CUMULATIVE MILD HEAD INJURY IN RUGBY: A COMPARISON OF COGNITIVE DEFICIT AND POSTCONCUSSIVE SYMPTOMATOLOGY BETWEEN SCHOOLBOY RUGBY PLAYERS AND NON-CONTACT SPORT CONTROLS

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TARYN BEILINSOHN

Rhodes University

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ABSTRACT

This study investigates the cumulative effects of concussive and subconcussive mild head injury on the cognitive functioning of schoolboy rugby players. A comprehensive battery of neuropsychological tests and a self-report postconcussive questionnaire were administered to top level schoolboy rugby players (n=47), and a non-contact sport control group of top level schoolboy hockey players (n=34). Group comparisons of the percentage of individuals with cognitive deficit were carried out between i) the schoolboy rugby and the schoolboy hockey players, ii) the rugby forward and the rugby backline players; iii) the rugby forward and the schoolboy hockey players and, iv) the rugby backline and the schoolboy hockey players. Results on the neuropsychological test battery did not provide any substantial evidence of a higher level of neuropsychological impairment in the rugby players relative to the control group, or in the rugby forward players relative to the rugby backline players. Results obtained on the postconcussive symptom questionnaire provided tentative indications that the rugby players do report a greater frequency of postconcussive symptomatology. The symptoms most frequently reported were being easily angered, memory problems, clumsy speech and sleep difficulties. It was hypothesized that the absence of cognitive impairment in the schoolboy rugby players compared with that noted for professional players was due to their younger age, relatively high IQ and education level and a less intensive level of physical participation in the sport, and hence less accumulated exposure to the game, thereby decreasing their exposure to mild head injuries. From a theoretical perspective, these pre-existing conditions were considered to act as protective factors against reductions in brain reserve capacity and concomitant susceptibility to the onset of neuropsychological dysfunction.

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CHAPTER ONE: INTRODUCTION

1.1. RESEARCH CONTEXT

This study forms part of a larger and ongoing research initiative into the cumulative effects of concussive and subconcussive brain injury in rugby. The research project was initiated in 1996 by Rhodes University in collaboration with the South African Rugby and Football Union (SARFU) and the South African Sports Science Institute in Cape Town. To date, the project has comprised two distinct phases. The initial phase compared the neuropsychological test performances of Springbok rugby players with those of a matched non-contact sport control group of professional cricket players (the Proteas). This data formed the basis of three separate research studies, which were analysed according to three levels of statistical analysis namely: i) a direct comparison of means for rugby and cricket groups (Ancer, 1999); ii) a comparison of rugby and cricket groups, relative to normative data (Reid, 1998); and iii) a comparison of the percentage of cognitive deficit and postconcussive symtomatology in rugby and cricket groups (Dickinson, 1998). Preliminary findings from these studies indicated the presence of deficits in concentration, attention and memory in the rugby players relative to the cricket players. Positional variation within rugby was also noted, with rugby forward players demonstrating disproportionately poor performances relative to backline players, on tests sensitive to diffuse brain damage.

While this initial study provided important baseline data, its findings were partially compromised by a number of methodological limitations that included a small sample and a problematic control group (many of the cricket players had a rugby-playing history). In an attempt to address these methodological weaknesses, a second phase of research was instituted, which compared the neuropsychological test performances of both Springbok rugby players and Under 21 national rugby players (thereby increasing the sample size) with a more appropriate matched non-contact control group of national hockey players (hockey players did not have a rugby playing history as both sports are winter sports). This study replicated the preceding one, in that data were analysed according to the same three levels of analysis previously noted, and findings from these studies corroborated earlier research. In light of these positive findings, it became evident that it might be necessary to give consideration to whether a similar pattern of cognitive deficits could be found in earlier stages of rugby participation, such as schoolboy rugby playing.

This pertinent issue, namely whether cognitive deficits would be evidenced amongst schoolboy rugby players, comprised the focus of the third and current phase of research. This study concentrated on a younger population of high school rugby players as its sample for analysis and compared the

neuropsychological test performances of a large sample of high school rugby players with a matched control group of high school hockey players. While the study utilised the first and third levels of statistical analyses previously employed (direct comparison of group means and comparisons of the percentage of cognitive deficit and frequency of postconcussive symptomatology), it differed from the former studies in that players were tested during the sport season. The rationale for this was that it would allow for the detection not only of permanent effects, but also the overlay of any acute effects sustained during the season. Given the importance of establishing the impact of participation in rugby at school level on scholastic performance and everyday functioning, it was hypothesized that testing later into the season would provide a more comprehensive estimate of the full extent of intellectual difficulties that would be relevant for scholastic performance. This was deemed to have potentially significant consequences for those scholars who were attempting to gain admission into demanding and competitive tertiary programs.

The focus for the present study will be on the third level of analysis, namely, a comparison of the percentage of cognitive deficit of school rugby and hockey players relative to normative data as well as a comparison of the frequency of postconcussive symptomatology for rugby and hockey players. It will replicate Dickinson's (1998) and Border's (2000) methodology; the strength of which lies in its provision of an analysis of the distribution of deficit among individual players, as distinct from average effects, which constitute the first level of analysis. This study will attempt to maintain a dual focus on both cognitive deficits and postconcussive symptomatology, following cumulative mild head injury.

1.2. RESEARCH QUESTION

While the neuropsychological sequelae of mild head injury have been extensively documented (for example, Barth et al., 1983; Gentilini, Nichelli, & Schoenhuber, 1989), there appears to be a relative lack of empirical studies on the cumulative effects of repeated mild head injury. This has particular relevance for athletes participating in contact sports, as the physical nature of the play predisposes these players to far greater risk for sustaining repeated mild head injuries.

Initially, neuropsychological research into contact sports focused predominantly on boxing. More recently, research studies have appeared that focus on the contact sports of soccer, American Football, Australian Rules Football and Rugby League (for example, Abreau, Templer, Schuyler, & Hutchison (1990); Barth, et al., 1989; Hinton-Bayre, Geffen, & McFarland, 1997; Maddocks & Saling, 1991), although these studies have generally been confined to the United States, Australia and Europe. With respect to Rugby Union, the only studies to this author's knowledge have been those

conducted in South Africa, where an attempt has been made to address the paucity of research in this area. Results from this growing body of research in South Africa have consistently demonstrated the negative effects of cumulative mild head injuries (Ancer, 1999; Bold, 1999; Border, 2000; Dickinson, 1998; Finkelstein, 1999; Reid, 1998; Shuttleworth-Jordan, Balarin, & Puchert, 1993). These findings have important implications for South Africa, as rugby is an integral part of South-African culture and national image, and is the sport most frequently encouraged and participated in, in both English and Afrikaans-medium schools. The immense popularity of this sport may result in schoolboys feeling that they have little choice regarding participation. Since this sport is one that has come to be associated with strength and endurance and tends to earn its participants popularity and respect, adolescent males in particular, may face the pressure of feeling socially excluded and judged by their peers should they demonstrate a reluctance to participate in it. This is particularly worrying since South-African incidence studies report that concussion is the single most common injury in schoolboy rugby, making up 20% of all injuries and that as instances of concussion often go unreported, the incidence rate may in fact be even higher (Roux, Goedecke, Visser, Van Zyl, & Noakes, 1987). Given this consideration, it is crucial that investigations into the neuropsychological effects of head injuries sustained at a school level be undertaken. The indication for such a study is evidenced by the lack of research in this area, as studies at school level have been restricted to incidence rates and have not focused on the cognitive-behavioral sequelae of cumulative mild head injury.

The research question that is posed is whether high school rugby players will exhibit cerebral dysfunction, evidenced by impaired performance on neuropsychological measures, as a result of repeated head trauma. Given the earlier findings of phases one and two of the project on professional players, it is hypothesized that high school rugby players will show cognitive impairment on those tests sensitive to diffuse brain damage and that rugby forward players will perform disproportionately poorer relative to rugby backline players on those same tests. It is further hypothesized that schoolboy rugby players will report a greater frequency of postconcussive symptomatology relative to schoolboy hockey players and that rugby forward players will report a greater frequency of postconcussive symptomatolgy relative to rugby backline players. The present study will be located in the theoretical framework of Brain Reserve Capacity theory (Satz, 1993), in order to elucidate its findings.

CHAPTER TWO: LITERATURE REVIEW

This chapter attempts to locate mild head injury within the spectrum of traumatic brain injury, and focuses on definition, incidence, pathophysiology, mechanisms of injury and research findings on the cognitive-behavioural sequelae associated with mild head injury. This is followed by a discussion of mild head injury in sport, with particular reference to neuropsychological findings in the contact sports of boxing, soccer, American Football, Australian Rules Football, Rugby League and Rugby Union. Finally, a theoretical context for understanding outcomes for mild head injury will be proposed, with a particular focus on the implications for the present research question.

2.1. MILD HEAD INJURY: UNDERSTANDING TERMINOLOGY

2.1.1. MAJOR TYPES OF HEAD INJURY

When discussing head injury, a distinction is usually drawn between open/penetrating head injuries and closed head injuries. This is because open and closed head injuries differ both in relation to the nature of the injury and to the neuropsychological impairments to which they tend to give rise. Open head injuries occur as a result of puncture wounds, missile fragments and low velocity bullets, which cause laceration of the scalp, perforation of the skull and laceration of brain tissue and result in significant tissue damage that is generally concentrated in the path of the object (Levin, Benton, & Grossman, 1982). These injuries tend to be rare, accounting for less than 10% of all reported head trauma in the civilian population (Richardson, 1990).

In contrast, closed head injuries, which are the focus of the present study, are more common and range in severity from mild to moderate to severe. They typically result from blunt trauma to the head (Richardson, 1990). This usually occurs as a result of the force of a moving object while the head is still or moving slowly (resulting in acceleration) or by decelerative forces which occur when the head and body are brought to a sudden standstill by contact with a slower moving or stationary object (Levin et al., 1982; Lezak, 1995; Richardson, 1990). While these accelerative and decelerative forces can result in focal lesions, causing localized effects, they more frequently result in diffuse damage which is associated with widespread disruption of neurological function and often includes impairments in attention, concentration and memory. Since rotational acceleration is viewed as the primary mechanism of brain damage in closed head injury (Bruno, Gennarelli, & Torg, 1987), mild head injuries can in fact occur without a direct blow to the head (Lezak, 1995; Sweeny, 1992 in Gasquoine, 1997). Furthermore, structural damage can occur in injuries associated with only a brief period of dazed consciousness (Alexander, 1985), the briefest period of unconsciousness

(Oppenheimer, 1968), and even in instances where there has been no loss of consciousness (Cantu, 1986; Evans, 1992).

2.1.2. CLASSIFICATION OF HEAD INJURY: INDICATORS OF SEVERITY

Severity is generally regarded as a defining factor and predictor of outcome in the assessment of head injury (Anderson, 1996). It is usually classified during the acute period of hospitalization and is made on the basis of the following symptoms: alterations in consciousness level, loss of consciousness and changes in orientation and memory (Satz et al., 1997). These will be briefly examined.

The Glasgow Coma Scale (GCS) is routinely used to assess the level of severity of both coma and impairment of consciousness and has proved to have predictive value in pointing to long-term outcome, both in terms of survival and ultimate levels of disability (Lishman, 1987). It is a classification system based on the presence and results of different degrees and durations of trauma, which assesses a patient's verbal, ocular and motor responses to simple stimuli (Teasedale & Jennet, 1974). Injuries are classified using a 15-point scale into three groups of severity: severe (3 to 8), moderate (9 to 12) and mild (13 to 15). While the GCS is a useful screening instrument for evaluating the depth of coma in severe head injury, it is not designed to quantify mild disturbances of consciousness or post-traumatic amnesia (PTA) associated with mild head injury (Levin, Eisenberg, & Benton, 1989). Furthermore, it has been argued that the GCS has crude scoring categories, and that two patients with the same score may not function at the same level (Eisenberg & Weiner, 1987 in Binder, 1997). GCS scores may also be compromised by several factors, including alcohol intake, drugs and metabolic alterations due to injuries not involving the brain. In addition, scores are taken on admission and do not account for later deterioration (Lezak, 1995).

One of the defining characteristics of mild head injury is a loss of consciousness (LOC) of less than 30 minutes (Evans, 1992; Rimel, Giordani, Barth, Boll &, Jane, 1981). However, as in the case of GCS, LOC is often regarded as a more useful predictor for severe head injuries, as mild head injury can occur in the absence of a loss of consciousness (Anderson, 1996; Cantu, 1996; Evans, 1992; Rutherford, Merret, & Mac Donald, 1977). According to Lishman (1987), the longer the duration of unconsciousness, the more probable it is that permanent damage has been sustained. This will manifest on neurological examination as raised intercranial pressure and the presence of blood in the cerebral spinal fluid. Similarly, Teasedale and Mandolow (1984, in King, 1997) argue that the longer the period of unconsciousness, the higher the degree of diffuse axonal injury. Lengthier periods of

unconsciousness are also likely to be followed by a considerable period of post-traumatic confusion and physical and mental long-term sequelae (Lishman, 1987).

The third indicator of severity, PTA, refers to the time from the moment of injury to the interval the patient becomes aware that he/she has regained consciousness. The latter corresponds to the time when the patient begins to retain a stable record of ongoing events (Walsh, 1985). According to McAllister (1992, in Busch & Alpern, 1998), this time period is one of confusion and disorientation, characterized by an inability to recall events, sequence time or learn new information. While Lishman (1987) argues that PTA is a valid and useful predictor of retrograde amnesia, and while findings have indicated a good correlation between duration of PTA and GCS scores (Evans, 1992; Levin et al., 1982), some authors maintain that PTA must be used with caution by the clinician (for example, Binder, 1997; Satz et al., 1997). This is because PTA relies heavily on the subjective judgment of the doctor taking the patient's history and as this is always retrospective, it is difficult to establish the exact duration of PTA (Gronwall & Wrightson, 1981 in Binder, 1997). PTA can be underestimated due to islands of memory (Gronwall & Wrightson, 1980 in King, 1997) or overestimated by including periods of natural sleep or impaired consciousness, due to alcohol, medication or drugs (Whitty & Zangwill, 1977 in King, 1997).

Despite some inherent weaknesses, GCS, LOC and PTA measures still remain the most useful indicators of severity and, at present, there is no biologically objective measure that quantifies the severity of neuropathology more accurately than these three measures (Alexander, 1995).

2.1.3. CLASSIFICATION AND DEFINITION OF MILD HEAD INJURY

While the above measures (GCS, LOC and PTA) provide useful predictors of outcome in instances of severe head injury, they cannot be applied with as much certainty to those of mild head injury, due to the transient and variable nature of its associated symptoms (Satz et al., 1997). Mild head injury thus remains the least understood of the degrees of head injury, even though it accounts for 50-70% of documented head injuries (Dicker, 1989). Inconsistencies in inclusion criteria and measures of severity have tended to produce conflicting results, thereby creating difficulty in achieving a uniform understanding of the neurobehavioral outcome of mild head injury. Many of these difficulties can be attributed to significant variability in the defining criteria for mild head injury. In his meta-analytic review, Binder (1997) notes that despite extensive research, there is still no accepted definition of mild head injury that has achieved widespread usage.

This lack of a uniform definition of mild head injury constitutes a major problem in both research and clinical practice and has led to a recent attempt by Esselman and Uomoto (1995, in Satz et al., 1997), in agreement with the recommendations of the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitative Medicine, to propose a definition of mild head injury. Mild head injury was thus defined as the presence of at least one of the following criteria: (a) a duration of loss of consciousness of 30 minutes or less with a GCS rating of 13-15; (b) any loss of memory for events immediately preceding or following the accident with PTA of less than 24 hours; (c) any change in the mental state at the time of the accident (e.g. dazed, disoriented, or confused); and (d) focal neurological deficits that may or may not be transient (e.g. double vision, loss of balance, taste or smell). While this definition appears to cover a broad range of severity, and encourages the investigation of patients without hospital admission, it has not escaped criticism. Satz et al. (1997) argue that a weakness of this definition lies in its usage of arbitrary and a priori cut-off points which are not empirically validated in order to designate grades of severity. Similarly, Kibby and Long (1996) argue that this definition creates an overlap between mild and moderate severity as several studies have defined mild traumatic brain injury as that occurring with PTA under one hour and moderate traumatic brain injury associated with PTA ranging from 1 to 24 hours. It is also noteworthy that this definition allows for the presence of a focal neurological deficit, which does not occur in all instances of mild head injury, particularly in its least severe forms.

While a number of researchers have attempted to define mild head injury (Alexander, 1995; Dikmen, McLean, & Tempkin, 1986; Rimel et al., 1981), Evans (1992) provides a definition for mild head injury which appears closely to approximate the recommendations proposed by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitative Medicine, specifically with reference to their first three criteria, as noted above. His defining criteria include: LOC of less than 30 minutes, PTA of less than 24 hours, alteration in mental state at the time of the accident and an absence of focal neurological deficits. Despite their similarity, it becomes evident that Evans' definition, in contrast to that of the above-mentioned Mild Traumatic Brain Injury Committee (of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitative Medicine), to some extent alleviates the problematic overlap between the categories of mild and moderate head injury. This is because by including a fourth criteria that excludes the presence of neurological deficits, Evans' definition can be seen to pertain more specifically to the category of mild head injury. Since Evans' defining criteria were applied to phases one and two of the research on professional players on the basis that they were initially formulated by Evans specifically for use in research contexts (Evans, 1992), it was decided

that they would be adopted for the purposes of the current study, both for their utility value and to maintain continuity between different stages of the research project.

Further complicating the whole issue of mild head injury is the term 'concussion" and its relationship to mild head injury. It is important to differentiate between the terms concussion and mild head injury for while they are often used interchangeably in the literature, the term "concussion" is generally used to refer to instances of a particular type of head injury, specifically, a closed head injury (i.e. blunt nonpenetrating head injury) or a whiplash injury (where there is no direct impact to the head), caused by rapid acceleration/deceleration inside the skull (Lezak, 1995). In the following section, the concept of concussion will be examined in more detail and the relationship of concussion to mild head injury will be further explicated.

2.1.4. CLASSIFICATION AND DEFINITION OF CONCUSSION

The assessment and defining criteria of concussion and its severity remain controversial (Anderson, 1996; Cantu, 1996; McCrory 1997). As with mild head injury, there is a lack of universal agreement on a standard definition of concussion. This is because concussion is understood to exist along a continuum of severity, ranging from direct head trauma as a result of a collision to the absence of a direct blow to the head when sufficient force is applied to the brain, as occurs in whiplash (Alexander, 1995). Much of the variation in definitions and classifications of the severity of concussion appear to stem from researchers' tendencies to differ in their use of measures such as duration of LOC, PTA and retrograde amnesia, making evaluation of epidemiological data difficult (Cantu, 1986). One definition that has gained a measure of acceptance as a working definition of concussion is the one proposed by the Committee on Head Injury Nomenclature of the Congress of Neurological Surgeons. They define it as "a clinical syndrome characterized by immediate and transient post-traumatic impairment of neural function, such as alteration of consciousness, disturbance of vision, equilibrium, etc., due to brainstem involvement " (1993 in Cantu, 1986, p. 70). However, even this definition has not escaped criticism, due to research findings of more protracted periods of recovery and the persistence of cognitive effects.

Lezak (1995) defines concussion as the effects of immediate disturbances in neurological functions created by the mechanical forces of rapid acceleration/deceleration of the brain inside the skull. Rutherford (1989, p. 217) regards it as an "acceleration/ deceleration injury to the head almost always associated with a period of amnesia, and followed by a characteristic group of symptoms such as headache, poor memory and vertigo". While both definitions lay emphasis on the accelerative/ decelerative forces that occur during concussion, other researchers maintain that rapid angular

acceleration alone is sufficient to set these forces in motion and that concussion does not therefore require a direct impact to the head (Boll, 1993; Evans, 1992; Gennarelli, 1987). Given that impairment is not always discernible and can manifest as a transient dizziness with momentary confusion and disorientation, various authors have attempted to take cognizance of this when proposing graded classification systems of severity (for example, Ommaya & Gennarelli, 1974).

The need for graded classification systems of severity has arisen from the fact that one of the most frequently encountered problems of sports medicine practitioners is the assessment of concussion. In an attempt to address this problem and provide better management guidelines for on-field injuries, various classification systems have been proposed (Bruno et al., 1987; Colorado Medical Society, 1991; Kulund, 1982; Maroon, Steele, & Berlin, 1980; McCrory, 1997; Nelson, Jane, & Grieck, 1984; Torg, 1982). None, however, have achieved as widespread usage as Cantu's (1986) classification system which, due to its simplicity and accessibility, tends to be the system with which most sports medicine practitioners are familiar with (see Table 2-1, below). This classification system utilizes the duration of LOC and PTA, in order to differentiate between mild, moderate and severe concussive injury.

Table 2-1. Cantu's Classification System of Severity of Concussion

Grade	Description and outcome
I (mild)	No LOC and PTA < 30 minutes
II (moderate)	LOC < 5 minutes and PTA 30 minutes to < 24 hours
III (severe)	LOC $\geq$ 5 minutes and PTA $\geq$ 24 hours

(Cantu, 1986)

While Cantu's system is a useful one, its limitations have been noted. According to McCrory (1997), not only does the scale lack scientific validation, but LOC may be difficult to detect if it is only momentary. Cantu (1992) himself has acknowledged this weakness, as he asserts that the Grade 1 concussion, which is usually the most common, is the most difficult to recognize and consequently to treat. Furthermore, as noted earlier, PTA is a limited measure as it can only be determined in retrospect and is thus of limited practical use in the on-field situation.

More recently, the Quality Standards Subcommittee of the American Academy of Neurology (1997, in McCrea, Kelly, Kluge, Ackley, & Randolph, 1997) has specifically called for the development of a standardized, systematic sideline evaluation for the immediate assessment of concussion in athletes. In their report, the committee list three grades of concussion and the management guidelines they

have officially adopted. Grade 1 concussions are characterized by transient confusion, with no LOC and usual concussion symptoms or mental status abnormalities that are resolved in less than 15 minutes. Grade 2 concussions are also characterized by transient confusion and no LOC but with concussional symptoms lasting for more than 15 minutes. Grade 3 concussions are those that entail any LOC, either brief or prolonged.

Despite these various attempts to provide clarity and uniformity, there is still some confusion regarding the defining criteria and grading of the severity of concussion. At a fundamental level, this appears to stem from a lack of precision in related terminology. References are made to cerebral concussive injury (Gennarelli, 1987), mild traumatic brain injury (Alexander, 1995), mild concussion (Bruno et al., 1987) and minor head trauma (Barth et al., 1983; Kibby & Long, 1996; Weight, 1998), and these terms are often used interchangeably. What is the most confusing, however, is the tendency of some authors to use the terms 'mild head injury' and 'concussion' synonymously. This has resulted in a lack of clarity surrounding these terms, making it difficult to establish whether mild head injury and concussion are referring to the same form of injury, or whether there are fundamental differences between them.

A review of the literature suggests that the term "concussion" has gained popular usage amongst sports health practitioners and sports coaches, who use it to describe instances of closed head injury commonly occurring on the sports field. These injuries range in severity from slight dazing and momentary confusion to a more prolonged period of unconsciousness, and in some instances, death (Ommaya & Gennarelli, 1974; Torg, 1992). In contrast, neuropsychologists have favoured the term "mild head injury" which appears to incorporate a much broader spectrum of injury, ranging from open head injuries to closed head injuries (see section 2.1.1, p. 4 for definitions of these terms). Since the focus of the current research is on head injuries that are categorized as mild in severity, the term mild head injury will be used in this thesis to denote instances of closed head injury. However, where specific authors have employed the terms concussion or mild concussive head injury (most notably in the neuropsychological research on contact sports), these terms will be used in order to refer accurately to these authors' studies. The term concussive will also be retained when discussing the self-reported sequelae of mild head injury (including cognitive, emotional and behavioural changes), as postconcussive symptomatology remains the most common term for these symptoms. Finally, to avoid any further confusion, it should be noted that where the term subconcussive head injury is employed, reference is being made to a blow to the head of which the effects are brief and usually indiscernible. Thus mild head injury as defined for the purposes of this study (see section 2.1.3, p. 7), involves LOC of 30 minutes or less, and broadly subsumes subconcussive head injuries, as well as the category of mild concussion as per Cantu's (1996) definition (see Table 2-1, p. 9).

2.1.5. INCIDENCE AND PREVALENCE OF MILD HEAD INJURY

Mild head injury is one of the most important public health concerns due to its high incidence and its frequently persisting symptomatology (Evans, 1992). It is a major source of acute neuropsychiatric morbidity, with almost all patients encountering cognitive, somatic and behavioral difficulties (Silver & McAllister, 1997). These neuropsychological consequences make substantial demands on the health care system, with the cost of care for cases of hospitalized mild head injury being estimated at greater than a billion dollars in the United States (King, 1997). Furthermore, the sequelae of mild head injury have considerable economic effects on the industrial sector, as patients are often unable to return to work within a reasonable time period following injury and their productivity may be compromised thereafter (Rimel, Giordani, Barth, Boll, & Jane, 1981).

To date, there do not appear to be any available epidemiological data on the incidence of head injury in South Africa. For this reason, discussion will be limited to reported figures from Britain and the United States. Epidemiological studies have estimated that the annual number of hospital admissions in Britain involving head injury is between 250 and 300 per 100 000 of the population (Jennet & McMillan, in King, 1997). United States figures are also high, reflecting a reported annual frequency of 327 000 hospitalized cases (Weight, 1998). Estimates of the proportion of these cases falling into the category of mild head injury vary from 75% (Kraus & Nourjah, 1989) to 90% (Lezak, 1995; Lishman, 1987). Some authors argue that epidemiological data is usually drawn from documented cases of head trauma in hospitalized settings, and since many patients do not report their injuries or seek medical care, the incidence rate is probably higher than reflected (Binder, 1985; Templer, Kasiraj, Trent & Trent, 1992; Weight, 1998).

About half the mild head injuries suffered in the United States occur in persons between the ages of 15 and 34 years. Males between the ages of 15 and 19 have the highest incidence of any demographically defined group, with the ratio of males to females being 2:1 (Annegers, Grabow, Kurland, & Laws, 1980 in Boll, 1983; Kraus & Nourjah, 1989). This high incidence level amongst males has been supported by other research findings. For example, a Swedish population study found that 21-26% of adult males reported a history of head injury while another study of Canadian high school students reported a prevalence of concussion of 37% for males and 23% for females (cited in Binder, 1997). While males have a high incidence rate of head injury, epidemiological data indicate that children represent the majority of incidences of head injury cases. Mild head injury is most prevalent in children between the ages of 5-14, with severe head trauma accounting for only 10% of all paediatric head trauma (Annegers, 1983 in Satz et al., 1997). Specifically, there is a peak area of incidence between the ages of three and eight, which is followed by a slight decrease to age 12 (Boll,

1983). The incidence rate for head injuries in the adolescent population is also very high - adolescents incur 185 to over 300, per 100 000 head injuries annually in the United States alone (Basset & Slater, 1989). The most common causes of mild head injury are sports injuries, falls, assaults and motor vehicle accidents (Rimel et al., 1981; Rutherford et al., 1977; Silver & McAllister, 1997). Risk factors include substance abuse, a pre-existing psychiatric disorder, a previous head injury and lower socio-economic status (Binder, 1987).

2.1.6. PATHOPHYSIOLOGY AND MECHANISMS OF CEREBRAL INJURY

Despite the fact that head injury is the most common cause of neurological disorders, the neuropathology and pathophysiology of brain damage remain poorly understood (Dacey, Vollmer, & Dikmen, 1993; Strich, 1961). However, the traditional view of mild head injury as an essentially reversible syndrome without detectable neuropathological sequelae has come to be challenged by animal studies and autopsy reports (for example, Oppenheimer, 1968; Strich, 1961). Not only have these studies provided evidence of specific neuropathological and neurochemical changes following head injury, but they have highlighted the occurrence of cerebral insult in instances of relatively mild head injury, even where there has been no direct impact to the head.

As noted earlier, closed head injury occurs along a continuum from severe to moderate to mild. For this reason, it has been argued that the mechanisms for understanding mild head injury, namely acceleration/deceleration, are essentially the same as those for severe head injury. However, as the accelerative/decelerative forces occurring during mild head injury are less severe, there is consequently less damage (Alexander, 1995). In the majority of cases, neuropathology occurs as a result of the movement of the brain within the skull (Lishman, 1987). Head rotation is considered the key factor in producing diffuse axonal injury, the primary neuropathology of mild head injury (Ommaya & Gennarelli, 1974; Strich, 1961). Rotational forces in the brain produce shear stresses as rapid and swirling movements of the brain within the cranium causing it to impact upon the bony protuberances of the skull. These shearing forces disrupt fragile structures running in the long axis of the brain and result in axonal tearing and neural degeneration. Axonal injury causes localized transport failures in the axon, leading to swelling of the axon with wallerian degeneration. The resulting vascular injury disrupts small veins that lead to petechial hemorrhages or local or focal edema (Alexander, 1995). The effect of rotational forces can also result in lesions in the parasagittal deep white matter spreading from the cortex to the brain stem (Oppenheimer, 1968). More specifically, autopsy reports indicate that the greatest zones of brain contusion appear to be in the frontal and temporal regions of the brain (Walsh, 1985). This is due to blows most commonly being received in the anterior quadrants (i.e. from the front). Damage to these areas may result in what has

been termed "frontal lobe syndrome", which is characterized by both cognitive changes, (including memory difficulties, attentional deficits, speech difficulties, decreases in verbal fluency and executive deficits) as well as personality/emotional changes, such as disinhibition, aggressiveness, depression, anxiety and irritability (Lezak, 1995; Walsh, 1985). Importantly, it has been noted that if the frontal lobes are damaged, subtle deficits due to frontal dysfunction could become manifest in closed head injury patients who appear to be recovered (Walsh, 1985).

Magnetic resonance imaging (MRI) and animal research show that it is the magnitude of acceleration imparted to the head and the direction of head motion which determines the amount of shear strain and the extent of diffuse axonal injury that occurs during a closed head injury. Where there is more force, there is greater injury (Kibby & Long, 1996). More specifically, it has been indicated that the magnitude of diffuse axonal injury is proportional to the deceleration force, although not in a simple linear manner (Alexander, 1995). It is the inertial force transmitted by sudden deceleration that causes diffuse axonal injury, as might occur in a moving athlete's head hitting a fixed object, such as the ground or another player. Recent animal studies using acceleration/deceleration induced head injuries resulting in momentary LOC and physiological reactions for less than 30 seconds, were found to produce changes in brain structure including degeneration of axons and their terminal arborizations in locations including reticular nuclei, vestibular nuclei and dorsal regions of the medulla (Adams, Graham, & Gennarelli, 1981, in Boll, 1983). These findings bear similarity to lesions discovered in post-mortem examinations of patients who had sustained mild head injuries (Oppenheimer, 1968). Further evidence implicating brain stem dysfunction has been reported by researchers who observed an abnormality of brain stem auditory functioning in a small group of patients with minor head injuries (Povlishock & Coburn, 1989). In this respect, Oppenheimer (1968) has emphasized that permanent damage in the form of microscopic destructive foci can be inflicted on the brain by what are regarded as trivial head injuries. According to Oppenheimer (1968) if such injuries were to be repeated, "one would anticipate that a progressive loss of tissue, and of nervous function, would occur " (p. 306).

Despite the above findings, Binder (1997) argues that results from animal studies and post-mortem examinations may not necessarily be generalizable to the rest of the population and so the neuropathological implications of mild head injury remain uncertain. He argues, furthermore, that experimental animal studies have failed to find evidence of the cumulative effects of repeated head trauma. Instead, there appears to be some evidence of axonal regrowth and neural regeneration (Povlishock & Coburn, 1989). According to Binder (1997), this is consistent with the neuropsychological improvement observed in prospective human studies. However, given that mild head injury has been termed a "quiet disorder" due to the subtle and sometimes elusive nature of its

effects (Boll, 1983, p. 74), it is important to note that what may not be immediately discernible can still have potentially adverse consequences which may only become apparent much later (Shuttleworth-Jordan, 1999).

Section Summary

In the spectrum of head injury, a distinction can be drawn between open head injuries and closed head injuries. Closed head injuries exist on a continuum, ranging from severe to moderate to mild. Mild head injury can refer to open or closed head injuries, and is generally defined as a LOC of less than 30 minutes with a duration of PTA (< 1 hour) and an absence of structural damage. There is no standard definition of concussion, although the term is generally applied to closed head injuries ranging in severity from mild to severe. Concussion is generally graded and classified into differing levels of severity, in order to assist sports practitioners with on field management of injuries. Concussions can result from a blow to the head but can also occur in the absence of direct contact with external objects, such as a whiplash injury. The primary neuropathology involved in mild closed head injury (and concussion) are accelerative/decelerative forces in the brain which result in diffuse axonal injury. Incidence rates of mild head injury are high, with males, children and adolescents falling into the highest risk category.

2.2. MILD HEAD INJURY: SEQUELAE AND OUTCOME

While the immediate neuropsychological sequelae of mild head injury have been well established, its course of recovery and final outcome remain controversial. This has been largely due to significant variability in research findings, with disparities in outcome creating confusion and limiting the ability of researchers to draw meaningful conclusions. It has been argued that one of the primary sources of this confusion is the failure of researchers to differentiate between the cognitive consequences of mild head injury (as objectively demonstrated) and the postconcussive symptoms (based on self-report), when studying recovery of function (Kibby & Long, 1996). In this regard, it is not always clear whether reports of postconcussive symptomatology include a reference to objectively demonstrated cognitive deficits or whether they refer exclusively to subjective symptoms reported by the patient. As outcome varies considerably between these two groups of sequelae, and as postconcussive symptoms can occur in the absence of objectively demonstrated cognitive deficit and is inversely related to the severity of injury (Kibby & Long, 1996), it becomes crucial to maintain differentiation between these two aspects of sequelae following mild head injury when attempting to understand research findings.

For the purposes of the present research, a distinction will be drawn between a) neuropsychological deficits, which refer to objective measurable cognitive deficits and b) postconcussive symptoms which are the subjective/self-reported symptoms following a mild head injury and which include subjective reports of cognitive, emotional and behavioural difficulties. Where possible, these two categories will be discussed separately, in order to highlight the significant differences in outcome and recovery.

2.2.1. RESEARCH FINDINGS OF NEUROPSYCHOLOGICAL DEFICITS FOLLOWING MILD CLOSED HEAD INJURY

The neuropsychological consequences of mild head injury have been extensively documented (for example, Barth et al., 1983; Bohnen, Jolles, & Twijnstra, 1992; Gentilini et al., 1989; Rimel et al., 1981). Research findings have identified specific areas of deficit following mild head injury. These include impairments in attention, memory, information processing, vigilance and reaction time. While research studies have indicated that these deficits are particularly evident in the early stages following a mild head injury, their long term, and possibly permanent effects, have not yet been fully established.

Gronwall and Wrightson (1974) were the first researchers to assert that a reduced rate of information processing was the primary dysfunction associated with mild head injury. In a groundbreaking study, they compared the performance of mild head injured patients with normative controls, using the Paced Auditory Serial Addition Test (PASAT), a test regarded as sensitive to subtle changes in rate of information-processing capacity. They found that an initial ability to process a limited number of items as quickly as the normal controls was followed by a marked deterioration in performance when the number of items were increased beyond a critical point. While the majority of patients showed recovery after 35 days and all had recovered by 54 days, the findings of the study indicated that a reduced rate of information processing could be an important contributing factor in the genesis of the postconcussive syndrome. They concluded that not only does mild head injury reduce the individual's capacity to process information but successive injuries could produce deficits in information storage and retrieval capacity (Gronwall & Wrightson, 1975). This finding has been consistent with the results of several other studies that have also documented a pattern of compromised information processing following mild head injury (Leininger, Gramling, Farrell, Kreutzer, & Peck, 1990; Levin & Eisenberg, 1979; McLean, Tempkin, Dikmen, & Wyler, 1983).

In attempting to clarify the relationship between mild head injury and reduced rate of information processing, Gronwall (1989) draws attention to the specific difficulties encountered when mild head

injured patients are required to process simultaneously more items of information than they can manage. She argues that their observed difficulty appears to stem primarily from levels of inattentiveness, distractibility and forgetfulness, all of which reflect attentional deficits. Parasuruman, Mutter and Molloy (1991) concur with this, but argue further that any attempts to measure attention must recognize that attention itself is not a unitary aspect of cognition but is made up of a variety of interacting processes, which include selective/divided attention and sustained attention/vigilance. They assert that an individual's attentional capacity allocation involves his/her ability to vary the amount of attention paid to a stimulus in response to information processing requirements. This notion of differential degrees of attention is supported by Gentilini et al.'s (1985) study. In this study, which initially focused on attention as a global faculty, the researchers found no significant differences between mild head injured (MHI) patients and controls at one month post trauma. However, when analyses were extended to incorporate separate measures of attention, evidence for a specific deficit in selective attention was found at one month and three months post-injury. These findings were supported by a later study in which slowed reaction on tests of distributed attention was noted at three months post-injury (Gentilini et al., 1989). Similarly, in a multi-centre study of mild head injury, Levin et al. (1987) found pervasive neurobehavioural impairment across all cognitive measures that suggested subacute disturbances in attention, memory and information processing efficiency. However, these disturbances were found to have resolved by one to three months post-injury. In a study using four measures of attention, McLean et al. (1983) found that MHI patients demonstrated impaired performance on two of the four measures (the Stroop Colour Test and the Selective Reminding Test) relative to the matched controls. Similarly to Levin et al.'s (1987) study, these impairments in attention and memory were found to be resolved at one month post injury. Dikmen, McLean and Tempkin's (1986) study also found significant differences between MHI patients and controls on measures of sustained attention and memory within the first month after injury. However, these differences were no longer evident at a one-year follow up assessment.

In contrast to the extensive investigations of the effects of mild head injury on attention, memory and information processing, the cognitive functions of vigilance and reaction time have received less attention. The absence of research in this area is significant, as both vigilance and reaction time incorporate aspects of attention, and could possibly play a role in determining attentional deficits in patients with mild head injury (Parasuruman et al., 1991). The importance of exploring all aspects of attention cannot be overemphasized for Binder Rohling and Larrabee (1997, p. 429) in their meta-analytic review of neuropsychological studies on mild head injury conclude that "measures of attention may be the most sensitive indicators of dysfunction associated with MHT". With regard to neuropsychological research on these modalities, Parasuruman et al. (1991) found that while vigilance performance remained unaffected under normal task conditions, there was evidence of its

impairment in task conditions requiring sustained effortful processing. Another study by MacFlynn, Montgomery, Fenton and Rutherford (1984) found slowed reaction time both immediately and six weeks following head trauma, although there was an improvement between six weeks and six months following the injury. Slowed reaction time is thought to reflect a diminution of information processing (MacFlynn et al., 1984).

As noted earlier, there is a lack of consensus about outcome and recovery following mild head injury. What is noteworthy in the above studies is that while they clearly illustrate the cognitive impairment following a head injury, their findings mostly indicate a recovery period of less than three months. Other researchers have argued that the course of recovery is far more protracted, with cognitive impairment being evidenced in patients months and even years following injury (for example, Bohnen & Jolles, 1992; Rimel et al., 1981). These researchers argue that studies which demonstrate rapid recovery (for example, McLean et al., 1983) as well as those that have failed to find support of cognitive impairment post mild head injury (for example, Gentilini et al., 1985), all suffer from methodological limitations that compromise their findings. These limitations include a lack of premorbid data (making it difficult to assess whether patients have returned to their premorbid level of functioning), a lack of control groups (to account for practice effects) or inadequate control groups, inadequate test batteries, differing definitions of mild head injury, differing length of follow up period and a lack of control for pre-existing risk factors (Binder, 1987; Bohnen & Jolles, 1992; McLean et al., 1983). Furthermore, it has also been argued that many of these studies utilize global measures such as the Wechsler Verbal or Performance Intelligence scores and that these measures are not always sensitive to subtle changes in information processing (Barth et al., 1983).

2.2.1.1. Research Findings of Persisting Neuropsychological Deficits Three or More Months Post Closed Mild Head Injury

The most substantive studies to report persisting deficits following mild head injury are those of Rimel et al. (1981) and Barth et al. (1983). In an exhaustive analysis of 538 MHI patients, Rimel et al. (1981) recorded impaired neuropsychological test performances on measures of attention, concentration, memory and judgement relative to normative controls at three months post-injury. Drawing on the same subject pool, Barth et al. (1983) provided a more extensive evaluation in comparing the test performances of 71 of these patients across a more comprehensive neuropsychological test battery at three months post injury. They found that a significant percentage of the patients were impaired in the areas of memory and visuospatial skills. Their study led them to conclude that memory deficits are secondary to difficulties with information processing. While both these studies utilized large and comprehensive test batteries, their major limitations were the absence

of a matched control group, an absence of premorbid data and a repeated measures control group to account for practice effects. In a more closely controlled study using a matched group of uninjured controls, Leininger et al. (1990) found evidence of impairments in the areas of reasoning, information processing and verbal learning at 22 months post-injury. Patients who lost consciousness during injury were found to have obtained test scores similar to those who experienced disorientation or confusion but no LOC.

In exploring the relationship between cognitive deficits and behavioural sequelae, Bohnen and Jolles (1992) argue that a limitation of most studies on mild head injury is that they have tended to make comparisons between head-injured patients and those who have not sustained a head injury, instead of a direct comparison between with patients with or without persisting subjective complaints. In order to address this, they compared patients with persisting subjective complaints after a mild head injury with patients with mild head injuries who were symptom free, and with normal controls. They used a visual computerized version of the Auditory Verbal Learning Test, the Stroop Colour Word Interference Test and a computerized divided attention task. Evidence of deficits was found on measures of selective and divided attention and information processing. The authors concluded that cognitive deficits may be present up to six months after mild head injury when subjective symptoms persist. These findings are consistent with both Leininger et al.'s (1990) and Dikmen et al.'s (1986) studies, which also found deficits on tests of divided and selective attention.

More recently, Klonoff and Lamb (1998) evaluated nine patients with persisting deficits after an average of three years post mild head injury. Despite their low test scores, the symptoms were attributed to significant psychiatric disability and/or malingering. However, a limitation of this study was its restricted sample size that limited the generalizability of the results. In another study that examined the impact of psychological factors on cognitive functioning, Raskin, Mateer and Tweeten (1998) assessed 148 MHI patients with reported persisting symptomatology using a neuropsychological battery and a personality measure. Results indicated impaired performance on measures of complex attention, working memory, verbal learning and time dependent tasks at 21 months, which was the mean time elapsed since the injury. No correlation was noted between emotional/personality factors and cognitive functioning, leading the researchers to conclude that there could be an organic basis for persistent neuropsychological deficits. The main limitation of this study was that it did not include a matched control group.

In a significant study which examined potential risk factors implicated in mild head injury and which illustrated the importance of task conditions in determining outcome, Ewing, McCarthy, Gronwall and Wrightson (1980) compared the performances of both university students reported to have made

a full recovery from a prior head injury (two years earlier) and a matched control group of students who had never sustained a head injury, under conditions of mild hypoxia. They found that the MHI group performed at a significantly lower level than the normal controls on a memory and vigilance task. This led them to assert that even where there appears to be a full recovery, mild head injury may leave a residual effect that impairs the ability to withstand another central nervous system stressor. They concluded that in all likelihood each concussive event destroys neurons, thereby diminishing the reserve availability and making the loss evident under the stress of further injury. The possibility that MHI patients demonstrate cognitive deficits which are only apparent in stressful conditions is also illustrated by the previously noted study (p. 16) by Parasuruman et al. (1991) in which the authors found that vigilance performance after mild head injury was comparatively normal under tests conditions that required automatic processing but fell short under test conditions that required effortful processing.

In their meta-analytic review of neuropsychological research on mild head injury in adults, Binder et al. (1997) argue that not only is the average effect of mild head injury on neuropsychological performance undetectable, but research findings are more likely to reflect false positive diagnoses of mild head injury. They maintain that while some findings suggest the presence of persisting neuropsychological deficits, they are unable to demonstrate causation and their results suggest weak association only between mild head injury and persisting neuropsychological deficits. Thus, they conclude that "clinicians will more likely be correct when not diagnosing brain injury than when diagnosing a brain injury in cases with chronic disability after MHT" (p. 241).

In responding to Binder et al's (1997) review, Shuttleworth-Jordan (1999) argues that there are a number of important issues, which these reviews do not consider and which are in danger of being neglected. Firstly, the absence of sequelae tends to be decontexualixed, which fails to address the issue that mild head injury may cause permanent brain damage and that this in itself becomes a risk factor for future impairment. Secondly, the emphasis in both reviews is on empirical findings. These are often based on mean scores, which are not reliable indicators where there is significant variability in a sample. More specifically, she argues that "increased variability for tasks sensitive to diffuse brain damage indicates that while some individuals may be well preserved following a mild head injury, there are a significant proportion of individuals who are not" (p. 24). This has been supported by Reid's (1998) study of the neuropsychological effects of mild head injury in rugby, which found significant variability between the rugby group and the control group. Thirdly, the studies reviewed have not examined longitudinal effects and so long-term and possibly permanent damage cannot be ruled out. Finally, it is argued that the null outcomes of the studies reviewed all concern the effects of

a single mild head injury, and so do not reflect the potential damage sustained by cumulative head trauma.

2.2.1.2. Research Findings of Neuropsychological Deficits in Child and Adolescent Populations Post Mild Closed Head Injury

"Mild head injury is a quiet disorder. It is common, typically bloodless and without call for significant medical intervention. It seems even more quiet because the noise it does make (its symptoms) is often attributed to other causes. Nevertheless, the disruption in coping capacity and attendant breakdown in usual behavioral patterns causes more psychosocial and academic-economic hardship than have begun to be appreciated" (Boll, 1983, p. 74).

Until recently, most research on head injury has focused on adult populations. It was not until Boll's (1983) review that mild head injury in children gained public health attention as an underinvestigated disorder with potentially serious cognitive and behavioural outcomes (Satz et al., 1997). While there have since been other reviews of head injury in children (for example, Beers, 1992; Satz et al., 1997), there appears to be a lack of consensus regarding the outcome of mild head injury in children. Researchers have struggled to draw meaningful conclusions from the respective studies, as they have suffered from a number of methodological limitations and have also been based largely on adult samples (Satz et al., 1997).

While the focus of the current research is on a late adolescent population (16-18 years), neuropsychological studies on adolescents have tended to include children in their focus and to date there are few neuropsychological studies which concentrate exclusively on adolescent samples. For this reason, neuropsychological research studies on both child and adolescent populations will be presented together in a brief review.

Studies on outcome in children and adolescents following mild head injury have identified cognitive sequelae, behavioural problems and poor functional outcome. In a five-year follow-up study, Klonoff, Low and Clark (1977) compared two groups of children: 131 younger than nine years old at the time of the injury, and 100 who were older than nine. The two groups were compared with four investigations, namely, neuropsychological function, neuropsychological status, EEG status and school progress. Results indicated immediate and pronounced effects in all areas for both groups. In addition, the researchers found an extended recovery over time and a varied rate of recovery for all four aspects. The main limitation of this study was that it did not provide any way to differentiate

between mild and severe injuries using cognitive, academic or behavioural outcomes (Satz et al., 1997).

In another study that examined neuropsychological function in adolescents following mild head injury, it was found that 20–25% demonstrated impairment in memory, language and somatosensation (Levin & Eisenberg, 1979a). In addition, a retrospective study of 51 children and adolescents found that 6-18% of those who had suffered a mild head injury demonstrated impairment on a number of psychological tests at one year post-injury (Winogron, Knights, & Bawden, 1984 in Basset & Slater, 1989). In a frequently cited neuropsychological study of 56 MHI children between the ages of 9 to 13, Gulbrandsen (1984) found deficits at six months post-injury when compared with normal controls. Differences tended to decrease with increasing age and increase with greater complexity of the tests. The author concluded that neuropsychological sequelae following concussion may be demonstrated even where there are few subjective complaints and no discernible lags in academic achievement.

Using a sample of 1 345 high school and 2 321 university students, Segalowitz and Lawson (1995) conducted a survey on the relationship between mild head injury and a variety of psychological and educational symptoms. They found significant relationships between the incidence of mild head injury and gender, sleep difficulties, social difficulties, handedness pattern and diagnoses of attention deficit, depression and speech, language and reading disorders. A more recent study by Ong, Chandran, Zasmani and Lye (1998) compared the neurobehavioural outcome of Malaysian children ages 6 to 12 with severe closed head injury, mild to moderate head injury and orthopaedic controls. Assessment measures included the Glasgow Outcome Scale, the Wechsler Intelligence Scale for Children (WISC-III), the Movement Assessment Battery for Children, the Wide Range Assessment of Learning and Memory and a standardized neurological examination six months post-injury. In addition, parental reporting of pre- and post-injury behaviour was documented using the Child Behaviour Checklist. Although most of the children appeared to have made a good physical recovery, there were cognitive, motor, memory and learning difficulties and behavioural problems concomitant with a deterioration in school performance compared to those with lesser, or no, head injury.

Not all studies, however, have provided support for cognitive impairment in children and adolescents following mild head injury. For example, a study by Levin and Eisenberg (1979b, cited in Levin et al., 1982) of 46 MHI young adults and adolescents found no impairments in verbal learning or memory when compared with a population of normal adolescents.

In response to studies of null outcomes, Basset and Slater (1989) suggest that the difficulty of evaluating and comparing the results of these studies stems largely from the fact that previous studies that incorporate a large age span do not present a separate analysis for adolescent patients. Furthermore, they do not consistently use matched control groups for age and education, nor have they employed consistent injury-test intervals. In order to address these limitations, Basset and Slater (1989) compared 29 adolescents who had sustained a mild closed head injury with a control group of healthy adolescents, as well as a group of adolescents who had sustained a severe closed head injury. An extensive neuropsychological battery was administered to all subjects that included the Wechsler Adult Intelligence Scale, Wechsler Memory Scale Logical Memory and Visual Reproduction subtests (immediate and delayed recall), the Buschke Selective Reminding Test, the Trail Making Test, the Wisconsin Card Sorting Test and the Controlled Oral Word Association Test. Results indicated impairments in the MHI group on measures of learning, abstraction and reasoning. The researchers argue that the pattern of performance deficits indicated are consistent with research findings that closed head injury results in diffuse damage to the frontal and temporal lobes, causing impairments in memory, learning and higher cognitive processes such as abstraction and reasoning. They conclude that adolescents who are recovering from even a mild head injury may be expected initially to experience difficulty on returning to school. Some of this difficulty is related to the task conditions they are required to work under, as the school classroom is regarded as presenting the scholar with a vast amount of complex material in an environment full of distraction. This becomes very significant in light of Ewing et al.'s (1980) study (section 2.2.1.1, p. 18) as it suggests that neuropsychological impairment may only become apparent under conditions of stress and increased pressure. This view is supported by Boll (1983), who argues that difficulties with information processing are more likely to manifest as difficulties in learning under complex and stressful situations, as well as a tendency to be overwhelmed by stress more easily than before.

When we stop to consider the obvious lack of self-report skills of children in making their difficulties known to teachers and parents, it becomes apparent just how easily the effects of mild head injury could go unreported. Similarly, adolescents, in their developmental need for independence and reluctance to be infantilized, may choose not to report the appearance of physical symptoms following a head injury. Thus the need for a sensitive awareness of the subtle manifestations of mild head injury and of the importance of prevention becomes even more crucial when dealing with child and adolescent populations.

In their meta-analytic review of neuropsychological studies of head injury in children, Satz et al. (1997) report that out of a total of 40 neuropsychological studies investigated, 18 null outcomes and nine indeterminate findings regarding outcome were recorded in contrast to 13 adverse findings.

Furthermore, they argue that the studies that indicate positive diagnoses of brain dysfunction are compromised by methodological weaknesses (including an overreliance on subjective reports, lack of control groups and longitudinal follow-up), thereby making it difficult to draw firm conclusions as to whether mild head injury is associated neuropsychological deficits. Like Binder et al. (1997), they note that while there is some evidence to suggest that mild head injury is associated with impairment in neuropsychological performances, the effects are transitory and small.

While Binder (1997) argues that there are other variables that affect outcome (including age, gender and educational status), Satz et al. (1997) emphasize the role of pre-existing risk factors in determining outcome in children following mild head injury. They conclude that as the studies reporting null outcomes have tended to be methodologically stronger, researchers and clinicians should favour a cautious acceptance of the null hypothesis as it relates to outcome in mild head injury. They back this conclusion by drawing attention to the inherent bias in journals, which are more likely to report significant findings rather than null results. Thus those results which would support the null hypothesis often go unpublished and uncited. Once again, as noted earlier (p. 19), Shuttleworth-Jordan's (1999) response to these reviews of null outcome is to point out their lack of focus on the longitudinal effects of mild head injury; their focus on mean scores which are unreliable when there is significant variability in a sample; their tendency to decontexualize the absence of sequelae and perhaps most importantly, their tendency to focus on the effects of a single mild head injury rather than on cumulative head trauma. This last factor is particularly significant in light of the fact that all the research studies discussed so far have not focused on individuals with a prior history of mild head injury. The following section will consider the issue of cumulative mild head injury and will explore some of the most prominent research studies in this area.

2.2.1.3. Cumulative Effects of Mild Head Injury

It has been frequently argued that one of the most disturbing features of mild head injury is that its effects are cumulative (De Villiers, 1987). However, while there is a substantial body of evidence indicating the presence of cognitive deficits following a single mild head injury, there are a limited number of studies on the cumulative effects of repeated head trauma.

Gronwall and Wrightson (1975) compared 20 young adults who had sustained a second mild head injury with a control group consisting of patients with a first mild head injury. They found that information processing ability on the PASAT was slowed in patients after a second or third mild head injury, when compared to a single mild head injury. Furthermore, those who had sustained several mild head injuries took longer to return to normal levels of functioning. While intellectual

performance eventually returned after two mild head injuries, the researchers argue that the effects of repeated mild head injuries are cumulative in that they delay the cognitive and subjective recovery from a new head injury. This finding was supported by a later study by Gronwall (1989) involving a series of studies using the PASAT. She found that mild head injury results in reduced information processing ability and that older patients and those patients with a previous head injury took longer to recover than those patients with a single episode of mild head injury.

This growing body of evidence indicating the risk of permanent residual cognitive impairment as a result of cumulative mild head injury has raised public health attention, due to the implication of such findings for the contact sports. This is because the nature of contact sports predisposes players to the risk of repeated injury, and more specifically, to multiple concussions. These implications will be later explored and discussed, with specific reference to current neuropsychological research findings in the section on mild head injury and the contact sports.

2.2.2. THE POSTCONCUSSIONAL SYNDROME (PCS)

2.2.2.1. Postconcussive Symptoms

While many MHI patients recover without apparent residual sequelae, a clinically significant proportion continue to report a group of particular symptoms which persist long after the normal period of recovery. The term "postconcussional syndrome" (PCS) has been coined to refer to the emergence and variable persistence of this group of symptoms following a head injury (Jacobson, 1995). The most frequently reported symptoms are headaches, dizziness, fatigue and memory loss. While these symptoms can occur in isolation or in combination, Anderson (1996) argues that they tend to fall into three broad areas which he highlights as follows: *Neurocognitive* - impaired attention and concentration, memory and learning disorders, reduced mental flexibility, slowed reaction time, impaired decision making, speech difficulties and mental fatigue; *Somatic* - headaches, dizziness, insomnia, loss of appetite, drowsiness, strabismus, menstrual irregularities, decreased noise tolerance, sensitivity to medication or alcohol, clumsiness and postural changes; *Neuropsychiatric* - depression, anxiety, emotional lability, lowered frustration tolerance, somatization, denial of symptoms, apathy, lack of spontaneity and personality changes.

Rutherford (1989) argues that a further distinction can be drawn between what he categorizes as the early and late symptoms of concussion (see Table 2-2 below, for the range of possible symptoms experienced). *Early* symptoms are those the patient complains of immediately after regaining full consciousness after the head injury. These may also be reported the following morning. *Late*

symptoms are those the patient reports a few weeks later, often at clinic visits. Rutherford (1989) argues that later onset symptoms are often caused by an increase in stress levels when the individual is required to return from the hospital environment to a more stressful home or work situation

Table 2-2 Early and Late Postconcussive Symptoms

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

(Rutherford, 1989)

Despite considerable variability between findings regarding the frequency of reported postconcussive symptomatology (Binder, 1987), there is a growing body of research that suggests that the PCS is not a brief phenomenon, but has a number of long-term consequences. While some studies have demonstrated a full recovery from postconcussive symptoms within three months of a mild head injury (Alves, Colohan, O'Leary, Rimel, & Jane, 1986; Dikmen et al., 1989; Evans, 1992; Levin et al., 1987), others have indicated that a significant proportion of patients experience persisting symptomatology from three months to one year post-injury. For example, Rimel et al. (1981) found that of 424 MHI patients, 79% complained of headaches and 59% of memory disturbances at three months post-injury. Of the patients who had been employed before the accident, 34% were unemployed three months later. In a multi-centre study, Levin et al. (1987) found that that at three months post-injury, 47% of patients reported headaches, 22% fatiguability and 22% dizziness. In their study of postconcussive symptoms following mild head injury, Bohnen et al. (1992) found a range of 16-49% of patients with persistent symptoms at six months and 1-50% with persistent symptoms at one year. This is consistent with Rutherford, Merrit and McDonald's (1977) study which found that out of a sample of 145 MHI patients, 15% had persisting symptomatology one year

after trauma. Similarly, Alves et al. (1986) noted that 46% of their sample of MHI patients were experiencing persisting symptomatology at one year post-injury.

While there appears to be a body of evidence indicating the long-term effects of the PCS in MHI patients, Satz et al. (1999), in their review of the studies on the PCS over the last 40 years, argue that the majority of studies lack the required methodology to determine whether the observed effects of mild head injury are due to a specific head injury, a more general injury or other factors. In order to assess this, they assert that it is necessary to utilize a study design with two control groups, both a mild non- head injury, and an injury free reference group. After reviewing the literature to determine the findings of any studies using this design, they note that few such studies exist and so appropriate studies have yet to be performed. They conclude their review by arguing that thus far there is no strong evidence for a specific effect for the PCS following mild head injury and should future studies find no effect, the construct of the PCS should be abandoned.

2.2.2.2. Postconcussive Symptoms versus Postconcussive Syndrome

Patients with the PCS comprise a heterogeneous population, displaying significant variability in both symptom presentation, persistence and recovery. Some patients report a single symptom which may remit shortly after the injury while others report an entire complex of symptoms which persist weeks or months post-injury. This variability had led some researchers (for example, Silver & McAllister, 1997; Szymanski & Linn, 1992) to conclude that there may be two groups of MHI patients, those who recover within a three month period and those who continue to experience persistent symptoms.

Inter-individual variability has also sparked a debate as to whether persistent symptoms are part of a cohesive syndrome or whether they simply represent a collection of loosely related symptoms resulting from a mild head injury. In this respect, Lishman (1987) argues that with its mixture of symptoms, lack of conceptual clarity and doubtful etiology, the PCS cannot be regarded as a true syndrome. Binder (1987) concurs, arguing that postconcussive symptoms are too non-specific and do not cohere with sufficient reliability to form a true syndrome. In addition, he argues that the presence of postconcussive symptoms is normal and common in the first few weeks post-injury and thus, asserts that the term should be reserved solely for instances of *persisting subjective symptomatology*, following cerebral insult. This view is supported by other researchers, who argue that many so-called postconcussive symptoms have high base rates in the normal population (for example, McLean et al., 1983).

It appears that one of the primary difficulties in defining symptoms as a syndrome is that it leads to them being regarded and treated as a single entity when, in fact, there may be multifactorial groupings of symptoms that form distinct symptom clusters (King, 1997). This view has gained some support from Levin et al.'s (1987) factor analysis of the PCS in 155 mild MHI patients. In this study, the researchers were able to isolate salient features of the PCS that could be used to identify subgroups of patients. Results revealed three clusters of patient groups, one group with predominantly cognitive and affective symptoms, another with somatic symptoms and a third with very mild or no PCS. In addition, a factor analysis of all the symptoms identified a cognitive-depressive factor that included complaints of depression, impaired recent and remote memory, poor concentration and impaired thinking (Levin et al., 1987). These results were supported by the findings of Dikmen, Temkin, and Armsden's (1989) study. Despite these findings, it should be noted that there are authors who continue to argue that that even the presence of a single symptom should be deemed significant as symptoms can occur both alone and in isolation (Evans, 1992, Gasquoine, 1997). However, it appears that, like mild head injury and concussion, the term "postconcussive syndrome" is rarely clearly defined and different authors refer to different symptoms when discussing the PCS (Lishman, 1987).

2.2.2.3. Postconcussional Disorder

More recently, the DSM IV has proposed a category of "Postconcussional Disorder". In light of research findings, this category remains temporary and in need of further refinement. The rationale behind its introduction is that it is thought to propose a common language for researchers and clinicians working in the field of mild head injury which will help to clarify definition and classification inconsistencies. The essential feature of the proposed Postconcussional Disorder is an acquired impairment in cognitive functioning, accompanied by a number of specific neurobehavioural symptoms, which occur as a direct result of a closed head injury of sufficient severity to result in significant cerebral concussion.

An examination of the criteria for Postconcussional Disorder (see Table 2.3 below) reveals that this category does not differentiate between neuropsychological deficits (objectively measured deficits) and postconcussive symptoms (subjectively reported symptoms) but collapses them to form one diagnosis. As noted earlier, it is important to maintain a distinction between these two categories, due to their differences in outcome and recovery. Hence the provisional DSM IV category of Postconcussional Disorder is not adopted within the present research, in that, as outlined in section 2.2 (p.14), objective neuropsychological deficits and subjectively reported postconcussive symptoms are dealt with as two separate entities.

Table 2-3. DSM-IV Research Criteria for Postconcussional Disorder.

A. A history of head trauma that has caused significant cerebral concussion.
Note: the manifestations of concussion include loss of consciousness, posttraumatic amnesia, and, less commonly, posttraumatic onset of seizures. The specific method of defining this criterion needs to be established for further research.

B. Evidence from neuropsychological testing or quantified cognitive assessment of difficulty in attention (concentrating, shifting focus of attention, performing simultaneous cognitive tasks) or memory (learning or recalling information).

C. Three (or more) of the following occur shortly after the trauma and last at least 3 months:
(1) become fatigued easily
(2) disordered sleep
(3) headache
(4) vertigo or dizziness
(5) irritability or aggression on little or no provocation
(6) anxiety, depression, or affective lability
(7) changes in personality (e.g., social or sexual inappropriateness)
(8) apathy or lack of spontaneity

D. These symptoms in Criteria B and C have their onset following head trauma or else represent a substantial worsening of pre-existing symptoms.

E. The disturbance causes significant impairment in social or occupational functioning and represents a significant decline from a previous level of functioning. In school-age children, the impairment may be manifested by a significant worsening in school or academic performance dating from the trauma.

F. The symptoms do not meet criteria for Dementia Due to Head Trauma and are not better accounted for by another mental disorder (e.g., Amnestic Disorder due to Head Trauma, Personality Change Due to Head Trauma).

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

2.2.2.4. Etiology of PCS: Models of Conceptualization

The etiology of the PCS remains controversial. Symptoms based on subjective self-report are not amenable to objective measures, thereby making quantification extremely difficult. This is further complicated by differences in definitions of mild head injury as well as methodological issues and problems found in studies investigating outcomes of mild head injury (Bohnen & Jolles, 1992). These difficulties have led to a debate as to whether the basis of the PCS is organic or psychogenic in nature. Those authors who contend that organic factors play an important role in the genesis and maintenance of the PCS argue that there is a close relationship between severity and location of injury, and the number and severity of postconcussive symptoms. For example, somatic symptoms are associated with vestibular and visual dysfunction, and fatigue with diffuse axonal injury. Although the notion of the PCS as a purely organic syndrome is no longer a widely held view, it has been argued that recent histological, neurophysiological and neuropsychological data point to the possibility of a specific neuropathological contribution to these symptoms in the cases of minor head

injuries (Barth et al., 1983; Binder, 1986). While the demonstration of early organic symptoms and pathology does suggest their etiological involvement in the emergence of the PCS, the utility of this model is limited by the persistence of postconcussive symptoms long after the resolution of organic changes following mild head injury.

In contrast to the organic model, proponents of the psychogenic conceptualization of the PCS argue that as persisting cerebral dysfunction and related symptomatology can occur in an absence of gross neurological complications, psychogenic/psychological factors must therefore be a contributing cause. Psychological factors posited include a genetic propensity to neuroses, depression, anxiety and major psychoses. Malingering and litigation have also been implicated, the latter being viewed as resulting in the scapegoat motive, whereby the injury is held responsible for premorbid difficulties and subsequent life events (Lishman, 1988 in Gasquoine, 1997). The difficulty, however, with establishing psychological factors is that while some psychiatric symptoms can be demonstrated to derive from organic brain lesions, the relationship with emotional and behavioral sequelae are more complex to determine.

In attempting to overcome the dichotomy between organic and psychogenic factors, Lishman (1988) has argued for the utility of an organic-psychogenic conceptualization of the PCS, whereby physiological factors are thought to contribute to the onset of the PCS while psychological factors are responsible for their persistence. This is consistent with Gronwall and Wrightson's (1974) view that the condition of the PCS begins with organic damage to the brain causing intellectual impairment, and the resulting loss of self-confidence leads to neurosis. This conceptual framework has led to the proposal of a "coping hypothesis", which suggests that postconcussive symptoms occur when environmental demands exceed cognitive capacities. Thus the fatigue and stresses involved in attempting to cope with environmental demands may exacerbate postconcussive symptoms, such as headaches and dizziness (Van Zomeren & Van den Burg, 1985 in Bohnen & Jolles, 1992). This is particularly the case when patients return to work and face complex activities whose failure may result in aggravation of the postconcussive symptoms and lead to confusion, depression and self-doubt (Gronwall & Wrightson, 1975). It has also been argued that in instances where patients lack an explanation for their symptoms, expectation often becomes a major source of symptom persistence. This can then result in reattribution of benign symptoms to the head injury, selective attention to them and anxiety that then reinforces expectation (Mittenberg et al., 1992 in Jacobson, 1995). Other factors that are thought to prolong postconcussive symptoms and cognitive disturbance are anxiety, depression and anger (Lishman, 1988 in Gasquoine, 1997). Families of patients may also play a contributing role to in patients'persisting symptomatology by reinforcing and sometimes encouraging patients' sick role. While the desire for compensation has been cited as a primary cause of persisting

symptomatology (Miller, 1961 in Binder, 1986), studies have found that resolution of compensation claims does not result in rapid cessation of postconcussive symptomatology (for example, Jacobson, 1969 in Binder, 1986).

Bohnen and Jolles (1992) maintain that both the organic and psychogenic models in isolation are inadequate for explaining the occurrence of the PCS and that an interaction of both viewpoints is necessary. They argue that persistent symptoms are maintained by an interdependent set of organic psychosocial and behavioral factors. However, Jacobson (1985) argues that even organic-psychogenic conceptualizations do not adequately explain chronic postconcussive symptoms. She argues for the necessity of a cognitive-behavioral perspective, which places emphasis on the patients' beliefs, appraisals and coping responses as direct influences on the severity of symptoms. In this regard, the PCS is understood to reflect the interaction between the individual's brain function, stress management abilities and perceived stress as cognitive behavioral and psychophysiological factors mediate the patient's coping response.

While it is difficult to determine the precise outcome of the PCS due to the high variability in the population, several risk factors for prolonged symptoms after a mild head trauma have been noted. These include: advanced age, lower educational level, premorbid constitutional differences, premorbid psychiatric history, female gender, previous head injuries, preinjury stressful events, genetic liability, alcohol abuse, lower socio-economic status, post traumatic anxiety, depression and fat embolism. Other factors such as undetected parenchymal lesions, litigation, pain and malingering have also been proposed as playing a role determining symptom duration (Binder et al., 1997).

2.2.2.5. Treatment

It has been argued that postconcussive symptoms originate from minor brain trauma, but are perpetuated by the failure of doctors and medical personnel to validate patients and to provide information and reassurance about their favourable prognosis (Kelly, 1975 in Jacobson, 1995). At present, MHI patients are given minimal or no instructions regarding the risk for development of neurobehavioral sequelae (Bohnen & Jolles, 1992). Early clinical intervention in the form of education and reassurance is regarded as crucial in reducing levels of disability. It does this by providing information, assessing the presence of subacute neurobehavioral deficits, informing the patient about graded resumption of activities following discharge and by providing follow-up (Jacobson, 1995). In some instances, pharmacotherapy, cognitive restructuring, physical therapy, and counselling are indicated. Ruff et al. (1989) state that, in order to prevent secondary psychological reaction to the trauma, it is essential that patients be advised within the first month following injury

against participating in activities that may be too complex for them to undertake. In this regard, cognitive test results are useful in developing individualized rehabilitation programs to help individuals assess whether they are ready to resume vocational or academic routines (Levin, 1990 in Bohnen & Jolles, 1992). In sum, management of the PCS is a combination of somatic medical treatments, psychological-psychiatric management and pragmatic occupational interventions. Importantly, treatment programs should focus on managing the patient in the setting of real life demands (Alexander, 1995).

2.2.1.6. Relationship between neurocognitive deficits and postconcussive symptoms

While studies focusing on neuropsychological deficits and postconcussive symptoms have produced differing results, the relationship between particular aspects of neuropsychological impairment and specific postconcussive symptoms is clear and twofold. According to King (1997), poor concentration and fatigue would be expected difficulties with a reduction in speed of information processing. Similarly, forgetfulness would be expected with measurable memory impairment. However, at the same time, it is likely that a memory deficit will result in a need to process greater amounts of information and attentional deficits will lead to more forgetfulness as less is encoded and therefore retained (King, 1997). Similarly, anxiety is known to disrupt concentration and complex mental operations while depression has been shown to disrupt cognitive operations such as concentration, memory and executive functions (Alexander, 1995). Gronwall and Wrightson (1974) found that the subjective elements were accompanied by objective changes in intellectual functioning, and that as intellectual functioning returns to normal, postconcussive symptoms resolve. While Gasquoine (1997) argues that symptoms are often underreported in the first year following head injury compared to neuropsychological deficits, others have found that complaints tend to remain unchanged even in the face of complete neuropsychological functioning recovery (for example, Ruff et al., 1989).

Section Summary

Neuropsychological sequelae following mild head injury are generally divided into objectively measurable cognitive deficits and subjectively reported postconcussive symptoms which include cognitive, emotional and behavioural changes. Research findings for both adult and child populations have indicated that the most common neuropsychological deficits following mild head injury are impairments in attention, memory, information processing, vigilance and reaction time. There is a lack of consensus, however, regarding the course of recovery, as some researchers suggest that recovery occurs within days or weeks (for example, McLean et al., 1983) while others' studies have

found evidence of cognitive impairment in patients months, and even years, post-injury (for example, Leininger et al., 1990). However, it appears that in the majority of cases, cognitive deficits do resolve within one month. This has led to the conclusion that a single uncomplicated mild head injury does not produce any permanent disabling neurobehavioral impairment in most patients who do not suffer from a pre-exiting psychiatric disorder or substance abuse. While the course of recovery from a single mild head injury remains somewhat controversial, there is a growing body of evidence that indicates the risk of permanent cognitive deficit as a result of cumulative mild head injury (for example, Gronwall, 1989; Gronwall & Wrightson, 1975).

While some patients recover rapidly from mild head injury with no indication of adverse effects, a clinically significant proportion continues to report a group of symptoms post-injury. While the term "postconcussional syndrome" (PCS) has been coined to describe this group of symptoms, there is still a lack of agreement as to whether such sequelae are best conceptualized as a syndrome or not. The most frequently reported postconcussive symptoms are headaches, dizziness, fatigue and memory difficulties. Similar to the findings of neuropsychological research of cognitive deficits, there is a lack of consensus regarding the course of recovery of the PCS. Some studies indicate that symptoms resolve within a month, while other show evidence of persisting symptomatology at one year post-injury. Both organic and psychogenic factors have been implicated in the genesis and maintenance of the PCS.

2.3. MILD HEAD INJURY IN CONTACT SPORTS

Neuropsychological findings of the potentially hazardous effects of a single mild head injury, and the risk of neuropsychological dysfunction associated with cumulative mild head injury, have both stimulated a growing interest in research into the contact sports. This is because these sports, (particularly boxing, wrestling, rugby and soccer), place their participants at increased risk of sustaining a head injury and cumulative brain trauma than do non-contact sports (Anderson, 1996; De Villiers, 1987; Lehman & Ravich, 1990; Warren & Bailes, 1998). While episodes of mild brain trauma are frequent in contact sports and comprise a significant percentage of all athletic injuries (Sturmi, Smith, & Lombardo, 1998), it is difficult to establish precise incidence rates for mild head injury. This is because athletes tend to underreport head injuries due to reasons such as a fear of letting down the team, being seen as weak, or reducing their probability of playing in the next game (Cantu, 1986). Despite underreporting, it has been estimated that mild head injury occurs at a rate of 250 000 per year in the contact sports in the United States (Cantu, 1998 in Wilberger, 1993) and this is by far the most common sports-related injury (Cantu, 1996; Warren & Bailes, 1998).

The majority of head injuries in contact sports occur when a moving head hits the ground or a relatively large and stationary object, for example, during scrumming, being tackled or carrying out a tackle in rugby and football, or a collision of heads during soccer or heading the ball. In all instances, the head motion may come to an abrupt halt, but relative movement of the brain continues with translational and rotational acceleration. This is the mechanism of cerebral injury which is responsible for loss of consciousness (Gleave, 1986). The degree of injury is dependent on a number of factors and will vary according to the athlete's equipment, baseline neck strength and ability to tense neck muscles, thereby absorbing or dissipating some of the impact (Sturmi et al., 1998).

What has become apparent in the field of contact sports is the failure of coaches and players to take cognisance of the degree of cerebral insult resulting from repeated head trauma. It has been estimated that 70% of players return to play after sustaining a mild head injury, when they face a very real risk of further concussions. This occurs despite warnings from management guidelines that a player should abstain from play for a period of one week if he/she is symptomatic after an injury (Cantu, 1986). It has been argued that coaches tend to allow concussed players to return to play as the effects of a mild head injury are not always discernible and may only become noticeable when players cannot remember the game. A common occurrence is the so called "ding", which occurs when a player sustains a concussion but does not register any pain and is judged fit to continue play. The player may only later become aware of the consequences of the "ding", when he/she experiences memory difficulties during the game (Yarnell & Lynch, 1973).

Cumulative mild head injury in contact sport has been associated with a high risk of adverse (potential) consequences. It has been well documented that a fatal outcome can result from repeated minor head injury occurring in rapid succession (Kelly, Nichols, & Filley, 1991; Saunders & Harbaugh, 1984). Saunders and Harbaugh (1984) were the first researchers to draw attention to the fact that a fatal brain swelling can occur in the setting of recent mild head injury followed by a second mild head injury, in athletes who are still symptomatic from the first injury. This has been termed the second impact syndrome of catastrophic head injury (SIS). It is generally thought that this syndrome is due to a sensitivity of the cerebral vasculature induced by the first injury. The second injury leads to autoregulatory dysfunction, cerebral vascular congestion and subsequent raised intercranial pressure, and potentially to death (Warren & Bailes, 1998). As this syndrome can occur after a Grade 1 concussion where there has been no loss of consciousness, it becomes vital that sports health practitioners are able recognize all grades of concussion and adhere strictly to appropriate management guidelines (Cantu, 1998).

Although the precise incidence rate is not known, SIS is thought to be more common than previous reports suggest. Furthermore, while it was originally thought to be confined to American Football players, its occurrence has now been recognized in other contact sports, although it may not be labelled as such. Alarmingly, SIS has a mortality rate approaching 50% and a morbidity rate of nearly 100%, making prevention of athletic-related head injury a major concern in sports management (Cantu, 1996). This rare but catastrophic outcome has led some researchers to conclude that "there is no such thing as a mild concussion" (Sturmi et al., 1998, p. 351).

Despite the high incidence of mild head injury in contact sport with its potentially adverse (and even fatal) outcomes, there are few neuropsychological studies on the long-term effects of mild head injury in contact sport. The need for more intensive neuropsychological research is of particular relevance in reducing the rate and severity of head injury in contact sport and for assessing the effects of cumulative mild head injury. This is because such research helps to determine the extent of this problem and plays a role in encouraging a greater awareness and appreciation amongst sports health practitioners of the risks inherent in these sports, with the ultimate aim of making the game as safe as possible. In this respect, professional contact sports have been regarded as providing an ideal opportunity for measuring cognitive functioning pre- and post-injury (Hinton-Bayre et al., 1997) and as a useful model for understanding mild head injury in the general population (Barth et al., 1989).

The following section will review the literature on the contact sports of boxing, soccer, American Football, Australian Rules Football, Rugby League and Rugby Union. It will begin by focusing on incidence rates for each of these sports. This will be followed by a review of neurological studies conducted in each area. Thereafter, the focus will be on neuropsychological research in the area. In this regard, studies pertaining to neuropsychological deficits (objective cognitive test results) will first be discussed and this will be followed by consideration of those studies pertaining to self-reported postconcussive symptomatology (including emotional, cognitive and behavioural changes).

2.3.1. BOXING

While neuropsychological studies on boxing have focused on moderate to severe trauma, there has been little investigation of the effects of cumulative head injury in boxing (Barth et al., 1989). A possible explanation for this is that the nature of boxing is more likely to result in moderate to severe head injury, being that as the primary goal is to render the opponent unconscious though successive blows to the head (Casson, Sham, Campbell, Tarlai & DiDomenico, 1982; McCunney & Russo, 1984). This goal is commonly referred to as the knockout and is considered the most common acute neurological injury that occurs during boxing (Jordan, 1987).

Martland (1928) was the first to identify the progressive syndrome referred to as "punch drunk", which manifests with mild confusion and ataxia, followed by speech and motor deficits, and culminates in a movement disorder similar to Parkinson's disease. The diffuse cerebral atrophy found in boxers is currently referred to as "chronic boxer's encephalopathy" (Serel & Jaros, 1962 in Barth et al., 1989), "dementia pugilistica" (Lampert & Hardman, 1984 in Barth et al., 1989) and "chronic traumatic encephalopathy" (Jordan, 1987). These terms refer to the chronic progressive consequences of brain tissue damage resulting from numerous subconcussive and concussive blows to the head during boxing (Barth et al., 1989).

While neurospsychological studies into the effects of mild head injury in professional boxers have identified cognitive deficits (for example, Drew & Templer, 1992), the neurological and neuropsychological consequences of amateur boxing remain less certain. It is important to note that these two divisions of boxing differ in many respects. Some of these differences include the duration of fights (amateur boxers have shorter fights), rules and regulatory policies, medical evaluation, supervision and protective devices (Barth et al., 1989, Brookes, Kupshik, Wilson, Gabraith, & Ward, 1987; Butler, Forsythe, Beverly, & Adams, 1993; Haglund & Eriksson, 1993; Jordan, 1987; Ruchinkas, Francis, &, Barth, 1997). Thus, for purposes of differentiation, neuropsychological research on professional and amateur boxing will be reported separately in this work. However, where any study has included both professional and amateur players in its sample, that study will be examined as research on professional players, which will be discussed first.

A study by Kaste et al. (1984) of 14 boxers (six professional and eight amateur) revealed cognitive impairment for all boxers on the Trail Making Test, with two of the professionals demonstrating signs of more severe impairment. This led the researchers to conclude that the effects of repeated concussions are cumulative and may result in irreversible brain damage. However, the study's findings were compromised by the absence of an adequate matched control group and by a small sample. Another study that sought to examine the cumulative effects of repeated head trauma found cerebral atrophy in five out of 10 professional boxers (Casson et al., 1982). However, as each boxer had not been knocked out more than twice, the researchers concluded that the damage recorded was not due to the number of knockouts but rather to multiple subconcussive blows to the head. In another study by Casson et al. (1984), 18 boxers (15 former and active professionals, and three amateurs) were subject to a neurological examination, an EEG, a CT scan and a number of neuropsychological measures, including the Trail Making Test, the Digit Symbol Test, the Bender Visual-Motor Gestalt designs and the Wechsler Memory Scale. Of the 15 professional players, 13 were found to have abnormal results on at least two of the tests. In addition, all the boxers were found to be impaired on at least one of the four neuropsychological measures.

In a neuropsychological study investigating the cognitive performance of active professional players, Drew, Templar, Schuyler, Newell and Cannon (1986) found impairment on the Finger Tapping Test relative to the control group. In addition, the researchers noted a high correlation between the number of professional bouts and lost matches, although no significant correlation between former amateur career and signs of brain injury was recorded. Similarly, in a neuropsychological study of 15 former and active professional boxers, Ross, Casson, Siegel and Cole (1987) found that the neuropsychological-test-impairment index used in the study correlated with the number of professional fights and increasing age. In addition, greater impairment was found on those tests that had a strong memory component, than those not heavily weighted for memory. The researchers concluded that the development of abnormal neuropsychological test scores might be the earliest and first signs of subtle chronic cerebral injury.

As noted earlier, research findings on amateur boxers have tended to be inconclusive. While some studies have found evidence of cognitive impairment as a result of cumulative head trauma sustained in boxing (for example McLatchie et al., 1987), others have not (for example, Brooks et al., 1987). McLatchie et al. (1987) studied 20 active amateur boxers and found impaired performance relative to controls on the Inglis Word Learning Test and on the copy and immediate recall of the Rey Figure, thereby indicating deficits in verbal learning and memory. Additionally, correlations were found between abnormal neurological examinations and increasing number of fights as well as between abnormal EEG and decreasing age. In a neuropsychological study, Heilbronner, Henry, and Carson-Brewer (1991) assessed 23 amateur boxers before and after a boxing match and found impairments on measures of verbal recall and incidental memory. In contrast to these studies, a neuropsychological study conducted by Brookes et al. (1987) that used tests of visual and verbal memory, attention, information processing and motor function and intellectual abilities on amateur boxers, found no significant differences between amateur boxers and controls. Similarly in another neuropsychological study that assessed the cognitive functioning of 86 amateur boxers compared to matched controls on three occasions – pre-bout, immediately post-bout and a follow up within two years, Butler et al. (1993) found no evidence of neuropsychological dysfunction due to boxing, either following a bout or a series of bouts, at follow up. The researchers also found no relationship between cognitive functioning and the number of previous contests or the number of head blows received during a bout.

Both of the above-cited studies found no evidence of cognitive damage as a result of cumulative head trauma and have been criticised for methodological weaknesses. These include an inadequate control group, a lack of premorbid data and an inappropriate sampling method. In a retrospective study, Haglund and Eriksson (1993) compared 50 former amateur boxers with two control groups,

comprising of soccer players and track and field athletes, and found no significant difference between the groups on any of the neuropsychological and neurological examinations. However, it was noted that the boxers had an inferior finger-tapping performance and a higher incidence of moderate EEG deviations, both of which may be indicative of slight brain dysfunction.

While there has been much neuropsychological research on the cumulative effects of mild head injury in boxing, the issue of postconcussive symptomatology in boxers has been largely underinvestigated. In this respect, Jordan (1987) argues that the true frequency of postconcussive symtomatology among boxers following a bout remains unknown. While some studies on boxing have incorporated a postconcussive symptomatology evaluation (for example, Kaste et al., 1982), there is a tendency amongst boxers to not report the occurrence of these symptoms, despite there being objective evidence. This is not really surprising for, as noted earlier (p. 32), there is a general tendency in the contact sports for players to underreport any difficulties they experience, due to their concern about being judged "weak" or unfit to play.

2.3.2. SOCCER

Soccer is one of the most widely played sports in the world, with an estimated 200 million registered participants (Matser, Kessels, Jordan, Lezak, & Troost, 1998). While it has been generally regarded as a relatively safe sport with a low injury rate (Dailey & Barsan, 1992; Nilsson & Roaas, 1978 in Abreau et al., 1990), this assumption is now being questioned due to the game's unique feature of heading. Heading refers specifically to the purposeful use of the head for advancing and controlling the ball's motion (Barnes, Kirkendall, McDermott, Jordan, & Garret, 1998; Boden, Kirkendall, & Garret, 1998). This occurs frequently in soccer, with the average player heading the ball up to 10 times a game (Green & Jordan, 1998). Cumulatively, it is estimated that if a soccer player plays 300 games during his/her soccer career, he/she will receive about 2000 blows as a result of heading (Tysvaer & Storli, 1989). Furthermore, because soccer is a contact sport, it also carries the risk for a number of other sport-related injuries of which a significant percentage (4-22%) is head and neck injuries. In soccer, head injuries can occur in two ways. Firstly, through major impact with a moving or immovable object (e.g. head, ground or goalpost) and secondly, through chronic injury as a result of repetitive minor impacts. Despite the concern this raises for cerebral damage as a result of cumulative mild head injury, there appears to be a dearth of studies investigating the impact of heading on neuropsychological functioning.

With regard to neurological research on the sequelae of head injuries sustained in soccer through heading, Tysvaer and Storli (1989) and Tysvaer, Storli and Bachen (1989) found higher rates of EEG

abnormalities in soccer players compared to matched controls. In addition, these disturbances were more pronounced among the younger players. They concluded that the high incidence of EEG disturbances was probably a result of a cumulative effect due to sustained mild head injuries. However, both studies were compromised by methodological flaws, including the absence of suitable control groups, a failure to control for other factors that could lead to central nervous system disturbances, and a failure to screen for previous head injuries and a history of substance abuse. In another neurological study designed to determine whether repetitive heading of the soccer ball leads to chronic encephalopathy, Jordan, Green, Galanty, Mandelbaum and Jabour (1996) compared 20 elite soccer players with a control group of elite track athletes using MRI measures and a head injury questionnaire. No significant differences were found between the two groups on both measures. One of the strengths of this study was that it took account of alcohol use and previous head injuries when analysing the results.

With regard to neuropsychological research on the sequelae of head injuries sustained in soccer, Abreau et al. (1990) compared the cognitive performances of 31 collegiate soccer players and 31 collegiate tennis players using the Ravens Progressive Matrices, Symbol Digit Modalities, Perceptual Speed Tests and the PASAT. While no significant differences were found between groups on the cognitive tests, a negative correlation between the number of games played and performance on the PASAT was recorded. This finding suggests compromised information-processing abilities as a result of cumulative mild head injury. However, due to this study's methodical limitations (a small sample size, a lack of both premorbid data and repeated post injury testing), the researchers concluded that their findings provided only tentative support for the presence of neuropsychological deficits due to cumulative head injuries.

In a further attempt to examine the relationship between heading and cumulative mild head injury, Tysvaer and Lochen (1991) compared the neuropsychological performance of 37 former soccer players with a control group of 31 tennis players on measures of general intelligence [Wechsler Adult Intelligence Scale Revised (WAIS – R), sustained attention, concentration and mental flexibility (Trailmaking Test], aphasia, sensory perceptual skills, motor functions and visual memory (Benton Visual Retention Test). Results indicated impaired performances for the soccer players on measures of attention, concentration, memory and judgement. In addition, a higher degree of neuropsychological impairment was found in players who were headers than non-headers. The researchers argue that these results may indicate some degree of permanent brain damage as a result of cumulative trauma from heading the ball and they conclude that the brain damage found is similar to that found in patients who have sustained mild head injuries.

A more recent study by Matser et al. (1998) compared the cognitive performance of 53 Dutch active professional players with a control group of 27 professional non-contact sports players (swimmers and runners). Results indicated impaired performances for soccer players on measures of planning, memory and visuoperceptual processing. An inverse relationship was found between soccer players' performances on memory, planning and visuperceptual tasks and the number of previous mild head injuries and frequency of heading the ball. In addition, forward and defensive players were found to be more vulnerable to cognitive impairment, compared with midfield players and goalkeepers. This is consistent with Tysvaer and Lochen's (1991) finding, which also indicated a higher degree of neuropsychological impairment in headers than in non-headers.

While some research studies provide support for cognitive impairment as a result of heading, other studies have failed to find evidence of brain trauma following head injury. Haglund and Erikkson (1993) used soccer players who were considered "typical headers" as a control group in a study comparing former amateur boxers, soccer players and athletes. No differences were found on the CT scan and MRI measures between all three groups. Barnes et al. (1989) and Boden et al. (1989) conducted concurrent studies to determine the concussion incidence in elite male and female soccer players. The results of both studies were consistent. Findings demonstrated that a) male soccer players had a higher incidence of concussion than female players; b) a male player had a 50% probability of sustaining a concussion; c) the most common injury mechanism was collision with another player and; d) that most mild head injuries in soccer are classified as Grade 1 concussions (as per Cantu's (1996) definition). In Boden et al.'s (1998) study, it was recorded that there was not a single case where concussion occurred by routine heading of the ball. This led them to conclude that if long-term encephalopothy changes exist in soccer players, they are more likely to result from concussion sustained by collision with another player than from cumulative subconcussive injury arising from heading the soccer ball. Both these studies were, however, compromised by their lack of a control group.

Recently, research has begun to focus on the relationship between head injury and the onset of dementia. In exploring the possibility of a connection between head injury sustained from soccer and the development of dementia, Spear (1995) has argued that head injury is frequently cited as an environmental cause associated with the development of Alzheimer's disease. He maintains that footballers may face an increased risk of developing this disease as a result of repeated head injuries from heading the ball. However, since the long-term effects of mild head injury have yet to be established, it cannot be concluded that soccer players are at increased risk of developing Alzheimer's disease. However, given the implications of Spear's argument, it is vital that this subject be a focus of future research.

With regard to postconcussive symptomatology, neuropsychological studies have found evidence of postconcussive symptomatology in soccer players. In the previously noted study by Abreau et al. (1990), the researchers found that a significant number of soccer players reported experiencing headaches, blurred vision, dizziness and passing out after a game. However, the researchers were unable to ascertain the period of recovery from reported symptomatology, in order to establish whether these symptoms have long-term effects. Tysvaer and Storli (1989) in their study of the neurological sequelae of heading the ball in soccer found that former professional players reported headaches, dizziness, irritability and memory impairment. Similarly, in a neurological and neuropsychological assessment of boxers that used soccer players as the control group, Thomassen et al. (1979) found that 70% of soccer players complained of persisting memory and concentration difficulties (cited in Baroff, 1998). In Jordan et al.'s (1996) study, the researchers found a correlation between reported symptoms and the number of prior acute head injuries amongst the soccer players. They concluded that reported symptoms appeared to be related more to acute head injuries received playing soccer than from heading the ball. In Barnes et al.'s (1998) study, the researchers found that headaches (54%), being dazed (31%) and dizziness (18.1%) were the most common symptoms reported by players after heading the ball. Of the players, 6.9% reported long-term sequelae such as recurrent headaches or vertigo. However, this study was compromised by a number of methodological limitations including its retrospective nature and small sample size.

2.3.3. AMERICAN FOOTBALL, AUSTRALIAN RULES FOOTBALL, RUGBY LEAGUE AND RUGBY UNION

The goal of these four sports is to score more points than the opponent. In Rugby League and American Football, this is done by carrying, passing, kicking and grounding a ball in the scoring zone at the far end of the field. In contrast, scoring in Australian Rules Football occurs when the ball is carried to the far end of the field and then kicked through the goalposts. Most head injuries in American Football, Australian Rules Football, Rugby League and Rugby Union occur as a result of stresses and impacts on the head and neck from tackling, scrumming and collisions between players (Shuttleworth-Jordan et al., 1993). Tackling is one manoeuvre that is common to all four sports and occurs when a running player is held and brought to the ground by another. This results in sudden deceleration, which can cause mild head injury. In Rugby League and Rugby Union only the player carrying the ball may be tackled, whereas in American Football and Australian Rules Football, players without the ball may be tackled or blocked. In Rugby League teams there are 13 players who are allowed six tackles with the ball. Once each set of tackles is completed, the ball is handed to the opposition team to begin its set of six tackles. This means that the same players are both offensive and defensive players, depending on which team has the ball.

Another difference between Rugby League and Rugby Union, and American Football and Australian Rules Football, is scrumming, which only occurs in the first two sports. In a scrum, the forward players link up and bend down in order to allow the ball to be put on the ground between them. Once the ball has been placed between them, the two teams have to try to push the opposing team off the ball, in order to gain possession of it for their team. Another source of mild head injury that occurs in Rugby Union alone, is during "rucks' and "mauls". These refer to situations in which the player who is carrying the ball is held down by the opposition while the forward and backline players of both teams climb into the ruck or maul (a group of players bent forward at the waist and holding each other), often headfirst, in order to gain possession of the ball. As this does not occur in Rugby League, it appears that Rugby Union players are at increased risk for sustaining mild head injuries than Rugby League players. Importantly, it is Rugby Union which is the focus of the present research.

2.3.3.1. American Football

Due to a paucity of controlled prospective studies on mild head injury in American Football, much of our understanding of this sport comes from epidemiological, descriptive, retrospective and case studies. Despite the lack of prospective neuropsychological studies, recent concern regarding mild head injury has generated interest in high school communities, with a focus on identification, recovery and return to practice. This is because the incidence of mild head injury in American Football is particularly high, with approximately 10% of all college football players sustaining a mild head injury over any given season and the majority of football players reporting one or more mild concussions during their careers (Barth et al., 1989). With regard to high-school football players, there is an estimated 15-20% risk of mild head injury each season, with more than 200.000 concussions occurring annually (Metzl, 1999; Warren & Bailes, 1998). Furthermore, players who sustain a single concussion are four to six times more likely to experience a subsequent concussion (Gerberich, Priest, Boen, Straub & Maxwell, 1983; Zemper, 1994 in Erlanger, Kutner, Barth, & Barnes, 1999).

In a retrospective study of 3 064 players from 103 Minnesota High Schools, Gerberich et al. (1983) found that 19% of players had suffered possible concussive mild head injury and that 60% of players who experienced loss of consciousness returned to play the same day. In a prospective study of head and neck injuries of 342 college football players over an eight-year period, Albright, Mcauley, Martin, Crowley and Foster (1985) established an incidence of 175 injuries per 100 players (29%) during the length of their study. Consistent with Barth et al.'s (1989) and Gerberich et al.'s (1983) findings, their study also found an increased risk of future injury associated with a prior history of

players who had sustained mild head injuries. They compared their performance to 12 non-injured controls using a cued reaction time task. In the first stage of the research, nine footballers tested within two weeks of sustaining a head injury demonstrated the same deficit as controls in the speed of their response to targets in an unexpected visual field. However, their responses to targets in the expected location showed only a minor benefit when compared with controls. On retesting a year later, their pattern of performance did not differ although overall reaction time had improved. The researchers conclude that a persistent consequence of mild head injury might be an inability to act speedily in response to expected spatial events.

To date, there does not appear to be any research on the presence of postconcussive symptomatology in Australian Rules Football players.

2.3.3.4. Rugby League

There is a high incidence of head and neck injuries in Rugby League. In a three year survey of 24 Rugby League teams in Australia, Seward, Orchad, Hazzard and Collinson (1993) found that concussion accounted for 8.5% of all Rugby League injuries. This finding is consistent with a study by Stephenson, Gissane and Jennings (1996), which investigated the incidence of injury in English professional league rugby players over four seasons. The authors found the most frequently injured site was the head and neck region (33.3% of all injuries), and that the forward players had a higher injury rate than the backline players. They concluded that high rates of injury in Rugby League may be due to the intensive amount of bodily contact in the game. Forwards players are at greater risk due to their being involved in more collisions and more repetitive body contact. This has been supported by other studies on injury rates in Rugby League (Gibbs, 1993; Gissane, Jennings, Cumine, Stephenson & White, 1997; McQillan, 1992).

To date, there are few studies on the neuropsychological consequences of cumulative mild head injury in Rugby League players. Rather, the focus of research has tended to be on the acute and subacute stages of a single mild head injury. A study by Hinton-Bayre et al. (1997) examined the sensitivity of certain tests of information processing to the effects of mild head injury in professional Rugby League players. The neuropsychological measures employed were the Symbol Digit Modalities Test, the Digit Symbol Substitution Test and the Speed of Comprehension Test. When compared to pre-injury baseline scores, results indicated that measures of speed of comprehension and information processing were impaired in the post-acute phase of mild head injury, whereas the untimed word-recognition task was not. This finding is consistent with other research studies (Barth et al., 1983; Maddocks & Saling, 1991). However, while this study utilized premorbid data and

control groups for practice effects, its findings were compromised somewhat by a restricted test battery and a failure to take into account the prior concussive and subconcussive history common to most professional contact sport players.

To this author's knowledge, there do not appear to be any studies investigating the presence of postconcussive symptomatology in Rugby League players.

2.3.3.5. Rugby Union

A review of the literature suggests that the incidence of concussion in Rugby Union may in fact be higher amongst schoolboy players than among adult players. This is certainly the experience identified in studies undertaken in South Africa. In this respect, Nathan, Goedecke and Noakes (1983) found that concussion accounted for 20% of all schoolboy rugby injuries, and that the incidence of injury increased with increasing age and level of competence. The researchers also found that, on average, 10% of schoolboy rugby players will sustain a concussion during the course of the season. In addition, it was noted that rugby forward players were found to have higher rates of injury than rugby backline players, and that injury was more likely to occur during a match than during a practice. This research was consistent with the findings of another study by Roux et al. (1987), which investigated the incidence of injury in schoolboy rugby at 26 high schools. Like the previous study, this study also found that top level players (A team) are especially injury prone and that injuries were more likely to be sustained during tackles and loose scrummaging, making mild head injury more common among eighthmen (forwards), followed by fly-halves (backs). While this study reported a slighter lower incidence of concussion (12%) than the previous one, the researchers note that there may be an underreporting of concussion injuries due to ignorance about the nature of the injury. They argue that the incidence rate could have actually been higher, as subconcussive blows which do not result in LOC may go unreported. It has been observed that this is often due to players' anxiety about being suspended from play, which might lead to them being excluded from the team (Shuttleworth-Jordan et al., 1993). This observation concurs with Macleod's (1993) comment that underreporting may also be due to a degree of collusion between rugby players, coaches and medical attendants who are reluctant to make a diagnosis of a concussion without objective evidence, due to recommendations by the International Rugby Football Board regarding the three week period of rest.

With regard to Rugby Union, it appears that all the neuropsychological research in this area has been conducted in South Africa. In a study exploring the effects of cumulative mild head injury in rugby, Shuttleworth-Jordan et al. (1993) compared 60 university rugby players (five of whom sustained a

mild head injury during the season) with 25 matched non-contact sport controls across a neuropsychological test battery, comprising of tests selected specifically for their sensitivity to the effects of diffuse brain damage. Neuropsychological measures included hand motor dexterity (Denckla Finger Tapping Test and Purdue Pegboard Test), short-term verbal memory (Digits Forwards), verbal new learning (Digit Supraspan) and working memory (Trail Making Tests Parts A and B, and Digits Backwards). The study focused on two aspects: i) an investigation of pre- and post-season differences between non-head-injured rugby players and matched controls and ii) an investigation of repeated test differences between the five concussed rugby players and matched controls assessed at pre-season, three days, one month, two months and three months post-injury. Results from the analysis of the pre-and postseason comparisons indicated that rugby players had impairments in working memory (Trailmaking Test Parts A and B), verbal new learning ability (Digit Supraspan) and hand motor dexterity (Purdue Pegboard and Purdue Bimanual tasks) relative to controls; a pattern of deficits typically associated with closed head injury caused by diffuse brain damage effects. While the rugby group scored significantly faster Finger Tapping scores (a pattern inconsistent with the general trend of the results), the authors note that the differences amount to fractions of a second and conclude that this test has little interpretative validity as it cannot be scored rigorously enough to ensure reliable differences. Further analysis of results indicated that the forwards players demonstrated greater impairment than the backline players. This was attributed to their participation in scrumming, which would predispose them to cumulative brain damage effects.
As the researchers decided to exclude any players who had reported more than one concussion in the previous three years, the results were considered to provide an estimate of permanent deficits in the rugby playing group, either due to previous concussions or as a result of unreported concussions during the season.

With regard to results of the repeated test measures, it was found that the five players who reported sustaining a mild head injury during that season, and who were followed up by repeat testing at three days, one month, two months and three months along with the matched controls, showed significant impairments in attention, verbal new learning, working memory and hand motor dexterity at three days post injury. At the one month interval, substantial recovery in the rugby group was noted, with further recovery indicated at the two month interval. However, by the three month interval, the concussed group did not evidence the same degree of practice effect as the control group on Digits Backward, Digits Difference, Digit Supraspan A and B, and the Finger Tapping Test Preferred and Non-Preferred Hand, thereby indicating that recovery was not yet complete on those measures. This pattern of impairment in the concussed rugby-playing group is highly comparable with the pattern of impairment recorded in the "non-concussed" rugby-playing group on the pre- and postseason comparisons.

In concluding, the authors point out that recognition of the presence of deficits in working memory, verbal new learning ability and speed of hand motor dexterity is vital in order to cope with ensuing reductions in scholastic and occupational abilities. They argue that these compromised higher cognitive functions have particular consequences for students who rely heavily on these exact skills to achieve optimal academic performance. Furthermore, they emphasize that the risks for Matric schoolboy rugby players are particularly high, as many will be attempting to obtain entrance into competitive and demanding tertiary programs. This is also true for borderline achievers, who may be in danger of failing pre-injury and for whom any further slight reduction in functioning may have a potentially disastrous outcome. They recommend that any player who has sustained a mild head injury should be advised to delay undertaking any task that would require optimal academic performance for at least three months post-injury.

While some of the strengths of this study were its use of baseline premorbid data, its repeat testing of control groups to account for practice effects and the use of neuropsychological tests reported to be sensitive to brain damage, its findings were somewhat compromised by the small sample of concussed players as well as a small test battery, which, while sensitive to the effects of diffuse brain damage, was still fairly limited.

More recently, in an attempt to build on Shuttleworth et al.'s (1993) study, a long-term research project was initiated in 1996 at Rhodes University, which aimed at investigating head injury in rugby. A central focus of this project included an examination of the cumulative effects of repeated head trauma, commonly sustained in rugby. As noted in Chapter one (p. 1), the initial phase of this project consisted of a comparison of neuropsychological test performances of professional rugby players (n=26) with a matched control group of professional cricket players (n=21). Data from this project was broken up into three separate research projects, namely (i) a direct comparison of group mean scores between rugby and cricket players (Ancer, 1999); (ii) a comparison of rugby and cricket mean scores, against available normative data (Reid, 1998) and, (iii) a comparison of the percentage of rugby and cricket players showing cognitive deficit across each test, as well as a comparison of the frequency of reported postconcussive symptomatology (Dickinson, 1998). The results of these three studies will be briefly discussed below.

Ancer's (1999) study revealed no mean score differences between rugby and cricket players, with the exception of results on tests of Finger Tapping which recorded significantly poorer performances by the cricket players relative to the rugby players. However, as previously noted, this test which assesses hand motor dexterity has little interpretative validity, since it cannot be scored rigorously enough to ensure reliable differences. Furthermore, these results can be affected by hand injuries,

which are commonly sustained in rugby. Ancer's study did, however, find evidence of significantly increased variability of scores for the rugby players compared with the cricket players on measures of working memory (Trail Making Test Part B), hand motor dexterity (Finger Tapping Test Non-Preferred hand) and visuoperceptual tracking (SAWAIS Digit Symbol Substitution Test). Similarly, Reid (1998) also found an absence of any significant differences in mean scores between rugby and cricket players. However, increased variability in the rugby players relative to the cricket players was noted on measures of visual memory [WMS Visual Reproduction (Delayed Recall)], visuoperceptual tracking and working memory (Trail Making Test Part B) and hand motor dexterity (Finger Tapping Test), while significant variability in the rugby players relative to the norms was noted for measures of recent visual memory [Digit Symbol Incidental Recall (Immediate)]. Additional analysis revealed that the forward players were disproportionately poor compared to the norms on measures of working memory (Digits Backward), visuoperceptual tracking (SAWAIS Digit Symbol Substitution and Trail Making Tests) and hand motor dexterity (Finger Tapping Test). Results of Dickinson's (1998) study indicated impairments in the rugby players relative to cricket players on comparisons of individual levels of cognitive deficit for measures of speed of information processing and attention (Trail Making Test), visuoperceptual tracking (SAWAIS Digit Symbol Substitution Subtest), as well as verbal and/or visual memory Digits Forward and Digit Symbol Incidental Recall). Like Reid's study, her results indicated that rugby forward players were more vulnerable to cognitive deficit in comparison to backline players on measures of visuoperceptual tracking (SAWAIS Digit Symbol Substitution Subtest) and working memory (Trailmaking Test Part B).

While these studies provided some support for the hypothesis that cognitive functioning is compromised by cumulative concussive and subconcussive injuries, their findings contained a number of methodological weaknesses. These included a small sample size and a problematic control group. More specifically, it was found that the cricket players were suffering from fatigue and a lack of motivation due to their having been tested postseason, in comparison to the rugby players who were tested pre-season. In addition, many of the cricket players were found to have had a history of rugby playing. It was felt that this feature may have contaminated the results and led to an underestimation of the cognitive deficit in rugby players.

In an attempt to build on this study and improve its methodology, a second stage of research was instituted. This study utilized a larger sample consisting of professional rugby players and Under 21 rugby players. Once again, data was broken up into three research projects, and the same three levels of analysis applied (direct comparison of group means, comparison of rugby means with normative data and comparisons of individual levels of cognitive deficit and frequency of postconcussive symptomatology).

The first level of analysis (Finkelstein, 1990) found a consistent pattern of poorer performance across all rugby groups relative to controls on measures of verbal fluency (Words in One Minute Unstructured Verbal Fluency Test), visuoperceptual tracking at speed (SAWAIS Digit Symbol Substitution Subtest) and working memory (Trail Making Test). The second level of analysis (Bold, 1999) revealed significant differences in the direction of poorer performance relative to the norms for all rugby playing groups and rugby forward players relative to rugby backline players on measures of working memory (Digits Backwards and Trail Making Test Part B), visual memory and rate of information processing [WMS Visual Reproduction (Immediate Recall)] and visuoperceptual tracking at speed (SAWAIS Digit Symbol Substitution Subtest). The third level of analysis (Border, 2000) found significant differences in levels of individual deficit between all groups on measures of rate of information processing (SAWAIS Digit Symbol Substitution Subtest), attention and concentration (Digits Forward), mental flexibility (Trail Making Test Part B), visual memory [(WMS Visual Reproduction (Delayed Recall)] and verbal new learning [(WMS Paired Associate Learning – Hard (Immediate Recall)]. Consistent with the other studies, Border (2000) found forward players' performances to be disproportionately poor, relative to backs on measures of recent visual memory [Digit Symbol Incidental Recall (Immediate)], verbal new learning [WMS Paired Associate Learning – Hard (Immediate Recall)], visuoperceptual tracking (SAWAIS Digit Symbol Substitution Subtest), working memory (Trail Making Test Part B) and hand motor dexterity (Finger Tapping Test). Thus Border's (2000) findings corroborate those of Dickinson's (1998) study which, as noted earlier, found differences in levels of individual deficit for speed of information processing, attention, visuoperceptual tracking and visual/verbal memory. Overall, it appears that the findings of phase two of the research project are consistent with, and corroborate, the earlier findings of phase one, thereby suggesting that rugby players, in particular rugby forward players, evidence signs of cognitive deficit, due to their exposure to cumulative mild concussive and subconcussive head injuries

Apart from the above cited research on Rugby Union and professional players in South Africa, there do not appear to be any studies examining the incidence and manifestation of postconcussive symptomatology in Rugby Union players. In their study exploring the cumulative effects of mild head injury in rugby on university players Shuttleworth-Jordan et al. (1993) found that headaches, nausea, visual disturbance, poor attention and concentration, anxiety, insomnia, fatigue, vomiting, weakness of limbs, loss of appetite, sensitivity to noise, restlessness, clumsiness and speech problems, such as slurring and stumbling, were present in the rugby players three days post concussion. There was, however, a gradual reduction in postconcussive symptomatoloy over the three-month period and, at three months, no symptoms were reported that were not part of the premorbid presentation. In Dickinson's (1998) study, which investigated the presence of postconcussive symptomatology in professional rugby players, the most significant postconcussive symptoms found to be present in the

rugby players relative to the controls were in the areas of anxiety, depression, irritability and low frustration tolerance (argumentativeness). Furthermore, Dickinson found that there was a greater frequency of the above mentioned symptoms reported by rugby forward players relative to backline players. These findings were corroborated by Border's (2000) study (which replicated Dickinson's study) and found the most significant postconcussive symptoms present in the rugby players relative to the controls to be in the areas of anxiety, depression and low frustration tolerance. In addition to these symptoms, his findings recorded a greater presence of impairment in the additional areas of social contact, memory, and sensitivity to noise and worry.

Thus, to date, neuropsychological research has focused primarily on adult, university and professional populations of Rugby Union players. Despite the high incidence of concussion in schoolboy rugby, there has been no neuropsychological research, to this author's knowledge, in this area. Given this consideration, it was therefore decided that this population was an important target for the next stage of research in the Rhodes Psychology Clinic's research project into the cumulative effects of mild head injury. In this respect, a decision was made to replicate the proceeding studies, while ensuring that the methodology of the study was more suited to a schoolboy population. To this end, the same test battery was used although modifications were made to make it more appropriate for a younger experimental group. Furthermore, the test battery was refined to include more sensitive measures of premorbid ability, an important variable when attempting to establish equivalence between groups. Finally, given the problems noted with the control group from the phase two of the research project (see p. 1), it was decided to use a control group of hockey players. This is because the two sports have corresponding seasons of play, thereby making it less likely that the hockey players would have participated in rugby.

Section Summary

The nature of the contact sports predispose their players to increased risks of sustaining cumulative mild head trauma. Injuries in contact sports commonly occur through acceleration/deceleration injuries to the head but can also occur in the absence of a direct blow to the head, such as through whiplash type injuries, that result from manoeuvres such as tackling. The incidence rate of mild head injury in contact sport is high and the consequences potentially catastrophic. In this respect, fatal outcomes have been documented from repeated mild head injury occurring in rapid succession.

While neuropsychological research of professional boxers has provided support for cognitive impairment as a result of mild head injury, as well as cumulative brain trauma from multiple injuries, studies of the neuropsychological effects of amateur boxing have produced ambiguous results.

Neuropsychological studies of soccer players have indicated that heading and mild head injuries resulting from collisions between players can result in cognitive impairment in players. With regard to American Football, Australian Rules Football, Rugby League and Rugby Union, neuropsychological research has produced variable findings. Research on American football has indicated neuropsychological deficits in the areas of information processing and attention. Research on Australian Rules football has indicated permanent visuospatial attention deficits as well as impairments in information processing speed, decision time and reaction time in the acute stages following a mild head injury. Research into Rugby Union has indicated permanent deficits commonly associated with diffuse brain damage in rugby players. More recently, research in South Africa has indicated impairments in visuoperceptual tracking, rate of information processing and attention. Positional variation has also been noted, with rugby forward players demonstrating greater levels of impairment than the rugby backline players.

There has been very little research on postconcussive symptomatology following mild head injury in contact sports. While some transient postconcussive symptoms have been identified in professional boxers following a bout, these symptoms are not reported by amateur boxers. Postconcussive symptoms in soccer players are believed to be related to the number of prior head injuries received. However, it is unclear as to whether this is due to repeated heading of the ball or a result of collisions between players. With regard to American Football, there has been some evidence indicating the presence of postconcussive symptomatology six to nine months following the end of the season. With regard to Rugby Union players, postconcussive symptomatology appears to take a period of three months to resolve, with a complete absence of symptoms at three months, despite continuing cognitive impairment. The most recent research has indicated that the most significant postconcussive symptoms were anxiety, depression, irritability and lowered frustration tolerance (argumentativeness).

Thus, it was decided that given the high incidence of concussion in Rugby Union as well as the clear findings indicating the presence of both neuropsychological deficits and postconcussive symptomatology, it was necessary to focus now on schoolboy rugby populations, particularly given the absence of research in this area. Such a study was considered vital as it would enable researchers to assess whether the cognitive deficits and self reported postconcussive symptomatology noted in professional Rugby Union players could be identified at this earlier level of participation. This in turn would encourage consideration of the ways in which this sport might be made safer at a school level, such as instituting the use of headgear as compulsory or stricter management guidelines for on-field injuries.

2.4. BRAIN RESERVE CAPACITY (BRC) THEORY

The present study draws on Satz's (1993) Brain Reserve Capacity (BRC) Theory in order to provide a theoretical context for the current research. Satz's (1993) theory postulates that each individual has a threshold factor that exists prior to the manifestation of symptoms resulting from disease in the central nervous system. This threshold factor corresponds to an amount of functional brain tissue at which normal functioning is maintained. According to Satz (1993), individual differences exist in terms of BRC capacity and it is this variability which serves as either a protective or a vulnerability factor. More specifically, Satz (1993) posits that a higher BRC will decrease the risk of functional impairment, as there is less likelihood of the individual demonstrating symptoms of neurological impairment. Conversely, a lower BRC is more likely to result in an individual demonstrating symptoms of neuropsychological impairment. Furthermore, any reduction in BRC due to neurological pathology will serve as a vulnerability factor, predisposing the individual to greater risk of functional impairment.

According to Satz, head injury and age are two key risk factors which contribute to the lowering of BRC reserve capacity. While the effects of early head trauma may not cause discernible functional impairment, aggregation during aging may cause neuronal attrition, resulting in a reduction of cerebral reserves and pushing the individual over a critical threshold into functional impairment. Satz (1993) highlights a number of other risk factors, which may serve to reduce BRC that include low education levels and IQ, gender effects, and high task challenge (i.e. a cognitive task of appropriate challenge). These factors may reduce BRC and increase the individual's vulnerability to symptom onset and functional impairment. Importantly, the relationship between task challenge and functional impairment has been demonstrated by research studies which have explored the impact of stressful task conditions on information processing, memory and vigilance (Ewing et al., 1980; Parasuruamn et al., 1991). With respect to gender, Satz (1993) does not specify the direction of difference, although according to Shuttleworth-Jordan (1999), male gender has been implicated in contributing to increased risk of cognitive impairment.

According to Jordan (1997), the central issue arising out of BRC theory is that there is a functional cut off point, which varies between individuals and depends on the presence of different vulnerability and protective factors. These pre-existing differential vulnerability factors will manifest in a variability of symptom presentation. Like Satz, she emphasizes the relationship between aging and vulnerability to symptom onset, arguing that the process of aging causes gradual reductions in BRC that can be equated with mild head injury effects. Furthermore, she argues that if the above mentioned protective and vulnerability factors are controlled for, the additional effect of a head injury

could potentially cause an individual to fall below the symptom threshold and manifest functional impairment. Thus, she hypothesizes that in the instance of cumulative mild head injury sustained in rugby, rugby players' exposure to successive concussive and subconcussive sustained mild head injuries relative to a control group, will lead to a reduction in BRC and so act as a risk factor in neuropsychological impairment. In addition, rugby forward players, who are more likely to sustain repeated mild head trauma after incurring more physical collisions during play, are also more likely to show a reduction in BRC and, therefore, increased vulnerability to neuropsychological impairment.

2.5. RESEARCH HYPOTHESES

Drawing on the empirical research reviewed above and the theoretical postulates of Satz's (1993) BRC theory, as explicated by Shuttleworth-Jordan (1999), the following hypotheses were posed.

(1) Among schoolboy rugby players there are likely to be higher percentages of individuals with cognitive deficit detected by those tests sensitive to diffuse brain damage, as well as a reported greater frequency of postconcussive symptomatology than amongst hockey players, due to their increased exposure to successive concussive and subconcusive head injuries. This is because the cumulative effects of such repeated head trauma would serve as a threshold lowering factor, reducing BRC and so causing increased vulnerability to neuropsychological impairment.

(2) Rugby forward players are more likely to demonstrate higher percentages of individuals with cognitive deficit recorded by tests sensitive to diffuse brain damage, as well as report greater frequency of postconcussive symptomatology, relative to rugby backline players. This is because the position and nature of playing the rugby forward players are more likely to result in increased physical collisions and, therefore, increased exposure to cumulative head trauma.

CHAPTER THREE: METHODOLOGY

The present study comprises the third phase of a larger ongoing research study investigating the cumulative effects of mild head injury in rugby players. This phase concentrated on a population of schoolboy rugby players and aimed to investigate levels of cognitive deficit and the frequency of self-reported postconcussive symptomatology of schoolboy rugby players. The rationale was an absence of research in this area, where incidence rates of concussion are markedly high. It was considered that such a study would help determine whether cognitive impairment as a result of mild head injury occurs at lower levels of participation in the sport and if so, to what extent the game could be made safer through the provision of management guidelines for on-field injuries.

3.1. PARTICIPANTS

The participants were drawn from three English-medium boys' high schools in Cape Town. The particular schools were selected for their traditional emphasis on rugby playing and their histories of excellence in the sport, both of which suggested an intensive and competitive level of participation. The sample consisted of Std 9, Matric and Post-Matric rugby players drawn from the top four teams and all currently active members of their teams. The rationale for the selection of older scholars was the length of their playing career; the assumption being that the majority of them would have played at least five years of rugby. It was presupposed that this feature, in combination with their top-team status and its high level of competition, would have predisposed players to increased risk of injury, thereby making them an ideal experimental group for investigating the cumulative effects of repeated head injuries. The control group for this study consisted of currently active hockey players from the top four teams who were likely to be equivalent with the rugby group for age, educational level, type of education and IQ. The rationale for using hockey players was that their utility as a control group had been previously confirmed by earlier research (Bold, 1998; Border, 2000; Finkelstein, 1998), which found that corresponding seasons of play (both rugby and hockey are winter sports) decreased the likelihood of hockey players having played rugby for any extensive period.

While a larger sample of 180 players was initially targeted (60 players from each school), the final sample for neuropsychological assessment comprised 96 players, due to refusal of consent. In order to mitigate against potentially confounding variables, exclusion criteria were applied which further reduced the sample size. The exclusion criteria for this study included: rugby and hockey players with a reported history of substance abuse; a neurological disorder, including any player with a prior moderate to severe head injury for any reason; a psychiatric/psychological disorder; a learning disorder; players 16 years of age but currently in Grade 10; and any players who had undergone a

recent psychometric assessment (to mitigate against potential practice effects). Additional excluding criteria were applied to the hockey players and included: hockey players who had played rugby for a considerable period (i.e. at least three years) and hockey players who had sustained a mild head injury within the three months prior to the assessments (to control for the presence of acute effects). Hockey players who reported a mild head injury (sport and non-sport related) prior to three months before the assessment were not excluded from the study. This was based on the rationale that a single mild head injury is unlikely to show detectable effects (Binder, 1997; Satz et al., 1997), and that the target of the present study was on cumulative mild head injuries expected to occur in rugby players. As noted in Chapter two (section 2.1.3, p. 7), mild head injury was defined according to Evans' (1992) criteria and included: LOC of less than 30 minutes; PTA of less than 24 hours and an absence of focal neurological deficits.

As a result of the exclusion criteria, the following participants were excluded: Rugby players - six exclusions (two learning disorders, one neurological disorder, one moderate head injury, one prior psychometric assessment, one learning disorder with accompanying neurological disorder); Hockey players - nine exclusions (three learning disorders, three moderate head injuries, one psychological disorder, two players in Grade 10).

In order to mitigate against potential pre-selection differences, measures were taken to ensure that both groups had equivalence on IQ level. A premorbid IQ was calculated by utilizing two separate measures; i) a prorated IQ score obtained from the calculated average of two Wechsler Adult Intelligence Scale – 3rd edition (WAIS-III) subtests, namely Vocabulary and Picture Completion and ii) an estimated IQ score derived from results on the National Adult Reading Test (NART). (The rationale for the selection of these three tests is discussed in section 3.5.1, pp. 62-63) As the two scores were found to approximate each other with no significant differences between groups and subgroups, results were combined to give an average total IQ estimate reflecting both IQ measures. This method was considered an improvement on the measures utilized in the earlier research on professional players (phases one and two), which consisted of the calculated average of the South African Wechsler Adult Intelligence Scale (SAWAIS) Vocabulary and Picture Completion subtests, because the addition of the NART incorporated a word recognition component - a cognitive faculty found to show resilience in the face of brain damage (Nelson, 1992). No players were excluded on the basis of estimated IQ, as the groups were found to be equivalent.

Given the importance of demographic data for estimating premorbid ability (Lezak, 1995), group mean comparisons and within group mean comparisons were obtained for the variables of *age*, *education level* and *estimated IQ*. An additional category of *"average grade 1999"* (which referred

to the average academic grade achieved for the proceeding year) was included in the demographic data to help determine levels of equivalence between players with regards to premorbid IQ. As premorbid ability is closely tied to academic achievement (Lezak, 1995), it was hypothesized that such a measure would approximate the IQ estimates obtained, thereby helping to establish equivalence across groups and subgroups.

The final sample for data analysis consisted of the following groups: Total Rugby (n=47) and Hockey Control (n=34) with additional subgroups of Rugby Forwards (n=28) and Rugby Backs (n=19). The demographic data of the two groups appear in Tables 3-1 to 3- 4 below.

Table 3-1. Demographic Data of Hockey and Rugby Players with Group Mean Comparisons for Age and Education

		Age				Education Level[1]			
	n	Mean	SD	Range	P-value	Mean	SD	Range	P-value
Total Rugby	47	17.3	0.7	16 - 18	0.07	10.8	0.6	10 - 12	0.46
Hockey Control	34	17.0	0.7	16 - 19		10.7	0.6	10 -12	

[1] Numbers of years of education completed.

Table 3-2. Demographic Data of Rugby Forwards and Rugby Backs with Group Mean Comparisons for Age and Education

		Age				Education Level[1]			
	n	Mean	SD	Range	P-value	Mean	SD	Range	P-value
Rugby Forwards	28	17.3	0.7	16 - 18	0.42	10.9	0.7	10 - 12	0.26
Rugby Backs	19	17.2	0.6	16 - 18		10.7	0.5	10 -11	

[1] Numbers of years of education completed.

Analysis of the demographic data indicated that there were no significant differences between Total Rugby and Hockey Control with respect to *age* ($p = 0.07$) and *educational level* ($p = 0.46$). Subgroup analyses also indicated no significant differences between Rugby Forwards and Rugby Backs with respect to *age* ($p = 0.42$) and *educational level* ($p = 0.26$). Thus overall the means for *age* and *educational level* were equivalent across groups and subgroups.

Table 3-3. Demographic Data of Hockey and Rugby Players with Group Mean Comparisons for Average Grade 1999 and Estimated IQ

		Average Grade 1999 (%)				Estimated IQ			
	n	Mean	SD	Range	P-value	Mean	SD	Range	P-value
Total Rugby	47	69.0	10.3	50 – 93	0.20	109.0	9.7	89.0 – 133.0	0.96
Hockey Control	34	72.2	11.6	50 - 97		109.1	8.6	90.5 – 129.5	

Table 3-4. Demographic Data of Rugby Forwards and Backs with Group Mean Comparisons for Average Grade 1999 and Estimated IQ

		Average Grade 1999 (%)				Estimated IQ			
	n	Mean	SD	Range	P-value	Mean	SD	Range	P-value
Rugby Forwards	28	70.5	10.1	55 – 93	0.23	110.0	10.3	89.5 – 133.0	0.42
Rugby Backs	19	66.8	10.5	50 - 86		107.6	9.0	89.0 – 119.0	

Analysis of the demographic data indicated that there were no significant differences between Total Rugby and Hockey Control with respect to *average grade 1999* ($p = 0.20$) and *estimated IQ* ($p = 0.96$). Subgroup analyses also indicated no significant differences between Rugby Forwards and Rugby Backs with respect to *average grade 1999* ($p = 0.23$) and *estimated IQ* ($p = 0.42$). Importantly, the data also indicate that both the rugby and hockey players constitute a high functioning population, with *estimated IQ* falling in the upper limits of the high average range (bordering on above average), for both the main groups and subgroups. This is commensurate with their a*verage grade 1999* scores, which are also above average and equivalent across both main groups and subgroups. There is also a consistent tendency for Rugby Forwards to be higher than Rugby Backs on *average grade 1999* (70.5 versus 66.8, respectively) and *estimated IQ* (110.0 versus 107.6, respectively), although in neither case is this difference significant, or even approaching significance.

Thus in sum, it appears that both the Total Rugby and Hockey Control groups, and the Rugby Forwards and Rugby Backs are all equivalent for the variables of age, educational level, school achievement and estimated premorbid level of intellectual functioning (estimated IQ). It can therefore be argued that the probability of these factors acting as confounding variables in this study is a highly unlikely one.

Table 3-5. Group Mean Comparisons of the Incidence of Reported MHI (including Sport and Non-Sport Injuries) in Rugby and Hockey Players

		MHI Sport [1]				MHI Non-Sport				Total MHI			
	n	Mean	SD	Range	p-value	Mean	SD	Range	p-value	Mean	SD	Range	p-value
Total Rugby	47	2.3	0.5	0 to 7		0.3	0.6	0 to 2		2.6	1.6	0 to 7	
Total Hockey	34	0.1	0.3	0 to 1	0.00 *	0.3	0.5	0 to 1	0.83	0.4	0.5	0 to 1	0.00 *

Significant Difference (* $p<0.05$)

[1] Where MHI Sport is reported, this refers to those injuries sustained by Rugby and Hockey Players in their respective sports.

Table 3-6. Group Mean Comparisons of the Incidence of Reported MHI (including Sport and Non-Sport Injuries) in Rugby Forwards and Rugby Backs

		MHI Sport[1]				MHI Non-Sport				Total MHI			
	n	Mean	SD	Range	p-value	Mean	SD	Range	p-value	Mean	SD	Range	p-value
Forwards	28	2.2	1.4	0 to 5		0.3	0.6	0 to 2		2.5	1.6	0 to 7	
Backs	19	2.4	1.7	0 to 7	0.74	0.3	0.6	0 to 2	0.74	2.6	1.7	0 to 7	0.85

[1] Where MHI Sport is reported, this refers to those injuries sustained by Rugby Players whilst playing rugby.

Analysis of the data indicated a significant difference between Total Rugby and Hockey Control for both reported sport-related MHI ($p = 0.00$), and total MHI ($p = 0.0$). Total Rugby reported a higher mean of sport-related MHI (2.3) in comparison to Hockey Control (0.1), and a higher mean for total MHI (2.6) in comparison with Hockey Control (0.4). While there were no significant differences between Rugby Forwards and Rugby Backs with regard to the above categories, Rugby Backs had a wider range for sport-related MHI (0 - 7) in comparison with Rugby Forwards (0-5).

3.2. CONSENT OF PARTICIPANTS

Before the study could proceed, permission had to be obtained from the Department of Education. Following this, the respective schools were contacted and preliminary meetings arranged between the researchers, the principals and the rugby and hockey coaches, to discuss the nature of the research and to establish interest in participation from the various schools. Permission was granted by all three principals to conduct the research on the condition that feedback would be provided by the researchers on completion of the data analysis. The researchers then met exclusively with the rugby and hockey coaches of each school to discuss the practical administration of the task and the issue of

scholar consent. As the scholars were legally constituted as minors, it was required that their parents sign a consent form on their behalf. Consent forms, along with a covering letter to parents, were distributed by the coaches to the scholars. In the instance of boarders, the coaches contacted the parents/legal guardian/s of the scholar telephonically and signed the consent form on their behalf. Given the scholars' legal status as minors, an option was added to the original consent form used in the earlier research on professional players (phases one and two), in which the scholar's parent or legal guardian could indicate whether or not they wished to be contacted by the researchers in the event that any pattern of impairment warranting medical or scholastic concern was found (see Appendix I). As noted earlier (see section 3.1, p. 54), 24 of the players abstained from participation in the study, with the number of non-participants evenly distributed across rugby and hockey playing groups. Reasons cited for refusal to participate were a demanding work and extra-mural schedule, and the fact of approaching examinations. The assessment began only once the consent forms were received.

3.3. PROCEDURE

In contrast to earlier studies of this series on professional players that utilized pre-season measures, all players were tested during the sports season. The rationale for this was that it would allow for the detection not only of permanent effects but the overlay of any acute effects of cumulative mild head injury in the rugby players, which could have implications for scholastic performance. All players were assessed between April and May 2000. Assessments were conducted individually at the respective schools. Each player was tested for approximately one and a half hours in an allocated school classroom, and testing took place after school hours. In order to assuage any anxiety provoked by the assessment situation, participants were briefed prior to the assessment on the nature and purpose of the research, and all queries and concerns were clarified. Participants were also informed that the data obtained during the assessment would be used for group research and publication purposes only, and that individual results would remain confidential and anonymous.

The testing was conducted by the research team, comprising two intern psychologists and three clinical psychologists (who were involved in phase two of the larger research study). All the researchers were trained by the same clinical neuropsychologist at Rhodes University (co-ordinator of the whole rugby research project) in the administration and scoring of the test protocols. To ensure uniformity, a preliminary meeting was held for the purposes of discussing test materials, instructions and procedures. All assessors were provided with standardized protocols that had attached standardized written instructions from the original test manual and/or Lezak. In addition, they were

supplied with an assessment schedule in order to ensure that tests were administered in the appropriate order, with specific time delays (see Appendix II).

3.4. QUESTIONNAIRES

Before testing began, participants were required to complete a demographic questionnaire that provided information deemed necessary for establishing whether a player was suitable to be included in the study for the final stage of data analysis (see Appendix III). The questionnaire included information on personal history (age, highest level of education, average grade of past academic year), sporting history (age at which scholar first played the respective sport, period of time played, positions played, reason for the choice of sport and use of head gear), previous head injuries, including instances of mild head injury (both sport and non-sport related) and exclusion criteria (see section 3.1, pp. 54-55). Following this, a symptom checklist containing 31 items was administered to assess the frequency of a range of residual post concussive symptoms suffered by players (see Appendix IV). The 31 questions were designed around 14 content areas (see Table 3-7 below) that were collated by Burbach, (1987), and drawn from a number of sources (Levin et al., 1987; Lezak, 1995; Lishman, 1988; Walsh; 1985). This questionnaire was also used in the first and second phases of the larger research project (Border, 2000; Dickinson, 1998).

TABLE 3-7. Content Areas of Postconcussive Symptomatology Questionnaire (PCSQ)

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

3.5. NEUROPSYCHOLOGICAL TEST BATTERY

Neuropsychological measures have demonstrated acute sensitivity in the detection of subtle cognitive and behavioural dysfunction in patients with mild head injury. Modelling on the previous research, a comprehensive neuropsychological battery was designed in order to test participants' current functioning across a spectrum of cognitive modalities typically found to be compromised in mild head injury. These included attention and concentration, verbal fluency, memory and learning, visuo-

perceptual tracking and hand motor dexterity (see Appendix V). In order to mitigate against the possibility of pre-selection differences in players that could contaminate the research findings, the battery also included a selection of tests which provided a measure of premorbid functioning for the respective players.

Attempts were made to utilize the best available normative data. The normative data used for most of the tests were derived from Shuttleworth-Jordan and Bode (1995), which established norms for an 18-25 year old South African university population. These normative data were considered the most appropriate available, as they closely matched the rugby and hockey groups in terms of other variables such as age, educational level and intellectual functioning. The exceptions to these were the norms used for the following eight tests: 1) the **WAIS - III Vocabulary, Picture Completion and Letter-Number Sequencing Subtests** - these three subtests were chosen as they have been shown to be good estimates of premorbid ability (Vocabulary and Picture Completion) or sensitive indicators of diffuse brain damage (Letter-Number Sequencing). Furthermore, it was considered that since the rugby and hockey players constituted a high functioning population, the use of the slightly higher standard of American norms was justified. Furthermore, as these norms were to be applied to both groups, they would be equally affected by any cultural bias; 2) the **National Adult Reading Test** - there were no South African-based norms available for this test. As with the above three subtests, it was considered that the use of American norms could be justified, given that the rugby and hockey players constitute a high functioning population; 3) the **Stroop Neuropsychological Screening Test** - there were no mean values available for this test, only percentile values. Since the rugby and hockey players were found to be equivalent on age, education, and premorbid IQ, it was considered that hockey players' means for this test provided a suitable norm against which rugby players could be compared; 4) the **Words in One Minute Unstructured Verbal Fluency Test** - school hockey player means for this test were used as the norm against which rugby players could be compared. This was due to the finding that the South African-based norms used in phases one and two were inflated when compared to their respective control groups, as well as compared to the control group of the current study, and could therefore obscure the results; and 5) the **'S' Words in One Minute Structured Verbal Fluency Test** - normative data for this test were utilized from Yeudall (1986), due to a lack of availability of South African-based norms.

The final battery included the following tests which are listed in order of administration: Sequential Finger Tapping Test; SAWAIS Digit Symbol Subtest; SAWAIS Digit Symbol Substitution Incidental Recall (Immediate); Trail Making Test (Parts A and B); Words in One Minute Unstructured Verbal Fluency Test; "S" Words in One Minute Structured Verbal Fluency Test; National Adult Reading Test; WAIS - III Vocabulary Subtest; SAWAIS Digit Symbol Incidental Recall (Delayed); SAWAIS

Digit Span Subtest; Wechsler Memory Scale (WMS) Visual Reproduction Subtest (Immediate Recall); WMS Paired Associate Learning Subtest (Immediate Recall); Stroop Neuropsychological Screening Test; WAIS - III Letter -Number Sequencing Subtest; WMS Visual Reproduction Subtest (Delayed Recall); WMS Paired Associate Learning Subtest (Delayed Recall); and the WAIS - III Picture Completion Subtest. Each test will now be described in detail in its specific category.

3.5.1. GENERAL INTELLECTUAL FUNCTIONING

As noted in Chapter two (section 2.2.1, p. 17), failures in past research endeavors to take account of premorbid levels of functioning have posed a serious limitation on the validity of results and the ability to make recommendations and generate hypotheses. This is because the absence of such measures prohibits the assessing of deficit levels in individual players. In order to enable the researchers to calculate a premorbid level of functioning, two subtests from the Weschler Adult Intelligence Scale Adult Intelligence Scale Revised – 3rd edition (WAIS - III), namely, the Vocabulary Subtest and Picture Completion Subtest were used.

3.5.1.1. Wechsler Adult Intelligence Scale Revised - 3rd edition (WAIS - III)

WAIS - III Picture Completion Subtest

This test consists of 25 stimulus pictures in which a single essential feature is missing. It differs from the SAWAIS Picture Completion subtest (used in phases one and two) in that the stimulus pictures are larger and in colour. Following instructions from the WAIS - III manual, the cards were presented individually and in order of increasing difficulty to the participant, who was then instructed to identify the most important missing part within a time limit of 20 seconds. This test measures visual reasoning and discrimination, as well as visual perceptual and verbal abilities. It is a good "hold" test, as it remains largely unaffected by the presence of diffuse brain damage. It is therefore considered a reliable indicator of premorbid ability (Lezak, 1995).

WAIS - III Vocabulary Subtest

This test was selected in the place of the SAWAIS Comprehension subtest (previously employed in phases one and two) as recent research has confirmed its status as the WAIS - III subtest that correlates the most with overall ability level (Rust, 2000), as well as the subtest least affected by a dementing process (Nelson, 1992). The test differs from the SAWAIS Vocabulary subtest in that the words which were formerly read out are now printed on six cards (four words per card). The cards were presented to the participant who was asked to identify the meaning of each word after the

researcher had stated the word out aloud. The test is failed after six consecutive incorrect responses. Where the participant gives a vague response, the researcher may query the response in order to elicit the participant's best performance. According to Lezak (1995), vocabulary scores often provide the best estimate of general premorbid ability level as this function demonstrates great resilience to the effects of diffuse cerebral damage.

3.5.1.2. National Adult Reading Test (NART) (Nelson, 1982)

This test is a reading test which provides a sensitive measure of word recognition (thus relying on previously acquired knowledge). Because the WAIS-III Vocabulary subtest requires oral definitions and so can be vulnerable to certain types of brain damage, reading tests scores are considered to provide better estimates of cerebral dysfunction (Lezak, 1995). As the NART consists of irregularly spelled words which do not conform to rules, the words cannot be correctly pronounced by guesses based on alphabetical structure. Thus correct pronunciation requires a previous familiarity with the words (Lishman, 1987).

This test was included in the battery as its IQ score correlates well with WAIS IQ scores and its increased sensitivity to premorbid vocabulary level permits more accurate prediction of premorbid ability for individuals with high average ability (Lezak, 1995). According to Nelson (1992), not only does reading ability correlate well with General IQ, but word-reading ability is maintained at a premorbid level in the face of a dementing process. For this reason, the NART has been shown to demonstrate potential as a criterion for group matching, when the matching is required on premorbid IQ levels. Furthermore, it was considered a good test for estimating IQ in a high-functioning population, as the words available in the list exceed the ceiling of the test and allow for estimation of IQ scores in the high average and superior range (Lishman, 1987).

In this test the participant was presented with a list of 50 phonetically irregular words (chosen for their rarity and therefore, unfamiliarity) and was instructed to read the list out aloud from top to bottom. The participant was warned that he may not recognize some of the words and was permitted to guess. The NART error score is the complete number of words incorrectly pronounced, which is then converted into an IQ score.

3.5.2. ATTENTION AND CONCENTRATION (MENTAL TRACKING)

3.5.2.1. SAWAIS Digit Span Subtest

This test consists of two parts, namely Digits Forwards and Digits Backwards. As the two tests tap different cognitive functions and are affected differently by brain damage, they are presented separately and reported and analysed individually.

3.5.2.2. Digits Forwards

This test is primarily a test of attention or "freedom from distraction", although it does also test immediate verbal memory (Lezak, 1995). The participant was required to repeat a sequence of numbers in the correct order, after the researcher had read them out at a rate of one number per second. Each sequence consists of two spans of equal length but different numbers and, if the participant was able to repeat one sequence of the trial, the next span (which contains one extra number) was then attempted. The test is failed only after the incorrect repetition of both trials of a span. The score is the longest span achieved. This test does not provide as sensitive a measure to diffuse brain damage as Digits Backwards, which tends to hold in the presence of cerebral injury (Lezak, 1995).

3.5.2.3. Digits Backwards

This test is very similar to Digits Forward, the only difference being that the participant was required to repeat the sequence of numbers in reverse order. As with Digits Forward, the test is failed with incorrect repetition of both trials and the score comprises of the longest span attained. This test involves double mental tracking and working memory (storing information while manipulating it mentally). As noted above, this test is more sensitive than the Digits Forwards subtest to the effects of diffuse brain damage, such as typically occurs in instances of closed head injury (Lezak, 1995).

3.5.2.4. WAIS - IIII Letter-Number Sequencing Subtest

This is a new WAIS - III subtest, which assesses working memory and attention, and hence (like other tests of working memory and attention e.g. Digits Backwards), it is likely to be sensitive to the effects of diffuse brain damage. However, as it is a new test yet to be fully evaluated, there is no final verification of this, to this author's knowledge. In this test, the participant was instructed to order sequentially a series of numbers and letters that are orally presented in a specified random order.

Each item consists of three trials and if the participant is able to sequence one span correctly, the test continues. The test is discontinued after failure of three trials of the same span.

3.5.2.5. Stroop Neuropsychological Screening Test (SNST) (Trenery, Grosson, DeBoe & Leber, 1989)

This test was included in the battery as it provides a good measure of concentration effectiveness and subtle attentional deficits (Lezak, 1995), while also tapping interference and speed (Mclean et al., 1983). Previous studies have reported evidence for deficits in selective attention in MHI patients (Gentilini et al, 1985; McLean et al., 1983) and, according to Binder (1997), measures of attention may be the most sensitive indicators of dysfunction associated with mild closed head injury.

There are two tasks involved in this test, namely the Colour Task and the Colour Word Task. The Colour Task is administered first and while this part is not formally interpreted, it is always administered because a) the normative data for the SNST were collected under the condition that both parts of the test were administered and b) the administration of the Colour Task may have a priming effect on the degree of interference reflected in the Colour Word Task. Thus in this study, whilst the Colour Task was administered according to standard administration procedure, it was decided not to include the data in the final data analysis.

Before administering the Colour Task the participant's ability to identify accuraately the four colours used in the SNST needs to be assessed. This is accomplished by asking the participant to identify the colours of common objects in the test setting. If participants cannot correctly identify the four colours, the researcher does not proceed with the administration of the test.

The Colour Task

Like the Digits Backwards subtest and the Trail Making Test Part B, this second part of the SNST is the part that is likely to be more sensitive to the effects of diffuse brain damage than the less challenging first part. During this test, the participant was presented with the test stimulus sheet, which consists of 112 colour names (namely, red, green, blue and tan) arranged in four columns of 28 names. The names are printed in one of four different colours of ink but no name is ever printed in its matching colour. The participant was required to read the words out aloud starting at the top of the column and moving on to the next column when he had finished, and to do this at as rapid a pace as possible. The participant was informed that if he made an error, he could self-correct. These spontaneous corrections are recorded for qualitative analysis. There is a time limit of 120 seconds

during which the task has to be completed. Following the administration of this test, the Colour Word Task is then presented.

The Colour Word Task

During this test, the participant was presented with a test stimulus sheet which is identical to the one employed in the Colour Task, except for the order of the colour names. The participant was then required to name aloud the colour of the ink in which the words are printed. In order to make sure that the participant fully understands the task, the researcher pointed to the first word and gave the participant the correct response. As with the Colour Task, the participant was told to perform as quickly as possible and instructed that if he made any errors, he could self-correct. As with the Colour Task, there is a time limit of 120 seconds. The Colour Word score is the primary score used in interpretation and consists of the number of correct responses, or number of items completed minus incorrect responses. Percentile and probability values are obtained from the appendix of the SNST manual.

3.5.3. VISUOPERCEPTUAL TRACKING

3.5.3.1. SAWAIS Digit Symbol Substitution Subtest

This test was retained in the test battery as opposed to the option of utilizing the more recent WAIS - III version because South African-based norms for the Digit Symbol Substitution Recall (based on the SAWAIS Incidental Recall) were available. This test is also quicker to administer as it shorter than the WAIS - III version. The test consists of three rows containing 67 open squares and a key comprising of nine different symbols that match each of the numbers. The first seven constitute a sample item, which the researcher completes in order to demonstrate the nature of the task. The participant was then instructed to fill in the blank squares with the symbol that is paired with the number in the key, as quickly and accurately as possible, and without omitting any blank squares. The participant was encouraged to continue if he paused to correct an error during the test. The number of blocks the participant completed in 90 seconds is the score achieved. Instructions were taken from the SAWAIS manual (1969).

This test is primarily a test of visuoperceptual tracking, although it also taps other cognitive functions, including sustained attention, response speed, motor persistence and visuomotor co-ordination (Lezak, 1995). This test is generally consistently sensitive to brain damage, regardless of

the locus of the lesion and even in instances where damage is minimal (Lezak, 1995). It is therefore useful in identifying diffuse brain damage, commonly associated with closed head injury.

3.5.3.2. **Trail Making Test** (Reitan, 1956)

This test is a test of complex visual scanning, motor speed and attention, and is thus highly sensitive to the effects of brain injury. It is administered in two parts, Part A and Part B.

Part A - The participant was instructed to draw lines to connect consecutively numbered circles (1 to 25) on a piece of paper, as quickly as possible and without lifting his pen from the paper. Before commencing the test, the participant was given a sample trial (numbers from 1 to 8) to complete, in order to practice, before proceeding to the test proper. If the participant made any errors during the test, these were pointed out and he was required to correct them before continuing. The score is the time taken to complete the trial.

Part B - The format and administration of this test is similar to Part A, with the exception that the participant was instructed to alternately join numbered and lettered circles. Part B involves complex visuoperceptual tracking, the ability to shift a response set and working memory, thus it is consequently particularly more sensitive than Part A to the effects of diffuse brain damage (Lezak, 1995).

3.5.4. VERBAL MEMORY

3.5.4.1. WMS Paired Associate Learning (Immediate Recall)

The version used was taken from Form 1 of the WMS manual, due to the availability of South African -based norms. The test consists of a series of 10 word pairs, comprising five easy pairs and five hard pairs. The easy pairs consist of words normally associated with one another while the hard pairs consist of words not normally associated with one another and thus, more difficult to learn. The researcher read out the sequence of pairs and then read out the first word only of each pair and the participant was instructed to recall the associated word. This procedure was repeated three times. This test measures two different activities, namely, old associate learning (which is required to recall the easy pairs) and new learning ability (which is required to recall the hard pairs). Consequently, the ability to recall the hard pairs is more susceptible to the effects of brain damage (Lezak, 1995). In order not to lose this distinction by combining the results into one composite score, this study reported and analysed the easy and hard scores separately.

3.5.4.2. WMS Associate Learning Subtest (Delayed Recall)

As delayed memory is typically more sensitive to the effects of diffuse brain damage (Lezak, 1995), a 20 minute delayed version of this test was administered. This makes the overall memory task more neuropsychologically sensitive. In this version, the first word of the pairs was read out and the participant was instructed to try and recall the associated word from the list of paired words read earlier.

3.5.5. VISUAL MEMORY

3.5.5.1. WMS Visual Reproduction (Immediate Recall)

The Form 1 version of the WMS manual (Weschler, 1945) was used for the administration and scoring of this test, due to the availability of South African-based norms. The test consists of three cards; Cards I and II contain one design each, while Card III has two designs on it. The participant was shown each card for 10 seconds and was then instructed to draw what he could remember of the design. According to Lezak (1995), this test is sensitive to the effects of head trauma and has been used to distinguish between patients with mild head injury and non-injured controls (Stuss et al., 1985).

3.5.5.2. WMS Visual Reproduction (Delayed Recall)

A delayed version of this test was administered. After a 20-minute interval, participants were given a clean sheet of paper on which they were instructed to draw the designs from memory. A delayed version was included, since delayed memory has consistently been shown to be more sensitive than immediate memory to the effects of diffuse brain damage (Lezak, 1995). This makes the overall memory task more neuropsychologically sensitive.

3.5.5.3. Digit Symbol Incidental Recall (Immediate)

This study used the short- form method of the Digit Symbol Incidental Recall (Shuttleworth-Jordan & Bode, 1995). After completion of the Digit Symbol Subtest, the researcher noted the last square the participant had filled in after the 90-second time limit has lapsed. The participant who was unable to complete the digit symbol substitutions up to the end of the second last row, was then instructed to do so. The participant was then given a sheet of paper marked with the numbers one to nine and empty

squares and instructed to fill in as many matching symbols as he could recall. This test taps various functions, including attention, planning, memory and information processing (Walsh, 1985). Its excellent discriminatory power in detecting cognitive deficit is well illustrated by research that has shown that that the Digit Symbol measure of incidental recall may assist in the differential diagnosis of Alzheimer's dementia and pseudodementia, as the test was found to be less affected in depressed patients than patients with organic dementias (Hart, Kwentus, Wade & Hammer, 1987 cited in Shuttleworth-Jordan & Bode, 1995). Thus, due to its sensitivity to the detection of diffuse brain damage typical in mild head injury, this test was included in the battery.

3.5.5.4. Digit Symbol Incidental Recall (Delayed)

A delayed recall version of this test was administered. After a 20-minute delay, the participant was handed a fresh sheet of paper marked with the numbers one to nine and instructed to fill in as many matching symbols he could recall. This test was included as delayed memory has shown to be more sensitive than immediate memory to the effects of diffuse brain damage (Lezak, 1995).

3.5.6. VERBAL FLUENCY

3.5.6.1. Words in One Minute Unstructured Verbal Fluency Test (Terman & Merril, 1973)

This is an unstructured test of verbal fluency. The participant was instructed to say as many different unconnected words as possible and as quickly as he could, excluding the use of proper nouns, the same word with a different suffix, constructed sentences or counting. The participant was given examples of the above mentioned, as well as examples of different unconnected words. This test also utilizes short-term memory indirectly in order to keep track of words already used. Verbal fluency has been shown to be a sensitive indicator of brain dysfunction, particularly frontal lobe damage (Lezak, 1995).

3.5.6.2. "S" Words in One Minute Structured Verbal Fluency Test

The same instructions were given as those in the Words in One Minute Structured Verbal Fluency test, except that the participant was instructed to use unconnected words starting with "S". According to Lezak (1995), fluency tests that require word generation using a given initial letter give the greatest scope to participants looking for a strategy to organize their search for words (in comparison to those that rely on random word generation), and are particularly difficult for participants unable to develop strategies of their own. As a verbal fluency test, it is sensitive to the effects of brain damage.

3.5.7. HAND MOTOR DEXTERITY

3.5.7.1. **Finger Tapping Test** (Denckla, 1973)

This test was chosen above other hand motor dexterity tests due to the availability of South African-based norms as well as the fact that it does not require any instrumentation, so making its administration simpler and quicker. The participant was instructed to place both elbows on the table and with one hand at a time, to touch each finger to the thumb, beginning with the index finger, as quickly as possible. The participant was afforded the opportunity to practice the sequence task, before commencement of the test proper. The score is the number of seconds taken to perform five sets of four taps. According to Lezak (1995), brain injury has a slowing effect on finger tapping rate, thereby indicating diffuse brain damage in the absence of any other physical impairment.

3.6. DATA PROCESSING

To ensure inter-rater reliability, all protocols were scored twice (once by each intern psychologist). Scoring was based on standardized instructions. All test protocols were cross checked by the two intern psychologists, in order to confer and reach agreement on tests that involved a subjective scoring component such as the WMS Visual Reproduction (Immediate and Delayed Recall).

Data for this third research phase were broken up for analysis to form two separate research projects:

1. A direct comparison of mean scores of Total Rugby versus Hockey Control players across all neuropsychological tests. In addition, the same comparative analyses were conducted on the following subgroups: Rugby Forwards versus Rugby Backs, Rugby Forwards versus Hockey Control and Rugby Backs versus Hockey Control. A corrrelational analysis was also included in order to ascertain whether a relationship existed between the number of reported mild head injuries recalled by active players their cognitive test performance.

2. A comparison of the percentage of rugby and hockey players with cognitive deficit relative to normative data, as well as a comparison of the frequency of reported postconcussive symptomatology were made. In addition, the same comparative analyses were conducted on the following subgroups: Rugby Forwards versus Rugby Backs, Rugby Forwards versus Hockey Control and Rugby Backs versus Hockey Control.

The focus of the present research was on the second set of comparisons, i.e. the percentage of cognitive deficit found and the frequency of reported postconcussive symptomatology. As noted in Chapter one (p. 2), the strength of this analysis lies in its provision of an analysis of the distribution of deficit among individual players, as distinct from average effects. This is particularly significant given the recent trend in the literature, which emphasizes that statistical significance is not equivalent to clinical significance and that a sole reliance on tests of statistical significance (e.g. statistical comparisons of means) in the understanding of neuropsychological data may actually confound conclusions drawn from neuropsychological research regarding brain-behaviour relations (for example, Donders, 2000; Zakzanis, 1998). The methodology employed in the present research circumvents the danger of such statistical artifacts and provides a clinically relevant set of data, i. e. the *number* of individuals with deficit as per analyses conducted for clinical purposes.

3.7. STATISTICAL ANALYSIS

This section comprises two components, the neuropsychological test results and the postconcussive symptomatology results. In this section, the statistical procedure for the calculation of the neuropsychological results will be discussed first, followed by that for the postconcussive symptomatology results.

3.7.1. NEUROPSYCHOLOGICAL TEST RESULTS

The first stage of data analysis involved calculating the level of deficit shown by each player on each of the tests administered. The level of deficit was determined according to the degree to which a test score deviated from the best existing normative data available at the time of analysis. Deficit was defined in terms of Dickinson's (1998) and Border's (2000) criteria (phases one and two of the research), and reported in the categories of 'none', 'mild' and 'moderate/severe' terms, relative to the extent to which test scores deviated from the normative data as follows:

None- the test score falls within 1 standard deviation of the norm
Mild- the test score is equal to or greater than 1 standard deviation of the norm but less than 2 standard deviations in the direction indicating poor performance
Moderate/Severe- the test score is equal to or greater than 2 standard deviations of the norm in the direction indicating poor performance

Based on these definitions, two separate sets of analyses were conducted comprising i) Individual Cognitive Test Deficit and ii) Total Cognitive Test Deficit

3.7.1.1. Individual Cognitive Test Deficit

After the level of deficit had been calculated, the number of players falling in each of the 3 specific categories (none, mild, moderate/severe) was determined for each test in the neuropsychological battery. This number was then translated into a percentage (%), which represented the proportion of participants in each group falling into each of the three categories (none, mild, and moderate/severe) for each test in the neuropsychological battery. The chi-square formula was used to compare the percentages of deficit present for each test between the different groups and subgroups. The tables showing these results and their page numbers are grouped under the heading **Individual Cognitive Test Deficit** in Chapter Four, p. 74.

3.7.1.2 Total Cognitive Test Deficit

Following this, the presence of cognitive deficit for players across *all cognitive tests* was determined (as distinguished from the first level of analysis which focuses on the percentage of players with cognitive deficit on *each test* in the battery) and reported in two categories: 1) **Mild Cognitive Deficit - Any** (the number of players with *mild* deficit as defined above using any one, or more, of the cognitive tests) and 2) **Moderate/Severe Cognitive Deficit - Any** (the number of players with *moderate to severe* deficit as defined using any one, or more, of the cognitive tests). The number of individuals per group with i) any mild and ii) any moderate/severe deficit across all tests was calculated and translated into a percentage which represented the proportion of participants in each group falling into each of these two categories. The chi-square formula was used to compare the percentages of deficit between the different groups and subgroups for each of these two categories. The tables of these results and their page numbers are grouped under the heading **Total Cognitive Test Deficit** in Chapter Four, p. 74.

3.7.2. POSTCONCUSSIVE SYMPTOMATOLOGY RESULTS

Within each group the number of players reporting each frequency level (never, sometimes, often) was established and then represented as a percentage (%), which represented the proportion of participants in each group falling into each of these three categories. The chi-square formula was used to compare the percentage of deficit in the different groups and subgroups. The tables and their page numbers are grouped under **Postconcussive Symptomatology Results Questionnaire** in Chapter Four, p. 76.

3.7.3. CHI-SQUARE COMPARITIVE ANALYSIS

The chi-square provides an appropriate procedure when comparing proportions of two separate groups with each other (Ferguson, 1988), as it can be used to test the significance of observed differences (Bless & Kathuria, 1993). It is not a parametric test and so, does not require any parametric conditions to be fulfilled, nor does it assume a normal distribution of the population and is therefore used for random independent samples or groups (Bless & Kathuria, 1993). Thus when making comparisons between the levels of deficit (or frequency of symptoms) of two independent groups, such as between the rugby and hockey players, it is an appropriate measure to use.

Results of the chi-square test were then interpreted in terms of two levels of significance: **significant** and **approaching significance**. The difference between the two main groups (Total Rugby versus Hockey Control) was regarded as **significant** when $p < \text{or} = 0.05$. The difference between the two main groups (Total Rugby versus Hockey Control) was regarded as **approaching significance** when $p > 0.05$ but < 0.15. Bonferroni adjustments were made to the significance levels because pairwise multiple comparison tests were performed between the three subgroups of Rugby Backs versus Rugby Forwards, Rugby Forwards versus Hockey Control and Rugby Backs versus Hockey Control. This was done in order to ensure that the overall level of significance did not exceed 0.05 (Miller, 1981). Following Bonferroni adjustments, results for the three subgroup comparisons (Rugby Forwards versus Rugby Backs, Rugby Forwards versus Hockey Control and Rugby Backs versus Hockey Control) were interpreted in terms of two levels of significance. Differences were regarded as **significant** when $p < 0.025$. Differences were regarded as **approaching significance** when $p > 0.025$ but < 0.075.

CHAPTER FOUR: RESULTS

The comparative results for a) the neuropsychological assessment and b) the postconcussive symptomatology will be grouped together and appear in tabular form at the end of the chapter (see Tables 4-10 to 4-32, pp. 80-96).

4.1. NEUROPSYCHOLOGICAL ASSESSMENT RESULTS

Neuropsychological assessment results will be divided into a) **individual cognitive test deficit** results and b) **total cognitive test deficit** results, and these will be reported separately in that order. The comparative results for this section will appear at the end of chapter in tabular form as illustrated below

Individual Cognitive Test Deficit

Total Rugby versus Hockey Control	Tables 4-1 to 4-6, pp. 80-81
Rugby Forwards versus Rugby Backs	Tables 4-7 to 4-12, pp. 82-83
Rugby Forwards versus Hockey Control	Tables 4-13 to 4-18, pp. 84-85
Rugby Backs versus Hockey Control	Tables 4-19 to 4-24, pp. 86-87

Total Cognitive Test Deficit

Total Rugby versus Hockey Control	Table 4-25, p. 88
Rugby Forwards versus Rugby Backs	Table 4-26, p. 88
Rugby Forwards versus Hockey Control	Table 4-27, p. 88
Rugby Backs versus Hockey Control	Table 4-28, p. 88

The results indicate the number (n) and percentage (%) of players across each level of deficit for all the cognitive tests in the respective modalities together with the chi (x^2) statistic. In Tables 4-3, 4-9, 4-15 and 4-21, there are instances of *No statistic* reported. This indicates that in those cases no participant in either of the groups exhibited any deficit and thus the statistical comparison was not applied.

Within both the above mentioned sections, neuropsychological assessment results will be reported in the following order: i) results indicating greater impairment in the rugby players relative to the hockey players; ii) results indicating greater impairment in the hockey players relative to the rugby players; iii) results indicating greater impairment in the rugby forward players relative to the rugby

backline players; and iv) results indicating greater impairment in the rugby backline players relative to the rugby forward players.

4.1.1. INDIVIDUAL COGNITIVE TEST DEFICIT

4.1.1.1. Significant Results

There were no significant differences in performance on any of the neuropsychological tests for the Total Rugby versus Hockey Control comparison, nor for the subgroup comparisons of Rugby Forwards versus Rugby Backs, Rugby Forwards versus Hockey Control and Rugby Backs versus Hockey Control.

4.1.1.2. Results Approaching Significance

There was one neuropsychological test in which differences approached significance in the direction of Total Rugby demonstrating greater impairment relative to Hockey Control (see Table 4-6, p. 81), which was the **Finger Tapping Test (Preferred Hand)** ($p = 0.0897$), where 74.5% of Total Rugby demonstrated no impairment compared with 88.2% of Hockey Control and 25.5% of Total Rugby demonstrated mild impairment compared with 8.8% of Hockey Control.

There was one neuropsychological test in which differences approached significance in the direction of Hockey Control demonstrating greater impairment relative to Rugby Forwards (see Table 4-17, p. 85), which was the **Structured Verbal Fluency Test** ($p = 0.0467$), where 79.4% of Hockey Control demonstrated no impairment compared with 96.4 % of Rugby Forwards and 26.0% of Hockey Control demonstrated mild impairment compared with 3.6 % of Rugby Forwards.

There were two neuropsychological tests in which differences approached significance in the direction of Rugby Backs demonstrating greater impairment relative to Rugby Forwards (see Table 4-10, p. 83) which were a) **Digit Symbol Incidental Recall -Immediate** ($p = 0.0624$), where 89.3 % of Rugby Forwards demonstrated no impairment compared with 63.2 % of Rugby Backs and 10.5 % of Rugby Backs demonstrated moderate/severe impairment while 0% of Rugby Forwards demonstrated impairment in the moderate to severe category, and b) **WMS Visual Reproduction Immediate Recall** ($p = 0.0510$), where 96.4 % of Rugby Forwards demonstrated no impairment compared with 73.7 % of Rugby Backs and 15.8 % of Rugby Backs demonstrated moderate/severe impairment while 0% of Rugby Forwards demonstrated impairment in the moderate to severe category.

4.1.2. TOTAL COGNITIVE TEST DEFICIT

4.1.2.1. Significant Results

There were no significant differences in the total cognitive test deficit category for the Total Rugby versus Hockey Control comparison, nor for the subgroup comparisons of Rugby Forwards versus Rugby Backs, Rugby Forwards versus Hockey Control and Rugby Backs versus Hockey Control.

4.1.2.2. Results Approaching Significance

There were no differences approaching significance from the total cognitive test deficit category in the direction of rugby players demonstrating greater impairment relative to hockey players. There were no differences approaching significance from the total cognitive deficit category in the direction of Rugby Forwards demonstrating greater impairment relative to Rugby Backs. There were no differences approaching significance from the total cognitive deficit category in the direction of Rugby Backs demonstrating greater impairment relative to Rugby Forwards

The was one result approaching significance in the direction of hockey players demonstrating greater impairment relative to Rugby Forwards (see Table 4-27, p. 88), which was in the **Cognitive Deficit-Moderate/ Severe** subcategory ($p = 0.0736$), where 47.1% of Hockey Control demonstrated moderate to severe cognitive deficit across any one or more neuropsychological tests in comparison with 25.0% of Rugby Forwards.

4.2. POSTCONCUSSIVE SYMPTOMATOLOGY QUESTIONNAIRE RESULTS

The comparative results for this section will appear at the end of the chapter after the neuropsychological results in tabular form as illustrated below:

Total Rugby versus Hockey Control	Table 4-29, pp. 89-90
Total Rugby Forwards versus Total Rugby Backs	Table 4-30, pp. 91-92
Total Rugby Forwards versus Hockey Control	Table 4-31, pp. 93-94
Total Rugby Backs versus Hockey Control	Table 4-32, pp. 95-96

In each case the results indicate the number (n) and percentage (%) of players within the three categories of frequency for the complete symptom list together with the chi (x^2) statistic. On item 6 (seizures) and 11 (sexual difficulties) across all four comparisons, there are instances of *No statistic*

reported. This indicates that in those cases no participant in either of the groups reported any symptomatology and thus the statistical comparison was not applicable.

Within this section postconcussive symptomatology questionnaire results will be reported in the following order: i) results indicating a greater frequency of reported postconcussive symptomatology amongst rugby players relative to hockey players; ii) results indicating a greater frequency of reported postconcussive symptomatology amongst hockey players relative to rugby players; iii) results indicating a greater frequency of reported postconcussive symptomatology amongst rugby forward players relative to rugby backline players; and iv) results indicating a greater frequency of reported postconcussive symptomatology amongst rugby backline players relative to rugby forward players.

4.2.1. Significant Results

There were no significant differences in the percentages of reported postconcussive symptomatology for the Total Rugby versus Hockey Control comparison and for the subgroup comparisons of Rugby Forwards versus Rugby Backs, Rugby Forwards versus Hockey Control and Rugby Backs versus Hockey Control.

4.2.2. Results Approaching Significance

There were four symptoms in which differences approached significance in the direction of rugby players reporting greater frequency of postconcussive symptomatology relative to hockey players. These were for 1) clumsy speech; 2) memory; 3) being easily angered and, 4) sleep difficulties (see Tables 4-29, 4-31 and 4-32, respectively, pp.89, 90, 93, and 96).

4.2.2.1 Clumsy Speech

For this symptom (see Tables 4-29 and 4-31, pp. 89, 93), 46.8 % of Total Rugby reported never experiencing clumsy speech in comparison with 67.6% of Hockey Control ($p = 0.0625$); and 53.2% of Total Rugby reported sometimes experiencing clumsy speech in comparison to 32.4% of the Hockey Control group. Similarly, 42.9% of Rugby Forwards reported never experiencing clumsy speech in comparison with 67.6% of Hockey Control ($p = 0.0501$) and 57.1% of Rugby Forwards reported sometimes experiencing clumsy speech in comparison with 32.4% of Hockey Control.

4.2.2.2. Memory

For this symptom (see Table 4-29, p. 89), 23.4% of Total Rugby reported sometimes experiencing memory problems in comparison to 11.8% of Hockey Control. ($p = 0.1170$).

4.2.2.3. Easily Angered

For this symptom (see Tables 4-29 and 4-32, pp. 90, 96), 44.7% of Total Rugby reported never experiencing being easily angered in comparison with 50.0% ($p = 0.0954$) of Hockey Control while 12.8% of Total Rugby report often experiencing being easily angered in comparison to no players in the Hockey Control group. Similarly, 15.8% of Rugby Backs reported being easily angered often in comparison to 0% of players in the Hockey Control group ($p = 0.0581$).

4.2.2.4. Sleep Difficulties

For this symptom (see Tables 4-29 and 4-31, p. .90, 94), 63.8% of Total Rugby reported never experiencing sleep difficulties in comparison with 81.8% of Hockey Control ($p = 0.0801$) and 36.2% of Total Rugby reported sometimes experiencing sleep difficulties in comparison with 18.2% of Hockey Control. Similarly, 60.7% of Rugby Forwards reported never experiencing sleep difficulties in comparison with 81.8% of Hockey Control ($p = 0.0670$) and 39.3% of Rugby Forwards reported sometimes experiencing sleep difficulties in comparison with 18.2 % of Hockey Control.

There were two symptoms in which differences approached significance in the direction of hockey players reporting greater frequency of postconcussive symptomatology relative to rugby players which were a) worry and 2) weakness in limbs (See Table 4-29, pp. 89-90).

4.2.2.5. Weakness in limbs

For this symptom (see Table 4-29, p. 89), 73.5% of Hockey Control reported never experiencing weakness in limbs in comparison with 89.4% of Total Rugby and 23.5% of Hockey Control reported sometimes experiencing weakness in limbs in comparison with 10.6% of Total Rugby

4.2.2.6. Worry

For this symptom (see Table 4-29, p. 90), 47.1% of Hockey Control reported never experiencing worry in comparison to 66.0% of Total Rugby ($p = 0.1301$) and 52.9% of Hockey Control reported sometimes experiencing worry in comparison with 31.9% of Total Rugby.

There were no postconcussive symptoms in which comparisons of the reported frequencies approached significance in the direction of rugby forward players reporting greater frequency of postconcussive symptomatology relative to rugby backline players. Neither were there any postconcussive symptoms in which comparisons of the reported frequencies approached significance in the direction of rugby backline players reporting greater frequency of postconcussive symptomatology relative to rugby forward players.

Neuropsychological assessment: TOTAL RUGBY versus HOCKEY CONTROL

Table 4-1. ATTENTION AND CONCENTRATION: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	TOTAL RUGBY			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forwards									
n	28	13	6	19	10	5			
%	59.6	27.7	12.8	55.9	29.4	14.7	0.122	2	0.9407
Digits Backwards									
n	33	11	3	23	7	4			
%	70.2	23.4	6.4	67.6	20.6	11.8	0.750	2	0.6872
Letter-Number Sequencing									
n	42	4	1	28	6	0			
%	89.4	8.5	2.1	82.4	17.6	0.0	2.169	2	0.3380
STROOP-CW									
n[1]	40	4	2	29	4	1			
%	87.0	8.7	4.3	85.3	11.8	2.9	0.29	2	0.8635

Table 4-2. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	TOTAL RUGBY			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution									
n	35	10	2	29	4	1			
%	74.5	21.3	4.3	85.3	11.8	2.9	1.417	2	0.4923
Trail Making Test A									
n	41	5	1	32	2	0			
%	87.2	10.6	2.1	94.1	5.9	0.0	1.343	2	0.5108
Trail Making Test B									
n	39	8	0	28	6	0			
%	83.0	17.0	0.0	82.4	17.6	0.0	0.005	1	0.9414

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

TEST	TOTAL RUGBY			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
WMS Associate Learning (Easy) Immed. Recall									
n	46	1	0	30	3	1			
%	97.9	2.1	0.0	88.2	8.8	2.9	3.369	2	0.1856
WMS Associate Learning (Hard) Immed. Recall									
n	43	4	0	33	1	0			
%	91.5	8.5	0.0	97.1	2.9	0.0	1.057	1	0.3040
WMS Associate Learning (Easy) Delayed Recall									
n	47	0	0	34	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[2]		
WMS Associate Learning (Hard) Delayed Recall									
n	42	5	0	33	1	0			
%	89.4	10.6	0.0	97.1	2.9	0.0	*1.70*	1	0.1917

[1] On the STROOP-CW, n = 46 for Total Rugby as one colour-blind rugby forward's result was not included in the analysis.

[2] Where No Statistic is reported, all subjects have no impairment thus rendering a statistical comparision null and void.

Continued overleaf

Neuropsychological Assessment: TOTAL RUGBY versus HOCKEY CONTROL (Continued)

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

TEST	TOTAL RUGBY			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall - Imm.									
n	37	8	2	24	7	3			
%	78.7	17.0	4.3	70.6	20.6	8.8	0.976	2	0.6139
Digit Symbol Substitution Incidental Recall - Del.									
n	42	4	1	28	5	1			
%	89.4	8.5	2.1	82.4	14.7	2.9	0.846	2	0.6549
WMS Visual Reproduction Immed. Recall									
n	41	3	3	28	5	1			
%	87.2	6.4	6.4	82.4	14.7	2.9	1.912	2	0.3844
WMS Visual Reproduction Delayed Recall									
n	42	3	2	28	5	1			
%	89.4	6.4	4.3	82.4	14.7	2.9	1.588	2	0.4521

Table 4-5. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	TOTAL RUGBY			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	35	11	1	28	5	1			
%	74.5	23.4	2.1	82.4	14.7	2.9	0.966	2	0.6169
Structured Verbal Fluency									
n	42	4	1	27	7	0			
%	89.4	8.5	2.1	79.4	20.6	0.0	3.072	2	0.2153

Table 4-6. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	TOTAL RUGBY			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Finger Tapping Test (Preferred Hand)									
n	35	12	0	30	3	1			
%	74.5	25.5	0.0	88.2	8.8	2.9	4.822	2	0.0897 ~
Finger Tapping Test (Non-Preferred Hand)									
n	29	17	1	27	6	1			
%	61.7	36.2	2.1	79.4	17.6	2.9	3.332	2	0.1890

Approaching Significance (~ 0.05<p<0.15)

Neuropsychological Assessment: RUGBY FORWARDS versus RUGBY BACKS

Table 4-7. ATTENTION AND CONCENTRATION: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY FORWARDS			RUGBY BACKS			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forwards									
n	17	9	2	11	4	4			
%	60.7	32.1	7.1	57.9	21.1	21.1	2.234	2	0.3273
Digits Backwards									
n	21	6	1	12	5	2			
%	75.0	21.4	3.6	63.2	26.3	10.5	1.199	2	0.5490
Letter-Number Sequencing									
n	25	2	1	17	2	0			
%	89.3	7.1	3.6	89.5	10.5	0.0	0.831	2	0.6601
STROOP-CW									
n[1]	22	3	2	18	1	0			
%	81.5	11.1	7.4	94.7	5.3	0.0	2.071	2	0.3550

Table 4-8. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY FORWARDS			RUGBY BACKS			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution									
n	20	6	2	15	4	0			
%	71.4	21.4	7.1	78.9	21.1	0.0	1.444	2	0.4858
Trail Making Test A									
n	25	3	0	16	2	1			
%	89.3	10.7	0.0	84.2	10.5	5.3	1.507	2	0.4706
Trail Making Test B									
n	23	5	0	16	3	0			
%	82.1	17.9	0.0	84.2	15.8	0.0	0.034	1	0.8531

Table 4-9. VERBAL MEMORY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY FORWARDS			RUGBY BACKS			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
WMS Associate Learning (Easy) Immed. Recall									
n	27	1	0	19	0	0			
%	96.4	3.6	0.0	100.0	0.0	0.0	0.693	1	0.4050
WMS Associate Learning (Hard) Immed. Recall									
n	26	2	0	17	2	0			
%	92.9	7.1	0.0	89.5	10.5	0.0	0.166	1	0.6833
WMS Associate Learning (Easy) Delayed Recall									
n	28	0	0	19	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[2]		
WMS Associate Learning (Hard) Delayed Recall									
n	25	3	0	18	1	0			
%	89.3	10.7	0.0	94.7	5.3	0.0	0.432	1	0.5110

[1] On the STROOP-CW, n = 27 for Rugby Forwards as one colour-blind rugby forward's result was not included in the analysis.

[2] Where No Statistic is reported, all subjects have no impairment thus rendering a statistical comparision null and void.

Continued overleaf

Neuropsychological Assessment: RUGBY FORWARDS versus RUGBY BACKS (Continued)

Table 4-10 . VISUAL MEMORY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY FORWARDS			RUGBY BACKS			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall - Imm.									
n	25	3	0	12	5	2			
%	89.3	10.7	0.0	63.2	26.3	10.5	5.548	2	0.0624 ~
Digit Symbol Substitution Incidental Recall - Del.									
n	27	1	0	15	3	1			
%	96.4	3.6	0.0	78.9	15.8	5.3	3.846	2	0.1462
WMS Visual Reproduction Immed. Recall									
n	27	1	0	14	2	3			
%	96.4	3.6	0.0	73.7	10.5	15.8	5.950	2	0.0510 ~
WMS Visual Reproduction Delayed Recall									
n	27	1	0	15	2	2			
%	96.4	3.6	0.0	78.9	10.5	10.5	4.192	2	0.1229

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

TEST	RUGBY FORWARDS			RUGBY BACKS			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	22	6	0	13	5	1			
%	78.6	21.4	0.0	68.4	26.3	5.3	1.746	2	0.4177
Structured Verbal Fluency									
n	27	1	0	15	3	1			
%	96.4	3.6	0.0	78.9	15.8	5.3	3.846	2	0.1462

Table 4-12. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY FORWARDS			RUGBY BACKS			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Finger Tapping Test (Preferred Hand)									
n	21	7	0	14	5	0			
%	75.0	25.0	0.0	73.7	26.3	0.0	0.010	1	0.9191
Finger Tapping Test (Non-Preferred Hand)									
n	19	9	0	10	8	1			
%	67.9	32.1	0.0	52.6	42.1	5.3	2.210	2	0.3313

Approaching Significance (~ 0.025<p<0.75), following Bonferonni's adjustments

Neuropsychological Assessment: RUGBY FORWARDS versus HOCKEY CONTROL

Table 4-13. ATTENTION AND CONCENTRATION: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY FORWARDS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forwards									
n	17	9	2	19	10	5			
%	60.7	32.1	7.1	55.9	29.4	14.7	0.877	2	0.6450
Digits Backwards									
n	21	6	1	23	7	4			
%	75.0	21.4	3.6	67.6	20.6	11.8	1.400	2	0.4965
Letter-Number Sequencing									
n	25	2	1	28	6	0			
%	89.3	7.1	3.6	82.4	17.6	0.0	2.614	2	0.2707
STROOP-CW									
n[1]	22	3	2	29	4	1			
%	81.5	11.1	7.4	85.3	11.8	2.9	0.642	2	0.7254

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

TEST	RUGBY FORWARDS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution									
n	20	6	2	29	4	1			
%	71.4	21.4	7.1	85.3	11.8	2.9	1.823	2	0.4020
Trail Making Test A									
n	25	3	0	32	2	0			
%	89.3	10.7	0.0	94.1	5.9	0.0	0.484	1	0.4868
Trail Making Test B									
n	23	5	0	28	6	0			
%	82.1	17.9	0.0	82.4	17.6	0.0	0.000	1	0.9828

Table 4-15. VERBAL MEMORY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY FORWARDS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
WMS Associate Learning (Easy) Immed. Recall									
n	27	1	0	30	3	1			
%	96.4	3.6	0.0	88.2	8.8	2.9	1.592	2	0.4511
WMS Associate Learning (Hard) Immed. Recall									
n	26	2	0	33	1	0			
%	92.9	7.1	0.0	97.1	2.9	0.0	0.589	1	0.4429
WMS Associate Learning (Easy) Delayed Recall									
n	28	0	0	34	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[2]		
WMS Associate Learning (Hard) Delayed Recall									
n	25	3	0	33	1	0			
%	89.3	10.7	0.0	97.1	2.9	0.0	1.537	1	0.2150

[1] On the STROOP-CW, n = 27 for Rugby Forwards as one colour-blind rugby forward's result was not included in the analysis.

[2] Where No Statistic is reported, all subjects have no impairment thus rendering a statistical comparision null and void.

Continued overleaf

Neuropsychological Assessment: RUGBY FORWARDS versus HOCKEY CONTROL (Continued)

Table 4-16. VISUAL MEMORY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY FORWARDS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall - Imm.									
n	25	3	0	24	7	3			
%	89.3	10.7	0.0	70.6	20.6	8.8	4.078	2	0.1302
Digit Symbol Substitution Incidental Recall - Del.									
n	27	1	0	28	5	1			
%	96.4	3.6	0.0	82.4	14.7	2.9	3.134	2	0.2087
WMS Visual Reproduction Immed. Recall									
n	27	1	0	28	5	1			
%	96.4	3.6	0.0	82.4	14.7	2.9	3.134	2	0.2087
WMS Visual Reproduction Delayed Recall									
n	27	1	0	28	5	1			
%	96.4	3.6	0.0	82.4	14.7	2.9	3.134	2	0.2087

Table 4-17. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY FORWARDS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	22	6	0	28	5	1			
%	78.6	21.4	0.0	82.4	14.7	2.9	1.242	2	0.5374
Structured Verbal Fluency									
n	27	1	0	27	7	0			
%	96.4	3.6	0.0	79.4	26.0	0.0	3.956	1	0.0467 ~

Table 4-18. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY FORWARDS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Finger Tapping Test (Preferred Hand)									
n	21	7	0	30	3	1			
%	75.0	25.0	0.0	88.2	8.8	2.9	3.642	2	0.1619
Finger Tapping Test (Non-Preferred Hand)									
n	19	9	0	27	6	1			
%	67.9	32.1	0.0	79.4	17.6	2.9	2.433	2	0.2962

Approaching Significance (~ 0.025<p<0.075), following Bonferonni's adjustments

Neuropsychological Assessment: RUGBY BACKS versus HOCKEY CONTROL

Table 4-19. ATTENTION AND CONCENTRATION: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY BACKS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forwards									
n	11	4	4	19	10	5			
%	57.9	21.1	21.1	55.9	29.4	14.7	0.620	2	0.7333
Digits Backwards									
n	12	5	2	23	7	4			
%	63.2	26.3	10.5	67.6	20.6	11.8	0.230	2	0.8912
Letter-Number Sequencing									
n	17	2	0	28	6	0			
%	89.5	10.5	0.0	82.4	17.6	0.0	0.482	1	0.4874
STROOP-CW									
n	18	1	0	29	4	1			
%	94.7	5.3	0.0	85.3	11.8	2.9	1.228	2	0.5413

Table 4-20. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY BACKS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution									
n	15	4	0	29	4	1			
%	78.9	21.1	0.0	85.3	11.8	2.9	1.315	2	0.5183
Trail Making Test A									
n	16	2	1	32	2	0			
%	84.2	10.5	5.3	94.1	5.9	0.0	2.270	2	0.3214
Trail Making Test B									
n	16	3	0	28	6	0			
%	84.2	15.8	0.0	82.4	17.6	0.0	0.030	1	0.8629

Table 4-21. VERBAL MEMORY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY BACKS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
WMS Associate Learning (Easy) Immed. Recall									
n	19	0	0	30	3	1			
%	100.0	0.0	0.0	88.2	8.8	2.9	2.418	2	0.2985
WMS Associate Learning (Hard) Immed. Recall									
n	17	2	0	33	1	0			
%	89.5	10.5	0.0	97.1	2.9	0.0	1.313	1	0.2518
WMS Associate Learning (Easy) Delayed Recall									
n	19	0	0	34	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
WMS Associate Learning (Hard) Delayed Recall									
n	17	2	0	33	1	0			
%	89.5	10.5	0.0	97.1	2.9	0.0	1.313	1	0.2518

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

Continued overleaf

Table 4-22 . VISUAL MEMORY: Comparison of the Percentage of Subjects with Cogntive Deficit.

TEST	RUGBY BACKS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall - Imm.									
n	12	5	2	24	7	3			
%	63.2	26.3	10.5	70.6	20.6	8.8	0.313	2	0.8551
Digit Symbol Substitution Incidental Recall - Del.									
n	15	3	1	28	5	1			
%	78.9	15.8	5.3	82.4	14.7	2.9	0.201	2	0.9044
WMS Visual Reproduction Immed. Recall									
n	14	2	3	28	5	1			
%	73.7	10.5	15.8	82.4	14.7	2.9	2.943	2	0.2296
WMS Visual Reproduction Delayed Recall									
n	15	2	2	28	5	1			
%	78.9	10.5	10.5	82.4	14.7	2.9	1.418	2	0.4922

Table 4-23. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY BACKS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	13	5	1	28	5	1			
%	68.4	26.3	5.3	82.4	14.7	2.9	1.351	2	0.5090
Structured Verbal Fluency									
n	15	3	1	27	7	0			
%	78.9	15.8	5.3	79.4	20.6	0.0	1.939	2	0.3794

Table 4-24. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Cognitive Deficit.

TEST	RUGBY BACKS			HOCKEY CONTROL			*x2*	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Finger Tapping Test (Preferred Hand)									
n	14	5	0	30	3	1			
%	73.7	26.3	0.0	88.2	8.8	2.9	3.340	2	0.1882
Finger Tapping Test (Non-Preferred Hand)									
n	10	8	1	27	6	1			
%	52.6	42.1	5.3	79.4	17.6	2.9	4.187	2	0.1233

Table 4-25. Comparison of Number of Rugby and Hockey Players with Any Cognitive Abnormality and Moderate to Severe Cognitive Abnormality on One or More Cognitive Tests

		Cognitive Abnormality - Any				Cognitive Abnormality - Moderate to Severe			
	n	Presence of Deficit (%)	*x2*	*df*	*p*-value	Presence of Deficit (%)	*x2*	*df*	*p*
Rugby	47	82.2				29.8			
Hockey	34	76.5	0.397	1	0.5288	47.1	2.524	1	0.1843

Table 4-26. Comparison of Number of Rugby Forwards and Rugby Backs with Any Cognitive Abnormality and Moderate to Severe Cognitive Abnormality on One or More Cognitive Tests

		Cognitive Abnormality - Any				Cognitive Abnormality - Moderate to Severe			
	n	Presence of Deficit (%)	*x2*	*df*	*p*-value	Presence of Deficit (%)	*x2*	*df*	*p*
Forwards	28	75.0				25.0			
Backs	19	94.1	2.645	1	0.1039	36.8	0.759	1	0.3837

Table 4-27. Comparison of Number of Rugby Forwards and Hockey Players with Any Cognitive Abnormality and Moderate to Severe Cognitive Abnormality on One or More Cognitive Tests

		Cognitive Abnormality - Any				Cognitive Abnormality - Moderate to Severe			
	n	Presence of Deficit (%)	*x2*	*df*	*p*	Presence of Deficit (%)	*x2*	*df*	*p*
Forwards	28	75.0				25.0			
Hockey	34	76.5	0.018	1	0.8930	47.1	3.202	1	0.0736 ~

Approaching Significance (~0.025 < p <0.075), following Bonferonni's adjustment

Table 4-28. Comparison of Number of Rugby Backs and Hockey Players with Any Cognitive Abnormality and Moderate to Severe Cognitive Abnormality on One or More Cognitive Tests

		Cognitive Abnormality - Any				Cognitive Abnormality - Moderate to Severe			
	n	Presence of Deficit (%)	*x2*	*df*	*p*	Presence of Deficit (%)	*x2*	*df*	*p*
Backs	19	94.1				36.8			
Hockey	34	76.5	2.429	1	0.1191	47.1	0.518	1	0.4717

Postconcussive Symptomology: TOTAL RUGBY versus HOCKEY CONTROL

Table 4-29. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	TOTAL RUGBY			HOCKEY CONTROL			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
1. Headaches										
n	19	25	3	18	15	1				
%	40.4	53.2	6.4	52.9	44.1	2.9	1.479	2	0.4774	
2. Eyesight										
n	40	4	3	29	3	2				
%	85.1	8.5	6.4	85.3	8.8	5.9	0.010	2	0.9949	
3. Hearing										
n	44	3	0	31	3	0				
%	93.6	6.4	0.0	91.2	8.8	0.0	0.171	1	0.6789	
4. Weakness in Limbs										
n	42	5	0	25	8	1				
%	89.4	10.6	0.0	73.5	23.5	2.9	4.023	2	0.1338	~
5. Clumsiness										
n	38	8	0	28	5	1				
%	82.6	17.4	0.0	82.4	14.7	2.9	1.440	2	0.4868	
6. Seizures										
n	47	0	0	34	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
7. Dizziness										
n	33	14	0	26	7	1				
%	70.2	29.8	0.0	76.5	20.6	2.9	2.132	2	0.3443	
8. Fatigue										
n	22	21	4	23	9	2				
%	46.8	44.7	8.5	67.6	26.5	5.9	3.492	2	0.1744	
9. Sensitivity to Noise										
n	42	5	0	28	6	0				
%	89.4	10.6	0.0	82.4	17.6	0.0	0.826	1	0.3635	
10. Hallucinations										
n	45	1	1	34	0	0				
%	95.7	2.1	2.1	100.0	0.0	0.0	1.483	2	0.4763	
11. Sexual Difficulties										
n	47	0	0	34	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
12. Speech Difficulties										
n	43	4	0	31	3	0				
%	91.5	8.5	0.0	91.2	8.8	0.0	0.002	1	0.9606	
13. Clumsy Speech										
n	22	25	0	23	11	0				
%	46.8	53.2	0.0	67.6	32.4	0.0	3.470	1	0.0625	~
14. Stutter										
n	43	4	0	32	2	0				
%	91.5	8.5	0.0	94.1	5.9	0.0	0.199	1	0.6558	
15. Slurred Speech										
n	35	11	1	30	3	0				
%	74.5	23.4	2.1	90.9	9.1	0.0	3.617	2	0.1639	
16. Memory										
n	36	11	0	28	4	2				
%	76.6	23.4	0.0	82.4	11.8	5.9	4.291	2	0.1170	~

Approaching Significance (~ 0.05<p<0.15)

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

Continued overleaf

Table 4-29. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire. (continued).

Question	TOTAL RUGBY			HOCKEY CONTROL			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
17. Attention/Concentration										
n	25	21	1	22	12	0				
%	53.2	44.7	2.1	64.7	35.3	0.0	1.601	2	0.4491	
18. Sustained Attention										
n	13	32	2	11	23	0				
%	27.7	68.1	4.3	32.4	67.6	0.0	1.594	2	0.4507	
19. Impatience										
n	19	24	4	14	17	3				
%	40.4	51.1	8.5	41.2	50.0	8.8	0.009	2	0.9953	
20. Irritability										
n	12	33	2	9	22	3				
%	25.5	70.2	4.3	26.5	64.7	8.8	0.762	2	0.6833	
21. Easily Angered										
n	21	20	6	17	17	0				
%	44.7	42.6	12.8	50.0	50.0	0.0	4.699	2	0.0954	~
22. Depressed										
n	23	24	0	19	15	0				
%	48.9	51.1	0.0	55.9	44.1	0.0	0.381	1	0.5369	
23. Social Contact										
n	0	3	44	0	1	33				
%	0.0	6.4	93.6	0.0	2.9	97.1	0.498	1	0.4805	
24. Restlessness										
n	30	14	3	23	10	1				
%	63.8	29.8	6.4	67.6	29.4	2.9	0.518	2	0.7718	
25. Sleep Difficulties										
n	30	17	0	27	6	0				
%	63.8	36.2	0.0	81.8	18.2	0.0	3.063	1	0.0801	~
26. Appetite Difficulties										
n	44	3	0	30	4	0				
%	93.6	6.4	0.0	88.2	11.8	0.0	0.724	1	0.3949	
27. Anxiety										
n	24	22	1	20	14	0				
%	51.1	46.8	2.1	58.8	41.2	0.0	1.083	2	0.5819	
28. Worry										
n	31	15	1	16	18	0				
%	66.0	31.9	2.1	47.1	52.9	0.0	4.079	2	0.1301	~
29. Argumentative										
n	14	25	8	9	21	4				
%	29.8	53.2	17.0	26.5	61.8	11.8	0.700	2	0.7048	
30. Short-tempered										
n	29	15	3	19	15	0				
%	61.7	31.9	6.4	55.9	44.1	0.0	3.076	2	0.2148	
31. Aggression										
n	40	4	3	31	3	0				
%	85.1	8.5	6.4	91.2	8.8	0.0	2.255	2	0.3238	

Approaching Significance (~ 0.05<p<0.15)

Postconcussive Symptomology: RUGBY FORWARDS versus RUGBY BACKS

Table 4-30. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	RUGBY FORWARDS			RUGBY BACKS			x2	df	*p*
	Never	Sometimes	Often	Never	Sometimes	Often			
1. Headaches									
n	14	12	2	5	13	1			
%	50.0	42.9	7.1	26.3	68.4	5.3	3.024	2	0.2205
2. Eyesight									
n	22	4	2	18	0	1			
%	78.6	14.3	7.1	94.7	0.0	5.3	3.124	2	0.2097
3. Hearing									
n	25	3	0	19	0	0			
%	89.3	10.7	0.0	100.0	0.0	0.0	2.175	1	0.1403
4. Weakness in Limbs									
n	26	2	0	16	3	0			
%	92.9	7.1	0.0	84.2	15.8	0.0	0.890	1	0.3454
5. Clumsiness									
n	21	7	0	17	1	0			
%	75.0	25.0	0.0	94.4	5.6	0.0	2.883	1	0.0895
6. Seizures									
n	28	0	0	19	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
7. Dizziness									
n	28	0	0	13	6	0			
%	71.4	28.6	0.0	68.4	31.6	0.0	0.049	1	0.8249
8. Fatigue									
n	15	11	2	7	10	2			
%	53.6	39.3	7.1	36.8	52.6	10.5	1.280	2	0.5272
9. Sensitivity to Noise									
n	26	2	0	16	3	0			
%	92.9	7.1	0.0	84.2	15.8	0.0	0.890	1	0.3454
10. Hallucinations									
n	27	0	1	18	1	0			
%	96.4	0.0	3.6	94.7	5.3	0.0	2.156	2	0.3403
11. Sexual Difficulties									
n	28	0	0	19	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
12. Speech Difficulties									
n	25	3	0	18	1	0			
%	89.3	10.7	0.0	94.7	5.3	0.0	0.432	1	0.5110
13. Clumsy Speech									
n	12	16	0	10	9	0			
%	42.9	57.1	0.0	52.6	47.4	0.0	0.434	1	0.5099
14. Stutter									
n	26	2	0	17	2	0			
%	92.9	7.1	0.0	89.5	10.5	0.0	0.166	1	0.6833
15. Slurred Speech									
n	20	7	1	15	4	0			
%	71.4	25.0	3.6	78.9	21.1	0.0	0.840	2	0.6571
16. Memory									
n	23	5	0	13	6	0			
%	82.1	17.9	0.0	68.4	31.6	0.0	1.189	1	0.2756

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

Continued overleaf

Table 4-30. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire. (continued).

Question	RUGBY FORWARDS			RUGBY BACKS			x2	df	*p*
	Never	Sometimes	Often	Never	Sometimes	Often			
17. Attention/Concentration									
n	15	13	0	10	8	1			
%	53.6	46.4	0.0	52.6	42.1	5.3	1.523	2	0.4670
18. Sustained Attention									
n	8	19	1	5	13	1			
%	28.6	67.9	3.6	26.3	68.4	5.3	0.097	2	0.9524
19. Impatience									
n	13	12	3	6	12	1			
%	46.4	42.9	10.7	31.6	63.2	5.3	1.926	2	0.3817
20. Irritability									
n	6	20	2	6	13	0			
%	21.4	71.4	7.1	31.6	68.4	0.0	1.828	2	0.4008
21. Easily Angered									
n	13	12	3	8	8	3			
%	46.4	42.9	10.7	42.1	42.1	15.8	0.277	2	0.8706
22. Depressed									
n	13	15	0	10	9	0			
%	46.4	53.6	0.0	52.6	47.4	0.0	0.174	1	0.6763
23. Social Contact									
n	0	3	25	0	0	19			
%	0.0	10.7	89.3	0.0	0.0	100.0	2.175	1	0.1403
24. Restlessness									
n	18	7	3	12	7	0			
%	64.3	25.0	10.7	63.2	36.8	0.0	2.571	2	0.2765
25. Sleep Difficulties									
n	17	11	0	13	6	0			
%	60.7	39.3	0.0	68.4	31.6	0.0	0.291	1	0.5895
26. Appetite Difficulties									
n	26	2	0	18	1	0			
%	92.9	7.1	0.0	94.7	5.3	0.0	0.067	1	0.7959
27. Anxiety									
n	15	12	1	9	10	0			
%	53.6	42.9	3.6	47.4	52.6	0.0	0.995	2	0.6081
28. Worry									
n	19	8	1	12	7	0			
%	67.9	28.6	3.6	63.2	36.8	0.0	0.959	2	0.6191
29. Argumentative									
n	8	15	5	6	10	3			
%	28.6	53.6	17.9	31.6	52.6	15.8	0.065	2	0.9682
30. Short-tempered									
n	17	9	2	12	6	1			
%	60.7	32.1	7.1	63.2	31.6	5.3	0.075	2	0.9633
31. Aggression									
n	25	1	2	15	3	1			
%	89.3	3.6	7.1	78.9	15.8	5.3	2.190	2	0.3345

Postconcussive Symptomology: RUGBY FORWARDS versus HOCKEY CONTROL

Table 4-31. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	RUGBY FORWARDS			HOCKEY CONTROL			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
1. Headaches										
n	14	12	2	18	15	1				
%	50.0	42.9	7.1	52.9	44.1	2.9	0.592	2	0.7440	
2. Eyesight										
n	22	4	2	29	3	2				
%	78.6	14.3	7.1	85.3	8.8	5.9	0.528	2	0.7680	
3. Hearing										
n	25	3	0	31	3	0				
%	89.3	10.7	0.0	91.2	8.8	0.0	0.063	1	0.8021	
4. Weakness in Limbs										
n	26	2	0	25	8	1				
%	92.9	7.1	0.0	73.5	23.5	2.9	4.077	2	0.1302	
5. Clumsiness										
n	21	7	0	28	5	1				
%	75.0	25.0	0.0	82.4	14.7	2.9	1.769	2	0.4129	
6. Seizures										
n	28	0	0	34	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
7. Dizziness										
n	20	8	0	26	7	1				
%	71.4	28.6	0.0	76.5	20.6	2.9	1.281	2	0.5271	
8. Fatigue										
n	15	11	2	23	9	2				
%	53.6	39.3	7.1	67.6	26.5	5.9	1.316	2	0.5179	
9. Sensitivity to Noise										
n	26	2	0	28	6	0				
%	92.9	7.1	0.0	82.4	17.6	0.0	1.508	1	0.2195	
10. Hallucinations										
n	27	0	1	34	0	0				
%	96.4	0.0	3.6	100.0	0.0	0.0	1.234	1	0.2666	
11. Sexual Difficulties										
n	28	0	0	34	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
12. Speech Difficulties										
n	25	3	0	31	3	0				
%	89.3	10.7	0.0	91.2	8.8	0.0	0.063	1	0.8021	
13. Clumsy Speech										
n	12	16	0	23	11	0				
%	42.9	57.1	0.0	67.6	32.4	0.0	3.838	1	0.0501	~
14. Stutter										
n	26	2	0	32	2	0				
%	92.9	7.1	0.0	94.1	5.9	0.0	0.040	1	0.8407	
15. Slurred Speech										
n	20	7	1	30	3	0				
%	71.4	25.0	3.6	90.9	9.1	0.0	4.219	2	0.1213	
16. Memory										
n	23	5	0	28	4	2				
%	82.1	17.9	0.0	82.4	11.8	5.9	2.040	2	0.3606	

Approaching Significance (~ 0.025<p<0.075), following Bonferonni's adjustments

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

Continued overleaf

Table 4-31. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire. (continued).

Question	RUGBY FORWARDS			HOCKEY CONTROL			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
17. Attention/Concentration										
n	15	13	0	22	12	0				
%	53.6	46.4	0.0	64.7	35.3	0.0	0.791	1	0.3738	
18. Sustained Attention										
n	8	19	1	11	23	0				
%	28.6	67.9	3.6	32.4	67.6	0.0	1.286	2	0.5257	
19. Impatience										
n	13	12	3	14	17	3				
%	46.4	42.9	10.7	41.2	50.0	8.8	0.321	2	0.8515	
20. Irritability										
n	6	20	2	9	22	3				
%	21.4	71.4	7.1	26.5	64.7	8.8	0.318	2	0.8532	
21. Easily Angered										
n	13	12	3	17	17	0				
%	46.4	42.9	10.7	50.0	50.0	0.0	3.851	2	0.1458	
22. Depressed										
n	13	15	0	19	15	0				
%	46.4	53.6	0.0	55.9	44.1	0.0	0.550	1	0.4585	
23. Social Contact										
n	0	3	25	0	1	33				
%	0.0	10.7	89.3	0.0	2.9	97.1	1.537	1	0.2150	
24. Restlessness										
n	18	7	3	23	10	1				
%	64.3	25.0	10.7	67.6	29.4	2.9	1.573	2	0.4554	
25. Sleep Difficulties										
n	17	11	0	27	6	0				
%	60.7	39.3	0.0	81.8	18.2	0.0	3.356	1	0.0670	~
26. Appetite Difficulties										
n	26	2	0	30	4	0				
%	92.9	7.1	0.0	88.2	11.8	0.0	0.375	1	0.5402	
27. Anxiety										
n	15	12	1	20	14	0				
%	53.6	42.9	3.6	58.8	41.2	0.0	1.300	2	0.5221	
28. Worry										
n	19	8	1	16	8	0				
%	67.9	28.6	3.6	47.1	52.9	0.0	4.565	2	0.1020	
29. Argumentative										
n	8	15	5	9	21	4				
%	28.6	53.6	17.9	26.5	61.8	11.8	0.595	2	0.7427	
30. Short-tempered										
n	17	9	2	19	15	0				
%	60.7	32.1	7.1	55.9	44.1	0.0	3.059	2	0.2166	
31. Aggression										
n	25	1	2	31	3	0				
%	89.3	3.6	7.1	91.2	8.8	0.0	3.091	2	0.2132	

Approaching Significance (~ 0.025<p<0.075), following Bonferonni's adjustments

Postconcussive Symptomology: RUGBY BACKS versus HOCKEY CONTROL

Table 4-32. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	RUGBY BACKS			HOCKEY CONTROL			x2	df	*p*
	Never	Sometimes	Often	Never	Sometimes	Often			
1. Headaches									
n	5	13	1	18	15	1			
%	26.3	68.4	5.3	52.9	44.1	2.9	3.528	2	0.1714
2. Eyesight									
n	18	0	1	29	3	2			
%	38.3	0.0	33.3	61.7	100.0	66.7	1.807	2	0.4051
3. Hearing									
n	19	0	0	31	3	0			
%	100.0	0.0	0.0	91.2	8.8	0.0	1.777	1	0.1825
4. Weakness in Limbs									
n	16	3	0	25	8	1			
%	84.2	15.8	0.0	73.5	23.5	2.9	1.090	2	0.5797
5. Clumsiness									
n	17	1	0	28	5	1			
%	94.4	5.6	0.0	82.4	14.7	2.9	1.582	2	0.4533
6. Seizures									
n	19	0	0	34	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
7. Dizziness									
n	13	6	0	26	7	1			
%	68.4	31.6	0.0	76.5	20.6	2.9	1.266	2	0.5309
8. Fatigue									
n	7	10	2	23	9	2			
%	36.8	52.6	10.5	67.6	26.5	5.9	4.719	2	0.0900
9. Sensitivity to Noise									
n	16	3	0	28	6	0			
%	84.2	15.8	0.0	82.4	17.6	0.0	0.030	1	0.8600
10. Hallucinations									
n	18	1	0	34	0	0			
%	94.7	5.3	0.0	65.4	0.0	0.0	1.824	1	0.1769
11. Sexual Difficulties									
n	19	0	0	34	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
12. Speech Difficulties									
n	18	1	0	31	3	0			
%	94.7	5.3	0.0	91.2	8.8	0.0	0.221	1	0.6379
13. Clumsy Speech									
n	10	9	0	23	11	0			
%	52.6	47.4	0.0	67.6	32.4	0.0	1.170	1	0.2795
14. Stutter									
n	17	2	0	32	2	0			
%	89.5	10.5	0.0	94.1	5.9	0.0	0.377	1	0.5394
15. Slurred Speech									
n	15	4	0	30	3	0			
%	78.9	21.1	0.0	90.9	9.1	0.0	1.481	1	0.2236
16. Memory									
n	13	6	0	28	4	2			
%	68.4	31.6	0.0	82.4	11.8	5.9	3.960	2	0.1381

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

Continued overleaf

Table 4-32. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire. (continued).

Question	RUGBY BACKS			HOCKEY CONTROL			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
17. Attention/Concentration										
n	10	8	1	22	12	0				
%	52.6	42.1	5.3	64.7	35.3	0.0	2.234	2	0.3273	
18. Sustained Attention										
n	5	13	1	11	23	0				
%	26.3	68.4	5.3	32.4	67.6	0.0	1.938	2	0.3795	
19. Impatience										
n	6	12	1	14	17	3				
%	31.6	63.2	5.3	41.2	50.0	8.8	0.888	2	0.6415	
20. Irritability										
n	6	13	0	9	22	3				
%	31.6	68.4	0.0	26.5	64.7	8.8	1.814	2	0.4037	
21. Easily Angered										
n	8	8	3	17	17	0				
%	42.1	42.1	15.8	50.0	50.0	0.0	5.691	2	0.0581	~
22. Depressed										
n	10	9	0	19	15	0				
%	52.6	47.4	0.0	55.9	44.1	0.0	0.052	1	0.8196	
23. Social Contact										
n	0	0	19	0	1	33				
%	0.0	0.0	100.0	0.0	2.9	97.1	0.570	1	0.4504	
24. Restlessness										
n	12	7	0	23	10	1				
%	63.2	36.8	0.0	67.6	29.4	2.9	0.806	2	0.6684	
25. Sleep Difficulties										
n	13	6	0	27	6	0				
%	68.4	31.6	0.0	81.8	18.2	0.0	1.219	1	0.2695	
26. Appetite Difficulties										
n	18	1	0	30	4	0				
%	94.7	5.3	0.0	88.2	11.8	0.0	0.603	1	0.4374	
27. Anxiety										
n	9	10	0	20	14	0				
%	47.4	52.6	0.0	58.8	41.2	0.0	0.646	1	0.4217	
28. Worry										
n	12	7	0	16	18	0				
%	63.2	36.8	0.0	47.1	52.9	0.0	1.268	1	0.2602	
29. Argumentative										
n	6	10	3	9	21	4				
%	31.6	52.6	15.8	26.5	61.8	11.8	0.436	2	0.8042	
30. Short-tempered										
n	12	6	1	19	15	0				
%	63.2	31.6	5.3	55.9	44.1	0.0	2.383	2	0.3037	
31. Aggression										
n	15	3	1	31	3	0				
%	78.9	15.8	5.3	91.2	8.8	0.0	2.522	2	0.2834	

Approaching Significance (~ 0.025<p<0.075), following Bonferonni's adjustments

CHAPTER FIVE: DISCUSSION

This chapter focuses on an examination of the research hypotheses in relation to the results of the statistical analyses. These results will be discussed in depth and compared with previous research findings in the area. Thereafter, the theoretical implications of the results will be explored, utilizing the conceptual framework of Satz's brain reserve capacity (BRC) theory where applicable and conclusions will be drawn. Finally, this study's methodological strengths and limitations will be assessed and recommendations for future research will be made.

5.1. AIMS AND HYPOTHESES OF THE CURRENT STUDY

The present study aimed to investigate the effects of concussive and subconcussive cumulative head injuries among schoolboy rugby players. This was achieved by comparing their neuropsychological test performances with those of a non-contact sports control group (schoolboy hockey players). An additional comparison was made between the schoolboy rugby players and schoolboy hockey players' results on a postconcussive symptom checklist. The participants for this study were all Post-Matric, Matric and Std 9 scholars from the top-level rugby and hockey teams of three English-medium boys' high schools. Participants were selected on the basis of a number of exclusion criteria, including the reported presence of learning difficulties, a neurological or psychiatric disorder, a history of substance abuse and a prior moderate to severe head injury for any reason. These criteria were applied in order to ensure that any impairments noted from this study in the rugby group could not be ascribed to any one of these other causes. Further attempts were made to ensure that the groups were equivalent on a number of potentially confounding demographic variables. In this respect, analyses revealed no significant differences between the schoolboy rugby and hockey players with regard to the variables of age, education level, highest grade achievement and estimated premorbid IQ. Furthermore, in terms of positional variation within the rugby group, analyses revealed no significant differences between the Rugby Forwards group and the Rugby Backs group with regard to the variables of age, education level, highest grade achievement and estimated premorbid IQ. Thus, it can be argued that in this study any differences in results noted between rugby and hockey players, and between rugby forwards and rugby backs, cannot be ascribed to the effects of any one of these demographic variables.

For the purposes of this study, it was hypothesized that schoolboy rugby players would demonstrate greater cognitive impairment on neuropsychological tests sensitive to the effects of diffuse brain damage as well as report a greater frequency of postconcussive symptomatology, relative to

schoolboy hockey players. This is because rugby players, due to the nature of the game, are more likely to be exposed to cumulative concussive and subconcussive mild head injuries. It was further hypothesized that rugby forward players would demonstrate greater cognitive impairment on neuropsychological tests sensitive to the effects of diffuse brain damage as well as report a greater frequency of postconcussive symptomatology, relative to rugby backline players. This is because forward players tend to be involved in more collisions and impacts (sustained during scrumming and tackling) than backline players, making them more likely to be exposed to mild head injuries. In both cases, these hypotheses were supported by prior studies on Rugby Union professional players, using a comparable test battery (for example, Border, 2000; Dickinson, 1998).

5.2. NEUROPSYCHOLOGICAL TEST RESULTS

5.2.1. INDIVIDUAL COGNITIVE TEST DEFICIT

As noted in Chapter four (p. 75), there were no significant differences in performance on any of the neuropsychological tests for the Total Rugby versus Hockey Control comparison, nor for the subgroup comparisons of Rugby Forwards versus Rugby Backs, Rugby Forwards versus Hockey Control and Rugby Backs versus Hockey Control. This suggests that rugby players, in particular forward players, do not demonstrate cognitive impairment as a result of cumulative head trauma. However, there were four test results in which comparisons of the level of deficit between groups and subgroups were *approaching significance*, which warrant further discussion below. These tests were Finger Tapping (Preferred Hand), Structured Verbal Fluency, Digit Symbol Substitution Incidental Recall (Immediate) and WMS Visual Reproduction (Immediate Recall). Where the percentage of impairment is utilized in the discussion, this figure will refer to a combined percentage including both the categories of "mild" and "moderate/severe" unless otherwise stated.

5.2.1.1. Finger Tapping Test (Preferred Hand)

For the Finger Tapping Test (Preferred Hand), there was one comparison which approached significance. When Total Rugby was compared with Hockey Control, 25.5% of Total Rugby demonstrated impairment compared with 11.7% of Hockey Control. This result was strengthened by the fact that the Finger Tapping Test (Non-Preferred Hand) result was in the same direction, with Total Rugby displaying a tendency towards greater impairment relative to Hockey Control. This test measures hand motor dexterity. According to Stuss et al. (1985), brain damage can in some instances have a slowing effect on finger tapping rate (cited in Lezak, 1995). As this is a timed test, bilateral slowing would be an indication of diffuse brain damage. Thus this finding would appear to indicate a

higher level of deficit amongst the rugby players with respect to hand motor dexterity. However, before any meaningful conclusions can be drawn from this result, there are other factors that need to be considered.

Firstly, it is important to note that this result is not consistent with the findings of previous research on university and professional rugby players, which noted superior hand motor functioning amongst the rugby players (for example, Ancer, 1999; Bold, 1999; Finkelstein, 1999). In these studies, an explanation offered for this finding was that the nature of the game requires coarse hand motor dexterity and thus necessitates the development of superior hand motor functioning, which may in turn compensate for some deficit (Shuttleworth-Jordan, 1993). In this respect, it can be argued that the reason for the difference in results between the present finding on schoolboy rugby players and previous research findings on professional rugby players is that schoolboy rugby players have not participated long enough in rugby to develop such superior hand motor skills. Thus, their results can be seen to provide tentative indicators of the presence of mild brain damage. However, at the same time, it is important to bear in mind that this test may not be rigorous enough for neuropsychological screening. As noted earlier (p. 45), Shuttleworth-Jordan (1993) has argued that the interpretive validity of this test is limited, in that the differences in results amounts to fractions of a second and thus the test cannot be scored rigorously enough to ensure consistent and reproducible differences. This is because the test involves manual scoring rather than the use of a mechanical device, thereby resulting in a lack of precision and accuracy. Thus, it could be that the conflicting results noted between the present and previous studies are reflecting the variability in administration, rather than the presence of impairment. So, at this stage, the finding of more impairment in the schoolboy rugby players relative to the hockey controls needs to be interpreted cautiously and can only be regarded as providing a marginal indication of cognitive impairment in the schoolboy rugby players for this particular function.

5.2.1.2. Structured Verbal Fluency

On the Structured Verbal Fluency Test, there was one comparison that approached significance. When Rugby Forwards were compared to Hockey Control, 3.6% of Rugby Forwards demonstrated impairment compared with 26.0 % of Hockey Control. This test assesses ease and speed of verbal productivity, a function often compromised after brain injury. It also indirectly utilizes recent short-term memory in order to keep track of words already used (Lezak, 1995).

This result appears to be in the opposite direction than expected, indicating a higher level of impairment in hockey players rather than rugby players for this function. Furthermore, it does not

corroborate research findings from phase two, which indicated a lowered performance relative to the norm for the Under 21 rugby group (Bold, 1999). As in the previous result, a possible explanation for this absence of cognitive impairment in the schoolboy rugby players may be their limited exposure to mild head injuries as a result of not having participated as intensively and for as lengthy periods in the sport as other research groups. However, if the result is examined in relation to the demographic data for each of these groups, it seems more likely that the reason for the absence of impairment amongst rugby players is due to the fact that the Rugby Forwards appear to be a particularly high functioning group. As noted earlier in Chapter three (p. 57), the Rugby Forwards had the highest upper limit on the demographic variable of *estimated IQ* in relation to both Hockey Control and Rugby Backs (133.0 versus 129.5, for Hockey Control and 119.0 for Rugby Backs), thereby indicating that they are showing a tendency towards being a particularly high-functioning group Thus, rather than demonstrating the existence of impairment in the hockey players, this result would appear to suggest that what is being evidenced here is normal variation amongst the hockey players, and that the Rugby Forwards are demonstrating a tendency towards well-developed verbal skills. Given that verbal ability is strongly correlated with General IQ, it may be argued that the Rugby Forwards' result on this test can be understood in relation to the sampling effect of their tendency to be a particularly high functioning subgroup.

5.2.1.3. Digit Symbol Incidental Recall (Immediate)

On the Digit Symbol Incidental Recall (Immediate) test, there was one comparison that approached significance. When Rugby Forwards were compared with Rugby Backs, 10.7% of Rugby Forwards demonstrated impairment compared with 36.8% of Rugby Backs. This test taps various functions, including attention, planning, memory and information processing (Walsh, 1985). It is a sensitive indicator of diffuse brain damage and has demonstrated discriminatory capacity in detecting the presence of cognitive impairment (Shuttleworth-Jordan & Bode, 1995). This result, however, occurs in the direction opposite to that hypothesized, indicating a higher level of impairment in Rugby Backs rather than Rugby Forwards on this test. In addition, it does not corroborate research findings from phase two that indicated a higher level of impairment in the Springbok rugby players relative to the controls and in the Springbok Forwards relative to the Springbok Backs (Border, 2000). Since this test is considered a sensitive indicator of diffuse brain damage, this result would appear to suggest a lack of cognitive impairment in the schoolboy rugby forward players. However, this suggestion must remain tentative, as it is possible that this is a function where impairment is not detectable at the lower level of the game but requires a lengthier period of intensive participation and greater exposure to the cumulative effects of mild head injury, before beginning to manifest.

Furthermore, if the results are considered in relation to the demographic data (see discussion above on verbal fluency), it is evident that the Rugby Forwards may be performing well due to their being a particularly high functioning group. As noted in Chapter three (p. 57), although not statistically significant, Rugby Forwards have higher scores than Rugby Backs for both the demographic variables of *average grade 99* (70.5 versus 66.8, respectively) and *estimated IQ* (range 89.0-133.0 versus 89.0-119.0, respectively). Thus, once again it may be argued that, rather than indicating impairment in the Rugby Backs for this particular function, this result simply demonstrates that the Rugby Forwards are reflecting their tendency to be a particularly high functioning group.

5.2.1.4. WMS Visual Reproduction (Immediate Recall)

On the WMS Visual Reproduction (Immediate Recall), there was one comparison that approached significance. When Rugby Forwards were compared with Rugby Backs, 3.6 % of Rugby Forwards demonstrated impairment compared with 26.3 % of Rugby Backs. This test assesses visual memory for designs, and is sensitive to the effects of brain trauma (Lezak, 1995). Once again, this is another finding which occurs in the opposite direction as hypothesized, indicating a higher level of impairment in Rugby Backs than in Rugby Forwards on this test. Furthermore, this result does not corroborate research findings from phase two, which indicated a poorer performance for both the rugby groups (Bold, 1999) relative to the hockey groups, as well as other studies in which this test has distinguished between MHI patients and controls (for example Stuss et al., 1985). However, as argued above, when these results are examined in relation to the demographic data, they become more meaningful in relation to the research hypotheses. This is since the tendency of Rugby Forwards to have higher scores than Rugby Backs for the demographic variables of *average grade 99* and *estimated IQ* suggests that the Rugby Forwards are a particularly high functioning subgroup. Thus rather than indicating impairment amongst the Rugby Backs for this function, the result appears to indicate that Rugby Forwards may demonstrate a tendency for relatively superior visual memory.

5.2.2. TOTAL COGNITIVE TEST DEFICIT

As noted in Chapter four (p. 76) there were no significant differences in the total cognitive test deficit category for the Total Rugby versus Hockey Control comparison, nor for the subgroup comparisons of Rugby Forwards versus Rugby Backs, Rugby Forwards versus Hockey Control and Rugby Backs versus Hockey Control. This would appear to indicate an absence of cognitive impairment for all rugby players relative to hockey players across all cognitive tests as well all rugby forward players relative to rugby backline players across all cognitive tests. Further, there was only one test result in

which comparison of the level of deficit between groups and subgroups was *approaching significance*, which warrants further discussion as follows.

There was a single comparison in which differences approached significance, which was in the subcategory of **Moderate/Severe Cognitive Deficit - Any**. When Rugby Forwards were compared with Hockey Control, 25.0% of Rugby Forwards demonstrated moderate to severe cognitive deficit across one or more neuropsychological tests in comparison with 47.1% of Hockey Control. This finding is in the opposite direction as expected, indicating a higher level of impairment in the Hockey Control group than in the Rugby Forwards. While this result would appear to indicate an absence of cognitive impairment in the Rugby Forwards, this finding is limited clinically. This is because a comparison of the results for Rugby Forwards versus Hockey Control for the subcategory of Mild Cognitive Test Deficit-Any indicates that a large percentage (up to 75%) of both groups have mild deficit on one or more neuropsychological tests. This means that what is being evidenced is a pattern of normal variation, as the large majority of both rugby and hockey players appear to show impairment on at least one test or more. Thus it is apparent that on a test battery of this type, it would not be statistically significant for an individual to demonstrate a moderate/severe cognitive deficit (see section 3.7.1, p. 72 for definition) on only a single test, without this necessarily implying the presence of brain damage. Thus in order to differentiate more accurately between individuals with brain damage and those without, it is necessary to utilize a more stringent cut-off point comprising at least two or three neuropsychological tests with the presence of deficit.

5.3. POSTCONCUSSIVE SYMPTOMATOLOGY RESULTS

As noted in Chapter four (p.77), there were no significant differences in the percentage of self–reported postconcussive symptomatology for the Total Rugby versus Hockey Control comparison, nor for the subgroup comparisons of Rugby Forwards versus Rugby Backs, Rugby Forwards versus Hockey Control and Rugby Backs versus Hockey Control. However, within the postconcussive symptomatology questionnaire there were six postconcussive symptoms in which comparisons of reported frequencies *approached significance*. These were a) clumsy speech; b) memory; c) being easily angered; d) sleep difficulties; e) worry and f) weakness in limbs. These will be discussed below. Where the actual percentage of individuals who report a symptom is used, this figure will refer to combined categories of both "sometimes" and "often".

5.3.1. CLUMSY SPEECH

There were two comparisons that approached significance for this symptom. When Total Rugby was compared with Hockey Control, 53.2% of Total Rugby reported experiencing this symptom in contrast to 32.4 % of Hockey Control. Similarly, when Rugby Forwards were compared with Hockey Control, 57.1% of Rugby Forwards reported experiencing this symptom in contrast with 32.4 % of Hockey Control.

Neuropsychological research on the contact sports has indicated that clumsy speech is a self-reported symptom of postconcussive symptomatology. According to Critchley (1957, in Jordan, 1987), boxers sometimes report transient speech difficulties following a bout, although this symptom soon resolves. More recently, Shuttleworth-Jordan et al. (1993) noted in their study of university rugby players that speech problems were reported three days post-injury, although they had resolved by one month post-injury. Not only is this current finding consistent with these studies, but it corroborates findings from phase two of the rugby research on professional players, which indicated a greater frequency of self-reported clumsy speech amongst both rugby groups (Springboks and Under 21) relative to the controls, and amongst rugby forward players relative to rugby backline players (Border, 2000). While this finding does not appear to be backed up by objective test findings on the verbal fluency tests (which indicated an absence of cognitive impairment in rugby players relative to controls), this does not necessarily weaken the significance of this result. Rather, it can be argued that despite a strong performance on objective cognitive tests as a result of being a particularly high functioning group, the Rugby Forwards, in particular, still experience and report difficulties in this area. This may imply that it is the subjectively reported symptoms following a mild head injury rather than the objective cognitive test results that are the first manifestations of cognitive impairment. In this respect it is possible that the objective tests may not be sensitive enough measures for detecting the presence of deficit in populations whose functioning may be high enough to compensate for any deficit present.

5.3.2. MEMORY

There was one comparison that approached significance for this symptom. When Total Rugby was compared with Hockey Control, 23.4% of Total Rugby reported experiencing this symptom in contrast to 17.7 % of Hockey Control.

Memory problems have been frequently documented in the neuropsychological literature on mild head injury in general (Barth et al., 1983; Basset & Slater, 1990; Rimel et al., 1980; Rutherford et al., 1977) and mild head injury in the contact sports (Barth et al., 1989, Critchley in Jordan, 1987;

Macciochi et al., 1996). Transient memory difficulties have been recorded in boxers following a bout, although they tend to resolve rapidly (Critchley, 1957 in Jordan, 1987). Consistent with this finding are the results of a study by Barth et al. (1989) that noted a considerable increase in memory problems 24 hours post-injury although this symptom returned to pre-season rates by 10 days post-injury. In the follow up study, Macciochi et al. (1996) also found a significant increase in reported memory problems 24 hours post-injury. Notably, however, the researchers found that, in contrast to other postconcussive symptoms that had resolved by 10 days post-injury, there was a slight increase in reported memory problems 24 hours post-injury. More recently, findings from phase one of the rugby research project on professional players have indicated a greater frequency of self-reported memory problems amongst the Springbok rugby players relative to the control group and most notably, in the Springbok Forwards (Border, 2000; Dickinson, 1998).

While this finding of a higher incidence of self-reported memory problems in the schoolboy rugby playing group is consistent with the previous research, it is not backed up by the objective cognitive tests, which found neither significant differences nor differences approaching significance in the direction of rugby players demonstrating greater impairment than hockey players on tests of visual and verbal memory. However, as noted earlier, the lack of corroboration from objective tests data may simply indicate that these tests are not sensitive enough to reveal subtle deficits in very high functioning groups. The fact that these high functioning groups continue to report difficulties, despite a superior performance on the cognitive tests, again indicates that it is the subtle self-reported difficulties that may become apparent first and perhaps only at a much later stage become verifiable with objective test data.

5.3.3. EASILY ANGERED

There were two comparisons that approached significance for this symptom. When Total Rugby was compared with Hockey Control, 92.6% of Total Rugby reported experiencing this symptom in contrast to 50.0 % of Hockey Control. Similarly, when Rugby Backs were compared with Hockey Control, 15.8% of Rugby Backs reported experiencing this symptom in contrast with 0% of players in the Hockey Control group.

In broad terms this finding of being easily angered, a feature for schoolboy rugby groups and not controls, is consistent with research findings of phase two, although in contrast to the previous research, the rugby forward players did not especially report a greater frequency of this symptom than the rugby backline players. However, the fact that the Total Rugby group have more of this reported symptom than the controls suggests that the trend is there for Rugby Forwards as well as Rugby

Backs, or it would not have shown up as approaching significance. Further, when being easily angered (i.e. showing lowered frustration tolerance) is considered together with the above noted symptoms of memory difficulties and clumsy speech, these appear to suggest a pattern of difficulties consistent with the pathophysiology of frontal-temporal lobe damage, typically associated with closed head injury (see section 2.1.6, p. 12). Here it was noted that autopsy reports have indicated that the greatest and commonest zones of brain contusion in closed head injury are in the frontal and temporal regions of the brain. Damage to these areas may result in "frontal lobe syndrome" that comprises both *cognitive* changes, including memory difficulties, attentional deficits, speech difficulties, decreases in verbal fluency and executive deficits as well as *personality/emotional* changes such as disinhibition, aggressiveness, depression, anxiety and irritability (Lezak, 1995; Walsh, 1985).

However, while the above argument provides some tentative support indicating a tendency towards a poor frustration tolerance in the rugby group, it is difficult to ascertain whether this is a direct effect of repeated exposure to mild head injuries or a consequence of pre-selected differences which cannot be ruled out in cross-sectional research, such as this study. In this respect, it should be noted that long-standing personality variables could also account for this result. Specifically, it may be argued that adolescents who are more extroverted and who adopt a more tougher "macho" exterior are more likely to be drawn in the first instance to and choose rugby over other sports. In contrast, the less aggressive nature of hockey might be a feature that attracts individuals with a more introverted, sensitive disposition. Further, it should be noted that rugby is, by its very nature, a more aggressive sport and thus as a consequence, rugby players may become socialized into adopting more aggressive modes of behaviour and expressing themselves in a less inhibited fashion. Thus, at this stage, it is not possible to draw any definite conclusions about the meaning of the frequency of this symptom in rugby players and any hypotheses proposed need to be made with extreme caution, especially in light of the fact that this result was only approaching significance.

5.3.4. SLEEP DIFFICULTIES

There were two comparisons that approached significance for this symptom. When Total Rugby was compared with Hockey Control, 36.2% of Total Rugby reported experiencing this symptom in contrast to 18.2% of Hockey Control. Similarly, when Rugby Forwards were compared with Hockey Control, 39.3% of Rugby Forwards reported experiencing this symptom in contrast with 18.2% of the Hockey Control group.

Insomnia has been documented following mild head injuries in general (McLean et al., 1983; Rutherford, 1989; Rutherford et al., 1977) as well as following mild head injuries in sport (Barnes et al., 1998; Shuttleworth-Jordan et al., 1993). In the Shuttleworth-Jordan et al. (1993) study on top

level university players, the researchers noted that this symptom was present three days post-injury although it had resolved by the one month follow-up. This finding corroborates findings from phase two of the rugby research on professional players in which a general trend was noted within both rugby groups (Springboks and Under 21) for the forward players to report a greater frequency of this symptom than the backline players (Border, 2000). This may suggest some positional variation due to the fuller contact role of the forward players. However, due to the fact that the schoolboy rugby research was performed during the playing season, and the lack of post-season measures of the current study, it is difficult to ascertain whether this is a chronic symptom as found in phase two (which was post-season to rule out acute effects) or whether it is a symptom which resolves quickly as noted by Shuttleworth-Jordan et al. (1993).

5.3.5. WORRY

There was one comparison that approached significance for this symptom. When Total Rugby was compared with Hockey Control, 34.0 % of Total Rugby reported experiencing this symptom in contrast to 52.9 % of Hockey Control.

Worry (anxiety) has been documented following mild head injuries in general (McLean et al., 1983; Rutherford, 1989; Rutherford et al., 1977) as well as mild head injuries in sport (Dickinson, 1998; Shuttleworth-Jordan et al., 1993). In their study on university players, Shuttleworth-Jordan et al. (1993) noted that anxiety was present three days post-injury but had resolved within one month. In contrast, findings from phase one of the research on professional rugby players recorded anxiety as present at least three months post-season. Positional variation was also noted with rugby forward players reporting experiencing anxiety more frequently than rugby backline players (Dickinson, 1998). Similarly, in phase two of the research, Border (2000) found a greater frequency of reported worry in rugby forward players relative to rugby backline players, and in Springbok forward players relative to Springbok backline players.

This study's finding is not consistent with the results of previous research as the result occurs in the opposite direction to that expected, with hockey players reporting a greater frequency of worry than rugby players. This finding appears to indicate that rugby players do not experience higher levels of anxiety relative to hockey players. However, a possible explanation for this unexpected finding is that the apparent absence of anxiety in rugby players is due to their underreporting this particular symptom. As noted earlier, rugby players may undergo social pressures, which lead to their adopting particular modes of behaviour and presentation that they regard as more fitting with the self-image accompanying the sport in which they participate. In this respect, it is feasible that rugby players have underreported this symptom due to their concern at being perceived as weak or "neurotic",

which would not be seen as fitting with a general image of themselves as confident and assertive individuals. A possible explanation for the difference in findings between this study and the previous research on professional players is that since the current sample consists of adolescents rather than adults, the issue of social image, from a developmental perspective, is more likely to be a prominent and sensitive one for them. However, as this result occurs in isolation, only tentative conclusions regarding this finding can be drawn at this stage.

5.3.6. WEAKNESS IN LIMBS

There was one comparison that approached significance for this symptom. When Total Rugby was compared with Hockey Control, 10.6% of Total Rugby reported experiencing this symptom in contrast to 23.5% of Hockey Control.

This result is in the opposite direction as expected, with hockey players reporting a greater frequency of weakness in the limbs relative to rugby players. This finding is, however, consistent with findings from phase two of the research with professional players where greater frequencies of this symptom were observed in the hockey players relative to the Springbok rugby players. As noted for the above symptom of worry, it is possible that this result may be due to an underreporting of this symptom by the rugby players. Due to a need to present with apparent great physical strength and stamina, rugby players may have been reluctant to report this symptom due to their possible concern of appearing unfit or weak. This hypothesis is further strengthened by the argument presented above, that rugby players may show a tendency not to report anxiety, lest they be viewed as being less masculine. The notion of preselected differences may also apply to the hockey players, for it is possible that individuals who perceive themselves as less tough and having less physical strength may be more likely to choose hockey over rugby, as the game is less physically demanding.

While the validity of this above finding may appear to be undermined by Satz's (1999) argument that without a research design using another trauma control group (see section 2.2.2.1, p. 26), one cannot argue that the observed postconcussive symptoms are due to cumulative head trauma, it can be argued that the so-called sports injury group is different from classical MHI patients in that the assessment of the group of schoolboy rugby and hockey players is not an assessment of individuals who identify themselves as having experienced trauma. Thus, in this instance, an "other injury" group is superfluous in the current research design and therefore, its absence does not weaken the proposed argument that the postconcussive symptoms that are being evidenced may well apply to cumulative mild head trauma.

5.4. IMPLICATIONS OF THE RESEARCH

Overall, the neuropsychological results (cognitive test data) of the present research do not appear to support previous research findings of cognitive deficit in rugby players. Specifically, previous research findings on professional players indicated a pattern of deficit in the functional areas of speed of information processing, attention and concentration, verbal and visual memory, working memory, verbal fluency and hand motor dexterity amongst the rugby players (Border, 2000; Dickinson, 1998). Furthermore, a consistent pattern of positional variation within the rugby group was recorded, with rugby forward players being more susceptible to cognitive impairment than rugby backline players. In contrast, results from the present study clearly demonstrate an absence of any pattern suggestive of cognitive impairment within the rugby playing group. Furthermore, there is no evidence to support the hypothesis that forward players demonstrate a disproportionately poorer cognitive performance relative to backs. With the exception of an isolated test result indicating a lowered performance in the schoolboy rugby players relative to hockey controls for the function of hand motor dexterity (Finger Tapping Test (Preferred Hand), there were no results that were significant or even approaching significance in the direction of rugby players demonstrating greater impairment in cognitive functioning relative to hockey players. While hand motor dexterity is one of the functions typically compromised in closed head injury, it was considered that this result in isolation had questionable meaning due to an unreliable scoring procedure and its lack of corroboration with research findings on professional and university rugby players, that have generally noted superior hand motor dexterity among the rugby players as well as the fact that this result was not significant, but only approaching significance Thus overall, the cognitive test results do not support the hypothesis that cumulative mild head trauma causes cognitive impairment in rugby players, or that forward rugby players are more susceptible to such impairment than backline players. This may be due to schoolboy rugby players having had a shorter and less intensive period of participation in the game, thereby making them less exposed to cumulative mild head injury than previous research groups. At most, it may be argued that what is being observed at this stage are the *beginnings* of a drop off in cognitive performance, and that perhaps this modality is one for which impairment is likely to manifest at an earlier stage.

In contrast, the results of the postconcussive symtomatology questionnaire clearly indicate a greater frequency of reported post concussive symptoms in the rugby players relative to the hockey players. As previously discussed, six symptoms were found to be approaching significance, of which four, namely, clumsy speech, easily angered, sleep problems and memory problems were in the expected direction of rugby players reporting greater frequency of postconcussive symptomatology relative to hockey controls. Together, these symptoms are conceptually consistent with the expected cluster of

symptoms associated with frontal and/or temporal damage, common in closed head injury and furthermore, are commensurate with the findings of previous research on professional players (Border, 2000; Dickinson, 1998), thereby adding strength to the current findings.

Methodologically, in contrast to the positive finding on the Finger Tapping Test, the cluster of postconcussive symptoms noted in this study appear to be more robust. As noted earlier, Satz (1999) has suggested that without properly controlled studies comprising of another other injury control group, no valid conclusions can be drawn regarding the PCS. However, this argument cannot be said to apply in the case of the present study where non-specific cumulative effects of multiple mild head injuries are being targeted i.e. it seems unlikely that these target groups are aware of having personally experienced trauma in the same way as an accident victim might. Therefore the symptoms elicited can probably be attributed to the effects of the head injuries sustained during playing rugby. Significantly, all the symptoms noted in this study were also recorded in phase two of the research (i.e. being easily angered, clumsy speech, sleep difficulties and memory difficulties).

In contrast to previous studies from phase one and two with professional rugby players, the postconcussive symptoms noted in this study were not supported by the cognitive test data as evidenced by the players' performances across a battery of neuropsychological tests. An explanation offered for this finding was that that the neuropsychological tests were perhaps not sensitive enough for the detection of deficit in a population of particularly high functioning schoolboys whose high IQs may compensate for any deficits that are present. It was further argued that schoolboy rugby players have not played rugby for as long nor as intensively as professional players and therefore, may begin to manifest the symptoms sought only much later in their careers. While it may be argued that the symptoms recorded in this study were only approaching significance and therefore are tentative indicators of neuropsychological dysfunction, the fact that they are consistent with the expected brain damage picture of frontal pathology and corroborate previous research findings, suggests that they need to be taken seriously as they may be the earliest manifestations evidenced proceeding to a later onset of cognitive impairment.

In this respect, it may be argued that while the negative effects are not overall as evident in the schoolboy rugby players as they are in the professional players utilized in past research, the lack of explicit symptomatic evidence of neuropsychological dysfunction does not necessarily imply an absence of structural brain injury. Symptoms that are not immediately evident may, in fact, be latent and may only arise later as a result of subclinical brain injury. In order to understand the seeming absence of neuropsychological dysfunction in the rugby players, it is necessary to move away from an empirical focus to locating the results within a theoretical framework, which allows for the

identification of patterns not always apparent and may provide a more clinically relevant understanding of the test results.

If we return to Satz's (1993) BRC theory as discussed earlier (see section 2.4, p. 52), it will be recalled that Satz posits a threshold factor (also referred to as a functional cut-off point), which exists prior to the manifestation of symptoms caused by disease in the central nervous system. According to this model, BRC thresholds differ between individuals, as certain factors act as protective or vulnerability factors, serving to increase or lower the BRC, and thereby decreasing or increasing the risk of functional impairment in an individual. According to Satz (1993), protective factors typically comprise younger age, high IQ and high educational levels. If we now examine the demographic characteristics of the research group, it is clear from the demographic data that the research group represents a population of young, relatively high functioning individuals with high average to superior IQs and a relatively high standard of education. It may thus be argued that these factors serve to preserve these individuals from neuropsychological dysfunction, allowing them to fall above the functional cut-off point, which manifests as an absence of symptoms of neuropsychological dysfunction. Furthermore, the application of exclusion criteria to this sample, including a learning disability, a neurological disorder and a prior moderate to severe head injury would also serve to increase BRC in this sample, as these three factors have all been identified by Satz as vulnerability factors which would cause a reduction in BRC, predisposing individuals to falling below the symptom threshold and thus demonstrating functional impairment.

In addition to the above vulnerability factors noted, Satz (1993) also argues that another risk factor that serves to lower brain threshold, is the effects of a high task challenge. This refers to the differing levels of difficulty of cognitive tests which become increasingly more demanding so as to increase the possibility of demonstrating functional impairment. If we examine the potential effects of this factor in relation to the neurospsychological results of this study, it is possible to argue that the tests used in this battery, while being sophisticated, were in essence not complex enough for such a high functioning population and that the use of more sensitive measures such as computerized tests of reaction time e.g. the Vienna Test System (Schuhfried, 1994) may reveal more pronounced deficits in the rugby players. Furthermore, the role of task conditions in affecting performance cannot be overemphasized. As noted earlier (see section 2.2.1.1, p. 18), Ewing et al. (1980) found that head-injured subjects performing under hypoxic conditions manifest cognitive impairment in contrast to non-head injured controls. Similarly, Parasuruman et al. (1991) found that vigilance in MHI patients remains unaffected under normal task conditions but becomes impaired in conditions under task conditions requiring effortful processing. Thus it is possible that an alternative environment of more stressful test conditions may have resulted in cognitive impairment becoming more discernible.

Finally, it must be noted once again, that the research sample comprised younger players who have not been playing as long or as intensively as the professional and university players' samples. Thus, having had a shorter rugby playing career, it is more than likely that these players will not have been exposed as much to mild head injuries as university and professional players and that it is only after an intensive long-term period of participation that the effects of cumulative mild head injury may become apparent. The marginal indications noted, particularly on the postconcussive symptoms, may indicate that the rugby group is approaching the threshold for symptom presentation that will occur if they take their rugby careers further at a university or professional level.

In sum, given all of the above these factors, it can be argued that that the schoolboy rugby players who are exposed to mild head injuries may have sustained brain injury resulting in reduced BRC but, that due to the presence of a number of protective factors, they are not evidencing any cognitive impairment or overt symptomatology at present. However, given the fact that subtle indications of self-reported postconcussive symptoms are already beginning to manifest, it is possible that should they continue to experience mild head injuries due to the participation in the game, that these, in combination with their natural aging, will cause these individuals to fall below the threshold point and begin to demonstrate symptoms of functional impairment. Thus it is not advisable at this point to rule out potentially harmful latent effects that may not be evident now, but may place these individuals at increased future risk for later onset neuropsychological dysfunction, with the increase of vulnerability factors such as older age.

5.5. CONCLUSION

The objective of this study was to investigate the effects of cumulative concussive and subconcussive mild head injury on schoolboy rugby players. Overall, the results indicated that schoolboy rugby players do not demonstrate increased cognitive impairment relative to schoolboy hockey players (the control group), as evidenced by their performances on a neuropsychological test battery, sensitive to the effects of diffuse brain damage. They do, however, manifest subtle indicators of postconcussive symptomatology with a greater frequency relative to the control group. The symptoms noted in this study were clumsy speech, memory difficulties, being easily angered and sleep difficulties. It was hypothesized that these symptoms might be the earliest manifestations of subclinical brain damage, which could manifest at a later stage, should they continue with their rugby career at a university and professional level. While the current findings are not consistent with previous research findings of cognitive deficit in professional players, the observation of increased postconcussive symtomatology in the schoolboy rugby players does corroborate research findings from the first and second phases of the research project. To this end, the present findings can be regarded as sufficiently robust to be

given serious consideration. It is thus essential that sports medicine practitioners, coaches, parents and rugby players be aware of the clear risks associated with exposure to cumulative concussive and subconcussive mild head injuries, so that prevention measures can be taken to maximise and ensure safety for on-field participation.

5.6. EVALUATION OF PRESENT RESEARCH

5.6.1. METHODOLOGICAL STRENGTHS

1) The present study utilized an adequate sample size (N = 82), which was an improvement on the sample sizes used in both phases one and two of the research with professional players. This larger sample allowed both for pairwise multiple comparison tests to be performed for a subgroup analysis as well as drawing more meaningful conclusions from the analysis.

2) The use of an appropriate control group comprising of non-contact sport (hockey) players, who were equivalent in terms of the variables of age, education, estimated IQ, thereby ensuring that any cognitive impairment noted could not be attributed to any one of these variables. The control group was also adequate for assessing the frequency of postconcussive symptomatology in players in that the group comprised a sports group, and not a "trauma" group, as per Satz's critique (see section 2.2.2.1, p. 26).

3) All groups (Total Rugby, Hockey Control, Rugby Forwards and Rugby Backs) were equivalent with regard to demographic data such as age, education, average grade and estimated IQ, thereby eliminating these potentially confounding variables. Exclusion criteria were also applied and strictly adhered to, in order to prevent any mitigating effects arising from further confounding variables. These included a reported history of substance abuse, a previous moderate to severe sport and non-sport related head injury and the reported presence of learning difficulties, a neurological disorder, and a psychiatric/psychological disorder.

4) A more sensitive method than that the one employed in phase two of the research on professional players for calculating premorbid IQ was utilized, based on the average of two WAIS - III subtests (Vocabulary and Picture Completion) and the National Adult Reading Test (NART). This was an improvement on phases one and two of the research that used only the calculated average of the SAWAIS Picture Completion and Comprehension subtests in order to calculate an estimated premorbid IQ. Furthermore, the addition of the National Adult Reading Test assisted in providing a more accurate measure of premorbid ability, as the test incorporates a word recognition component, a cognitive faculty found to show resilience in the face of brain damage (Nelson, 1992).

5) An extensive test battery was used, which employed a variety of tests sensitive to the effects of brain damage. This battery was a more refined and updated version of the battery used in phases one and two. In this respect, the SAWAIS Comprehension and Picture Completion subtests were replaced with the more recent WAIS - III Vocabulary and Picture Completion subtests, and the WAIS-III Letter-Number Sequencing Subtest and the Stroop Neuropsychological Screening Test (SNST) were added to the test battery as they provided measures of attention and concentration, and are therefore sensitive indicators of diffuse brain damage.

6) The combination of an analysis of neuropsychological test data with results of self-reported postconcussive symptomatology has been shown to be a powerful method of providing cross validation between objectively measured cognitive deficit and self-reported symptoms in order to determine whether the findings for each are supported by the other. The importance of focusing on both is that one might be more sensitive than the other, which tentatively appeared to be the case in this study.

7) The comparison of individual players to normative data and the calculation of individual levels of deficit allows for a more sensitive discrimination analysis than the comparison of group means, as it provides a picture of individual variation within groups and enriches interpretation of results. As noted earlier (section 3.6, p. 71), this is particularly significant given the recent call for the use of research methods that have relevance to clinical rather than statistical significance. Since a sole reliance on tests of statistical significance (e.g. statistical comparisons of means) in the understanding of neuropsychological data may actually confound conclusions drawn from neuropsychological research regarding brain-behaviour relations (for example, Donders, 2000; Zakzanis, 1998), the current methodology circumvents this problem of such statistical artifacts and provides a clinically relevant set of data i.e. the *number* of individuals with deficit resulting from analyses conducted for clinical purposes.

5.6.2. METHODOLOGICAL WEAKNESSES

1) This research is a cross-sectional study of potential brain trauma from cumulative mild head trauma in rugby. For this reason, it difficult to make attributions of causality as it possible that the outcome in this study may reflect pre-existing cognitive patterns and preselected differences between the groups i.e. it is impossible to rule out the possibility of personality variables being a factor that could account for the differences between groups. However, with regard to the postconcussive

symptoms, the results did provide support for previous research findings, and are also conceptually consistent with the picture of frontal-temporal pathology.

2) The schools selected for this study were all elite privileged schools where the academic standard is high and the average grade appears to fall in the above average range. This limits the generalizability of these findings as it is not possible to determine whether the same pattern of results would be evidenced in disadvantaged schools where intellectual functioning may be more varied and the standard of education lower due to a lack of resources.

5.7. RECOMMENDATIONS FOR FUTURE RESEARCH

1) A self-report questionnaire for administration to parents and schoolteachers expanding on any deficits recorded in players' self-report questionnaires, in order to investigate negative effects in everyday scholastic and occupational functioning, should be drawn up and used.

2) A longitudinal study is required in order to explore the long-term, and possibly permanent effects of mild head injuries. It would be useful to follow schoolboys into their university careers and beyond, in order to determine if any changes have occurred and to try to identify which players are at risk for a earlier onset of neuropsychological dysfunction

3) The use should be made of more sensitive test measures such as computerized neuropsychological testing e.g. Vienna Test System (Schuhfried, 1994), incorporating tests of reaction time. In addition, the testing of individuals should be done under more stressful task conditions such as hypoxic states or any conditions that serve to increase task challenge. In this way it may be possible to detect latent effects which may not be apparent under less challenging conditions.

4) Replication studies on more of the same types of schools (elite privileged schools) in order to determine whether these results are confirmed and strengthened.

5) Future studies should be performed at less advantaged schools aimed at assessing cognitive impairment as a result of cumulative mild head injury in order to determine the incidence rates of concussion as well to provide educational support in the management of on field injuries and strategies for making the game as safe as possible.

6) Studies should be conducted for investigating and assessing the efficacy of headgear and other protective measures in preventing brain trauma incurred as a result of cumulative head injury. This

would be for the purpose of determining whether such protective gear should be compulsory for rugby at a school level.

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Rhodes University - Department of Psychology

NEUROPSYCHOLOGICAL ASSESSMENT RESEARCH CONSENT FORM

As legal guardian of_____, I hereby give permission for him to undergo a neuropsychological assessment for research purposes.

I understand the following:

1. The assessment will be conducted by a Clinical Psychologist (or training Clinical Psychologist) especially schooled in the practical administration of the research questionnaire and tests;

2. The assessment takes 1 ½ to 2 hours, and takes the form of a series of questions and a variety of verbal and written intellectual tests which are not harmful, and which are usually quite enjoyable for the testee;

3. The testing will not interfere with the scholars' academic programme;

4. Individual results will be totally confidential and remain anonymous - they will not be made available to parents, the school or the scholar himself (except under the conditions referred to in paragraph 7 below);

5. The results will be in the form of group data which will allow the researchers to make a comparison between the scores of scholars who are intensively involved in the contact sport of rugby and those who are not;

6. As is regularly done in the dissemination of scientific knowledge, results of the group comparisons may be used for publication purposes at scientific conferences, in journals, books, and in the media;

7. In the unlikely event that the researchers discover a pattern of results which might give cause for medical or scholastic concern, they are willing to discuss this with the parent(s)/ guardian(s) of the scholar concerned - please indicate whether, in this event, you would like them to contact you by placing a ✔ in one of the boxes below:

Yes: I would like them to contact me		**No: I would not like them to contact me**	

NAME: _____

SIGNED:_____ DATE:_____

NEUROPSYCHOLOGICAL TESTING

ASSESSMENT SCHEDULE

Testee: _____ **Date:** _____

Time	Test
	1. Consent form
	2. Pre-assessment questionnaire
	3. Symptom checklist
	4. Finger Tapping Test
	5. Digit Symbol *including* INCIDENTAL RECALL
	6. Trail Making A and B
	7. Words-in-a-Minute
	8. "S" Words-in-a-Minute
	9. National Adult Reading Test (NART)
	1. Vocabulary
	11. Digit Symbol DELAYED RECALL
	BREAK
	12. Digit Span
	13. WMS - Designs - IMMEDIATE RECALL
	14. WMS - Paired Associate Learning - IMMEDIATE RECALL
	15. Stroop Neuropsychological Screening Test
	16. Letter-number Sequencing
	17. WMS - Designs - DELAYED RECALL
	19. WMS - Paired Associate Learning - DELAYED RECALL
	18. Picture Completion

FINGER TAPPING TEST

Testee's Name:_____

Requirements: stop watch

TIMED: Time to perform 20 taps (5 sets of 4 taps) per hand

Time Limit: No

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

"I would like you to repeat this test using your other hand. Practice that. Are you ready? Go..."

SCORE:

Preferred hand: (RH / LH) _____seconds

Non-preferred hand: _____seconds

Notes or Observations:

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

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.

AAM
AME .....

Datum
Date .....

SLEUTEL
KEY

1	2	3	4	5	6	7	8	9
—	⩘	⊐	L	∪	O	∧	X	=

VOORBEELD SAMPLE								TOETS BEGIN TEST BEGINS																			
?	1	3	1	2	4	3	5	3	1	2	1	3	2	1	4	2	3	5	2	3	1	4	6	3			
	5	4	2	7	6	3	5	7	2	8	5	4	6	3	7	2	8	1	9	5	8	4	7	3			
;	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 Number correct	120' 90'	Aantal half korrek Number half correct	120' 90'	TOTAAL TOTAL	120' 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

Requirements: test sheets (4 pages)
pencil
Stop watch

TIMED

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.

(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, without lifting your pencil, and do it as fast as you can.

Record time

TRAIL B:

SAMPLE - Draw a line to join the circles consecutively by alternating between 1 and A, as fast as you can.

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

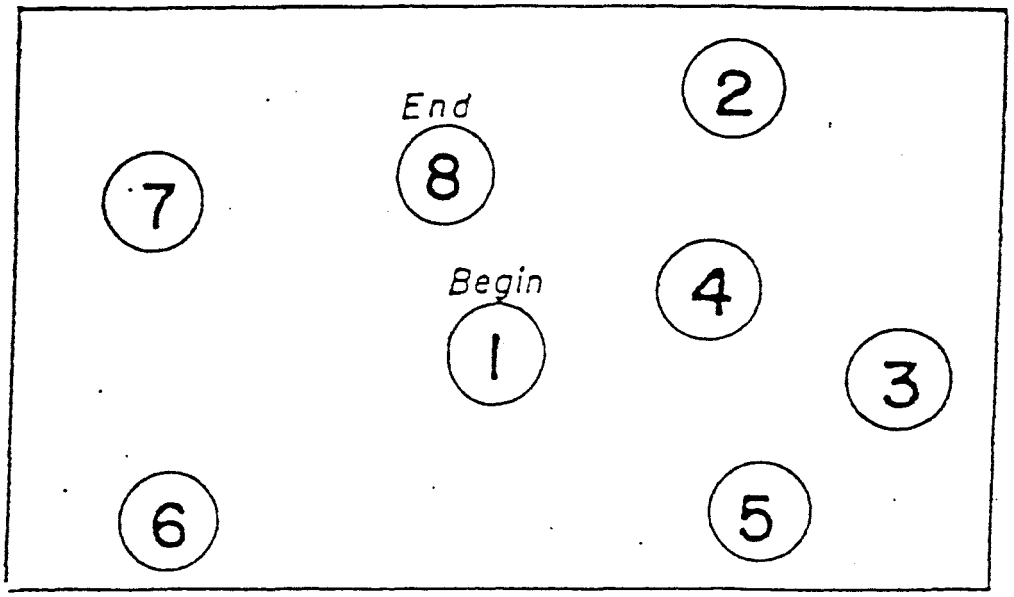
Draw a line to join the circles consecutively by alternating between 1 and A, as fast as you can.

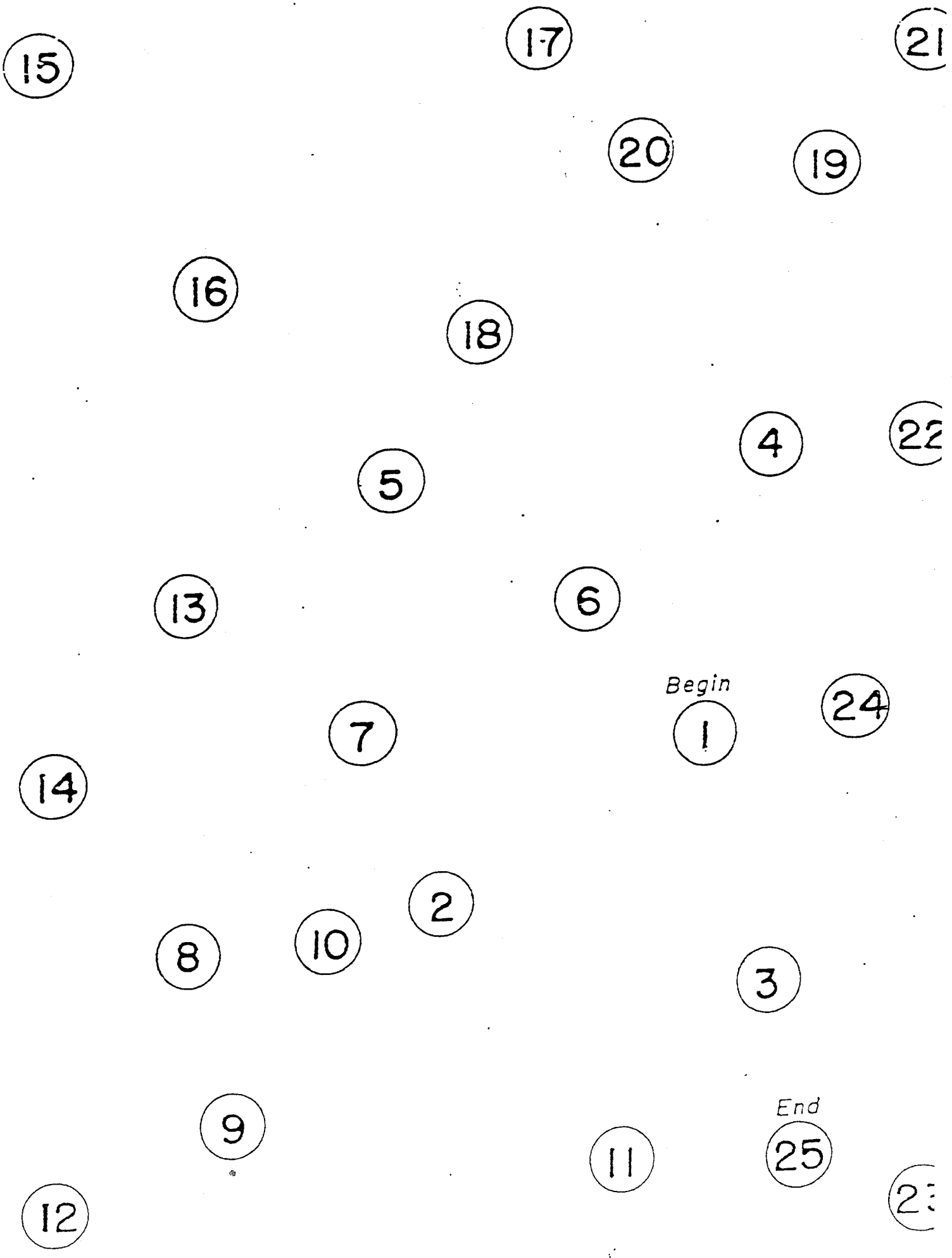
(Note: If subject makes mistake, **don't stop timing**: point out mistake and subject carries on).

TRAIL MAKING

Part A

SAMPLE

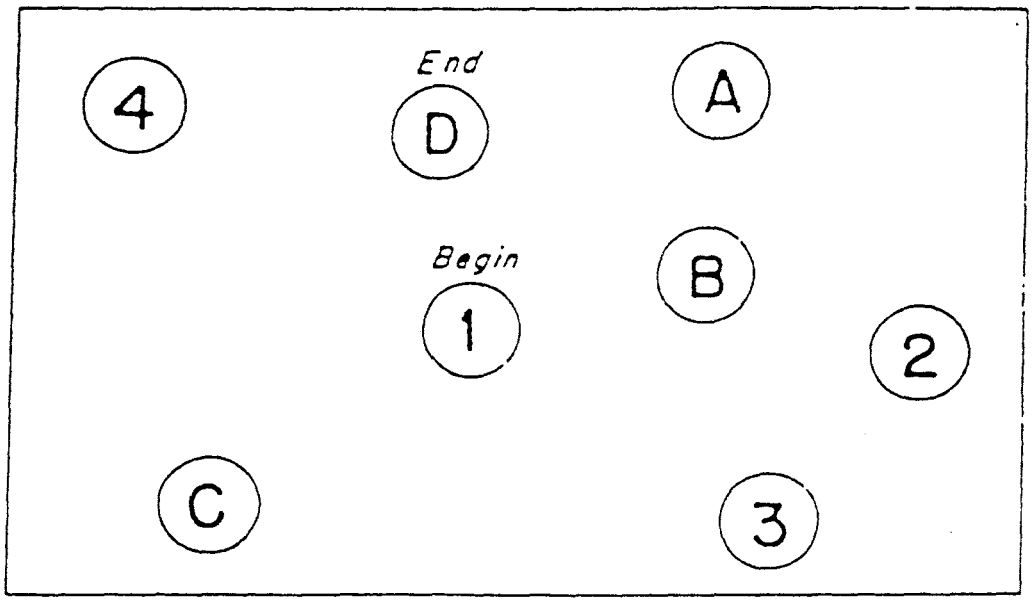


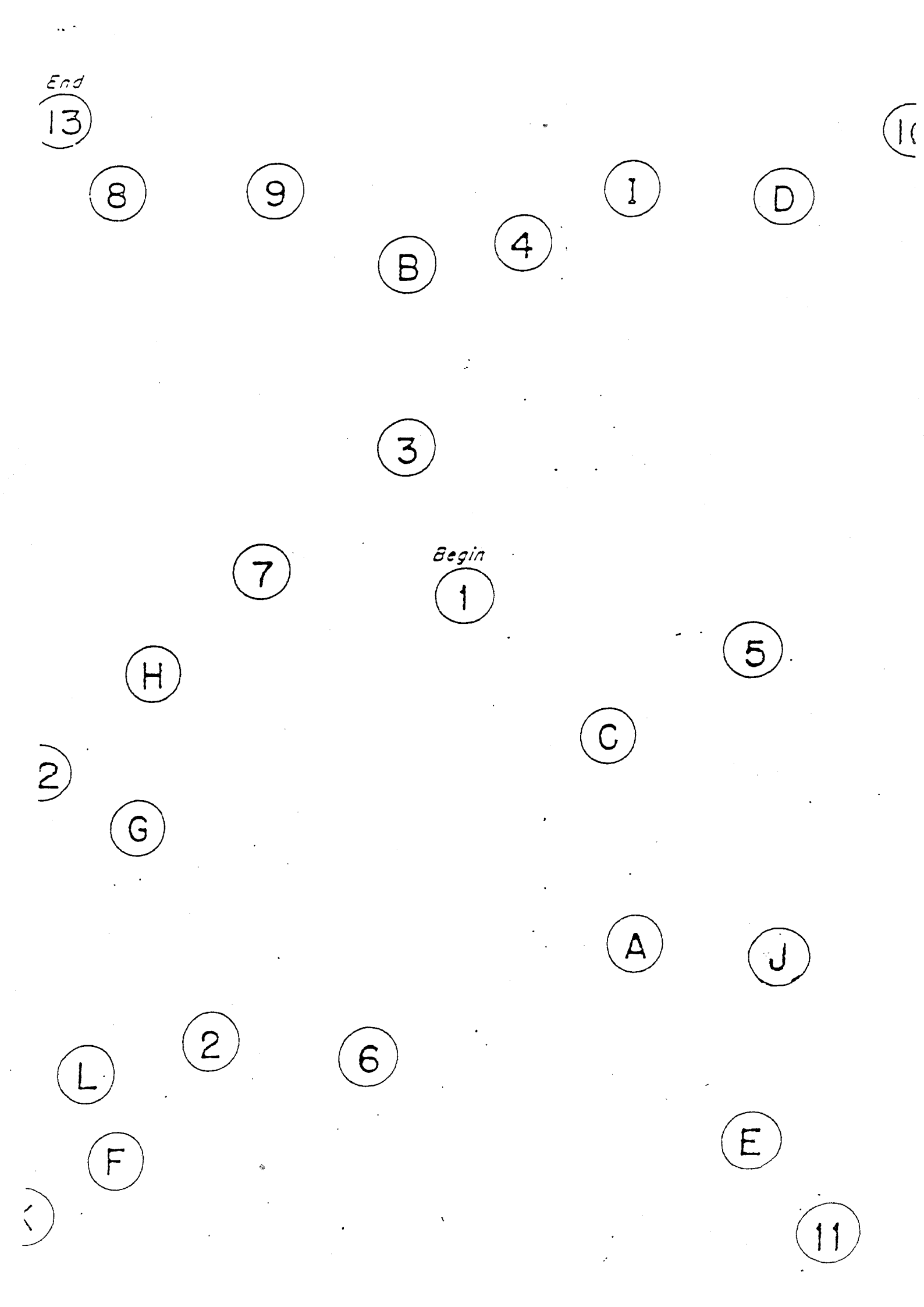

15
17
21
20
19
16
18
4
22
5
13
6
Begin
1
24
7
14
2
8
10
3
9
End
25
11
12
23

TRAIL MAKING

Part B

SAMPLE




End
13
8
9
I
D
4
B
3
7
Begin
1
5
H
C
2
G
A
J
2
6
L
F
E
11

WORDS-IN-A-MINUTE

Testee's Name:_____

Requirements: stop watch

TIMED

Time Limit: 1 minute

Instruction: *The subject can do this test in Afrikaans if that is their first language.*

"I would like you to say as many different words as you can think of. You must say the words as fast as you can and I will count them. You can say any words except proper nouns like a person's name or the name of a city. For example, you cannot say Mary or Jane or Grahamstown. You also cannot use different versions on one word. For example, if you say sing, you cannot also say singing, sings or sang. Counting or sentences are also not allowed. In other words I am asking you to say different, unconnected words such as, picture, carpet, music, dog, sky, building, grass and so on. Do you understand? Just keep going, I will tell you to stop after one minute. Go."

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

///// ///// ///// ///// ///// ///// ///// /////

///// ///// ///// ///// ///// ///// ///// /////

///// ///// ///// ///// ///// ///// ///// /////

SCORE:_____

Notes or Observations:

"S" WORDS-IN-A-MINUTE

Testee's Name: _____

Requirements: stop watch

TIMED

Time Limit: 1 minute

Instruction: *The subject can do this test in Afrikaans if that is their first language.*

"Now I would like you to say as many words as you can think of that begin with the letter "S". You must say the words as fast as you can and I will count them. Remember that you can say any words except proper nouns like a person's name or the name of a city. For example, you cannot say Susan or Sarah or Scotburgh. You also cannot use different versions on one word. For example, if you say sing, you cannot also say singing, sings or sang. Counting or sentences are also not allowed. In other words I am asking you to say different, unconnected words all starting with the letter "S". Do you understand? Just keep going, I will tell you to stop after one minute. Go."

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

///// ///// ///// ///// ///// ///// ///// /////

///// ///// ///// ///// ///// ///// ///// /////

///// ///// ///// ///// ///// ///// ///// /////

SCORE:

Notes or Observations:

NART Testee's Name: _____

Requirements: Word Card / Pencil

Instructions: "I want you to read slowly down this list of words starting here." *Indicate* ACHE. "After each word please wait until I say **'next'** before reading the next word. I must warn you that there are many words that you probably won't recognise, in fact most people don't know them, so just have a quess at these, O.K.? Go ahead."

If the participant fails to wait, repeat this instruction. The participant should be encouraged to attempt every word and instructed to guess where necessary. Reinforce all responses, for example "That's fine, good". *The participant may change a response but if more than one version is given, they must decide which is their final choice. Record errors on answer sheet.*

	Pronounciation	**Error**
CHORD	körd	_____
ACHE	āk	_____
DEPOT	dep'o	_____
AISLE	īl	_____
BOUQUET	bo͞ok'a, bo͞okā', bōkā'	_____
PSALM	säm	_____
CAPON	kā'pn	_____
DENY	di-nī	_____
NAUSEA	nö'si-e, nö'zhe	_____
DEBT	det	_____
COURTEOUS	kûrt'yes	_____
RAREFY	rār'-i-fī	_____
EQUIVOCAL	i-kwiv'e-kl	_____
NAÏVE	nä-ēv	_____
CATACOMB	kat'e-ko͞om	_____
GOALED	jāld	_____
THYME	tīm	_____
HEIR	ār	_____
RADIX	ra'diks	_____
ASSIGNATE	as'-ig-nāt	_____
HIATUS	hī-ā'tes	_____
SUBTLE	sut'l	_____
PROCREATE	prō'kri-āt	_____
GIST	jist	_____
GOUGE	gowj	_____

	Pronounciation	**Error**
SUPERFLUOUS	so͞o-pûr'flo͝os-es sū-pûr'flo͝o-es	_____
SIMILE	sim'ili	_____
BANAL	ben-al'	_____
QUADRUPED	kwod'ro͝o-ped	_____
CELLIST	chel'ist	_____
FACADE	fa-säd'	_____
ZEALOT	zel'et	_____
DRACHM	dram	_____
AEON	ē'on	_____
PLACEBO	ple-sē'bō	_____
ABSTEMIOUS	ab-stē'mi'es	_____
DÉTENTE	dā-tât (Fr.)	_____
IDYLL	id'il, id'el	_____
PUERPERAL	pū-ûr'per-el	_____
AVER	e-vûr'	_____
GAUCHE	gō sh	_____
TOPIARY	tō'pi-e-ri	_____
LEVIATHAN	le-vī'e-then	_____
BEATIFY	bi-at'i-fi	_____
PRELATE	prel'it	_____
SIDEREAL	sī-dē'ri-el	_____
DEMESNE	di-mān', di-men'	_____
SYNCOPE	sing'ke-pē	_____
LABILE	lā'bīl	_____
CAMPANILE	kam-pan-ē'lā, kam-pan-ē'lē	_____

TOTAL ERROR SCORE _____

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		

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.)	

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: _____

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

NAAM
NAME .....

Datum
Date .....

SLEUTEL
KEY

1	2	3	4	5	6	7	8	9

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: _____

Digits Backwards: _____

Digits Difference: _____ (Forwards minus Backwards)

WMS : VISUAL REPRODUCTION - *IMMEDIATE RECALL*

Testee's Name:_____

Requirements: 3 cards
stop watch / count in head
pencil
1 piece A4 paper

TIMED viewing

Time Limit: 10" viewing per card

Instructions: *All drawings to be drawn on one piece of A4 paper.*
Cards 1 and 2: "I am going to show you a drawing. You will have just 10 seconds to look at it. Then, I shall take it away and let you draw it from memory. Don't begin to draw until I say "Go". Ready? *Expose card: 10 seconds.* Go."

Card 3: "Here is one that is a little harder. This card has 2 designs on it. I want you to look at them both carefully - again you will have only 10 seconds to look at the card, then I shall take it away and let you make both drawings; the one on the left side - here (*pointing to space in which subject is to make drawing*) and the right one - here (*pointing*). Ready? *Expose card: 10 seconds.* Go."

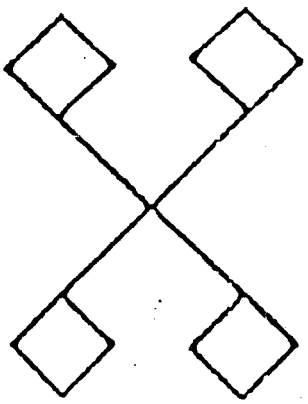
SCORE:

Card 1: _____

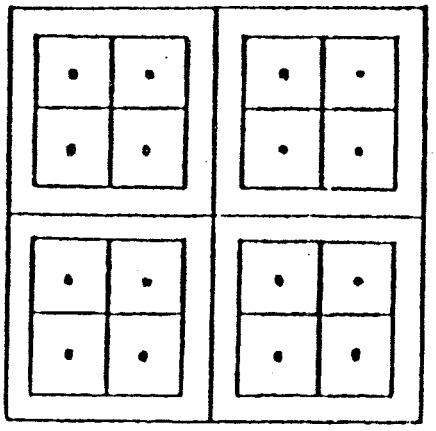
Card 2: _____

Card 3: _____

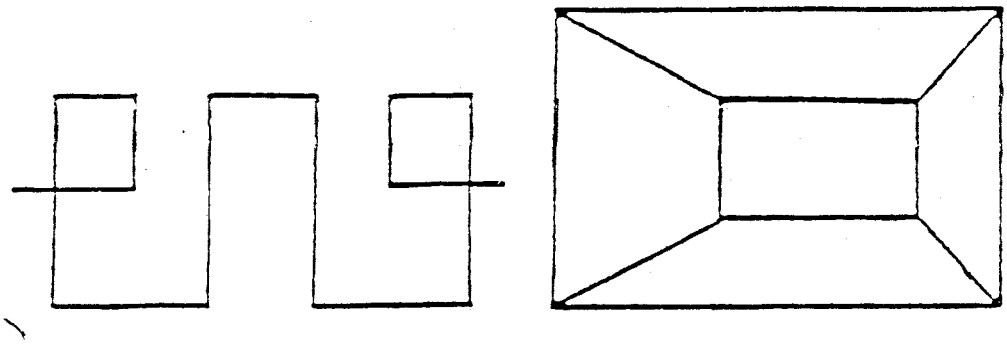
Notes or Observations:



Card A
W-M-S I



Card B



Card C
W-M-S I

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:

First Recall		Second Recall		Third Recall	
TOTAL	____ ____	TOTAL	____ ____	TOTAL	____ ____

Easy:			Hard:		
	1.	____		1.	____
	2.	____		2.	____
	3.	____		3.	____
	A Total	____		B Total	____

Score: A/2 + B = ____

Read 1 pair every 2 seconds.

First Presentation			Second Presentation			Third Presentation		
Metal	-	Iron	Rose	-	Flower	Baby	-	Cries
Baby	-	Cries	Obey	-	Inch	Obey	-	Inch
Crush	-	Dark	North	-	South	North	-	South
North	-	South	Cabbage	-	Pen	School	-	Grocery
School	-	Grocery	Up	-	Down	Rose	-	Flower
Rose	-	Flower	Fruit	-	Apple	Cabbage	-	Pen
Up	-	Down	School	-	Grocery	Up	-	Down
Obey	-	Inch	Metal	-	Iron	Fruit	-	Apple
Fruit	-	Apple	Crush	-	Dark	Crush	-	Dark
Cabbage	-	Pen	Baby	-	Cries	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	Second Recall	Easy	Hard	Third Recall	Easy	Hard
North	____		Cabbage		____	Obey		____
Fruit	____		Baby	____		Fruit	____	
Obey		____	Metal	____		Baby	____	
Rose	____		School		____	Metal	____	
Baby	____		Up	____		Crush		____
Up	____		Rose	____		School		____
Cabbage		____	Obey		____	Rose	____	
Metal	____		Fruit	____		North	____	
School		____	Crush		____	Cabbage		____
Crush		____	North	____		Up		____
TOTAL	____	____	**TOTAL**	____	____	**TOTAL**	____	____

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

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

Score: A/2 + B = ____

STROOP NEUROPSYCHOLOGICAL SCREENING TEST

Testee's Name: _____

Requirements:

- Card with Pictures
- Form C Stimulus Sheet
- Form C-W Stimulus Sheet
- Stopwatch
- Form C Stimulus Record Form
- Form C-W Stimulus Record Form
- Pencil

Time Limit: 120'' (2 mins) per task

Instructions: **Screening for Colour Naming:** Show subject card with pictures. Then say: "Can you tell me what each of these colours are?" If the subject says BROWN where TAN is indicated, explain to subject that for the purposes of this test, the colour they have identified as BROWN will be called TAN.

Colour Task: "On this page are some words. I would like you to read these words aloud as quickly as you can, starting at the top of this first column. When you finish this column, go to the top of the next column and so on *(point to the top of the columns and indicate that the subject should read all the columns in the same manner).* Read the words aloud as quickly and as accurately as you can. If you make a mistake, just correct yourself and keep on going. Ready? Begin."

Colour-Word Task: "Here is a page with more words on it. This time, I would like you to name aloud the colour of the ink - RED, BLUE, GREEN, or TAN *(point to words printed in these colours)* - in which the word is printed. Go as quickly as you can, going down the columns just as you did before. For this first one you would say "RED". Understand? If you make a mistake, just correct yourself and keep on going. Name the colour of the ink as quickly and as accurately as you can. Ready? Begin."

Remember: Subjects may not cover up a part of any of the words in an attempt to reduce the interference effect, neither can they pick up the stimulus sheet in an attempt to facilitate responding but must leave it on the flat surface.

SCORING (for both tasks):

Record correct responses by making a check mark next to the item as shown on the Record Form. Record incorrect responses by entering an X next to the item. If the subject gives an incorrect response and corrects it spontaneously, mark a C next to that item.

Remember: If subjects give BROWN as a response instead of TAN, this will still be considered a correct answer.

SCORE:	Colour Task	Colour-Word Task
Number of responses	_____	_____
Incorrect Responses	_____	_____
Score	_____	_____
Percentile		_____

Form C Stimulus Sheet

BLUE	RED	TAN	RED
GREEN	GREEN	RED	TAN
TAN	TAN	TAN	RED
RED	BLUE	BLUE	TAN
GREEN	GREEN	TAN	BLUE
BLUE	BLUE	RED	GREEN
GREEN	TAN	GREEN	RED
BLUE	GREEN	RED	BLUE
RED	TAN	BLUE	RED
BLUE	BLUE	TAN	TAN
TAN	GREEN	RED	GREEN
RED	BLUE	GREEN	TAN
TAN	GREEN	RED	BLUE
GREEN	RED	TAN	RED
BLUE	BLUE	BLUE	BLUE
TAN	GREEN	TAN	RED
GREEN	TAN	GREEN	GREEN
RED	RED	TAN	RED
TAN	TAN	BLUE	BLUE
RED	GREEN	TAN	TAN
TAN	TAN	BLUE	BLUE
RED	RED	GREEN	GREEN
GREEN	BLUE	RED	BLUE
RED	RED	GREEN	RED
TAN	GREEN	TAN	BLUE
BLUE	RED	RED	TAN
GREEN	TAN	GREEN	BLUE
TAN	BLUE	BLUE	GREEN

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Form C-W Stimulus Sheet

BLUE	GREEN	RED	GREEN
GREEN	BLUE	GREEN	TAN
RED	RED	BLUE	RED
TAN	BLUE	TAN	TAN
GREEN	TAN	RED	BLUE
BLUE	RED	TAN	TAN
RED	GREEN	BLUE	GREEN
TAN	TAN	TAN	RED
RED	GREEN	RED	GREEN
BLUE	BLUE	BLUE	RED
RED	RED	RED	BLUE
TAN	TAN	TAN	GREEN
BLUE	GREEN	BLUE	TAN
TAN	RED	GREEN	BLUE
RED	BLUE	TAN	GREEN
BLUE	GREEN	BLUE	RED
GREEN	RED	TAN	GREEN
TAN	GREEN	BLUE	TAN
GREEN	BLUE	RED	GREEN
TAN	TAN	GREEN	BLUE
RED	GREEN	BLUE	TAN
BLUE	RED	GREEN	BLUE
RED	TAN	BLUE	GREEN
TAN	BLUE	GREEN	RED
RED	TAN	RED	BLUE
TAN	RED	GREEN	GREEN
GREEN	TAN	TAN	RED
TAN	GREEN	RED	BLUE

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Form C Responses–Color Task

1 BLUE_____	29 RED_____	57 TAN_____	85 RED_____
2 GREEN_____	30 GREEN_____	58 RED_____	86 TAN_____
3 TAN_____	31 TAN_____	59 TAN_____	87 RED_____
4 RED_____	32 BLUE_____	60 BLUE_____	88 TAN_____
5 GREEN_____	33 GREEN_____	61 TAN_____	89 BLUE_____
6 BLUE_____	34 BLUE_____	62 RED_____	90 GREEN_____
7 GREEN_____	35 TAN_____	63 GREEN_____	91 RED_____
8 BLUE_____	36 GREEN_____	64 RED_____	92 BLUE_____
9 RED_____	37 TAN_____	65 BLUE_____	93 RED_____
10 BLUE_____	38 BLUE_____	66 TAN_____	94 TAN_____
11 TAN_____	39 GREEN_____	67 RED_____	95 GREEN_____
12 RED_____	40 BLUE_____	68 GREEN_____	96 TAN_____
13 TAN_____	41 GREEN_____	69 RED_____	97 BLUE_____
14 GREEN_____	42 RED_____	70 TAN_____	98 RED_____
15 BLUE_____	43 BLUE_____	71 BLUE_____	99 BLUE_____
16 TAN_____	44 GREEN_____	72 TAN_____	100 RED_____
17 GREEN_____	45 TAN_____	73 GREEN_____	101 GREEN_____
18 RED_____	46 RED_____	74 TAN_____	102 RED_____
19 TAN_____	47 TAN_____	75 BLUE_____	103 BLUE_____
20 RED_____	48 GREEN_____	76 TAN_____	104 TAN_____
21 TAN_____	49 TAN_____	77 BLUE_____	105 BLUE_____
22 RED_____	50 RED_____	78 GREEN_____	106 GREEN_____
23 GREEN_____	51 BLUE_____	79 RED_____	107 BLUE_____
24 RED_____	52 RED_____	80 GREEN_____	108 RED_____
25 TAN_____	53 GREEN_____	81 TAN_____	109 BLUE_____
26 BLUE_____	54 RED_____	82 RED_____	110 TAN_____
27 GREEN_____	55 TAN_____	83 GREEN_____	111 BLUE_____
28 TAN_____	56 BLUE_____	84 BLUE_____	112 GREEN_____

Form C-W Responses – Color-Word Task

1 RED_____	29 BLUE_____	57 BLUE_____	85 TAN_____
2 BLUE_____	30 TAN_____	58 TAN_____	86 RED_____
3 GREEN_____	31 GREEN_____	59 RED_____	87 GREEN_____
4 BLUE_____	32 RED_____	60 GREEN_____	88 BLUE_____
5 RED_____	33 BLUE_____	61 TAN_____	89 TAN_____
6 TAN_____	34 GREEN_____	62 RED_____	90 GREEN_____
7 BLUE_____	35 BLUE_____	63 GREEN_____	91 RED_____
8 RED_____	36 GREEN_____	64 BLUE_____	92 TAN_____
9 TAN_____	37 RED_____	65 GREEN_____	93 BLUE_____
10 GREEN_____	38 TAN_____	66 TAN_____	94 GREEN_____
11 BLUE_____	39 BLUE_____	67 BLUE_____	95 RED_____
12 RED_____	40 RED_____	68 GREEN_____	96 TAN_____
13 TAN_____	41 BLUE_____	69 RED_____	97 RED_____
14 BLUE_____	42 TAN_____	70 BLUE_____	98 GREEN_____
15 GREEN_____	43 RED_____	71 RED_____	99 RED_____
16 RED_____	44 TAN_____	72 GREEN_____	100 BLUE_____
17 TAN_____	45 BLUE_____	73 BLUE_____	101 RED_____
18 GREEN_____	46 RED_____	74 TAN_____	102 BLUE_____
19 BLUE_____	47 GREEN_____	75 GREEN_____	103 TAN_____
20 RED_____	48 BLUE_____	76 BLUE_____	104 GREEN_____
21 TAN_____	49 TAN_____	77 RED_____	105 RED_____
22 GREEN_____	50 GREEN_____	78 TAN_____	106 TAN_____
23 BLUE_____	51 RED_____	79 GREEN_____	107 BLUE_____
24 GREEN_____	52 TAN_____	80 RED_____	108 TAN_____
25 TAN_____	53 GREEN_____	81 TAN_____	109 RED_____
26 BLUE_____	54 TAN_____	82 BLUE_____	110 BLUE_____
27 TAN_____	55 BLUE_____	83 GREEN_____	111 GREEN_____
28 RED_____	56 RED_____	84 BLUE_____	112 TAN_____

LETTER-NUMBER SEQUENCING

Testee's Name: _____

Requirements: Record Form
pencil

Not timed

Instructions: **Practice Items:** "I am going to say a group of numbers and letters. After I say them, I want you to tell me the numbers first, in order, starting with the lowest number. Then tell me the letters in alphabetical order. For example, if I say B - 7, your answer should be 7 - B. The number goes first, then the letter. If I say 9 - C - 3, then your answer should be 3 - 9 - C, the numbers in order first, then the letter in alphabetical order. Let's practice."

Administer all practice trials. For each Practice Item and item trial, say each combination at a rate of one number or letter per second.

6 - F	**(6 - F)**
G - 4	**(4 - G)**
3 - W - 5	**(3 - 5 - W)**
T - 7 - L	**(7 - L - T)**
1 - J - A	**(1 - A - J)**

If the subject makes an error on any Practice Item, correct them and repeat instructions as necessary. Even if the subject fails all Practice Items, continue with the test.

P.T.O for Item Trials.

WMS VISUAL REPRODUCTION *DELAYED RECALL*

Testee's Name: _____

Requirements: 3 cards [not shown to P]
pencil
1 piece A4 paper

Not timed

Instructions: *All drawings to be drawn on one piece of A4 paper.*
"Earlier you memorised designs off cards presented to you for 10 seconds. I would like to see how many of those designs you can remember and draw now."

SCORE:

Card 1: _____

Card 2: _____

Card 3: _____

Notes or Observations:

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 = ____

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 From 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)	