in press).

SAWAIS Digit Symbol Incidental Recall (Delayed) – after a 20-minute delay, the participant was handed another sheet of paper with the digits printed on it and was requested to fill in as many of the corresponding symbols as they could remember. Although most people remember as many or almost as many digit-symbol pairs on delayed recall as on immediate recall, this is not usually the case in instances of diffuse brain damage, as delayed memory tends to be more sensitive to the effects of such damage than immediate memory (Lezak, 1995).

3.5 DATA PROCESSING

Scoring of test protocols was conducted by the core research team according to standardised methods (stated in section 3.2). To ensure inter-rater reliability, the scores were crosschecked amongst the core research team and then randomly checked by the supervising trained clinical psychologist to ensure accuracy of scoring and continuity of scoring standards within the ongoing research study. Cape Town rugby and field hockey samples were then combined with the Grahamstown samples to create enlarged samples.

Data analysis involved a direct comparison of group mean scores across each neuropsychological test for: (i) Total Rugby versus Total Field Hockey (non-contact sport control group); and (ii) Rugby Forwards versus Rugby Backs.

3.6 STATISTICAL ANALYSIS

For each neuropsychological test, means were calculated to allow for comparisons to be made between the groups (Total Rugby versus Total Field Hockey; and Rugby Forwards versus Rugby Backs). As opposed to using two-tailed t-tests to compare the means of the groups (as was done in the earlier school research), one-tailed t-tests (which predicted poorer performance by the Rugby players and the Rugby Forwards) were utilised in this research project. One-tailed t-tests are contingent upon the researcher's decision, regarding directionality of findings, prior to running the tests. Where this is possible, one-tailed t-tests are more powerful than two-tailed t-tests as the probability of a difference in one specified direction is half that of the same amount of difference in either direction (Runyon & Haber, 1980). Hence, use of one-tailed t-tests reduces the possibility of a Type II error (i.e. the failure to detect real differences when there are differences). The prediction of poorer performance by

Rugby players and the Rugby Forwards is based on: (i) theoretical postulates of BRC theory (Satz, 1993) that each mild brain insult lowers cognitive reserve, thereby rendering Rugby players and Rugby Forwards more vulnerable to cognitive impairment than Field Hockey players and Rugby Backs respectively, and (ii) results of previous Rugby Union research, which revealed the presence of significant cognitive deficit among Rugby players, specifically Rugby Forwards, on those neuropsychological tests known to be sensitive to mild head injuries (Shuttleworth-Edwards et al., in press).

Finally, the t-test analysis used pooled sample variances if homogeneity of the variances was appropriate, and separate sample variances if heterogeneity of the variances was appropriate. The latter required the use of the Smith-Satterthwaite Procedure t-test so that the means could be validly compared (Runyon & Haber, 1980).

CHAPTER FOUR: RESULTS

Statistically significant results of the group mean comparisons will be presented in the following order: Total Rugby versus Total Field hockey, and Rugby Forwards versus Rugby Backs.

4.1 COMPARISON OF GROUP MEANS ACROSS ALL NEUROPSYCHOLOGICAL TESTS

As described in the methodology chapter, data were analysed using one-tailed t-tests, which predicted poorer performance by the rugby players versus the field hockey players, and the forwards players relative to the backs. The t-test analysis used pooled sample variances if homogeneity of the variances was appropriate, and separate sample variances if heterogeneity of the variances was appropriate. The results of the statistical analyses conducted for the present study are presented at the end of this chapter (see tables 4.1 and 4.2, pp. 51-52).

4.1.1 Total Rugby Versus Total Field Hockey (see table 4.1, p. 51)

For the group mean comparison of Total Rugby versus Total Field Hockey (non-contact sport control group), a significant difference was found on the Digit Symbol Substitution Subtest ($p = 0.049$), as well as on Trail Making Test (Part A) ($p = 0.011$), both in the direction of poorer performance by the Total Rugby group. Trail Making Test (Part B) showed a tendency to be slower for Total Rugby compared to Total Field Hockey, but was not statistically significant ($p = 0.267$).

4.1.2 Rugby Forwards Versus Rugby Backs (see table 4.2, p. 52)

For the group mean comparison of Rugby Forwards versus Rugby Backs, a significant difference was found on Digits Backwards ($p = 0.047$) in the direction of poorer performance by the Rugby Backs. In contrast, comparisons of group mean scores of WMS Associate Learning Subtest – Hard (Delayed Recall) revealed significantly poorer performance by the Rugby Forwards ($p = 0.046$) compared to the Rugby Backs. In addition, Digit Symbol Substitution Subtest and Trail Making Test (Part A) are both lower in the direction of poorer performance by the Rugby Forwards relative to the Rugby Backs, although not reaching significance ($p = 0.126$ and $p = 0.065$ respectively).

Table 4.1: Comparison of Means across Total Rugby and Total Field Hockey

Test	Rugby			Field Hockey			t-statistic	p-value
	n	Mean	SD	n	Mean	SD		
DF	78	7.18	1.36	58	6.85	1.12	-1.523	0.065
DB	78	5.22	1.45	58	5.28	1.37	0.248	0.403
ALE (I)	78	8.70	0.55	58	8.69	0.41	-0.076	0.470
ALH (I)	78	9.01	2.63	58	9.19	2.37	0.406	0.343
ALE (D)	78	5.95	0.27	58	5.97	0.18	0.390	0.349
ALH (D)	78	3.56	0.86	58	3.57	0.96	0.077	0.470
DSS	78	50.20	8.64	58	52.66	8.49	1.659	0.049 *
DSS (IR)	78	7.27	1.60	58	6.79	1.86	-1.614	0.055
DSS (D)	78	7.02	1.78	58	6.60	1.88	-1.318	0.095
TMT A	78	27.78	7.74	58	24.85	6.56	-2.332	0.011 *
TMT B	78	56.40	16.31	58	54.66	15.83	-0.624	0.267

* Significant Difference ($p < 0.05$)

Table 4.2: Comparison of Means across Rugby Forwards and Rugby Backs

Test	n	Forwards Mean	SD	n	Backs Mean	SD	t-statistic	p-value
DF	45	7.33	1.28	33	6.97	1.45	-1.180	0.121
DB	45	5.44	1.57	33	4.91	1.22	<i>-1.697</i>	0.047 *
ALE (I)	45	8.62	0.67	33	8.79	0.33	1.380	0.086
ALH (I)	45	8.67	2.86	33	9.47	2.25	1.353	0.090
ALE (D)	45	5.91	0.36	33	6.00	0.00	<i>1.665</i>	0.052
ALH (D)	45	3.42	0.97	33	3.74	0.67	<i>1.705</i>	0.046 *
DSS	45	49.22	8.99	33	51.49	8.09	1.156	0.126
DSS (IR)	45	7.50	1.37	33	6.97	1.85	-1.462	0.074
DSS (D)	45	7.21	1.55	33	6.76	2.04	-1.106	0.136
TMT A	45	26.63	6.77	33	29.30	8.74	1.529	0.065
TMT B	45	55.70	16.34	33	57.32	16.46	0.433	0.333

* Significant Difference ($p < 0.05$)

Italicised t-statistic values reflect use of separate sample variances.



CHAPTER FIVE: DISCUSSION

5.1 CONTEXT OF THE STUDY

The present study forms part of a larger and ongoing research study investigating the effects of cumulative mild head injuries on rugby players. Initially, *professional* rugby players and a comparison group of national cricket players were individually assessed using a wide neuropsychological test battery and a self-report measure of post-concussive symptoms. Results were analysed via direct comparisons of rugby versus cricket group mean scores (Ancer, 1999). National field hockey players were then assessed via the same assessment procedures and used as an alternative control group to the cricket players, and Springbok rugby versus field hockey group mean scores were compared (Finkelstein, 1999). Additionally, the national rugby sample was enlarged to incorporate the national Under 21 rugby squad, and results of a wide neuropsychological test battery of the combined rugby sample (Springboks and Under 21 players) were compared to those of the national field hockey sample via direct comparisons of rugby versus field hockey group mean scores (Finkelstein, 1999).

At the *professional level*, results of *visuoperceptual tracking* tasks were consistently low, with significant deficit being apparent on Digit Symbol Substitution Subtest (Copy and Delayed Recall), as well as on Trail Making Test (Part B), with the rugby group demonstrating poorer cognitive test performance than the field hockey group. The unstructured verbal fluency task (Words-in-One-Minute) also revealed significant deficit amongst the rugby players relative to the field hockey players, whereas the hand-motor dexterity task (Finger Tapping Test – non-preferred hand) showed better performance by the rugby players relative to the field hockey players.

With regard to rugby forwards versus rugby backs at the professional level, results of *visuoperceptual tracking* tasks were also consistently low in the direction of poorer cognitive test performance by the rugby forwards relative to the rugby backs. These tests included the Digit Symbol Substitution Subtest (Copy and Delayed Recall) and Trail Making Test (Parts A and B). Rugby forwards also displayed worse test performance than rugby backs on the hand-motor dexterity task (Finger Tapping Test – non-preferred hand), as well as on one attention and concentration task (Digits Backwards) and on one verbal memory task (WMS Associate Learning Subtest –

Hard – Delayed Recall) (Finkelstein, 1999). These results provide support for the presence of significant cognitive deficiency associated with mild closed head injury amongst professional rugby players, with impairment being particularly pronounced amongst the rugby forwards.

Next, an analysis at the *school level* was conducted using the same basic assessment procedure to compare the cognitive test performance and post-concussive symptoms of top-level schoolboy rugby players (from rugby-playing schools in Cape Town) to top-level schoolboy field hockey players from the same schools (non-contact sport controls). Data analysis was the same as that conducted at the professional level, where rugby versus field hockey group mean scores were compared (Ackermann, 2000). Results of this research revealed one significant finding on a verbal memory task (WMS Associate Learning Subtest – Hard – Delayed Recall) in the direction of poorer test performance by the rugby group relative to the field hockey group (Ackermann, 2000). Unlike findings on professional rugby players, this research at the school level did not reveal statistically significant results on visuoperceptual tracking tasks (Digit Symbol Substitution Subtest and Trail Making Test – Parts A and B).

For rugby forwards versus rugby backs at the school level, there were no significant differences. Ackermann (2000) suggests that her contrasting findings (compared with the professional players where forwards performed more poorly than backs) may have been influenced by the fluidity of school level positions, which are possibly not as entrenched as they are at the professional level. Yet, due to a relatively small number of participants in Ackermann's (2000) school sample, and the lack of school level research to corroborate her findings on rugby forwards versus rugby backs, it was considered premature for the purposes of the present study's hypotheses to suggest that school level forwards would not display more deficit than school level backs.

5.2 REVISITING THE AIMS, METHODS AND HYPOTHESES OF THE STUDY

The present study introduced a *second phase at the school level* in order to enlarge the size of the original school sample. The aim of the study was to ascertain whether a high school rugby-playing population would be susceptible to the effects of rugby-related cumulative mild head injuries, as was evidenced by prior research on

professional rugby players. The study employed a neuropsychological test battery comprised of tests known to be sensitive to the effects of diffuse brain damage associated with mild closed head injury. In particular, those tests that were revealed as particularly sensitive in the prior studies were targeted, namely visuo-perceptual tracking and memory tests. Participants were selected from a rugby-playing school in Grahamstown and exclusion criteria were applied on the basis of a reported history of substance abuse, any neurological or psychiatric/psychological disorders, previous diagnosis of a learning disorder, and/or any previous moderate to severe head injury sustained for any reason. The exclusion procedure minimised the possibility of any differences between the groups being attributable to these factors.

With regard to demographic data, no significant differences were noted on estimated premorbid IQ and level of education between Total Rugby and Total Field Hockey, or between Rugby Forwards and Rugby Backs. Although Total Rugby and Total Field Hockey were equivalent with regard to age, a significant difference was noted on this variable between Rugby Forwards and Rugby Backs, with the Forwards group being marginally older (mean age = 17.38) compared to the Backs (mean age = 17.03). However, considering that age norms tend to span a decade, substantial effects on cognition would not be expected by this discrepancy since the age ranges are well within a five-year span (age range for Rugby Forwards = 15-19; age range for Rugby Backs = 16-18). As a result, any observed differences between the groups cannot easily be accounted for by the variable of age.

Data analysis for this study involved a direct comparison of group mean scores across each neuropsychological test for Total Rugby versus Total Field Hockey, and for Rugby Forwards versus Rugby Backs. These groups represent the combined Grahamstown and Cape Town school rugby and field hockey samples. Data were analysed using one-tailed t-tests that predicted the directionality of findings based on the results of prior Rugby Union research, as well as on the theoretical postulates of Brain Reserve Capacity (BRC) theory (Satz, 1993), as follows.

It was hypothesised that top-level school *rugby players* would perform more poorly on certain cognitive tests known to be sensitive to the effects of diffuse brain damage compared to matched *non-contact sport controls*. This hypothesis was based on

results of prior Rugby Union research that consistently revealed significant cognitive impairment amongst professional rugby players compared to non-contact sport controls (Shuttleworth-Edwards et al., in press). The rationale for this hypothesis is contextualised within the theory of BRC (Satz, 1993). Due to the nature of the game, rugby players are potentially more likely to be exposed to sustaining concussive and sub-concussive head injuries than are field hockey players. These injuries in turn are likely to cause neuronal damage that serves to lower the critical threshold at which functional symptomatology manifests. This is likely to lead to lowered BRC, rendering rugby players more vulnerable to neuropsychological impairment, particularly on tests sensitive to subtle neurocognitive dysfunction.

Additionally, it was hypothesised that *rugby forwards* would perform more poorly on certain cognitive tests known to be sensitive to the effects of diffuse brain damage compared to *rugby backs*. This hypothesis was based on the premise that research supports that the positioning of rugby forwards on the field (who tend to be more involved in physical collisions and impacts) are more susceptible to sustaining concussive and sub-concussive blows than are rugby backs. Such injuries serve to lower the critical threshold at which functional symptomatology becomes apparent, which in turn lowers BRC, thereby rendering rugby forwards more vulnerable to neuropsychological dysfunction than rugby backs, particularly on tests sensitive to mild cognitive impairment. This hypothesis was proposed on results of prior Rugby Union research that showed significant neuropsychological deficit amongst professional rugby forwards relative to rugby backs at university and professional levels (Shuttleworth-Edwards et al., in press; Shuttleworth-Jordan et al., 1993). Although the finding was not replicated by preliminary research at the school level (Ackermann, 2000), this study still adopted the original hypothesis (i.e. that rugby forwards are likely to show more cognitive deficit than rugby backs) based on the above-mentioned research results and on epidemiological findings that forwards sustain more head injuries than backs (for example, Bird et al., 1998).

5.3 DISCUSSION OF RESULTS

Results of the group mean comparisons will be separated into Total Rugby versus Total Field Hockey and Rugby Forwards versus Rugby Backs, and discussed accordingly.

5.3.1 Group Mean Comparisons for Total Rugby Versus Total Field Hockey

The analysis of group mean comparisons revealed significant findings on two tests of *visuoperceptual tracking*, both in the direction of poorer test performance by the Total Rugby group relative to the Total Field Hockey group. These tests include the Trail Making Test (Part A) and the Digit Symbol Substitution Subtest, both of which require information processing at speed. These findings show a measure of consistency with the findings of both national studies, where in both instances results of these two tests of visuoperceptual tracking were impaired, displaying poorer test performance by the rugby players compared to the field hockey players (Shuttleworth-Edwards et al., in press). Although at the national level it was Trail Making Test (Part B), and not Part A, that was significantly lowered, results of the present study reveal a trend in the same direction (i.e. Total Rugby showing worse test performance compared to Total Field Hockey on Trail Making Test – Part B). The significance of results on Trail Making Test (Part A) is an unexpected finding as it would be anticipated that cognitive deficit would show up earlier on Trail Making Test (Part B) as it is more difficult than Part A as well as more susceptible to the effects of diffuse brain damage (Lezak, 1995; Spreen & Strauss, 1991). Consideration of the mean scores on Trail Making Test (Part A) reveals that the differences are subtle, rendering it possible that the lack of significance on Part B may be an artefact of fairly subtle differences that are obscured by more elaborate standard deviations on this timed task. Additionally, it may be possible that the young cohort of the present study would be more affected by the novelty of the task than would older professional players, and potentially vulnerable people tend to show up sooner on an easier novel task than on a more difficult task like Trail Making Test (Part B) (Lezak, 1995).

The significant result on Digit Symbol Substitution Subtest, which is lower in the hypothesised direction, is consistent across all the national studies (Shuttleworth-Edwards et al., in press). Furthermore, this test has been shown to be sensitive across

all severities of brain injury (Russell, 1979). Research has shown that Digit Symbol Substitution Subtest is consistently the single most sensitive indicator of diffuse brain damage for a wide spectrum of neuropathological conditions, a finding that has been replicated specifically in sensitivity to traumatic closed head injury (for example, Correll, Brodowski & Rokosz, 1993; Crawford, Johnson, Mychalski & Moore, 1997). Moreover, neuropsychological studies into mild head injury in rugby and/or football-related sports have consistently found impaired performance in mild head injured players relative to controls on the Digit Symbol Substitution Subtest (or similar tests of speed of information processing) (for example, Barth et al., 1989; Hinton-Bayre et al., 1997; Macciocchi et al., 1996; Maddocks and Saling, 1996). That this test has elicited the presence of neurocognitive impairment amongst the Total Rugby group relative to the Total Field Hockey group not only provides support for the original hypothesis, but it corroborates the sensitivity of the Digit Symbol Substitution Subtest in detecting diffuse brain damage in a mildly head injured population.

Of note is that results of the earlier school research reveal trends in the same direction as those revealed by the present study, on both Trail Making Test (Parts A and B) and Digit Symbol Substitution Subtest, albeit not statistically significant (Ackermann, 2000). This suggests that even with a small sample, deficits in visuospatial tracking that involves working at speed are already becoming evident at the school level, a finding that has been corroborated via the enlarged sample of the present study. According to research, information processing at speed has shown to be a cognitive function readily affected by mild head injury and among those functions imperative for academic success (for example, Barth et al., 1989; Collins et al., 1999; Erlanger et al., 1999; Evans, 1992).

Whereas Ackermann (2000) elucidated a significant difference on WMS Associate Learning Subtest – Hard (Delayed Recall) in the direction of worse test performance by the rugby players compared to the field hockey players, this was not corroborated by the findings of the present extended study in respect of the Total Rugby group. Further research therefore needs to be conducted to confirm whether WMS Associate Learning is indeed a test sensitive to the effects of diffuse brain damage in a high school rugby-playing population.

5.3.2 Group Mean Comparisons for Rugby Forwards Versus Rugby Backs

With respect to group mean comparisons, the analysis revealed a significant finding on WMS Associate Learning – Hard (Delayed Recall), lower in the direction of the hypothesis (i.e. Rugby Forwards worse than Rugby Backs), and a significant finding on Digits Backwards in the opposite direction to the hypothesis (i.e. Rugby Backs worse than Rugby Forwards). Both these tests assess *verbal memory* and both are particularly susceptible to diffuse brain damage. The ability to recall hard pairs is related to new learning ability and is therefore more sensitive to the effects of brain damage than the recall of easy pairs. Digits Backwards taps working memory function, which is known to be highly sensitive to diffuse brain damage (Lezak, 1995).

In keeping with the hypothesis that forwards players are more likely to exhibit cognitive deficits than backline players, the finding on *WMS Associate Learning – Hard (Delayed Recall)* of forwards worse than backs is consistent with previous Rugby Union research at the national level, where rugby forwards were found to perform more poorly than rugby backs (Finkelstein, 1999).⁵ Although at the school level in the present extended study this test did not show up as defective in the rugby group as a whole, it does appear that there were problems for a substantial proportion of forwards players in this regard. The result on this test is congruent with literature stating that delayed memory is more vulnerable than immediate memory to the effects of diffuse brain damage and is therefore more likely to manifest on neuropsychological tests (Lezak, 1995).

However, the finding on *Digits Backwards* is not in the direction of the hypothesis of the present study (of forwards worse than backs) in that Rugby Backs performed more poorly than Rugby Forwards. This result points to the conclusion that, for Rugby Forwards versus Rugby Backs, the predicted directionality of findings cannot be sustained. Furthermore, it is of note that Rugby Forwards and Rugby Backs were equivalent with respect to concussion incidence (see table 3.4, p. 42), which suggests very similar exposure of the two positions to mild head injury on the rugby field. This is in contrast to the comparison between Total Rugby and Total Field Hockey, where the rugby group had a significantly higher concussion incidence than the field hockey group. Taken together, the contradictory findings on the cognitive tests,

implying that neither group are exclusively performing worse, as well as reported equivalence of concussion incidences between the forwards and backs, may be understood as follows. First, as Ackermann (2000) suggested, it is possible that at the school level positions are not well established (i.e. groups will change positions over time as they find their strengths) and therefore positional variation is not yet distinguishable on the basis of neuropsychological test results as there is no consistent direction in respect of the identified deficit across the tests. Second, it is feasible that the nature of the game at the school level is different compared to that at the professional level, as follows.

In personal communication with a former school level rugby coach and former Border provincial rugby player, it was made apparent that school level rugby players are less heavy than university level or professional players, which renders schoolboy players more mobile and the game more fluid as they do not use their weights for competitive advantage, but rather pass the ball frequently. Hence players are less involved in physical collisions and impacts. Additionally, at the school level there is minimal difference between the weights of the forward and backline players, so both groups are equally mobile and forwards are not necessarily used for physically abrasive play. In contrast, university level and professional players are heavier, with forwards weighing considerably more than backs. As a result, the game is slower and players “use their weight” to “wear down” their opponents, with forwards being utilised for “crash balls” and backs being kept for mobility. This playing strategy is mostly absent amongst school level players who are all (regardless of position) coached to be mobile and to pass the ball rather than to hold onto it and to “crash” into other players as is done at the university and professional levels. The result is that both forwards and backs at the school level are possibly exposed to the potential for sustaining mild head trauma.

Of note, however, is that for the only two tests that revealed deficits for the total school rugby versus the field hockey players, namely *Digit Symbol Substitution Subtest* and *Trail Making Test (Part A)*, forwards showed a strong tendency to be worse than backs ($p = 0.126$ and $p = 0.065$ respectively, see table 4.2, p. 52). Therefore, one explanation for the fact that performance on Digits Backwards goes in

the opposite direction to the hypothesis (of forwards being worse than backs) is that this result was an artefact of chance.

Thus, in sum, it is feasible that the significant findings on WMS Associate Learning – Hard (Delayed Recall), in the direction of forwards worse than backs, and on Digits Backwards, in the direction of backs worse than forwards, may be a reflection that some individuals in the cohort are starting to exhibit subtle cognitive deficit, which is being tapped by both these tests of verbal memory that are known to be sensitive to the effects of diffuse brain damage (Lezak, 1995), but that these individuals are neither exclusively forwards nor backs. According to Leathem & Body (1997), verbal memory ability is claimed to be the only discriminator of mild cognitive impairment in a mostly mild head injured adolescent population. Moreover, a neuropsychological study on amateur versus professional boxers revealed how verbal memory was the only impaired cognitive modality amongst the amateur players (Casson et al., 1982). However, the fact that *several* tests showed strong trends in the direction of forwards being worse than backs (WMS Associate Learning – Hard – Delayed Recall, Digit Symbol Substitution Subtest and Trail Making Test – Part A) raises the possibility that forward players are more susceptible to cumulative mild head injury already at the school level, and that the contradictory finding on Digits Backwards is an isolated result due to chance. Therefore, at this stage there is still not enough evidence to rule out the hypothesis that the cognitive functioning of forward players may be worse than that of backline players at the school level, and needs to be further researched.

5.4 CONCLUSIONS AND THEORETICAL IMPLICATIONS OF THE RESEARCH FINDINGS

Results of the present extended school study provide tentative support for the hypothesis that school level rugby players are more susceptible to the effects of cumulative concussive and sub-concussive head injuries than are non-contact sport controls. This support gains strength by virtue of the fact that, although only two tests revealed significant deficits for rugby players versus field hockey players, one of these tests was the Digit Symbol Substitution Subtest that is typically the most sensitive to diffuse brain damage. However, results do not entirely support the hypothesis that cognitive deficit is more readily observed in the cognitive test

performance of school level rugby forwards compared to rugby backs. These findings are explicable in terms of Satz's (1993) theory of BRC.

Taking into consideration the fact that all the participants in the study were equivalent with respect to estimated premorbid IQ and level of education, and arguably broadly equivalent with respect to age (see discussion on p. 40), and that these variables can function as protective or vulnerability factors that may differentially affect neuropsychological functioning (Satz, 1993), it can be assumed that the observed differences between Total Rugby and Total Field Hockey cannot be attributed to these factors, but rather to the additive effect of concussive and sub-concussive head injuries incurred while playing rugby. According to Satz (1993), there is a critical brain threshold that must be sustained before an individual will display functional impairment. It would appear that the measurable cognitive impairment amongst the rugby players relative to the field hockey players is a manifestation of the rugby players' lowered cognitive reserves, as they have begun to dip below the critical threshold at which impairment becomes visible, with each blow having had a deleterious effect on functioning. The fact that this cognitive dysfunction was only identified by two tests, Trail Making Test (Part A) and the particularly sensitive Digit Symbol Substitution Subtest, may suggest that at the school level the young age of the players, or the high quality of education being received, may act as protective factors in the face of repetitive cerebral insults that appear mild, but the effects of which manifest on tests that provide sufficient challenge so as to force cognitive functioning below critical threshold limits (as pointed out on p. 56 that Trail Making Test – Part A may be more sensitive in the younger age group than Part B). The Digit Symbol Substitution Subtest and the Trail Making Test are both timed tasks, with the timed element providing a considerable degree of challenge. Moreover, the Digit Symbol Substitution Subtest is particularly challenging, as superior performance on this test requires a complex interaction of cognitive functions (Groth-Marnat et al., 2000; Lezak, 1995).

A study by Gronwall, Wrightson & McGinn (1997) on the effects of mild head injury on preschool children showed how, immediately after injury, there were no differences between the cognitive functioning of children and that of controls. However, four to eight months later deficits became visible. The authors suggested

that their results were an indication that mild brain injury had not affected the children's already established skills, but that mildly head injured children failed to develop specific skills (such as visuospatial skills) consequent on mild head trauma. Such a finding is pertinent in that, although the participants in the present study are older than the participants in the Gronwall et al. (1997) study, they represent a bridging group of ages 15-19 (see earlier discussion, p. 10). As a result, some of the younger participants of less than 17 years of age may not have reached full cognitive maturation and may consequently experience cognitive skills deficit due to lowered BRC subsequent to mild cerebral insults incurred while playing rugby. As there were insufficient numbers of participants to examine age groups separately, it is not possible to say with certainty, on the basis of the current research results, whether age did play a significant role. Therefore, further research needs to be conducted that investigates the specific effects of age on the cognitive test performances of schoolboy rugby players.

The lack of consistent observable differences between the Rugby Forwards and the Rugby Backs on neuropsychological test results suggests that, at the school level, the differential effect of positional variation that was clearly evident at the professional level was not apparent. Hence, it is not possible to state that, at the school level, rugby forwards are more exposed to cumulative mild head insults than are rugby backs, which would have implications for their cognitive reserves. Therefore, any observed cognitive impairment between rugby forwards and rugby backs on neuropsychological tests cannot be distinguished along these lines. The differences that were noted on WMS Associate Learning – Hard (Delayed Recall) and on Digits Backwards may be a reflection that some individuals in the enlarged cohort are starting to manifest mild deficit as a result of repetitive concussive and sub-concussive blows sustained as a result of rugby, but not necessarily as a result of their playing positions, with the former serving to lower their cerebral reserve capacity. However, the findings on WMS Associate Learning – Hard (Delayed Recall) together with strong trends on Digit Symbol Substitution Subtest and Trail Making Test (Part A) that reveal worse cognitive test performance by the forwards compared to the backs, may be an indication that the finding on Digits Backwards (of backs worse than forwards) was an artefact of chance. Therefore, with regard to future research at

the school level, the comparison of differences between rugby forwards and rugby backs needs to be further researched.

5.5 EVALUATION OF THE RESEARCH STUDY

The methodological strengths of the study include the following:

1. The present study utilised an enlarged sample size ($n = 79$) of school level rugby players that combined existing Cape Town schoolboy rugby and field hockey samples with Grahamstown samples. The larger sample size served to strengthen the internal validity of the research design, as well as the generalisability of the findings of the present study and the indications of similar studies on the effects of cumulative mild head injuries in rugby players (see Shuttleworth-Edwards et al., in press).
2. The neuropsychological test battery employed in the present study was a reduced version of that utilised in the earlier phase of the school research, while still measuring abilities across a focused spectrum of cognitive modalities. The tests included were those used for the estimation of premorbid IQ as well as those that were shown to be particularly sensitive to the effects of diffuse brain damage associated with mild closed head injury in prior research on Rugby Union (Shuttleworth-Edwards et al., in press).
3. The effects of potentially confounding variables were controlled for via the application of exclusion criteria (these included a reported history of substance abuse, any neurological or psychiatric/psychological disorders, previous diagnosis of a learning disorder, and/or any previous moderate to severe head injury sustained for any reason).
4. The calculation of estimated premorbid IQ was based on two neuropsychological tests, namely the WAIS-III Vocabulary Subtest and the WAIS-III Picture Completion Subtest. This served to strengthen the research design in that estimated premorbid IQs were utilised in matching the groups, which eliminated IQ as a potentially confounding variable.
5. Data analysis involved direct comparison of group mean scores between a top rugby-playing group and matched top non-contact sport controls. The comparative nature of this study where participants were compared to matched controls rather than to normative data is considered preferential, as norms are

usually derived from a more general population of individuals. Therefore, for the purposes of this study, norm comparisons may not have been ideal.

6. As opposed to using two-tailed t-tests to compare the means of the groups (as was done in the earlier school research), one-tailed t-tests (which predicted poorer performance by the Rugby players and the Rugby Forwards) were utilised in this study. This prediction was based on theoretical postulates of BRC theory (Satz, 1993) as well as on results of previous Rugby Union research (Shuttleworth-Edwards et al., in press). Where such a prediction is possible, one-tailed t-tests are more powerful than two-tailed t-tests as the probability of a difference in one specified direction is half that of the same amount of difference in either direction (Runyon & Haber, 1980). Hence, the use of one-tailed t-tests reduced the possibility of a Type II error.
7. Research findings were contextualised within a sound theoretical framework, namely BRC theory (Satz, 1993), thereby specifying indications of the outcome and providing a framework for understanding the results.

The methodological limitations of the study include the following:

1. As the study focused solely on group mean comparisons, individual variations were necessarily obscured. In other words, there may have been a small population of individuals with deficit that would go undetected in this mode of analysis.
2. It is not possible to generalise the findings of the study to younger or lower level schoolboy rugby players, nor to the South African high school rugby-playing population as a whole. The participants in the study were adolescents drawn from top-level high school rugby teams affiliated to elite, advantaged schools.
3. The cross-sectional nature of this research design is limiting in that it is not possible to completely rule out pre-existing differences. However, it is unlikely that the differences on Digit Symbol Substitution Subtest, showing up virtually in isolation with the groups being equivalent (with regard to IQ, education and broadly to age), are due to pre-existing differences as this test is typically one of the most sensitive tests to diffuse brain damage.

5.6 RECOMMENDATIONS FOR FUTURE RESEARCH

The recommendations for future research are the following:

1. A prospective study is necessary to investigate the persisting and long-term effects of cumulative mild head injuries on the cognitive functioning and development of schoolboy rugby players. This should entail neuropsychological screening at the high school level to obtain baseline measurements of cognitive functioning, with follow-up of the same individuals throughout their rugby playing careers to ascertain whether clinically evident deficits manifest over time with the addition of further injuries and increased age. Such a study would also assist in determining at which point, during a player's career, positional variation becomes visible on the basis of neuropsychological test results.
2. Studies examining the effects of secondary stressors, such as hypoxia (Ewing et al., 1980) or fatigue (Jordan, 1997), on mildly head injured schoolboy rugby players should be conducted to further determine the impact of concussion on the cognitive functioning of these individuals.
3. Neuropsychological studies need to be carried out on younger and/or more disadvantaged schoolboy rugby players from lower level teams to ascertain the extent to which repetitive concussion affects the cognitive functioning of these populations.
4. A study in which rugby players with a history of learning disability and/or previous moderate to severe head injuries are analysed as a separate group relative to similarly compromised non-contact sport controls is necessary, as the BRC of these individuals is already vulnerable, leaving them at greater risk for the deleterious effects of subsequent cumulative mild head injury.
5. Recently developed computerised tests of cognitive functioning have shown to be more sensitive to subtle cognitive impairment (particularly in reaction time) caused by mild head injury than traditional paper and pencil neuropsychological tests (Bleiberg, Kane, Reeves, Garmoe & Halpern, 2000). The use of such tests in the evaluation of concussed school level rugby players would possibly be beneficial in detecting subtle cognitive impairment that may be missed by traditional neuropsychological tests. Additionally, since computerised testing is less time- and labour-intensive than traditional tests (Wojtys et al., 1999), a larger number of participants may be tested via this

method than is usually the case using paper and pencil tests, which in turn may serve to strengthen the generalisability of the research findings.

6. Qualitative research studies are recommended in order to explore the extent to which the nature of the game of rugby (at various levels of play) affects the exposure of forwards and backs to mild head trauma. This would facilitate an understanding of playing strategies at the school level that appear to prevent increased exposure of forwards to head trauma.

REFERENCES

- Abreau, F., Templer, D.I., Schuyler, B.A. & Hutchison, H.T. (1990). Neuropsychological assessment of soccer players. *Neuropsychology*, 4, 175-181.
- Ackermann, T.R. (2000). *Minor "dings" – major effects? A study into the cognitive effects of mild head injuries in high school rugby*. Unpublished Master's thesis, Rhodes University, Grahamstown.
- Alexander, G.E., Furey, M.L., Grady, C.L., Pietrini, P., Brady, D.R., Mentis, M.J. & Schapiro, M.B. (1997). Association of premorbid intellectual function with cerebral metabolism in Alzheimer's disease: Implications for the cognitive reserve hypothesis. *American Journal of Psychiatry*, 154 (2), 165-172.
- American Academy of Pediatrics Policy Statement (1988). Recommendations for participation in competitive sports. *Physical Sports Medicine*, 16, 165-167.
- Ancer, R. (1999). *A study on mild head injury in rugby: Cognitive test profiles of professional rugby and cricket players*. Unpublished Master's thesis, Rhodes University, Grahamstown.
- Anderson, S.J. (1996). Sports-related head injuries: A neuropsychological perspective. *Sports Medicine*, September, 23-27.
- Anderson, V.A., Catroppa, C., Morse, S.A. & Haritou, F. (1999). Functional memory skills following traumatic brain injury in young children. *Pediatric Rehabilitation*, 3 (4), 159-166.
- Anderson, V.A., Morse, S.A., Klug, G., Catroppa, C., Haritou, F., Rosenfeld, J. & Pentland, L. (1997). Predicting recovery from head injury in young children: A prospective analysis. *Journal of the International Neuropsychological Society*, 3, 568-580.
- Bailes, J.E. & Cantu, R.C. (2001). Head injury in athletes. *Neurosurgery*, 48 (1), 26-45.
- Baker, R.J. & Patel, D.R. (2000). Sports related mild traumatic brain injury in adolescents. *Indian Journal of Pediatrics*, 67 (5), 317-321.
- Barth, J.J., Alves, W.M., Ryan, T.V., Macciocchi, S.N., Rimel, R.W., Jane, J.A. & Nelson, W.E. (1989). Mild head injury in sports: Neuropsychological sequelae and recovery of function. In H.S. Levin, H.M. Eisenberg & A.L. Benton (Eds). *Mild head injury* (pp. 257-275). New York: Oxford University Press.
- Barth, J.J., Macciocchi, S.N., Giordani, B., Rimel, R., Jane, J.A. & Boll, T.J. (1983). Neuropsychological sequelae of minor head injury. *Neurosurgery*, 13 (5), 529-533.

- Basso, M.R. & Bornstein, R.A. (2000). Estimated premorbid intelligence mediates neurobehavioral change in individuals infected with HIV across 12 months. *Journal of Clinical and Experimental Neuropsychology*, 22 (2), 208-218.
- Bennett, T.L. & Raymond, M.J. (1997). Mild brain injury: An overview. *Applied Neuropsychology*, 4 (1), 1-5.
- Bernhardt, D.T. (2000). Football: A case-based approach to mild traumatic brain injury. *Pediatric Annals*, 29 (3), 172-176.
- Binder, L.M. (1986). Persisting symptoms after mild head injury: A review of the postconcussive syndrome. *Journal of Clinical and Experimental Neuropsychology*, 8 (4), 323-346.
- Binder, L.M. (1997). A review of mild head trauma. Part II: Clinical implications. *Journal of Clinical and Experimental Neuropsychology*, 19, 432-457.
- Binder, L.M., Rohling, M.L. & Larrabee, G.J. (1997). A review of mild head trauma. Part I: Meta-analytic review of neuropsychological studies. *Journal of Clinical and Experimental Neuropsychology*, 19 (3), 421-431.
- Bird, Y.N., Waller, A.E., Marshall, S.W., Alsop, J.C., Chalmers, D.J. & Gerrard, D.F. (1998). The New Zealand rugby injury and performance project: V. Epidemiology of a season of rugby injury. *British Journal of Sports Medicine*, 32, 319-325.
- Bleiberg, J., Garmoe, W., Halpern, E., Reeves, D. & Nadler, J. (1997). Consistency of within-day and across-day performance after mild brain injury. *Neuropsychiatry, Neuropsychology, and Behavioural Neurology*, 10, 247-253.
- Bleiberg, J., Halpern, E., Reeves, D. & Daniel, J.C. (1998). Future directions for neuropsychological assessment of sports concussion. *Journal of Head Trauma Rehabilitation*, 13, 36-44.
- Bleiberg, J., Kane, R.L., Reeves, D.L., Garmoe, W.S. & Halpern, E. (2000). Factor analysis of computerized and traditional tests used in mild brain injury research. *The Clinical Neuropsychologist*, 14 (3), 287-294.
- Blumbergs, P.C., Scott, G., Manavis, J., Wainwright, H., Simpson, D.A. & McLean, A.J. (1994). Staining of the amyloid precursor protein to study axonal damage in mild head injury. *Lancet*, 344, 1055-1056.
- Boden, B.P., Kirkendall, D.T. & Garrett, W.E. (1998). Concussion incidence in elite college soccer players. *American Journal of Sports Medicine*, 26 (2), 238-241.
- Bohnen, N. & Jolles, J. (1992). Neurobehavioral aspects of postconcussive symptoms after mild injury. *The Journal of Nervous and Mental Disease*, 180, (11), 683-692.

- Bohnen, N., Jolles, J. & Twijnstra, A. (1992). Neuropsychological deficits in patients with persistent symptoms six months after mild head injury. *Neurosurgery*, 30 (5), 692-696.
- Boll, T.J. & Barth, J. (1983). Mild head injury. *Psychiatric Developments*, 3, 263-275.
- Brooks, N., Kupshik, G., Wilson, L., Galbraith, S. & Ward, R. (1987). A neuropsychological study of active amateur boxers. *Journal of Neurology, Neurosurgery, and Psychiatry*, 50, 997-1000.
- Brown, S.J., Fann, J.R. & Grant, I. (1994). Postconcussional disorder: Time to acknowledge a common source of neurobehavioural morbidity. *Journal of Neuropsychiatry and Neurosciences*, 6, 15-22.
- Butler, R.J. (1994). Neuropsychological investigation of amateur boxers. *British Journal of Sports Medicine*, 28, 187-190.
- Butler, R.J., Forsythe, W.I., Beverly, D.W. & Adams, L.M. (1993). A prospective controlled investigation of the cognitive effects of amateur boxing. *Journal of Neurology, Neurosurgery, and Psychiatry*, 56 (10), 1055-1061.
- Casson, Sham, Campbell, Tarlau and Didomenico (1982). Neurological and CT evaluation of knocked-out boxers. *Journal of Neurology, Neurosurgery, and Psychiatry*, 45, 170-174.
- Catroppa, C. & Anderson, V. (1999). Attentional skills in the acute phase following pediatric traumatic brain injury. *Child Neuropsychology*, 5 (4), 251-264.
- Centers for Disease Control and Prevention. (1997). Sports-related recurrent brain injuries – United States. *MMWR*, 46, 224-227.
- Coffey, C.E., Saxton, J.A., Ratcliff, G., Bryan, R.N. & Lucke, J.F. (1999). Relation of education to brain size in normal aging: Implications for the reserve hypothesis. *Neurology*, 53, 189-196.
- Collins, M.W., Grindel, S.H., Lovell, M.R., Dede, D.E., Moser, D.J., Phalin, B.R., Nogle, S., Wasik, M., Cordry, D., Daugherty, M.K., Sears, S.F., Nicolette, G., Indelicato, P. & McKeag, D.B. (1999). Relationship between concussion and neuropsychological performance in college football players. *JAMA*, 282 (10), 964-970.
- Correll, R.E., Brodgerski, S.E. & Rokosz, S.F. (1993). WAIS performance during the acute recovery stage following closed-head injury. *Perceptual and Motor Skills*, 76, 99-101.
- Crawford, J.R., Johnson, D.A., Mychalkiw, B. & Moore, J.W. (1997). WAIS-R performance following closed-head injury: A comparison of the clinical utility of summary IQs, factor scores, and subtest scatter indices. *The Clinical Neuropsychologist*, 4, 345-355.

- Cremona-Meteyard, S.L. & Geffen, G.M. (1994). Persistent visuospatial attention deficits following mild head injury in Australian Rules Football players. *Neuropsychologia*, 32, 649-662.
- Cullum, C. & Thompson, L.L. (1997). Neuropsychological diagnosis and outcome in mild traumatic brain injury. *Applied Neuropsychology*, 4 (1), 6-15.
- Daniel, J.C., Olesniewicz, M.H., Reeves, D.L., Tam, D., Bleiberg, J., Thatcher, R. & Salazar, A. (1999). Repeated measures of cognitive processing efficiency in adolescent athletes: Implications for monitoring recovery from concussion. *Neuropsychiatry, Neuropsychology, Behavioural Neurology*, 12 (3), 167-169.
- Dikmen, S., McLean, A. & Temkin, N. (1986). Neuropsychological and psychosocial consequences of minor head injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, 49, 1227-1232.
- Dikmen, S.S., Temkin, N. & Armsden, G. (1989). Neuropsychological recovery: Relationship to psychosocial functioning and post concussional complaints. In H.S. Levin, H.M. Eisenberg & A.L. Benton (Eds). *Mild head injury* (pp. 229-244). Oxford: Oxford University Press.
- Di Scianfani, V., Westley Clark, H., Tolou-Shams, M., Bloomer, C.W., Salas, G.A., Norman, D. & Fein, G. (1998). Premorbid brain size is a determinant of functional reserve in abstinent crack-cocaine and crack-cocaine-alcohol-dependent adults. *Journal of the International Neuropsychological Society*, 4, 559-565.
- Donders, J. & Strom, D. (2000). The effect of traumatic brain injury on children with learning disability. *Pediatric Rehabilitation*, 1 (3), 179-184.
- Erichsen, J.E. (1882). *On concussion of the spine: Nervous shock and other obscure injuries of the nervous system in their clinical and medico-legal aspects*. New York: Bermingham.
- Erlanger, D.M., Kutner, K.C., Barth, J.T. & Barnes, R. (1999). Neuropsychology of sports-related head injury: Dementia Pugilistica to Post Concussion Syndrome *The Clinical Neuropsychologist*, 13 (2), 193-209.
- Evans, E.W. (1992). The postconcussion syndrome and the sequelae of mild head injury. *Neurologic Clinics*, 10(4) 815-847.
- Ewing, R., McCarthy, D., Gronwall, D. & Wrightson, P. (1980). Persisting effects of minor head injury observable during hypoxic stress. *Journal of Clinical Neuropsychology*, 2, 147-155.
- Ewing-Cobbs, L., Prasad, M., Fletcher, J.M., Levin, H.S., Miner, M.E. & Eisenberg, H.M. (1998). Attention after pediatric traumatic brain injury: A multidimensional assessment. *Child Neuropsychology*, 4 (1), 35-48.

- Fields, R.B. (1991). *The effects of head injuries on older adults*. Paper presented at the 99th Annual Convention of the American Psychological Association, San Francisco.
- Finkelstein, M. (1999). *The scrum-down on brain damage effects of cumulative mild head injury: A comparison of group mean scores between national rugby players and non-contact sport controls*. Unpublished Master's thesis, Rhodes University, Grahamstown.
- Flanagan, S. (1999). Psychiatric management of mild traumatic brain injury. *The Mount Sinai Journal of Medicine*, 66 (3), 152- 159.
- Gaetz, M., Goodman, D. & Weinberg, H. (2000). Electrophysiological evidence for the cumulative effects of concussion. *Brain Injury*, 14 (12), 1077-1088.
- Gaetz, M. & Weinberg, H. (2000). Electrophysiological indices of persistent postconcussion symptoms. *Brain Injury*, 14 (9), 815-832.
- Garraway, W.M., Lee, A.J., Hutton, S.J., Russell, E.B.A.W. & Macleod, D.A.D. (2000). Impact of professionalism on injuries in rugby union. *British Journal of Sports Medicine*, 34, 348-351.
- Gennarelli, T.A. (1996). The spectrum of traumatic axonal injury. *Neuropathology and Applied Neurobiology*, 22, 509-513.
- Gibbs, N. (1993). Injuries in professional rugby league: A three-year prospective study of the South Sydney professional rugby league football club. *The American Journal of Sports Medicine*, 21 (5), 696-700.
- Gronwall, D. (1989). Cumulative and persisting effects of concussion on attention and cognition. In H.S. Levin, H.M. Eisenberg & A.L. Benton (Eds.). *Mild head injury* (pp. 153-162). New York: Oxford University Press.
- Gronwall, D. & Sampson, H. (1974). *The psychological effects of concussion*. Auckland: Auckland University Press.
- Gronwall, D. & Wrightson, P. (1974). Delayed recovery of intellectual function after minor head injury. *Lancet*, 2, 605-609.
- Gronwall, D. & Wrightson, P. (1975). Cumulative effect of concussion. *Lancet*, 2, 995-997.
- Gronwall, D., Wrightson, P. & McGinn, V. (1997). Effect of mild head injury during the preschool years. *Journal of the International Neuropsychological Society*, 3, 592-597.
- Groth-Marnat, G., Gallagher, R.E., Hale, J.B. & Kaplan, E. (2000). The Wechsler intelligence scales. In G. Groth-Marnat (Ed). *Neuropsychological assessment in clinical practice* (pp. 129-194). New York: John Wiley & Sons, Inc.

- Gulbrandsen, G.B. (1984). Neuropsychological sequelae of light head injuries in older children 6 months after trauma. *Journal of Clinical Neuropsychology*, 6, 257-268.
- Haglund, Y. & Eriksson, E. (1993). Does amateur boxing lead to chronic brain damage? A review of some investigations. *American Journal of Sports Medicine*, 21, 97-109.
- Heilbronner, R.L., Henry, G.K. & Carson-Brewer, M. (1991). Neuropsychologic test performance in amateur boxers. *American Journal of Sports Medicine*, 19 (4), 376-380.
- Hinton-Bayre, A.D., Geffen, G.M., Geffen, L.B., McFarland, K.A. & Friis, P. (1999). Concussion in contact sports: Reliable change indices of impairment and recovery. *Journal of Clinical and Experimental Neuropsychology*, 21 (1), 70-86.
- Hinton-Bayre, A.D., Geffen, G. & McFarland, K. (1997). Mild head injury and speed of information processing: A prospective study of professional rugby league players. *Journal of Clinical and Experimental Neuropsychology*, 19, 275-289.
- Hughenoltz, H., Stuss, D.T., Stethem, B.A. & Richard, M.T. (1988). How long does it take to recover from a mild concussion? *Neurosurgery*, 22, 853-858.
- Jordan, A.B. (1997). *The Shuttle effect: The development of a model for the prediction of variability in cognitive test performance across the adult life span*. Doctoral Dissertation, Rhodes University, Grahamstown.
- Jordan, B.D. (2000). Chronic traumatic brain injury associated with boxing. *Seminars in Neurology*, 20 (2), 179-185.
- Jordan, S.E., Green, G.A., Galanty, H.L., Mandelbaum, B.R. & Jabour, B.A. (1996). Acute and chronic brain injury in United States national team soccer players. *American Journal of Sports Medicine*, 24 (2), 205-210.
- Jordan, B.D., Matser, E.J.T., Zimmerman, R.D. & Zazula, T. (1996). Sparring and cognitive function in professional boxers. *Physician Sports Medicine*, 24, 87-94.
- Kaufman, A.S., McLean, J.E. & Reynolds, C.R. (1991). Sex, race, residence, region, and education differences on the 11 WAIS-R subtests. *Journal of Clinical Psychology*, 44, 231-248.
- Kelly, J.P. & Rosenberg, J.H. (1997). Diagnosis and management of concussion in sports. *Neurology*, 48, 575-580.
- Kraus, J.F., & Nourjah, P. (1989). The epidemiology of mild head injury. In H.S. Levin, H.M. Eisenberg & A.L. Benton (Eds). *Mild head injury* (pp. 8-22). New York: Oxford University Press.

- Kutner, K.C., Erlanger, D.M., Tsai, J., Jordan, B. & Relkin, N.R. (2000). Lower cognitive performance of older football players possessing apolipoprotein E epsilon4. *Neurosurgery*, 47 (3), 651-657.
- Leathem, J.M. & Body, C.M. (1997). Neuropsychological sequelae of head injury in a New Zealand adolescent sample. *Brain Injury*, 11 (8), 565-575.
- Leininger, B.E., Gramling, S.E., Farrell, A.D., Kreutzer, J.S. & Peck, E.A. (1990). Neuropsychological deficits in symptomatic minor head injury patients after concussion and mild concussion. *Journal of Neurology, Neurosurgery, and Psychiatry*, 53, 293-296.
- Levin, H.S., Benton, A.L. & Grossman, R.G. (1982). *Neurobehavioral consequences of closed head injury*. Oxford: Oxford University Press.
- Levin, H.S. & Eisenberg, H.M. (1979). Neuropsychological impairment after closed head injury in children and adolescents. *Journal of Pediatric Psychology*, 4, 389-402.
- Levin, H.S., Mattis, S., Ruff, R.M., Eisenberg, H.M., Marshall, L.F., Tabaddor, K., High, W.M. & Frankowski, R.F. (1987). Neurobehavioural outcome following minor head injury: A three-center study. *Journal of Neurosurgery*, 66, 234-243.
- Lezak, M.D. (1995). *Neuropsychological Assessment* (3rd ed.). New York: Oxford University Press.
- Lishman, W.A. (1988). *Organic psychiatry: The psychological consequences of cerebral disorder* (3rd ed.) Oxford: Blackwell Scientific Publications.
- Lord-Maes, J. & Obrzut, J. (1996). Neuropsychological consequences of traumatic brain injury in children and adolescents. *Journal of Learning Disabilities*, 29 (6), 609-617.
- Macciocchi, S.N., Barth, J.T., Alves, W., Rimel, R.W. & Jane, J.A. (1996). Neuropsychological functioning and recovery after mild head injury in collegiate athletes. *Neurosurgery*, 39 (3), 510-514.
- Maddocks, D. & Saling, M. (1996). Neuropsychological deficits following concussion. *Brain Injury*, 10, 99-103.
- Maddocks, D.L., Saling, M.M. & Dicker, G.D. (1995). A note on normative data for a test sensitive to concussion in Australian Rules Footballers. *Australian Psychologist*, 30, 125-127.
- Maroon, J.C., Lovell, M.R., Norwig, J., Podell, K., Powell, J.W. & Hartl, R. (2000). Cerebral concussion in athletes: Evaluation and neuropsychological testing. *Neurosurgery*, 47, 659-672.
- Martland, H.S. (1928). Punch-drunk. *JAMA*, 19, 1103-1107.

- Matser, J.T., Kessels, A.G.H., Jordan, B.D., Lezak, M.D. & Troost, J. (1998). Chronic traumatic brain injury in professional soccer players. *Neurology*, 51, 791-796.
- Matser, E.J.T., Kessels, A.G., Lezak, M.D., Jordan, B.D. & Troost, J. (1999). Neuropsychological impairment in amateur soccer players. *JAMA*, 282, 971-973.
- Matser, J.T., Kessels, A.G.H., Lezak, M.D. & Troost, J. (2001). A dose-response relation of headers and concussions with cognitive impairment in professional soccer players. *Journal of Clinical and Experimental Neuropsychology*, 23 (6), 770-774.
- McLatchie, G., Brooks, N., Galbraith, S., Hutchison, J.S.F., Wilson, L., Melville, I. & Teasdale, E. (1987). Clinical neurological examination, neuropsychology, electroencephalography and computed tomographic head scanning in active amateur boxers. *Journal of Neurology, Neurosurgery, and Psychiatry*, 50, 96-99.
- McLean, A., Temkin, N., Dikmen, S. & Wyler, A.R. (1983). The behavioral sequelae of head injury. *Journal of Clinical Neuropsychology*, 5, 361-376.
- Mortimer, J.A. & Pirozzolo, F.J. (1985). Remote effects of head trauma. *Developmental Neuropsychology*, 1, 215-229.
- Nathan, M., Goedecke, R. & Noakes, T.D. (1983). The incidence and nature of rugby injuries experienced at one school during the 1982 rugby season. *South African Medical Journal*, 64, 132-137.
- Newcombe, F., Rabbitt, P. & Briggs, M. (1994). Minor head injury: Pathophysiological or iatrogenic sequelae? *Journal of Neurology, Neurosurgery, and Psychiatry*, 57, 709-716.
- Oddy, M. (1993). Head injury during childhood. *Neuropsychological Rehabilitation*, 3 (4), 301-320.
- Oppenheimer, D.R. (1968). Microscopic lesions in the brain following head injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, 31, 299-306.
- Pettersen, J.A. & Skelton, R.W. (2000). Glucose enhances long-term declarative memory in mildly head-injured varsity rugby players. *Psychobiology*, 28, 81-89.
- Pinkston, J.B., Gouvier, W.D. & Santa Maria, M.P. (2000). Mild head injury: Differentiation of long-term differences on testing. *Brain & Cognition*, 44 (1), 74-78.
- Powell, J.W. & Barber-Foss, K.D. (1999). Traumatic brain injury in high school athletes. *JAMA*, 282, 968-963.

- Quinn, N.P., Rossor, M.N. & Marsden, C.D. (1986). Dementia and Parkinson's disease – Pathological and neurochemical considerations. *British Medical Bulletin*, 42, 86-90.
- Ramirez, J.R. (1998). Attentional deficits in youth boxing: Effects of repeated mild closed head injuries. *Dissertation Abstracts International: Section B: The Sciences & Engineering*, 58 (9-B), 5138.
- Raskin, S.A., Mateer, C.A. & Tweeten, R. (1998). Neuropsychological assessment of individuals with mild traumatic brain injury. *The Clinical Neuropsychologist*, 12 (1), 21-30.
- Reid, I., Shuttleworth-Jordan, A.B., Ancer, R. Dickinson, A., Radloff, S. & Jakoet, I. (1999). To scrum or not to scrum: First report from the RU-SARFU rugby head injury study. In Shuttleworth-Jordan, A.B. (Chair), *Blowing the whistle on the hazards of head injury in rugby and football*. Symposium for presentation at the 22nd mid-year annual meeting of the International Neuropsychological Society, Durban, South Africa.
- Reitan, R.M. (1956). *The Trail Making Test: Manual for Administration, Scoring and Interpretation*. Indiana: Indiana Medical Centre Indiana University.
- Richardson, J.T.E. (1990). *Clinical and neuropsychological aspects of closed head injury*. London: Taylor & Francis.
- Rimel, R.W., Giordani, B., Barth, J.T., Boll, T.J. & Jane, J.A. (1981). Disability caused by minor head injury. *Neurosurgery*, 9, 221-228.
- Roth, M. (1986). The association of clinical and neurological findings and its bearings on the classification and etiology of Alzheimer's disease. *British Medical Bulletin*, 42, 42-50.
- Rothweiler, B., Temkin, N.R. & Dikmen, S.S. (1998). Aging effect on psychosocial outcome in traumatic brain injury. *Archives of Physical and Medical Rehabilitation*, 79 (8), 881-887.
- Roux, C., Goedecke, R., Visser, G.R., van Zyl, W.A. & Noakes, T.D. (1987). The epidemiology of schoolboy rugby injuries. *South African Medical Journal*, 71, 307-313.
- Ruchinskias, R.A., Francis, J.P. & Barth, J.T. (1997). Mild head injury in sports. *Applied Neuropsychology*, 4, 43-49.
- Runyon, R.P. & Haber, A. (1980). *Fundamentals of behavioral statistics* (4th ed.). Reading, Massachusetts: Addison-Wesley Publishing Company.
- Russell, E.W. (1979). Three patterns of brain damage on the WAIS. *Journal of Clinical Psychology*, 35, 611-620.

- Ryan, J.M. & McQuillan, R. (1992). A survey of rugby injuries attending an accident and emergency department. *Irish Medical Journal*, 85 (2), 72-73.
- Santrock, J.W. (2002). *Life-span development*. (8th ed.). New York: McGraw-Hill.
- Satz, P. (1993). Brain reserve capacity on symptom onset after brain injury: A formulation and review of evidence for threshold theory. *Neuropsychology*, 7 (3), 273-295.
- Satz, P. (2001). Mild head injury in children and adolescents. *Current Directions in Psychological Science*, 10 (3), 106-109.
- Satz, P., Zaucha, K., McCleary, C., Light, R., Asarnow, R. & Becker, D. (1997). Mild head injury in children and adolescents: A review of studies (1970-1995). *Psychological Bulletin*, 122 (2), 107-131.
- Schmand, B., Smit, J.H., Geerlings, M.I. & Lindeboom, J. (1997). The effects of intelligence and education on the development of dementia. A test of the brain reserve hypothesis. *Psychological Medicine*, 27 (6), 1337-1344.
- Selby, M.J. (2000). Overview of neurology. In G. Groth-Marnat (Ed). *Neuropsychological assessment in clinical practice: A guide to test interpretation and integration*. New York: John Wiley & Sons, Inc.
- Shuttleworth-Edwards, A.B. (in press). Fine tuning of the digit symbol paired associate recall test for practitioner purposes in clinical and research settings. *The Clinical Neuropsychologist*.
- Shuttleworth-Edwards, A.B., Border, M., Reid, I. & Radloff, S. (in press). Additive effects of concussion in South African rugby union: A case of a little becoming too much? In Lovell, M.R., Echemendia, R.E., Barth, J.T. & Collins, M.W. (Eds.). *Sports Neuropsychology*. The Netherlands: Swets.
- Shuttleworth-Jordan, A.B. (1999). *When a little becomes too much: A prospective theoretical context for cumulative mild head injury. A response to apparently null outcomes*. Paper presented at the 22nd Annual Meeting of the International Neuropsychology Society, Durban, South Africa.
- Shuttleworth-Jordan, A.B., Balarin, E. & Puchert, J. (1993). *Mild head injury effects in rugby: Is playing the game really worth the cost?* Paper presented at the International Neuropsychological Society's 16th European Conference, Island of Madeira (Portugal).
- Shuttleworth-Jordan, A.B. & Bode, S.G. (1995). Taking account of age related differences on digit symbol and incidental recall for diagnostic purposes. *Journal of Clinical and Experimental Psychology*. 17 (3), 439-448.
- Snoek, J.W., Minderhoud, J.M. & Wilmsink, J.T. (1984). Delayed deterioration following mild head injury in children. *Brain*, 107, 15-36.

- Sosin, D.M., Snizek, J.E. & Thurman, D.J. (1996). Incidence of mild and moderate brain injury in the United States, 1991. *Brain Injury*, 10 (1), 47-54.
- South African Wechsler Adult Intelligence Scale (SAWAIS) Manual*. (1969). Johannesburg: National Institute for Personnel Research, Human Sciences Research Council.
- Spear Bassett & Slater (1990). Neuropsychological function in adolescents sustaining mild closed head injury. *Journal of Pediatric Psychology*, 15 (2), 225-236.
- Spree, O. & Strauss, E. (1991). *A compendium of neuropsychological tests: Administration, norms, and commentary*. New York: Oxford University Press.
- Statistics on the 150 affiliated National Associations of Fifa (1982). *Fifa News*, 235, 538.
- Stewart, W.F., Gordon, B., Selnes, O., Bandeen-Roche, K., Zeger, S., Tusa, R.J., Celentano, D.D., Shechter, A., Liberman, J., Hall, C., Simon, D., Lesser, R. & Randall, R.D. (1994). Prospective study of central nervous system function in amateur boxers in the United States. *American Journal of Epidemiology*, 139, 573-588.
- Symmonds, C. (1962). Concussion and its sequelae. *Lancet*, i, 1-5.
- Tellier, A., Della Malva, L.C., Cwinn, A., Grahovac, S., Morrish, W. & Brennan-Barnes, M. (1999). Mild head injury: A misnomer. *Brain Injury*, 13 (7), 463-475.
- Thornhill, S., Teasdale, G.M., Murray, G.D., McEwan, J., Roy, C.W. & Penny, K.I. (2000). Disability in young people and adults one year after head injury: Prospective cohort study. *British Medical Journal*, 320, 1631-1635.
- Tysvaer, A.T. & Lochen, E.A. (1991). Soccer injuries to the brain. *American Journal of Sports Medicine*, 19, 56-60.
- Unverzagt, F.W., Hui, S.L., Farlow, M.R., Hall, K.S. & Hendrie, H.C. (1998). Cognitive decline and education in mild dementia. *Neurology*, 50, 181-185.
- Uzzell, B.P. (1999). Mild head injury: Much ado about something. In N.R. Varney & R.J. Roberts (Eds). *The evaluation of mild traumatic brain injury*. London: Lawrence Erlbaum Associates.
- Walton, G.L. (1883). Possible cerebral origin of the symptoms usually classed under 'railway spine'. *Boston Medical Surgery Journal*, 109, 337-340.
- Wechsler, D. (1945). A standardized memory scale for clinical use. *The Journal of Psychology*, 19, 87-95.

- Wechsler, D. (1997). *Wechsler Adult Intelligence* (3rd ed.): *Administration and scoring manual*. San Antonio, Texas: The Psychological Corporation: Harcourt Brace and Company.
- Winogron, H., Knights, R. & Bawden, H (1984). Neuropsychological deficits following head injury in children. *Journal of Clinical Neuropsychology*, 6, 269-286.
- Witol, A. & Webbe, F. (1993). *Neuropsychological deficits and soccer*. Paper presented at the annual meeting of the National Academy of Neuropsychology, Phoenix, Arizona, October 29.
- Wojtys, E.M., Hovda, D., Landry, G., Boland, A., Lovell, M., McCrea, M. & Minkoff, J. (1999). Concussion in sports. *The American Journal of Sports Medicine*, 27, 676-687.
- Wrightson, P. (1989). Management of disability and rehabilitation services after mild head injury. In H.S. Levin, H.M. Eisenberg & A.L. Benton (Eds). *Mild head injury* (pp. 245-256). New York: Oxford University Press.
- Wrightson, P. & Gronwall, D. (1998). Mild head injury in New Zealand: Incidence and persisting symptoms. *New Zealand Medical Journal*, 111 (1062), 99-101.
- Zhang, M., Katzman, R., Salmon, D., Jin, H., Cai, G., Wang, Z., Qu, G., Grant, I., Yu, E., Levy, P., Klauber, M.R. & Liu, T. (1990). The prevalence of dementia and Alzheimer's disease in Shanghai, China: Impact of age, gender and education. *Annals of Neurology*, 27, 428-437.

APPENDIX I

Letter of Consent



ST ANDREW'S COLLEGE

HEADMASTER: ANTONY CLARK, M.A. (Cantab), H.D.E. (Rhodes)

P.O. BOX 182
GRAHAMSTOWN
6140
SOUTH AFRICA

TEL: (046) 622 7214

(046) 622 7267

FAX: (046) 636 1806

9 May 2001

Professor A Edwards
Rhodes University
GRAHAMSTOWN
6140

Dear Professor Edwards

PARTICIPATION IN RESEARCH ON CONCUSSION IN SCHOOL RUGBY

I hereby give consent for the Rugby and Hockey pupils at St Andrew's College to be involved in your study on concussion in school rugby. Please be assured that we are totally behind research of this nature, and you will receive our full support.

Yours sincerely

MIKE FENNELL
ACTING DEPUTY HEADMASTER



Member of the Independent Schools Council
Member of the Conference of Heads of
Independent Schools in South Africa



APPENDIX II

Demographic Questionnaire

RHODES UNIVERSITY PSYCHOLOGY CLINIC
Pre-assessment Questionnaire

NAME: _____ FIRST LANGUAGE: _____

AGE: _____ D.O.B: _____

SCHOOL STANDARD: _____ SPORT: RUGBY / HOCKEY

TEAM: _____ CURRENT POSITION: _____

MOST FREQUENTLY PLAYED PAST POSITION: _____

GENERAL HISTORY

QUESTION 1

Have you ever failed a standard at school? Yes No

If Yes, when? _____ For what reason? _____

What was your overall grade in 2000? _____

How many of your subjects did you take on Standard Grade _____ / Higher Grade? _____

QUESTION 2

Have you ever experienced learning difficulties or required remedial classes? Yes No

If Yes, what was the problem? _____

QUESTION 3

Have you ever experienced neurological problems (e.g. seizures, tremors, stroke)?

Yes No

If Yes, what was the problem? _____

QUESTION 4

Have you ever suffered from a psychological/psychiatric disorder (e.g. depression, anxiety, attention deficit or hyperactivity) which needed the help of a psychologist/psychiatrist? Yes No

If Yes, what was the problem? _____

QUESTION 5

Are you currently taking any form of medication? Yes No

If Yes, please specify: _____

QUESTION 6

Do you smoke?	Yes	No
If Yes, how much? _____		

QUESTION 7

Do you drink alcohol?	Yes	No
If Yes, how much? _____		
Have you ever felt that you should cut down on your drinking?	Yes	No

QUESTION 8

Do you use any other substances?	Yes	No
If Yes, specify type and frequency of use: _____		

QUESTION 9

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 10

Rugby Players

At what age did you first start playing rugby? _____

For how many consecutive years have you been playing rugby? _____

Have you ever used protective headgear while playing rugby? Yes No

If Yes, specify type and duration _____

Hockey Players

At what age did you first start playing hockey? _____

For how many consecutive years have you been playing hockey? _____

Rugby and Hockey Players

What made you choose the sport you are currently playing? _____

Have you ever played any other sport for a lengthy period of time?

(For rugby players, check whether they have participated in BOXING and SOCCER)

(For hockey players, check whether they have participated in RUGBY, BOXING and SOCCER)

Yes No

If Yes, specify sport and time period played: _____

QUESTION 11 (Rugby players only)

How many times can you remember sustaining a head injury or concussion during a game of rugby, including occasions when you were knocked or "dinged" so hard that you felt dazed, confused and/or disorientated, even though you continued to play in the game? (*Note to examiners: Try to ascertain the specific incidents of injury, beginning with the most recent, followed by other incidences in as consecutive an order as the subject can recall.*)

If Yes, specify date/s: Injury 1 _____ Injury 2 _____

Injury 3 _____ Injury 4 _____ Injury 5 _____

Injury 1

What caused the injury/concussion? _____

Were you dazed, confused and/or disorientated? 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 taken off the field? Yes No

If Yes, for how long? _____

Were you hospitalised? Yes No

If Yes, for how long? _____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If Yes, please specify: _____

Injury 2

What caused the injury/concussion? _____

Were you dazed, confused and/or disorientated? 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 taken off the field? Yes No

If Yes, for how long? _____

Were you hospitalised? Yes No

If Yes, for how long? _____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes

No

If Yes, please specify: _____

Injury 3

What caused the injury/concussion? _____

Were you dazed, confused and/or disorientated?

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 taken off the field?

Yes

No

If Yes, for how long? _____

Were you hospitalised?

Yes

No

If Yes, for how long? _____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes

No

If Yes, please specify: _____

Injury 4

What caused the injury/concussion? _____

Were you dazed, confused and/or disorientated?

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 taken off the field?	Yes	No
If Yes, for how long? _____		
Were you hospitalised?	Yes	No
If Yes, for how long? _____		
Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?		
	Yes	No
If Yes, please specify: _____		

Injury 5

What caused the injury/concussion? _____

Were you dazed, confused and/or disorientated?	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 taken off the field?	Yes	No
If Yes, for how long? _____		
Were you hospitalised?	Yes	No
If Yes, for how long? _____		
Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?		
	Yes	No
If Yes, please specify: _____		

QUESTION 12

What other injuries have you sustained while playing rugby (e.g. hand injuries, sprains, fractures)?

Please specify: _____

QUESTION 13

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

Yes

No

If Yes, specify which sport/s and date/s:

Injury 1 _____ Injury 2 _____ Injury 3 _____

Sport _____ Sport _____ Sport _____

Injury 1

What caused the injury/concussion? _____

Were you dazed, confused and/or disorientated?

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 removed from the game?

Yes

No

If Yes, for how long? _____

Were you hospitalised?

Yes

No

If Yes, for how long? _____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes

No

If Yes, please specify: _____

Injury 2

What caused the injury/concussion? _____

Were you dazed, confused and/or disorientated?

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 removed from the game?	Yes	No
If Yes, for how long? _____		
Were you hospitalised?	Yes	No
If Yes, for how long? _____		
Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?		
	Yes	No
If Yes, please specify: _____		

Injury 3

What caused the injury/concussion? _____

Were you dazed, confused and/or disorientated?	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 removed from the game?	Yes	No
If Yes, for how long? _____		
Were you hospitalised?	Yes	No
If Yes, for how long? _____		
Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?		
	Yes	No
If Yes, please specify: _____		

APPENDIX III

Assessment Schedule and Neuropsychological Test Battery

NEUROPSYCHOLOGICAL TESTING
ASSESSMENT SCHEDULE

Name: _____

Date: _____

Tester: _____

Time Test

1. Demographic Questionnaire
2. Postconcussive Symptom Checklist
3. Neuropsychological Tests:
 - 3.1 WMS Associate Learning – Immediate Recall
 - 3.2 Digit Symbol Substitution
 - 3.3 Digit Symbol Substitution Incidental Recall – Immediate
 - 3.4 Trail Making Test – Parts A and B
 - 3.5 Picture Completion
 - 3.6 WMS Associate Learning – Delayed Recall
 - 3.7 Digit Symbol Substitution Incidental Recall – Delayed
 - 3.8 Digit Span – Forwards
 - 3.9 Digit Span – Backwards
 - 3.10 Vocabulary

WMS : ASSOCIATE LEARNING - IMMEDIATE RECALL

Testee's Name: _____

Requirements: Lists of words [below, or on answer sheet]

NOT TIMED

Instruction: "I am going to read you a list of words, 2 at a time. Listen carefully, because after I am finished I shall want you to remember the words that go together. For example, if the words were EAST-WEST; GOLD-SILVER; then when I would say the word EAST, I would expect you to answer (*pause*) WEST. And when I say the word GOLD, you would of course, answer (*pause*) SILVER. Do you understand?"

"Now listen carefully to the list as I read it." *P.T.O. for list of words.*

SCORE:

<u>First Recall</u>	<u>Second Recall</u>	<u>Third Recall</u>
TOTAL _____	TOTAL _____	TOTAL _____

Easy: 1. _____
2. _____
3. _____
A Total _____

Hard: 1. _____
2. _____
3. _____
B Total _____

Score: $A/2 + B =$ _____

Read 1 pair every 2 seconds.

First Presentation

Metal - Iron
 Baby - Cries
 Crush - Dark
 North - South
 School - Grocery
 Rose - Flower
 Up - Down
 Obey - Inch
 Fruit - Apple
 Cabbage - Pen

Second Presentation

Rose - Flower
 Obey - Inch
 North - South
 Cabbage - Pen
 Up - Down
 Fruit - Apple
 School - Grocery
 Metal - Iron
 Crush - Dark
 Baby - Cries

Third Presentation

Baby - Cries
 Obey - Inch
 North - South
 School - Grocery
 Rose - Flower
 Cabbage - Pen
 Up - Down
 Fruit - Apple
 Crush - Dark
 Metal - Iron

Wait 5 seconds before beginning to test the recall and then wait at least 5 seconds before moving onto the next pair.

First Recall

Easy Hard

North _____
 Fruit _____
 Obey _____
 Rose _____
 Baby _____
 Up _____
 Cabbage _____
 Metal _____
 School _____
 Crush _____

TOTAL _____

Easy: 1. _____
 2. _____
 3. _____
 A Total _____

Second Recall

Easy Hard

Cabbage _____
 Baby _____
 Metal _____
 School _____
 Up _____
 Rose _____
 Obey _____
 Fruit _____
 Crush _____
 North _____

TOTAL _____

Hard: 1. _____
 2. _____
 3. _____
 B Total _____

Third Recall

Easy Hard

Obey _____
 Fruit _____
 Baby _____
 Metal _____
 Crush _____
 School _____
 Rose _____
 North _____
 Cabbage _____
 Up _____

TOTAL _____

Score: A/2 + B = _____

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 $1\frac{1}{2}$ 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 $1\frac{1}{2}$ 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.

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 $\mathcal{N}$, subjects sometimes use the letter "N". Grant $\frac{1}{2}$ -point for each such reversal.

On the other hand,

L	for	L	
V	or	U	for U
U	for	H	
and 3, >	or	7	for □

do not score.

Maximum Score: 67

DIGIT SYMBOL SUBSTITUTION

Testee's Name: _____

Requirements: Test sheet
 Pencil
 Stop Watch

TIMED

Time Limit: 90 seconds (1 minute 30 seconds)

Instructions: *Place the Digit Symbol sheet in front of the subject and indicate the key at the top.*
 *“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 boxes, but this time they only have the numbers at the top and the spaces below 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 fill in the 2-sign*). The next is a 1, 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 they subject begins erasing or correcting an incorrect solution tell him to leave it out and go on with the rest.

IMPORTANT:

Make a note of how many the subject completes in 1½ minutes but allow him to finish up to the end of the second last horizontal line (or 42 blocks from the beginning of the test). If the subject has passed this point during the test then carry on with incidental recall.

- 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	U	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*	Aantal half korrek	120*	TOTAAL	120*
Number correct	90*	Number half correct	90*	TOTAL	90*

DIGIT SYMBOL SUBSTITUTION – INCIDENTAL RECALL

Testee's Name: _____

Requirements: Test sheet
 Pencil

NOT TIMED

Instructions: *Place the Digit Symbol Incidental recall sheet in front of the subject.*
 “See how many of the symbols used in the previous test you are able to remember. There is no time limit and you can do them in any order you wish.”

SCORE:

Number remembered correctly: _____

- 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

Adult and Child

RHODES UNIVERSITY PSYCHOLOGY CLINIC

Name: _____

Clinician: _____

Date: _____

Requirements: test sheets (4 pages: adult or child)
pencil
Stop watch

TIMED Time to complete each trail

Time Limit: No

Instructions:

TRAIL A:

SAMPLE:

"Draw a line to connect the circles consecutively from 1 to 8, without lifting your pencil, as fast as you can."

TEST:

Showing the subject the test sheet and pointing out the first 3 or 4 circles which must be joined give the following instruction:

"Now draw a line to connect the circles consecutively from 1 to 25 (*children: 1 to 15*), without lifting your pencil, and do it as fast as you can."

Record time taken to join all the circles in the correct order.

TRAIL B:

SAMPLE

"Draw a line to join the circles consecutively by alternating between numbers and letters starting with 1 then A. Do it as fast as you can."

TEST:

Showing the subject the test sheet and pointing out the first 3 or 4 circles which must be joined give the following instruction:

"Draw a line to join the circles consecutively by alternating between numbers and letters starting with 1 then A and finishing with 13 (*children: finish with 8*). Do this as fast as you can."

Record time taken to join all the circles in the correct order.

Note: If subject makes mistake, don't stop timing; point out mistake and subject corrects the error and carries on.

SCORE:

Adult/Child

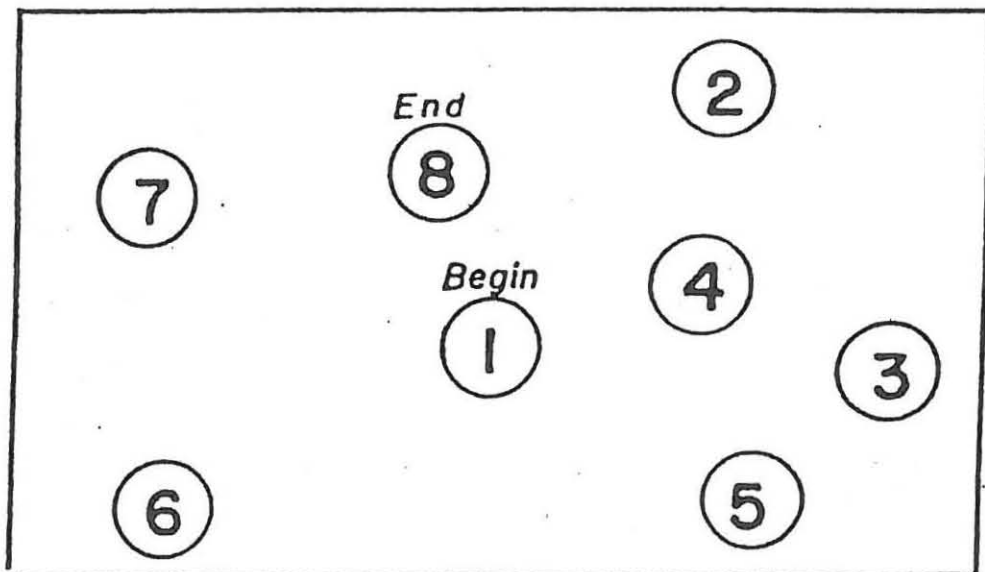
Trail A: _____

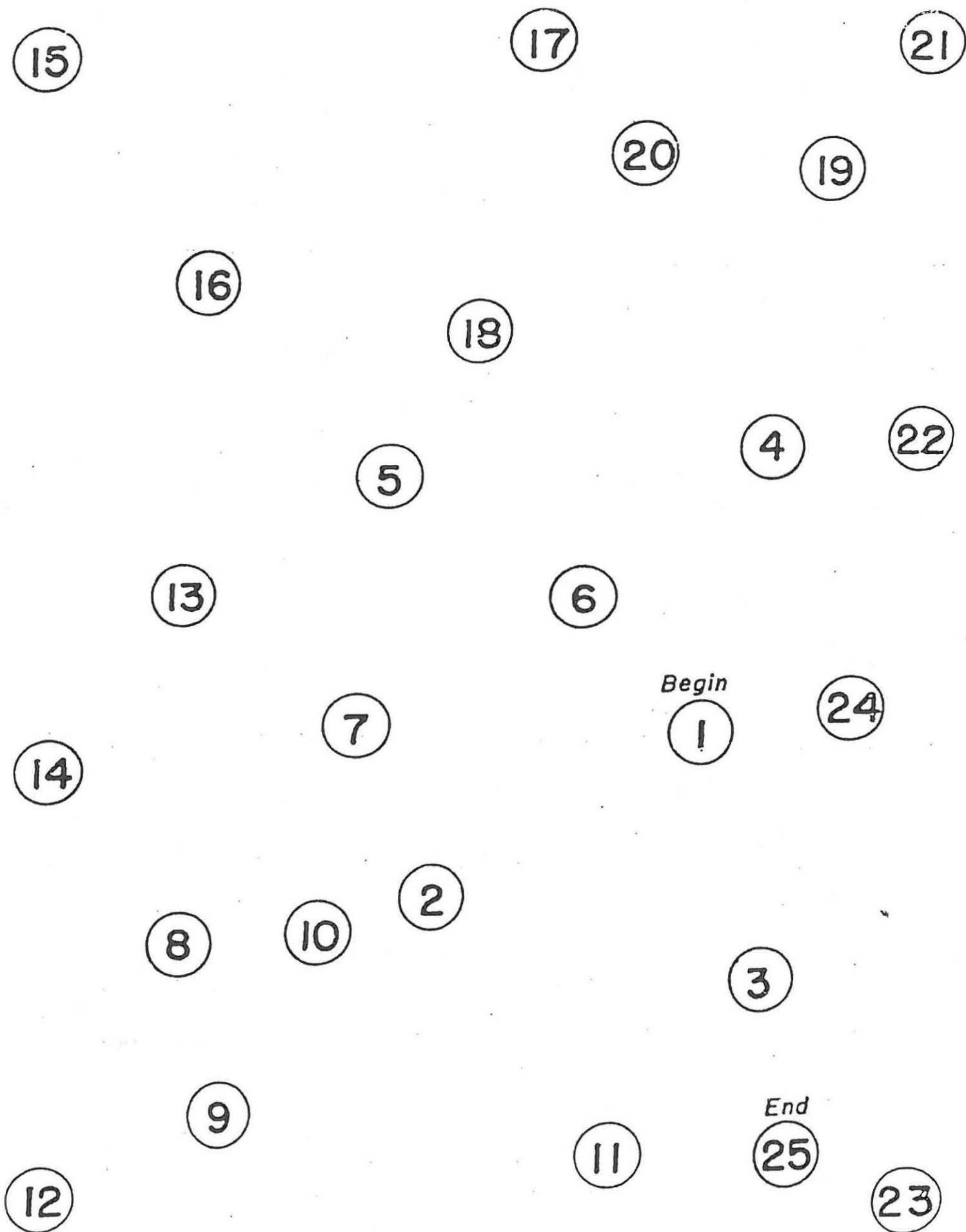
Trail B: _____

TRAIL MAKING

Part A

SAMPLE

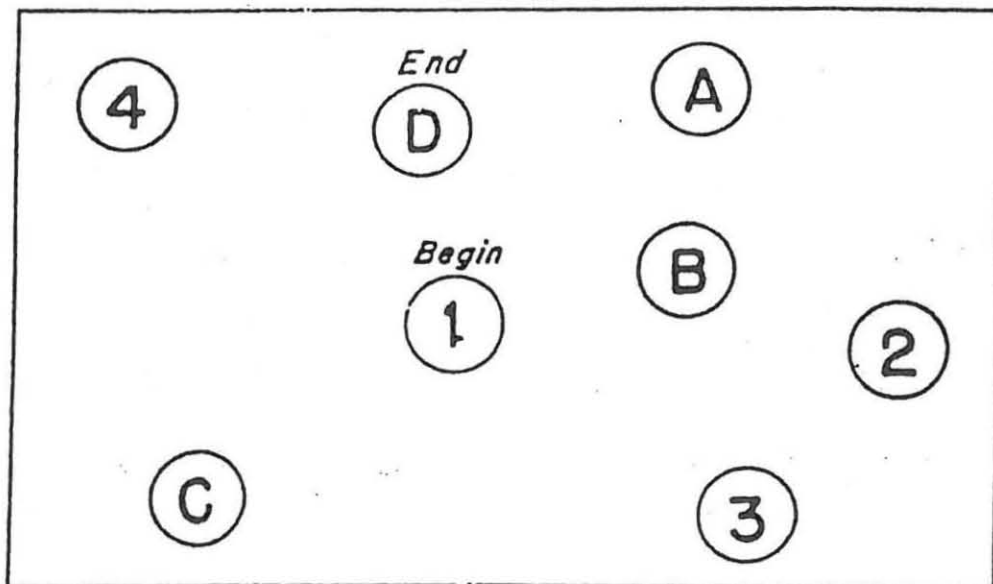




TRAIL MAKING

Part B

SAMPLE



End

13

10

8

9

I

D

B

4

3

Begin

1

7

H

5

C

12

G

A

J

L

2

6

E

11

F

PICTURE COMPLETION

Testee's Name: _____

Requirements: Picture Completion Items
 Pencil
 Stopwatch

Time limit: 20" per card to respond

Instructions: "I am going to show you some pictures in which there is some important part missing. Look at each picture and tell me what is missing?"

Start: Start on Item 6. If subject obtains perfect scores (1 point) on Items 6 and 7, give full credit for Items 1-5. If subject scores 0 on either Items 6 or 7, administer Items 1 - 5 in *reverse* sequence until the subject obtains perfect scores (1 point) on *two* consecutive items.

Place Picture Completion Items in front of subject, starting at Item 6 and say:

"Now, look at this picture. What important part is missing?"

Continue with succeeding items saying:

"Now, what is missing in this one?"

If the participant fails Items 6 or 7, point and say:

"You see the doorknob/the bridge or nose piece is missing." No other "teaching" may be offered on any other item.

Each of the following prompts may only be used once:

If the subject merely names the object pictured rather than the missing part, say:

"Yes, but what's missing?"

If the subject mentions a part that is off the page (e.g., the hand that holds the pitcher in Item 8), note the response on the Record Form and say:

"Something is missing in the picture. What is it that is missing?"

If the subject mentions an unessential missing part (e.g., the life jacket in Item 18), note the response on the Record Form and say:

"Yes, but what is the most important part that is missing?"

Record the response verbatim on the response form below.

Discontinue: Discontinue after five consecutive scores of 0.

Item	Response	Score (0 or 1)
1. Comb		
2. Table		
3. Face		
4. Briefcase		
5. Train		
6. Door		
7. Glasses		
8. Pitcher		
9. Pliers		

Item	Response	Score (0 or 1)
10. Leaf		
11. Pie		
12. Jogging		
13. Fireplace		
14. Mirror		
15. Chair		
16. Roses		
17. Knife		
18. Boat		

Item	Response	Score (0 or 1)
19. Basket		
20. Clothing		
21. Lockers		
22. Cow		
23. Tennis Shoes		
24. Woman		
25. Barn		
Total Raw Score (Maximum = 25)		

WMS ASSOCIATE LEARNING DELAYED RECALL

Testee's Name: _____

Requirements: Lists of words [below, or on answer sheet]

NOT TIMED

Instruction: "Remember the pairs of words I read you earlier. I want you to see how many pairs you remember."

First Recall	Easy	Hard
North	_____	
Fruit	_____	
Obey		_____
Rose	_____	
Baby	_____	
Up	_____	
Cabbage		_____
Metal	_____	
School		_____
Crush		_____
TOTAL	_____	_____

SCORE:

Delayed recall = _____

DIGIT SYMBOL SUBSTITUTION – DELAYED RECALL

Testee's Name: _____

Requirements: Test sheet
 Pencil

NOT TIMED

Instructions: *Place the Digit Symbol Incidental recall sheet in front of the subject.*
 "I would like to see how many of the symbols used in the earlier test you are still able to remember. There is no time limit and you can do them in any order you wish."

SCORE:

Number remembered correctly: _____

- Λ. SYFERS VERVANG DEUR SIMBOLE.
X. DIGIT SYMBOL SUBSTITUTION. - DELAYED

NAAM
NAME

Datum

Date

SLEUTEL
KEY

1	2	3	4	5	6	7	8	9

SA WAIS DIGIT SPAN

Testee's Name: _____

Requirements: SA WAIS Manual, p 29 [or below]
SA WAIS record form [or below]
pencil

Not timed

Instruction: **DIGITS FORWARD:**

"I am going to say some numbers. Listen carefully and when I have finished say them right after me." *Say the numbers in an even tone, one number per second.*

They fail the test after the incorrect repetition of both trials of a span. At this point the Digits Forward 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 one set of 9 correct but fail both sets of 10, their score is 9. If they get 12 digits forward correct - then improvise until you have established their span - ie. until they fail twice in a row.

- | | | |
|-----|------------------------------------|------------------------------------|
| 3. | 5, 8, 2 | 6, 9, 4 |
| 4. | 6, 4, 3, 9 | 7, 2, 8, 6 |
| 5. | 4, 2, 7, 3, 1 | 7, 5, 8, 3, 6 |
| 6. | 6, 1, 9, 4, 7, 3 | 3, 9, 2, 4, 8, 7 |
| 7. | 5, 9, 1, 7, 4, 2, 3 | 4, 1, 7, 9, 3, 8, 6 |
| 8. | 5, 8, 1, 9, 2, 6, 4, 7 | 3, 8, 2, 9, 5, 1, 7, 4 |
| 9. | 7, 5, 8, 3, 6, 3, 2, 7, 9 | 4, 2, 7, 3, 1, 8, 1, 2, 6 |
| 10. | 6, 1, 9, 4, 7, 3, 5, 2, 9, 4 | 4, 7, 3, 9, 1, 2, 8, 3, 2, 7 |
| 11. | 7, 4, 8, 6, 4, 9, 5, 8, 5, 3, 1 | 2, 6, 4, 9, 7, 3, 6, 1, 8, 5, 3 |
| 12. | 8, 2, 5, 3, 7, 4, 6, 9, 2, 5, 3, 6 | 1, 7, 3, 6, 9, 5, 7, 2, 8, 4, 1, 8 |

P.T.O. for Digit Supraspan A and B.

DIGITS BACKWARD

"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 say(wait for them to say 9 - 2 - 6)."

The test is failed after 2 consecutive failures of a span on Digits Backwards, and the score is the highest backwards span achieved.

- | | | |
|----|---------------------------|---------------------------|
| 2. | (2, 4) | (5, 8) |
| 3. | 2, 8, 3 | 4, 1, 5 |
| 4. | 3, 2, 7, 9 | 4, 9, 6, 8 |
| 5. | 1, 5, 2, 8, 6 | 6, 1, 8, 4, 3 |
| 6. | 5, 2, 9, 4, 1, 8 | 7, 2, 4, 8, 5, 6 |
| 7. | 8, 1, 2, 9, 3, 6, 5 | 4, 7, 3, 9, 1, 2, 8 |
| 8. | 4, 7, 2, 6, 9, 1, 5, 8 | 7, 2, 8, 1, 9, 6, 5, 3 |
| 9. | 2, 8, 4, 1, 7, 9, 5, 4, 6 | 8, 6, 9, 3, 5, 7, 1, 4, 2 |

SCORE:

Digits Forwards: _____

Supraspan A: _____

Supraspan B: _____

Digits Backwards: _____

Digits Difference: _____ (Forwards minus Backwards)

VOCABULARY

Testee's Name: _____

Requirements: Vocabulary Cards
 Sample responses
 Record Form
 Pencil

Instructions: "In this section, I want you to tell me the meaning of some words. Now listen carefully and tell me what each word I say means. Are you ready?"

Start: Start on Item 4. If subject obtains perfect scores (2 points) on Items 4 and 5, give full credit for Items 1-3. If subject scores 0 or 1 on either Items 4 and 5, administer Items 1 - 3 in *reverse* sequence until the subject obtains perfect scores (2 points) on *two* consecutive items.

Locate Vocabulary card with Item 4 on it and place it in front of the subject. Simultaneously point to and say: "Tell me what _____ means."

Record the response verbatim on the Record Form. Use the Sample Responses as scoring guidelines. If the subject's response is unclear or too vague you may say: "Tell me more about it" or "Explain what you mean".

Discontinue: Discontinue after *six* consecutive scores of 0.

Item	Response	Score (0, 1, or 2)
1. Bed		
2. Ship		
3. Penny		
4. Winter		
5. Breakfast		
6. Repair		
7. Assemble		

2. Vocabulary *(continued)*

Item	Response	Score (0, 1, or 2)
8. Yesterday		
9. Terminate		
10. Consume		
11. Sentence		
12. Confide		
13. Remorse		
14. Ponder		
15. Compassion		
16. Tranquil		
17. Sanctuary		
18. Designate		
19. Reluctant		
20. Colony		
21. Generate		
22. Ballad		
23. Pout		
24. Plagiarize		
25. Diverse		
26. Evolve		
27. Tangible		
28. Fortitude		
29. Epic		
30. Audacious		
31. Ominous		
32. Encumber		
33. Tirade		
Total Raw Score (Maximum = 66) (Include credit for Items on previous page.) 