THE SCRUM-DOWN ON BRAIN DAMAGE EFFECTS OF CUMULATIVE MILD HEAD INJURY IN RUGBY

A COMPARISON OF GROUP MEAN SCORES BETWEEN NATIONAL RUGBY PLAYERS AND NON-CONTACT SPORT CONTROLS

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ABSTRACT

The present study comprises the second phase of a larger and ongoing research study investigating the brain damage effects of cumulative mild head injury in rugby. The purpose of this study was to determine whether cumulative mild head injury sustained in the game of rugby would cause brain injury as evidenced by impaired performance on sensitive neuropsychological tests. Participants were Springbok professional rugby players (n = 26), Under 21 rugby players (n = 19), and a non-contact sport control of national hockey players (n = 21). Comparisons of performance were carried out across a spectrum of neuropsychological tests for the three rugby groups (Total Rugby, Springbok Rugby, and Under 21 Rugby) versus the performance of the non-contact sport control group (Hockey Control), as well as comparisons of performance for the subgroups of Rugby Forwards versus Rugby Backs. Comparisons revealed a consistent pattern of poorer performance across all rugby groups relative to the performance of the controls on tests highly sensitive to the effects of diffuse brain damage. Within rugby group comparisons (Forwards versus Backs) showed significantly poorer performance for Total Rugby Forwards and Springbok Rugby Forwards relative to the performance of the respective Total Rugby Backs and Springbok Rugby Backs on sensitive, as well as on somewhat less sensitive, neuropsychological tests. The performance of Under 21 Rugby Forwards relative to Under 21 Rugby Backs demonstrated similar trends. Brain reserve capacity theory was used as a conceptual basis for discussing the implications of these findings.

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CHAPTER ONE: INTRODUCTION

1.1. RESEARCH CONTEXT

This project forms part of a larger and ongoing research study investigating the brain damage effects of cumulative mild concussive and sub-concussive head injury in rugby. The study was initiated in 1997 by Rhodes University in collaboration with the South African Rugby and Football Union (SARFU) and the South African Sports Science Institute in Cape Town. The initial phase of the research compared the cognitive performance of Springbok professional rugby players on a wide variety of neuropsychological measures with the cognitive performance of a matched non-contact sport control group consisting of Protea professional cricket players. The data were analysed in three separate research projects (Dickinson, 1998; Ancer, 1999; and Reid, 1998).

The present study comprises the second phase of the larger research study and expands on the first phase by comparing the cognitive performance of Springbok professional rugby players and Under 21 national rugby players with the performance of a matched non-contact sport control group consisting of national hockey players. Utilising the combined data from both the first and second phases of the study, three further research projects emerged, including the present study (Bold, 1999; Border, 1999; and Finkelstein, 1999).

1.2. RESEARCH QUESTION

Increasing attention is being paid to mild closed head injury, partly due to the high incidence with which it occurs and partly due to the relatively high morbidity associated with the condition (Dikmen, McLean, & Tempkin, 1986; Rimel, Giordani, Barth, Boll, & Jane, 1981). However, most of the studies have tended to focus on the effects of a single uncomplicated mild head injury and there is a paucity of studies examining the neuropsychological sequelae of *cumulative* mild concussive and sub-concussive head injury.

Athletes participating in contact sports (e.g. boxing, soccer, and rugby) are at particular risk of sustaining repeated mild closed head injuries. However, until recently, mild head injuries in sport were regarded as inconsequential and the possible cumulative effects of concussive and sub-concussive mild head injuries were not viewed as particularly relevant (Anderson, 1996;

Barth et al., 1989). Around the world, very little research has focused specifically on the permanent effects of mild head injury in rugby. In Australia, a few studies have been conducted (Hinton-Bayre, Geffen, & McFarland, 1997; Maddocks & Saling, 1991), and some studies have been initiated in South Africa (Shuttleworth-Jordan, Balarin, & Puchert, 1993 and the present research). In order to further the limited research in this area, the present study aims to investigate the neuropsychological sequelae of the likely history of cumulative mild closed head injury in a specific group of athletes involved in a contact sport, namely rugby players.

Broadly, the research question being addressed is whether cumulative mild head injury sustained in the game of rugby will cause brain injury as evidenced by impaired performance on sensitive neuropsychological tests. The question will be addressed by making group comparisons of the performance of top-level rugby players with the performance of non-contact sport controls across a spectrum of neuropsychological tests known to be sensitive to the effects of closed head injury. It is hypothesised that the performance of the rugby playing group, and the subgroup of rugby forward players, is likely to be impaired on certain cognitive tasks known to be sensitive to diffuse brain damage relative to the respective non-contact sport controls, and the subgroup of rugby back-line players. Whereas head injury research has typically been empirically based, the present study provides a theoretical context in the form of Brain Reserve Capacity (BRC) theory (Satz, 1993) in order to elucidate the findings.

CHAPTER TWO: LITERATURE REVIEW

2.1. UNDERSTANDING MILD CLOSED HEAD INJURY – CLASSIFICATION, INCIDENCE, AND PATHOPHYSIOLOGY

2.1.1. TYPES OF HEAD INJURY (OPEN/PENETRATING VERSUS CLOSED HEAD INJURY)

The two types of commonly occurring head injury are open/penetrating head injury and closed head injury. The majority of head injuries are closed in that the skull remains intact and the brain is not exposed. In the case of open head injuries, the skull is penetrated, for instance, by missiles or other penetrating objects (Lezak, 1995). Open and closed head injuries differ in terms of the nature of the injury and also the pathophysiological processes that occur as a result of damage to the brain. *Open/penetrating head injuries* occur due to damage from puncture wounds, missile fragments, and low-velocity bullets which tend to produce "clean" wounds in that significant tissue damage tends to be concentrated in the path of the penetrating object (Levin, Benton, & Grossman, 1982). In cases of open head injuries, behavioural changes and specific cognitive deficits can usually be traced to the site of the lesion (although some patients with such injuries may show impairments more typical of diffuse brain damage). In addition, the severity of injury tends to be determined by the depth of penetration and the loss of brain tissue in that area (Levin et al., 1982).

The focus of the present study is on *closed head injuries*, which involve blunt trauma to the head (Richardson, 1990). With this type of head injury, brain damage usually occurs in two phases - the primary and the secondary injuries. The primary injury involves the damage that occurs at the time of impact. The blow at the point of impact is called coup, while contrecoup lesion refers to a contusion in the area opposite the blow. Coup and contrecoup lesions cause discrete impairment of those functions mediated by the cortex at the site of the lesion. Another type of brain damage that occurs in closed head injury results from the combination of translatory force and rotational acceleration of the brain within the bony structure of the skull. This can lead to shearing (discussed in more detail in section 2.1.6). The secondary injury comprises the effects of the physiological processes set in motion by the primary injury. Among the most well-known pathophysiological processes which cause secondary damage are haemorrhages and their sequelae (tissue swelling, and alterations in blood volume and blood flow) (Lezak, 1995).

Diffuse brain injury is associated with widespread disruption of neurological function, and impairments of memory functions, attention, concentration, and mental slowing tend to be associated with such injury. Diffuse brain damage in closed head injury occurs as a result of acceleration/deceleration forces acting on the brain as a whole (Galbraith, 1986). Rotational acceleration is viewed as the primary injury mechanism for diffuse brain injuries (Bruno, Gennarelli, & Torg, 1987). In this regard, Binder (1986) maintains that direct impact to the head is not needed to cause concussion and rotational injuries are in fact sufficient. While the extent of the diffuse damage is dependent on the severity of the trauma, it is known that structural brain damage occurs even with those injuries associated with the briefest period of unconsciousness (Oppenheimer, 1968). Furthermore, repeated mild head injuries have been demonstrated to have a cumulative effect (Gronwall & Wrightson, 1975; Casson et al., 1984).

2.1.2. CLASSIFICATION AND INDICATORS OF SEVERITY OF HEAD INJURY

Severity of head injury is regarded as a defining factor and a predictor of outcome when examining head trauma (Anderson, 1996). Head injury is conventionally understood as a continuum, ranging from mild (minor blows resulting in no overt symptoms) through moderate to severe (causing prolonged coma or death) head injury (Anderson, 1996; Boll, 1983; De Villiers, 1987; Plum & Posner, 1982 in Warren & Bailes, 1998). The severity of a head injury is usually classified by the following symptoms: alterations in level of consciousness; duration of unconsciousness; and changes in orientation and memory (Satz et al., 1997).

The *Glasgow Coma Scale* (GCS)[1] is the most frequently used instrument to measure severity of injury in the acute stage. This scale classifies injuries in three groups of severity (mild, moderate, severe) using a scale of 0-15, assessing verbal, motor, and ocular responses to simple stimuli. Severity is classified as severe (GCS score of 3-8), moderate (GCS score of 9-12), and mild (GCS score of 13-15). However, although the GCS is efficient in evaluating 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). Controversy surrounds the use of *loss of consciousness* (LOC) as an indication of degree of injury. While mild head injury is typically defined with LOC of less than 30 minutes (Rimel et al., 1981), it has been argued that injury can occur without the LOC (Cantu, 1996; Rutherford, Merrett, & MacDonald, 1977). The third commonly used indicator of degree of injury is *post-traumatic amnesia* (PTA) which is a period of confusion,

[1] GCS is a clinical scale devised by Teasdale & Jennett (1974) to assess the depth and duration of impaired consciousness and coma. The reader is referred to the article written by these authors for further information concerning the scale and its application.

disorientation, and inability to recall events, sequence time, or learn new information (McAllister, 1992 in Busch & Alpern, 1998). However, the validity of short PTA (in the case of mild head injury) is problematic, because assessment is difficult and estimation of the duration of PTA depends on the subjective judgement of the doctor taking the patient's history (Rutherford et al., 1977). Furthermore, estimates of PTA tend to be based on self-report of symptoms, which may be unreliable, particularly if reported after a lapse of time (Satz et al., 1997).

2.1.3. CLASSIFICATION AND DEFINITION OF MILD HEAD INJURY

Whereas the classification of *severe* head injury is relatively clear, the classification and definition of *mild* head injury both for research purposes and for clinical treatment remains difficult/ambiguous (Binder, 1986; Bohnen & Jolles, 1992; Satz et al., 1997; Williams, Levin, & Eisenberg, 1990). Significant diversity exists in the literature with regard to criteria for inclusions of participants in research studies and definitions of concussion and mild head injury. For example, studies conducted by Dikmen et al., 1986; McLean, Temkin, Dikmen, & Wyler, 1983; Rimel et al., 1981; and Rutherford et al., 1977 have all differed on measures such as length of PTA, structural skull damage, and GCS scores. It is thought that the inconsistencies in the extent of neurobehavioural recovery reported in various studies may be accounted for by this failure to adopt uniform inclusion criteria for mild closed head injury across research studies (Williams et al., 1990). This is discussed in greater detail in section 2.2.2.3.

However, since the publication of Binder's earlier review (Binder, 1986), a more general agreement as to the definition of mild head trauma has been reached (Binder, 1997). Research on mild head trauma has frequently employed the GCS (Teasdale & Jennett, 1974) scores of 13 to 15. In addition, other consensual criteria have been utilised, including length of LOC (less than 30 minutes), brief or no hospitalisation, normal neuroimaging data, and the absence of acute neurological abnormalities (e.g. hemiparesis, aphasia) (Binder, 1997). Although PTA has demonstrated predictive utility in research settings, Binder (1997) recommends that the clinician should use this measure with great caution, as it is not prospectively assessed and retrospective assessment of PTA length tends to be unreliable.

"Mild" or "minor" head injury is generally defined as a relatively brief period of unconsciousness or PTA, an absence of structural damage to the skull or brain, and GCS of greater than 13 on admission (Binder, 1986). More recently, as recommended by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (Satz et al., 1997), mild head injury is

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 of 13-15 following the loss of consciousness; (b) any loss of memory for events immediately preceding or proceeding the accident with a PTA for 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 (e.g. double vision, loss of balance, taste, or smell) that may or may not be transient. Because this definition encompasses a broad range of injury severity, it increases the prevalence rate of mild head injury and also encourages investigation of patients without hospitalisation stays (Satz et al., 1997). However, this definition is also criticised. "Although this approach has much clinical appeal, it also suffers from the use of arbitrary and a priori cut points to designate grades of severity along the distribution of head injury that lack empirical verification at this time" (Satz et al., 1997, p. 128). Further, according to Anderson (1996), the validity and reliability of the GCS along with the period of PTA in assessing mild head injury, remains questionable.

Alexander (1995) and Evans (1992) have used similar criteria to define mild head injury as the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (Satz et al., 1997). On the basis of the criteria recommended by Alexander (1995), Evans (1992), and Satz (1997), the present study employed the following characteristics to define mild head injury: LOC of less than 30 minutes; PTA less than 24 hours; and no evidence of physical damage.

2.1.4. CLASSIFICATION AND DEFINITION OF CONCUSSION

As with mild head injury, assessment and definition of concussion, as well as classification of severity of concussion remains difficult and controversial (Anderson, 1996; Cantu, 1986, 1996, 1998a; McCrory, 1997; Nelson, Jane, & Gieck, 1984). One definition of concussion that has gained significant acceptance by researchers in the field and is frequently used as a working definition, is that description proposed by the Committee on Head Injury Nomenclature of the Congress of Neurological Surgeons (Cantu, 1995; McCrory, 1997). This Committee defines concussion as "a clinical syndrome characterized by immediate and transient posttraumatic impairment of neural function, such as alteration of consciousness, disturbance of vision, equilibrium, etc, due to brain stem involvement" (1966, in Cantu, 1986, p. 76). It is generally recognised that such a syndrome may or may not include loss of consciousness (Sturmi, Smith, & Lombardo, 1998). Another commonly cited definition of concussion is provided by Rutherford (1989, p. 217) who defines concussion as "an acceleration/deceleration injury to the head almost always associated with a period of amnesia, and followed by a characteristic group of symptoms such as headache, poor memory, and vertigo".

Ommaya & Gennarelli (1974) developed an hypothesis which defined cerebral concussion as "a graded set of clinical syndromes following head injury wherein increasing severity of disturbance in level and content of consciousness is caused by mechanically induced strains affecting the brain in a centripetal sequence of disruptive effect on function and structure. The effects of this sequence always begin at the surfaces of the brain in the mild cases and extend inwards to affect the diencephalic-mesencephalic core at the most severe levels of trauma" (p. 637). Flowing from this definition, they produced a proposed classification of the grades of cerebral concussion in which three of six grades of concussion did not involve loss of consciousness (see Table 2-1). The lower grades (I to III) of cerebral concussion are especially common in contact sports such as American football and boxing.

Table 2-1. Ommaya & Gennarelli's Classification of Grades of Cerebral Concussion

Grade	Description	Outcome
I	Confusion	Normal consciousness without amnesia
II	Confusion → confusion + amnesia	Normal consciousness with PTA only or Normal consciousness with PTA + retrograde amnesia (RGA)
III	Confusion + amnesia	Normal consciousness with PTA + RGA
IV	Coma (paralytic) → confusion + amnesia	Normal consciousness with PTA + RGA
V	Coma	Persistent vegetative state
VI	Coma	Death

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

According to Ommaya & Gennarelli's classification, the grades of severity occur due to the initial impact or impulse which then results in shear strain of the nerve fibres in the brain. This notion is in agreement with that proposed by Rutherford (1989) who defined concussion as an 'acceleration/deceleration injury to the head'.

Since Ommaya & Gennarelli's classification of concussive syndromes, a number of authors have variously classified and defined concussion (e.g. Bruno et al., 1987; Cantu, 1986; Kelly et al., 1991; Maroon, Steele, & Berlin, 1980; Nelson et al., 1984; Ommaya & Gennarelli, 1974; Rutherford, 1989). These classifications/definitions differ on important measures such as presence and/or duration of LOC, PTA, and retrograde amnesia, thereby making evaluation of epidemiological data difficult (Cantu, 1986).

In 1986, Cantu developed a practical scheme for grading severity of a concussion in athletes, which has been widely used by sports medicine clinicians, in particular, and is relevant to any discussion dealing with concussion in athletes (see Table 2-2).

Table 2-2. Cantu Classification System Describing 3 Grades 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 ≥ 5 minutes and PTA ≥ 24 hours

Cantu, 1986

Of the 3 grades of concussion, Grade 1 concussion is the most difficult to recognise and consequently manage (Cantu, 1992, 1996, 1998a). Although this classification system is regularly employed, a practical difficulty associated with this injury scale is that LOC may be difficult to detect if it is very brief, and hence basing a player's return to play on this measure is problematic (McCrory, 1997). Further, while PTA is an important prognostic measure in severe brain injury, this has not been shown for mild brain injury (Gronwall, 1989).

The definition of concussion seems even more difficult to resolve than that of mild head injury, and is complicated by the tendency of authors to use these terms equivalently. Clearly, in light of the definitions of concussion cited above (e.g. Ommaya & Gennarelli, 1974; Cantu, 1986), mild concussion cannot be used as equivalent to mild head injury. However, the various descriptions of mild concussion do fall within the definition of mild head injury in terms of the classification systems proposed by both Ommaya & Gennarelli (1974) and Cantu (1986). The term sub-concussive head injury is also frequently mentioned and refers to a blow to the head which goes unnoticed externally (i.e. there is no visible outcome). Thus, for the purposes of this paper, both mild concussive and sub-concussive head injuries will be understood to be subsumed under the heading of mild head injury.

2.1.5. EPIDEMIOLOGY OF MILD HEAD INJURY

Mild head injury is a major medical and public health concern because of its high incidence (Evans, 1992; Satz et al., 1997). Approximately 80% of all mild head injuries are related to sport injuries, motor vehicle accidents, and falls (Rimel et al., 1981; Rutherford et al., 1977). The incidence of mild head injury varies between 130 and 208 hospitalisations per 100,000 inhabitants per year, accounting for at least 75% of all brain injuries (Kraus & Nourjah, 1989). However, many people who have incurred a mild head injury do not seek treatment and hence it is difficult to determine accurately the incidence of mild head injury (Binder, 1986, 1997; Rimel et al., 1981). Regardless, it is believed that the reported incidence (i.e. injury count based on hospital records) should be adjusted in an upward direction (Templer, Kasiraj, Trent, & Trent, 1992).

Studies have consistently reported a high incidence of traumatic head injury in young male adults (2:1 ratio) (Evans, 1992; Levin et al., 1982; Minderhoud & Van Zomeren, 1984 in Bohnen & Jolles, 1992; Templer et al., 1992), usually of below average socioeconomic status (Templer et al., 1992). One explanation for this difference between male and female injury rates can be understood in terms of differences in alcohol consumption (Minderhoud & Van Zomeren, 1984 in Bohnen & Jolles, 1992). Approximately 50% of all patients are between the ages of 15 and 34 (Evans, 1992).

2.1.6. NEUROPATHOLOGY AND PATHOPHYSIOLOGY OF MILD HEAD INJURY

Although head injury is the most common cause of neurological disorders, the pathology and pathophysiology of brain damage are poorly understood (Strich, 1961; Dacey, Vollmer, & Dikmen, 1993). This lack of understanding is particularly evident in the area of non-impact head injuries, as opposed to impact injuries which tend to be well described in the literature (Anderson, 1996). However, it is accepted that cerebral brain damage may occur without impact to the head (and entirely through exposure to acceleration forces) (Busch & Alpern, 1998), and in the absence of LOC or PTA (Sweeny, 1992 in Anderson, 1996). In most cases, the neuropathology in impact and non-impact head injuries can be accounted for by the movement of the brain inside the skull (Anderson, 1996).

Diffuse axonal injury (DAI) is the primary neuropathology of traumatic brain injury and results from shearing forces generated in the brain by sudden deceleration. The inertial force transmitted by the sudden deceleration causes DAI – the more force, the greater the injury (Alexander, 1995). Sheer-strain is seen to be a primary mechanism responsible for neural damage and subsequent behavioural dysfunction in mild head injuries (Holburn, 1943). According to the sheer-strain model, acceleration-deceleration injuries (i.e. whiplash) result in axonal tearing and neural degeneration in certain ascending and descending tracts of the brain stem (Anderson, 1996). Such an injury may be sustained when a rugby player is tackled from behind. It is proposed that unconsciousness occurs when sheer-strain is caused by rapid movements of the brain within the cranium, the consequence being the stretching and tearing of fibres, along with impact damage (Lishman, 1987; Stritch, 1961). The neuronal damage is accompanied by small haemorrhages from ruptured blood vessels scattered throughout the cerebral white matter and lower brain structures (Lezak, 1995). It is assumed that axonal degeneration in the brain stem will have a disruptive effect on cortical arousal and consequently on cognitive performances (Gentilini et al., 1985).

Several investigations have produced results supporting brain stem axonal degeneration as a result of acceleration-deceleration injuries (Barth et al., 1983). Mild head injury tends to be

associated with less severe accelerative-decelerative forces (e.g. blunt head trauma) (Satz et al., 1997). In this regard, Oppenheimer (1968), using post-mortem examinations of persons who had sustained mild head injuries, found certain pathological changes (namely microscopic lesions of the cerebral white matter). This evidence suggests that permanent damage in the form of microscopic lesions can be imposed on the brain by seemingly trivial head injuries. Furthermore, Oppenheimer commented that if such injuries were to be repeated (as may be the case in an unsuccessful boxer), "one would anticipate that a progressive, cumulative loss of tissue, and of nervous function, would occur" (p. 306). However, contrary to the above-mentioned theory of greater force leading to greater injury, Blumbergs et al. (1994) in examining mild head injury in adults found there to be a continuity of pathophysiology from severe head injury to mild head injury using the concept of diffuse axonal injury. Despite this continuity of pathophysiology, studies into mild head injury have found more variable outcomes than the clear pattern of poor outcomes associated with severe head injury (Satz et al., 1997).

2.2. SEQUELAE FOLLOWING MILD CLOSED HEAD INJURY

2.2.1. NEUROPHYSIOLOGICAL SEQUELAE OF MILD CLOSED HEAD INJURY

With respect to neurophysiological consequences of mild head injury, a number of neuroimaging and neurophysiological measures have been used to determine deficit post-injury. According to Bohnen & Jolles (1992), there is a great need for precise quantification of impairment, especially when one considers medico-legal difficulties. In this regard, certain neuroimaging measures such as *magnetic resonance imaging* (MRI) have proved useful. MRI has been found to be a superior technique to *computed tomography* (CT) scanning. MRI is more sensitive than CT scan in evaluating mild head injury and is better able to detect lesions in the brain that may be of particular significance for neurobehavioural outcome, especially in the frontotemporal region (Evans, 1992). The presence of neurophysiological evidence of brain damage following a mild head injury is complex in that while such an injury may result in the findings of an abnormal *encephalogram* (EEG), patients with documented brain damage/disorientation may have normal EEGs (Binder, 1986). Hence, EEGs may prove to be redundant in the objective assessment of mild head injury sequelae. However, it has been quite consistently found that athletes with prior mild head injuries (i.e. cumulative mild head injuries) do have an increased incidence of abnormal EEG recordings (e.g. Tysvaer, Storli, & Bachen, 1989; Ross, Casson, Siegal, & Cole, 1987).

Positron emission tomography (PET) has recently become available as a new imaging modality. Unlike CT and MRI which are mostly concerned with brain anatomy, PET allows in vivo study of brain function (Szymanski & Linn, 1992). There do not appear to be any studies which have used PET in mild head injury to date. Furthermore, *single-photon emission computerised tomography* (SPECT) has been used to evaluate functional metabolic abnormalities post-mild head injury and it is believed that SPECT may provide some valuable insights into the physiological mechanisms of such head trauma (Szymanski & Linn, 1992). While it is felt that neuroimaging studies using CT and MRI have not significantly contributed towards our understanding of the pathophysiology of mild head injury sequelae, it is thought that techniques which reflect brain functions (such as PET and SPECT) will be more beneficial (Szymanski & Linn, 1992). Another objective measure for assessment of outcome of mild head injury is the *psychophysical assessment of tolerance to light and sound.* In a study conducted by Bohnen et al. (1991 in Bohnen & Jolles, 1992), the researchers found the patients with mild head injury had a significantly reduced tolerance to sound and light stimuli when compared with the nonconcussed controls 3 to 6 days post-injury.

2.2.2. NEUROPSYCHOLOGICAL SEQUELAE OF MILD CLOSED HEAD INJURY

Neurophysiological sequelae of mild head injury have been established via the use of various neuroimaging and neurophysiological measures, as mentioned above. Neuropsychological sequelae of mild head injury tend to take two forms in that they can be established either by means of subjective reports of postconcussive symptoms or by means of objective formal neuropsychological assessment. The first form (postconcussive symptoms) will be cursorily discussed, while the latter form (formal neuropsychological assessment) will be examined in more detail as it is more directly relevant to the present study.

2.2.2.1. The Postconcussive Syndrome (PCS)

Although there is no consensus as to the definition of PCS (Binder, 1997), this syndrome is generally understood to refer to numerous signs and symptoms (including cognitive, emotional, and somatic complaints) that may occur in isolation or in combination usually following mild head injury (Binder, 1986; Bohnen & Jolles, 1992; Evans, 1992). PCS can develop without there having been a loss of consciousness (Evans, 1992). Frequently occurring self-reported symptoms include: headache; dizziness; irritability; anxiety; depression; sleep disturbance; blurred vision; noise sensitivity; easy fatigability; and concentration and memory difficulties (Barth et al., 1989; Binder, 1986; Bohnen & Jolles, 1992; Dikmen et al., 1986; Evans, 1992; McLean et al., 1983; Rutherford et al., 1977).

Because PCS symptoms are based on subjective reports, quantification or conceptualisation of these complaints is very difficult for researchers. This has led to an ensuing debate concerning the psychogenetic (i.e. psychological causes including premorbid personality problems and/or blatant malingering) versus physiogenetic origin of the complaints (Binder, 1986; Rutherford et al., 1977). In this regard, Bohnen & Jolles (1992) maintain that either of these approaches in isolation is inadequate in explaining the occurrence of persisting PCS, and rather that an interaction of the two viewpoints is more useful. Since PCS is not a specific area of investigation for the present study, the concept has only been cursorily reviewed. The reader is referred to the comprehensive papers of Evans (1992) and Szymanski & Linn (1992) for a more exhaustive discussion of PCS.

2.2.2.2. Objective Neuropsychological Sequelae of Mild Closed Head Injury

Whereas PCS is based on subjective reports of symptomatology, objective neuropsychological measures have also been employed to evaluate cognitive and behavioural dysfunctions in patients with mild head injury (Bohnen & Jolles, 1992). Related to this, it has been noted that while the neuropsychological sequelae of severe head injury are well known and widely accepted, the outcome of mild head injury is not as well established and the presence of cognitive-behavioural sequelae due to mild head injury remains controversial (Barth et al., 1983; Bohnen & Jolles, 1992; Raskin, Mateer, & Tweenen, 1998; Segalowitz & Lawson, 1995). Over the past few decades, a number of studies investigating the neuropsychological sequelae of mild head injury have been conducted. Many studies in this area have shown that mild closed head injury leads to measurable cognitive deficits (e.g. Gentilini, Nichelli, & Schoenhuber, 1989; Gulbrandsen, 1984; Levin et al., 1987; Parasuraman, Mutter, & Molloy, 1991; Rimel et al., 1981). In this regard, impairments in information processing (e.g. Gronwall & Wrightson, 1974, 1975; Levin & Eisenberg, 1979; MacFlynn, Montgomery, Fenton, & Rutherford, 1984; Szymanski & Linn, 1992), memory (e.g. Barth et al., 1983; Bassett & Slater, 1990; Rimel et al., 1981; Ruff et al., 1989; Rutherford et al., 1977), language (e.g. Levin & Eisenberg, 1979; Segalowitz & Lawson, 1995), and visuospatial problem solving ability (e.g. Levin & Eisenberg, 1979) have been noted after mild closed head injury. Further, deficits in attention and concentration have frequently been found to accompany mild closed traumatic brain injury (e.g. Gentilini et al., 1985; Gronwall & Wrightson, 1974; Parasuraman et al., 1991; Rimel et al., 1981; Rutherford et al., 1977; Szymanski & Linn, 1992). Albeit not specifically related to mild head injury, Lezak (1995) reports that motor slowing has frequently been exhibited in patients suffering head injury. Furthermore, Lezak holds that closed head injury can cause diffuse damage to the frontal and temporal lobes with accompanying dysfunction in memory, learning, and higher cognitive processes (e.g. abstraction and reasoning).

2.2.2.3. Course of Recovery Following Mild Closed Head Injury

The recovery process after mild brain injury tends to be controversial (Binder, 1986). However, there is generally agreement that various postconcussion symptoms (e.g. reduced speed of information processing, memory impairment, complaints of poor attention and concentration) tend to be present in the first 1-3 months after the mild head injury (Szymanski & Linn, 1992). It seems that most of the controversy centres around the aetiology of persistent deficits. In this regard, some authors assert that cognitive sequelae should be mild and resolve within 1 to 3 months after the head injury (e.g. Dikmen et al., 1986; Gentilini et al., 1985; Levin et al., 1987). However, other researchers have reported a more protracted course of recovery for mild head injury patients (e.g. Bohnen, Jolles, & Twijnstra, 1992; Leininger, Gramling, Farrell, Kreutzer, & Peck, 1990; Rimel et al., 1981). Although most of mild head injury patients may not suffer persisting neuropsychological dysfunction (Binder, Rohling, & Larrabee, 1997), there is clear evidence supporting the fact that a subgroup of individuals are definitely affected (Leininger et al., 1990).

Research Supporting the Rapid Resolving of Neuropsychological Deficit One to Three Months Post-Mild Closed Head Injury. Gronwall & Wrightson (1974) were the forerunners in asserting that a reduced rate of information processing was an important factor in the formation of the postconcussive syndrome (PCS). Using the Paced Auditory Serial Addition Test (PASAT) (a measure of divided attention and complex mental tracking/rate of information processing), these authors found that concussed patients were able to process a limited number of items as quickly as normal controls. However, with increasing items the patient reached a critical point, resulting in fall off in performance and further divergence from that of the controls as more items were added. This finding relates to the theory of Brain Reserve Capacity proposed by Satz (1993), which is discussed in more detail in section 2.5. In sum, Gronwall & Wrightson (1974) found recovery in the majority of their patients after 35 days and recovery in all patients after 54 days, as well as a correlation between PASAT performance (rate of information processing) and the presence of symptoms.

McLean et al. (1983) found impaired performance on the Stroop Colour (interference/ distractibility test) and the Selective Reminding (recent memory test) Tests in mildly head injured patients compared with matched controls 3 days post-injury, but this impairment was no longer evident at 1 month follow-up. The authors viewed this result as being indicative of recovery. A study by Gentilini et al. (1985) found a general trend towards lowered cognitive performance, with evidence of a specific deficit in selective attention in mild head injury patients. Similarly to McLean et al. (1983), these researchers found evidence supporting the tenet that structural damage post-mild head injury tends to recover within 1 month after the

injury. Results from the Dikmen et al. (1986) study indicated that at 1 month post-injury the performances of the mild head injury subjects were not impaired in the clinical sense, although they were slightly lower than those of the uninjured group. Furthermore, at 1 year post-injury, no significant differences were evident on the neuropsychological measures.

In a three-centre study of minor head injury, Levin et al. (1987) found significant neurobehavioural impairment at 1 week post-mild head injury, indicating the presence of subacute disturbances of attention, memory[2], and speed of information processing. By 1 to 3 months post-injury they found that most patients had shown cognitive recovery to within the range of matched controls. A methodological constraint of this study was the employment of a small sample size in terms of the total number of patients tested on all 3 occasions. Furthermore, because practice effects were not controlled for and group (as opposed to individual) comparisons were made, these authors do not assume that the cognitive recovery noted is complete at 3 months. Levin et al.'s (1987) neuropsychological results are consistent with those of McLean et al. (1983), Gentilini et al. (1985), and Dikmen et al. (1986). However, these studies also share an important methodological constraint in that they all failed to utilise baseline premorbid data. This omission limits the definitive claim that the patients have in fact returned to their premorbid level of functioning. Furthermore, McLean et al. (1983), Gentilini et al. (1985), and Dikmen et al. (1986) emphasise the lack of statistically significant differences between subjects and controls at 1 month after mild head injury, but tend to overlook the tendency for the subjects to perform more poorly across neuropsychological tests than the uninjured controls. Finally, although all of these studies found fairly rapid resolution of neuropsychological deficit one to three months post-mild head injury, these findings must be cautiously interpreted in the context of methodological constraints and the unknown long-range or delayed effects of mild head injury.

Research Supporting the Persistence of Neuropsychological Deficit Three to Twenty-two Months Post-Mild Closed Head Injury. In a seminal prospective study by Rimel et al. (1981), an exhaustive analysis was conducted on 538 mild head injury patients. These researchers found impaired neuropsychological test performance (cognitive deficits in the areas of attention, concentration, memory, and judgement) at 3 months post-injury follow-up in the group of mild head injury patients compared with normative standards.

[2] The reader is referred to the study by Ruff et al. (1989). Utilising the same sample, definition of minor head injury, and data obtained from the Levin et al. (1987) study, Ruff et al. investigated more specifically the recovery of memory after minor head injury.

Utilising the same subject pool as Rimel et al. (1981), Barth et al. (1983) conducted neuropsychological evaluations on 71 patients with minor head injury 3 months post-injury. They found that a significant proportion of the patients evidenced cognitive impairment (memory, attention and visuospatial deficits). These authors postulated that the memory deficits were secondary to problems with information processing capacity. Although a comprehensive test battery was used in both these studies, a number of methodological limitations were noted. A major weakness was the absence of a matched control group, which made it difficult to ascertain the contribution of the head injury as contrasted to the contribution of other factors (e.g. premorbid characteristics, method of participant selection) which could impact on neuropsychological functions. Furthermore, these authors did not exclude patients with pre-existing conditions involving the central nervous system (including prior head injuries) or neuropsychiatric disorders. Thus, although both the studies conducted by Rimel et al. (1981) and Barth et al. (1983) found persistent neuropsychological and psychosocial difficulties at 3 months post-mild head injury, these studies were weakened by their methodological flaws.

A well-controlled study by Leininger et al. (1990) found that, relative to uninjured controls, mild head injury patients had deficits in the areas of reasoning, information processing, and verbal learning 1 to 22 months post-injury. Thus, they concluded that some patients seem to suffer enduring neuropsychological impairments. In accordance with these findings, Bohnen et al. (1992) found that mild head injury patients with persistent PCS performed significantly poorer than controls on certain neuropsychological tests. These authors attempted to control for a methodological flaw noted in other studies (namely the tendency to compare head-injured patients with those who had not suffered a head injury) by comparing neuropsychological deficits in patients with persistent PCS 6 months after an uncomplicated mild head injury with (i) patients with mild head injuries who did not have PCS and (ii) healthy controls. Their neuropsychological test battery comprised the following tests: a visual, computer-assisted version of the Auditory Verbal Learning Test (memory task); the Stroop Colour Word Interference Test; and a computerised divided attention task. Bohnen et al. (1992) found neuropsychological deficits on tests of attention (divided and selective) and information processing in those patients with PCS 6 months post-uncomplicated mild head injury. Interestingly, they also found that patients who had recovered from an uncomplicated mild head injury (i.e. did not report PCS symptoms) did not differ in cognitive functioning from healthy control subjects.

A recent study by Klonoff & Lamb (1998) investigated 9 mild head injury patients who presented with chronic and unusually severe deficits on average *3 years* post-mild head

injury. The authors found significant neuropsychological deficits on testing. In addition, there was evidence of significant psychiatric disability and/or malingering in each patient, which seemed to provide an explanation for the lowered neuropsychological test performance and also support for the psychogenicity of PCS. This latter finding is in support of the study by Barth et al. (1983), which found that a premorbid history of emotional problems and/or the presence of secondary gain might contribute to persistent postconcussive symptomatology. However, a major methodological flaw of the Klonoff & Lamb study was the restricted sample size, which limits the generalisability of the results. In another up-to-date study, Raskin et al. (1998) tested 148 patients with prolonged symptomatology following mild head injury on a comprehensive neuropsychological battery, which included a measure of personality. The mean time elapsed since the trauma was 21 months. Results showed that the performance of patients with mild traumatic brain injury was significantly impaired (when compared to normative data) on measures of complex attention, working memory, verbal learning, and especially on time-dependent tasks. It was found that cognitive performance was related to demographic variables of gender and age, but not to education, educational status, length of loss of consciousness, or length of time post-injury. Further, although the authors acknowledged that emotional factors could complicate neuropsychological assessment, they failed to find a direct relationship between depression (and other emotional or personality variables) and cognitive functioning, suggesting that persistent cognitive deficit has an organic aetiology. The main methodological weakness of the Raskin et al. study was the failure to include a matched control group.

Commentary on Reviews of Null Outcome. Recently, Binder et al. (1997) conducted a meta-analytic review of available prospective and quasi-prospective neuropsychological research on mild head trauma in adults (which they narrowed down to only eight studies). Their data suggest that attention measures are the most sensitive indicators of dysfunction associated with mild head trauma. Although these authors did find neuropsychological evidence of persisting cognitive deficit post-mild head trauma, the strength of the association is weak and no causation is shown. Based on their estimate of the prevalence of persistent neuropsychological problems after mild head trauma, the researchers suggest that false positive diagnoses of brain dysfunction are likely. They conclude that neuropsychologists are more likely to be correct when diagnosing no brain injury than when diagnosing the presence of a brain injury.

Similarly, Satz et al., (1997) recently produced an extensive review comprising 40 studies examining mild head injury in children over a 25-year period (1970 to 1995) and reported 13 adverse, 18 null, and 9 indeterminate findings with respect to outcome. These researchers

found that the studies reporting null outcomes tended to be much stronger methodologically than the other studies, thereby leading to the recommendation of a cautious acceptance of the null hypothesis as it relates to neuropsychological outcome in mild head injury. Satz et al. (1997) also comment on the tendency of journals to favour studies that report significant (i.e. reject the null hypothesis) rather than null results. This finding implies that while research reporting neuropsychological impairment following mild head injury tends to be published, many more unpublished null findings may in fact miss citation. Further, the authors caution against the application of these findings to adults, as they claim that children and adults differ significantly in terms of aspects of life experience which would consequently impact differentially on the course of recovery post-mild head injury. This latter comment is in agreement with Gulbrandsen (1984), who remarks that research has demonstrated that the effects of head injuries in children differ (medically and psychologically) from those noted in adults.

In a response to the recent Binder et al. (1997) and Satz et al. (1997) reviews, Shuttleworth-Jordan (1999) comments that while these authors fully recognised the lowering of brain reserve capacity (BRC) due to pre-existing risk factors, they did not acknowledge the acquisition of a new risk factor in the form of a silent brain injury. Shuttleworth-Jordan uses the notion of BRC threshold theory (formulated by Satz, 1993) to argue that it is fallacious to take null effects following a mild head injury to be absolute indicators. With respect to this, she maintains "that what is absent initially, may subsequently evolve into much (in the way of symptomatic presentation)" (p. 11). In support of Shuttleworth-Jordan, Reid (1998) (see p.32) in his investigation of the cumulative effects of head injury in rugby players found evidence of a significant variability between the rugby group and the control group, which invalidates the 'null' indications of average effects. Thus, in effect, there are significant differences between groups in variability of outcome. The large meta-reviews of Binder et al. (1997) and Satz et al. (1997) describe research which tends to be only concerned with mean scores (and not with significant variability between groups). Hence, with respect to mild head injury, Shuttleworth-Jordan (1999, p. 24) 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, whereas there are a significant proportion of individuals who are not".

In conclusion, some studies have shown the presence of neuropsychological and psychosocial difficulties persisting 3 to 22 months after mild head injury (Barth et al., 1983; Bohnen et al., 1992; Leininger et al., 1990; Rimel et al., 1981). However, a number of other studies have found that there are *no* indications of decreased cognitive functioning approximately 1 month

after mild head injury (Dikmen et al., 1986; Gentilini et al., 1985; Levin et al., 1987; and McLean et al., 1983). Bohnen et al. (1992, p. 692) explains that these "conflicting results may be because of the heterogeneity of the subjects, the different time intervals after injury, the sensitivity of the selected cognitive tests in detecting posttraumatic brain dysfunction, and the appropriateness of the control group". In this regard, Boll (1983) comments that even though negative reactions (in terms of both cognitive and emotional functions) are often not apparent, one cannot assume that neurological impairment and consequent psychological changes have not taken place. Furthermore, while the studies cited above suggest that reduced speed of information processing, attention deficits, and memory problems are amongst the most common neuropsychological sequelae of mild head injury, they also imply that not all mild head injury patients display significant difficulties in all of these areas. Finally, what is most apparent from the above-mentioned studies is the variability in mild head injury outcome (Shuttleworth-Jordan, 1999).

2.3. CUMULATIVE MILD CLOSED HEAD INJURY

A disturbing feature of mild concussive or sub-concussive head injury is that the effects may be cumulative (De Villiers, 1987; Gronwall & Wrightson, 1975). However, the findings of the studies cited above are concerned in the main with people with *no* history of prior head injury. This section will introduce studies investigating the neuropsychological sequelae of cumulative mild closed head injury[3]. In addition, Satz's (1993) theory of brain reserve capacity (BRC), which provides a theoretical framework for understanding the effects of cumulative mild head injury, will be discussed.

2.3.1. NEUROPSYCHOLOGICAL SEQUELAE OF CUMULATIVE MILD CLOSED HEAD INJURY

Gronwall & Wrightson (1975) provide compelling evidence supporting the hypothesis that the effects of mild head injuries may be cumulative, indicating that the time course of recovery is increasingly prolonged after successive insults which are believed to inflict progressive diffuse axonal injury. Using the Paced Auditory Serial Addition Task (PASAT), these researchers compared 20 young adults after a second mild head injury with a matched control group comprised of first mild head injury patients. These authors found that while a single mild head injury reduces intellectual performance temporarily, a second mild head

[3] The negative outcome of cumulative effects of many relatively minor head injuries has been documented in studies on *athletes* exposed to repeated concussions (e.g. boxers and rugby players). These studies will be discussed in section 2.4.

injury reduces it even further and the reduction continues for a longer duration. In addition, they found that while the intellectual performance eventually returns to normal after two mild head injuries, the effects of repeated mild head injuries tend to be cumulative, and each person has a limit beyond which recovery is not complete. These results are commensurate with those of Gronwall (1989), who conducted a series of research studies using the PASAT and found that mild head injury resulted in a reduction in information processing ability. Furthermore, this author found that most of the participants in the young adult group had recovered to a normal level by four to six weeks post-injury, but that older individuals (ages 40-49) and individuals with a previous head injury tended to take longer to recover.

Another much-cited research study conducted by Ewing, McCarthy, Gronwall, & Wrightson (1980) demonstrated that after a mild head injury with apparent full recovery, there is evidence of a residual effect which results in increased vulnerability to a second central nervous system (CNS) stressor, such as hypoxia or a further head injury. Thus, the implication is that each mild head injury destroys neurons, thereby diminishing the reserve available and rendering the loss evident under the stress of further injury.

2.4. MILD CLOSED HEAD INJURY IN CONTACT SPORT

Until this point, mild closed head injury and cumulative mild closed head injury in the general population have been discussed. The remainder of this chapter will focus on what is understood to be a very special case of cumulative mild closed head injury, namely that which occurs in contact sport (Binder, 1997).

The mild head injury is the most common sports-related head injury (Cantu, 1996; Davis & McKelvey, 1998; Warren & Bailes, 1998), and is estimated to occur at a rate of 250 000 per year in contact sports (Cantu, 1988 in Wilberger, 1993). Athletes participating in contact sports (e.g. boxing, American football, Australian rules football, wrestling, ice hockey, soccer, martial arts, and rugby) are at particular risk of sustaining head injuries (both mild and severe) (Lehman & Ravich, 1990; Sturmi et al., 1998; Warren & Bailes, 1998). However, until recently, mild head injuries in sport were regarded as inconsequential and the neuropsychological sequelae (including the cumulative effects of mild head injuries) following such traumas were not viewed as particularly relevant (Anderson, 1996; Barth et al., 1989; Shuttleworth-Jordan, Balarin, & Putchert, 1993). Many mild head injuries go unreported by athletes, who tend to minimise their symptoms in order to be declared fit to

return to the game, thereby making clinical assessment difficult (Anderson, 1996; Barth et al., 1989; Cantu, 1986; Kelly et al., 1991; Saunders & Harbaugh, 1984; Sturmi et al., 1998; Wilberger, 1993). Furthermore, because most cases of sports-related mild head injury go unreported, it is difficult to assess accurately the prevalence of such injuries (Anderson, 1996; Maddocks & Saling, 1991).

2.4.1. MECHANISM AND OUTCOME OF CEREBRAL INJURY IN SPORT

According to Cantu (1996), concussion (clearly referring to the entire spectrum of classification ranging from mild to severe) may occur as a result of a direct trauma to the head (e.g. in collisions or falls) or as a result of a sufficiently great application of a force to the brain (indirect trauma) as in a whiplash injury. Most head injuries are caused by the moving head hitting the ground or another relatively large and stationary object, for example, being tackled or carrying out a tackle at rugby and collision of heads at soccer (Gleave, 1986). The head comes to an abrupt halt, but the relative movement of the brain continues with translational and rotational acceleration. This results in mild concussion and, in more severe cases, an additional loss of consciousness. In rugby and soccer, a closed head injury with mild concussion from acceleration/deceleration stresses is the most frequently occurring type of head injury.

It has been adequately reported in the literature that repeated minor head injuries occurring in short succession may result in a fatal outcome (Kelly et al., 1991; Saunders & Harbaugh, 1984). Saunders & Harbaugh (1984) were the first to document that fatal brain swelling may occur in the setting of a recent minor head trauma followed by a second minor head trauma in athletes who are still symptomatic from the first injury. This is known as the *second impact syndrome* (SIS) of catastrophic head injury. The SIS is a potentially fatal (although rare) condition which has been documented in American football players, although it may occur in a variety of contact sports. The SIS can occur after a grade I (mild) concussion[4] (as well as after a more severe head injury) and hence it is important to be able to identify all grades of concussion (Cantu, 1996, 1998). In this regard, Kelly et al. (1991) demonstrated in their case report that this syndrome may occur in settings of a mild head injury without any loss of consciousness. Although its cause is unknown, the SIS is thought to involve disordered cerebral vascular autoregulation (Cantu, 1998a; Green & Jordan, 1998). However, no controlled case studies have been conducted to identify risk factors for the SIS and the concept rests entirely on the understanding of anecdotal reports (McCrory, 1997). Regardless, the SIS remains a catastrophic condition, with a mortality rate of nearly 50% and

[4] The reader is referred back to section 2.1.4 which deals with the classification of concussion.

a morbidity rate of nearly 100%, and thus the importance of prevention cannot be understated (Cantu, 1998). Sturmi et al. (1998) maintain that if one takes into account the catastrophic outcome of the SIS, then the actual existence of 'mild concussion' must be seriously questioned.

2.4.2. NEUROPSYCHOLOGICAL ASSESSMENT OF MILD CLOSED HEAD INJURY IN SPORT

Traditionally, the medical diagnosis of mild head injury and the assessment of recovery in athletes has been subjectively based on the presence of clinical symptoms (e.g. headache, dizziness, nausea) and signs (e.g. loss of consciousness, and PTA) (Barth et al., 1989). However, recently, there has been an increased interest in the use of neuropsychological tests as objective measures of recovery from mild head injury in sport (Maddocks & Saling, 1991). In this regard, tests of speed of information processing (e.g. the Digit Symbol Subtest from the Wechsler Adult Intelligence Scale - Revised) have been shown to be sensitive to the effects of mild head injury in American football (Barth et al., 1989) and Australian Rules footballers (Maddocks & Saling, 1991). Neuropsychological assessment may assist in diagnosis and may also provide an objective index of recovery and hence may assist in decisions regarding return to contact sport (Maddocks, Saling, & Dicker, 1995). The remainder of this literature review will report on neuropsychological studies of outcome in contact sport, with particular reference to the contact sports of boxing, soccer, American Football, Australian rules football, and finally the central focus of this research, namely rugby.

2.4.3. NEUROPSYCHOLOGICAL SEQUELAE FOLLOWING MILD CLOSED HEAD INJURY IN BOXING

While significant attention has been paid to the study of moderate and severe head trauma, there is generally a paucity of research into *mild* cumulative head injury in boxing (Barth et al., 1989). Boxing differs from other contact sports in that it aims to render opponents unconscious and helpless through successive blows to the head (Casson, Sham, Campbell, Tarlau & DiDomenico, 1982; Haglund & Eriksson, 1993; McCunney & Russo, 1984; Ross et al., 1987). The most common acute neurological injury in boxing is the knockout, which is equivalent to a mild head injury (Jordan, 1987). Controversy surrounds the relationship between boxing and brain damage (Brooks, Kupshik, Wilson, Galbraith, & Ward, 1987). Studies of professional boxers have provided evidence suggesting the presence of a relationship between brain damage and boxing, and that increased numbers of fights increases the likelihood of damage. However, studies investigating the neurological and neuropsychological effects of amateur boxing have produced ambiguous results. Before

embarking on a discussion of research in this area, it is necessary to point out that amateur boxing differs from professional boxing in the duration of fights, rules and regulatory policies, medical evaluation, and protective devices (Brooks et al., 1987; Butler, Forsythe, Beverly, & Adams, 1993; Haglund & Eriksson, 1993; Jordan, 1987).

Martland (1928) provided the earliest description on neurological, cognitive, and behavioural impairment occurring from boxing. He described this as the "punch drunk" syndrome of cumulative head trauma in boxers and noted neurological symptoms such as mild confusion and unsteady gait early on, progressing to increased speech and motor deficits, as well as upper-extremity and head tremors. This progressive encephalopathy would eventually be characterised by a movement disorder (similar to Parkinson's Disease) involving an extremely unsteady gait and significant mental decline. The diffuse cerebral atrophy which frequently occurs in boxers has been called "chronic boxers' encephalopathy" (Serel & Jaros, 1962), "dementia pugilistica" (Lampert & Hardman, 1984) (both in Barth et al., 1989), and "chronic traumatic encephalopathy" (Jordan, 1987). The punch drunk syndrome is noted in about 25% of professional boxers and typically occurs 7 to 35 years after the start of a fighter's career (Haglund & Eriksson, 1993; Ross et al., 1987). This syndrome seems to be directly related to the length of the boxer's career, frequency of participation, age of exposure (retirement age from boxing), and a certain genetic predisposition (Green & Jordan, 1998).

Research Studies of Mild Closed Head Injury in Professional Boxing. In boxing, the cumulative effects of multiple blows to the head (not necessarily causing knockouts) contributes to the severity of head injury. This point is illustrated in a study by Casson et al. (1982), which assessed 10 professional boxers post-mild head injury using EEG, CT, and neurological examinations. Testing results revealed the following: one boxer had an abnormal neurological examination; two had abnormal EEGs; and five demonstrated abnormal CT scans with mild to moderate cerebral atrophy. Because no boxer had been knocked out more than twice, the authors suggested that these detected abnormalities were caused by multiple sub-concussive blows to the head (as opposed to the number of knockouts).

Using a comprehensive neuropsychological test battery, Drew, Templar, Schuyler, Newell, & Cannon (1986) conducted a study investigating neuropsychological performance of active professional boxers and found impairment in finger tapping performance relative to the controls. These researchers also found a strong correlation between both the number of professional bouts and lost fights, and the boxer's performance deficits. However, no significant correlation was found between former amateur career and signs of brain injury. In

accordance with Drew et al. (1986), Ross et al. (1987) found a correlation between neuropsychological test impairment index and the number of professional fights. Ross et al. compared the cognitive performance of 15 former and active professional boxers on a neuropsychological battery (including the Trail Making Test, the Digit Symbol Test, the Wechsler Memory Test, and the Bender Gestalt Test) with established normative data for the general population. They found that performance was more impaired on those neuropsychological tests more heavily weighted for memory than on the non-memory tests of the battery. The researchers also found a significant correlation between poor test performance and the number of bouts fought as well as a correlation between poor test performance and increasing age. A neuropsychological study by Kaste et al. (1982) of 14 boxers (8 amateur and 6 professional) revealed brain damage in both professional and amateur boxers (although it seemed to be both less frequent and less advanced in the latter group), providing further support for the cumulative effects of repeated mild head injuries. This is consistent with the findings by Gronwall & Wrightson (1975). Methodological weaknesses of these studies include the absence of an adequate matched control group and small sample numbers.

Research Studies of Mild Closed Head Injury in Amateur Boxing. The McLatchie et al. (1987) study found that the sample of 20 amateur boxers performed significantly poorer than the controls on several neuropsychological measures (e.g. on the Inglis Word Learning Test and on the copy and immediate recall of the Rey Figure), indicating deficits in verbal learning and memory. Similarly, a study by Heilbronner, Henry, & Carson-Brewer (1991) found mild changes in cognitive functions (impairments in verbal recall and incidental memory) in 23 amateur boxers assessed immediately post-boxing match.

However, other researchers (e.g. Brooks et al., 1987; Butler et al., 1993) have found no evidence of significant neuropsychological deficit in amateur boxers (as compared with matched controls), nor any correlation between poor cognitive test performance and number of bouts fought (i.e. a cumulative effect). Both these studies have a number of methodological weaknesses, though, including the lack of an adequate control group and the use of a non-random sampling method (those who refused to participate may have had a high awareness of their impairments). In addition, most of the boxers in the Butler et al. (1993) study were very young (mean age = 16.7) and inexperienced which might make extrapolation of the results to amateur boxing at a more experienced (elite) level problematic. Haglund & Eriksson (1993) conducted a retrospective study of 50 former amateur boxers compared with two control groups (soccer players, and track-and-field athletes) and found no signs of serious chronic brain damage among any of the groups studied. However, there were moderate EEG

deviations among boxers and, neuropsychologically, the boxers had inferior finger-tapping performance which may be indicative of slight brain dysfunction. The above-mentioned studies of amateur boxers, although not devoid of methodological flaws, are methodologically superior to the studies of professional boxers in that they include matched controls and more sensitive standardised neuropsychological tests.

2.4.4. NEUROPSYCHOLOGICAL SEQUELAE FOLLOWING MILD CLOSED HEAD INJURY IN SOCCER

Soccer has been considered by some to be a relatively safe sport with a low rate of (minor) injuries and a low incidence of head injuries relative to more contact-oriented sports (e.g. American football and rugby) (Dailey & Barsan, 1992). However, this has been called into question more recently due to heading of the ball. Soccer is unique because the head may be purposefully used to advance, control, and strike the ball (Barnes et al., 1998; Boden, Kirkendall, & Garrett, 1998), with the average player heading the ball up to ten times per game (Jordan, Green, Galanty, Mandelbaum, & Jabour, 1996). Soccer has been classified as a contact/collision sport by the American Academy of Pediatrics (in Green & Jordan, 1998), and hence acute traumatic brain injury is a concern in soccer. Head injuries potentially occur in two different ways: (1) either through major impact with another object (e.g. head, elbow, boot, ground, or goalpost) which causes an acute head injury; or (2) through chronic injury as a result of repetitive, minor head impacts with the ball which may lead to cumulative encephalopathy (Jordan et al., 1996). Despite the purposeful heading of the ball and the contact nature of soccer, there is a paucity of studies investigating the risk of injury from heading a soccer ball (Green & Jordan, 1998). Furthermore, there has been a tendency to largely ignore the incidence of acute brain injury in the sport (Green & Jordan, 1998).

Studies that have been conducted, and are currently gathering momentum, are as follows. A study by Abreau, Templer, Schuyler, & Hutchison (1990) compared the neuropsychological performance of 31 college soccer players with 31 tennis players (controls) using a neuropsychological battery consisting of the Raven Progressive Matrices, Symbol Digit Modalities Test, Perceptual Speed Test, and PASAT. Although no significant group differences were found on neuropsychological tests, the soccer players demonstrated a significant negative correlation between number of games played and performance on the PASAT, suggesting compromised information processing ability as a result of cumulative mild head injury. However, the methodological limitations (e.g. small sample size and the failure to utilise premorbid data or repeated post-injury testing) led the researchers to conclude that their results provide only tentative support for neuropsychological deficits as a result of cumulative blows to the head received during soccer. Concordant with Abreau et al.

(1990), Tysvaer & Lochen (1991) demonstrated that heading of the soccer ball may cause cumulative brain damage. The latter-mentioned researchers conducted a neuropsychological study of 37 former soccer players compared with a control group (20 hospitalised patients) using an extensive neuropsychological test battery (including the WAIS with 10 subtests, Trail Making Test – Parts A and B, a modification of the Halstead-Wepman-Reitan aphasia screening test, tests of sensory-perceptual functions, motor tests, tests of hemisphere dominance, and the Benton Visual Retention Test – Form C). They found that the soccer players demonstrated mild to severe deficits in attention, concentration, memory, and judgement, strongly indicating the cumulative effect of repeated head traumas. Furthermore, the headers had a higher degree of neuropsychological impairment (20%) than nonheaders (8%), although the difference was not statistically significant. Furthermore, using neurological and EEG examinations, Tysvaer et al. (1989) and Tysvaer & Storli (1989) demonstrated a higher incidence of EEG abnormalities in soccer players compared to matched controls and concluded that these abnormalities were probably due to the cumulative effect of repeated head injuries. From the studies cited above, Tysvaer and his colleagues found that the dysfunctions evidenced during the investigations did not influence the players' normal daily activity and social adjustment (Tysvaer, 1992). However, these studies do have a number of methodological problems, including the absence of suitable control groups and the failure to control for factors which could lead to central nervous system disturbances, such as history of alcohol abuse and prior concussive episodes (e.g. due to non-sport related injuries such as motor vehicle accidents etc.).

Recently, a well-controlled study by Matser, Kessels, Jordan, Lezak, & Troost (1998) compared the cognitive performance of 53 active professional Dutch soccer players with the performance of a control group (27 elite non-contact sport athletes – elite swimmers and runners) using an extensive neuropsychological test battery and a comprehensive interview. The test battery included tests proven to be sensitive to cognitive changes incurred during contact sports, namely Raven Progressive Matrices Test, Wisconsin Card Sorting Task, PASAT, Digit Symbol Test, Trail Making Test A and B, Stroop Test, Bourdon-Wiersma Test, subtests of the Wechsler Memory Scale (Associate Learning, Logical Memory, and Visual Reproduction), Complex Figure Test, 15-Word Learning Test, Benton's Facial Recognition Task, Figure Detection Test, Verbal Fluency Test, and the Puncture Test. The researchers found that the soccer players performed poorer on verbal and visual memory, planning, and visuoperceptual processing tasks compared with the controls. In addition, an inverse relationship was found between performance on certain tasks (namely memory, planning, and visuoperceptual tasks) and the number of mild head injuries incurred in soccer along with the frequency of heading the ball. Further, forward and defensive players (classified as headers)

performed significantly poorer on Figure Detection, Complex Figure Test (Immediate and Delayed Recall), Logical Memory, and Visual Reproduction compared with midfield players and goalkeepers (classified as nonheaders). These findings are in accordance with those of Tysvaer & Lochen (1991), who demonstrated increased neuropsychological impairment in headers than in nonheaders.

The validity of heading as a cause of brain injury has been questioned by a few researchers (e.g. Haglund & Eriksson, 1993 and Jordan et al., 1996). In this regard, Barnes et al. (1998) and Boden et al. (1998), conducting separate studies to determine the incidence of mild head injury in elite soccer players, found the following: male soccer players have a higher incidence of mild head injury than female soccer players; the most common mechanism of injury is collision with another player; and the majority of mild head injuries are classified as grade 1 mild concussions (i.e. confusion without amnesia). Based on their findings, both sets of researchers maintained that any long-term encephalopathic changes in soccer players are due to mild head injuries from the player's head being struck (e.g. via a collision with another player) as opposed to repetitively heading the soccer ball. Hence, they questioned the association between repeated heading and neurological dysfunction which had been reported by other studies (e.g. Tysvaer & Lochen, 1991; Tysvaer & Storli, 1989; Tysvaer et al., 1989). However, the absence of a control group limits the generalisability of their results.

Interesting research was carried out by Spear (1995), who reviewed available studies to determine whether or not a link existed between playing football (soccer) and the risk of developing dementia. He comments that head injury is the environmental cause most consistently associated with Alzheimer's disease (Gentleman & Roberts, 1991 in Spear, 1995) and further, that footballers have an increased risk of recurrent mild head injuries than the general population due to head contact with the ball/other players. While it is known that severe head injury can lead to amyloid deposition, which in turn can cause pathological changes similar to those seen in Alzheimer's disease, the long-term pathological effects of mild head injuries are not well described. Hence, it is not yet known whether footballers have an increased risk of developing dementia. However, Spear raises this as a strong possibility.

2.4.5. NEUROPSYCHOLOGICAL SEQUELAE FOLLOWING MILD CLOSED HEAD INJURY IN RUGBY/FOOTBALL-RELATED SPORTS

Rugby/football-related sports occur in various forms, such as American football, Australian Rules football, rugby league football, and rugby union[5]. Although there are differences between the various forms of rugby/football-related sports, these tend to be subtle, and the number of shared commonalities justifies grouping them. Regardless of the form, all rugby/football-related sports are regarded as contact sports which have a high incidence of head and neck injuries (Gibbs, 1993; Seward, Orchard, Hazard, & Collinson, 1993). Mild head injuries occur as a result of stresses and impacts on the head and neck during scrumming, tackling and collisions between players (Shuttleworth-Jordan et al., 1993). Each of the above-mentioned rugby/football-related sports, in particular rugby union (hereafter referred to as rugby) which is the focus of this research, will be discussed in some detail with respect to incidence as well as neuropsychological outcome of mild head injury.

2.4.5.1. American Football

According to Wilberger (1993), the occurrence of repeated injury in football is very common, and the incidence of minor head injury may be up to five times higher than that in boxers. Nevertheless, as with boxing and soccer, there has been a paucity of controlled studies investigating football-related mild injury and, in particular, the neuropsychological sequelae of repeated mild head injury.

Incidence of Mild Head Injury in American Football. Barth et al. (1989) conducted a study investigating the incidence of mild head injury in football players in the United States of America and found that 10% of the college football players had experienced at least one mild head injury. Further, 42% of the players had a history of at least one mild head injury prior to this study, with 22% (of the 42%) having reported two or more (i.e. repeated) prior minor head traumas. Players with the most head injuries tended to play in the offensive line position (23.2%) and tackling and blocking tended to be the primary activities of the players when injured. Gerberich, Priest, Boen, Straub, & Maxwell (1983) found a reported mild head injury incidence of 19% in high school football players and that players with a prior history of mild head injury (including a loss of consciousness), had a risk of mild head injury four times greater than that of other players. Furthermore, the players were predominantly engaged in tackling and blocking activities at the time of mild head injury and hence these techniques involving the use of the head may be an important risk factor. Albright, Mcauley, Martin, Crowley, & Foster (1985) conducted a prospective study of 342 college football players over

[5] See Appendix V for a more detailed outline of the similarities and differences between the various forms of rugby/football-related sports.

an eight-year period and found that 29% of the players sustained head injuries during the eight seasons, with 87% of these being mild head injuries. Further, they found that offensive linemen had the highest frequency of head injuries when compared with defensive linemen. It was also noted that once the first injury had occurred, the probability of the individual incurring a future head (or neck) injury escalated to 42% and that 24% of the players had a recurrent injury to the head (or neck) in the same season as the original injury.

Neuropsychological Sequelae Following Mild Head Injury in American Football. Barth et al. (1989) conducted a four-year prospective study using a sample of 2350 football players at 10 American universities. Their brief neuropsychological test battery (including the Trail Making Test A and B, Aaron Smith's Symbol Digit Test, and the PASAT) was administered pre-season, 24 hours post-injury, 5 days and 10 days post-trauma, and at post-season. In order to control for test practice effects, the researchers included a group of college student controls and a group of players with orthopedic injuries. Barth et al. (1989) found that single mild head injury in football players caused deficits in the areas of attention and information processing when assessed within 24 hours of the trauma. However a pattern of rapid recovery seemed to occur over the following 5 to 10 days, in that no significant difference was noted between football players and student controls when the raw scores of the two groups were compared. Macciocchi, Barth, Alves, Rimel, & Jane (1996) provided a more recent account of the preliminary results reported by Barth et al. (1989). In their sample of 183 collegiate football players, Macciocchi et al. found that although the players with a single head injury demonstrated impaired performance when compared with the matched student control group, this impairment resolved within 5 days in the majority of players. Furthermore, head injured players demonstrated significant improvement between 24 hours and 5 days, and between 5 and 10 days. Deficits in both sustained auditory attention (PASAT) and visuomotor speed (Trail Making Test and Digit Symbol Test) were noticed, although these impairments were predominantly evident in players' failure to demonstrate improved performance over time, as opposed to impairment relative to baseline skill levels. Thus, the authors confirmed their initial clinical impression (see Barth et al., 1989) that neuropsychological dysfunction occurs after a *single* mild head injury in football and tends to be limited and brief in duration. However, although premorbid data and the repeated testing of matched control groups were employed to control for practice effects, there were still a number of methodological weaknesses. These researchers employed a limited neuropsychological test battery and, in focusing on single reported mild head injury, failed to consider previous mild or sub-concussive head injuries thereby limiting the generalisability of the findings to players who suffer multiple head traumas. In addition, they tended to focus on the acute or sub-acute

phase of single reported mild head injury and thus failed to enable the effective monitoring of a recovery curve or of residual symptoms.

Wilberger et al. (1991 in Wilberger, 1993) evaluated 62 high school football players who had sustained two mild head injuries within a single season using a neuropsychological test battery (comprising Wechsler Memory Scale, Selective Reminding Test, PASAT, Symbol Digit Modality, Stroop, and Trail Making Test) which was administered at 24 hours and 30 and 90 days post-mild head injury. They found that the initial neuropsychological tests were abnormal in most of the players, although these scores had normalised in all players at one month (with the exception of the PASAT, the Symbol Digit Modality, and the Stroop Test). At 3 months, the same tests showed continuing abnormalities in some of the players.

2.4.5.2. Australian Rules Football and Rugby League Football

Incidence of Mild Head Injury in Australian Rules Football and Rugby League Football. Australian Rules football is classified as a contact sport and hence injuries occur frequently (Maddocks et al., 1995). Amongst professional players, it has been found that nearly 25% of all injuries are to the head and neck region, and 5% of total injuries are mild head injuries (Dicker et al., 1986 in Maddocks et al., 1995). Seward et al. (1993) found that the most common injuries in rugby league and rugby union were head/facial lacerations (11% and 20%) followed by mild head injury (8% and 5%). They also noted that in these two rugby codes, minor head and neck injuries were more common, particularly in forwards. Gibbs (1993) reported a similar rate and distribution of injuries in Australian professional rugby league players as Seward et al. (1993), with rugby league demonstrating a high incidence of injury, with 5.7% of the total injuries being head injuries. Further, Gibbs found that the forwards (those players involved in more repetitive physical collisions) had more injuries than would have been expected, while the backs had fewer injuries than expected (the author did not provide a breakdown as to how many of these injuries involved head traumas). In a study of English professional rugby league players, Stephenson, Gissane, & Jennings (1996) found that the highest injury rates (33% of all injuries) took place in the head and neck region. This is higher than the figure of 5.7% quoted by Gibbs (1993) above. However, this difference may, in part, be accounted for by Stephenson et al.'s decision to include minor injuries. This study also demonstrated that the player being tackled is more likely to be injured (46.3%) due to being forcibly hit by other players and that forwards experienced greater rates of injury than backs. Finally, injury rates were shown to be higher at the highest standards of play.

Neuropsychological Sequelae Following Mild Head Injury in Australian Rules Football and Rugby League Football. In an attempt to account for the methodological problems in

previous American football studies (e.g. Barth et al., 1989), Maddocks & Saling (1991) obtained baseline (pre-injury) measures in a sample of 130 Australian Rules Football players using a neuropsychological test battery comprising the PASAT, Digit Symbol Substitution Test, and Four-Choice Reaction Time (involving measures of Decision Time and Movement Time). 10 players who subsequently suffered mild head injury were re-tested 5 days post-injury. An age-and education-matched control group of umpires was included in the study and assessed on two corresponding occasions. The researchers found impaired performances post-mild head injury on the Digit Symbol Substitution Test and Decision Time measures, indicating deficits in information processing and slowed decision and reaction time. A study by Maddocks et al. (1995) demonstrated no significant difference between the mild head injured and non-mild head injured professional Australian Rules football players on a test shown to be sensitive to mild head injury (i.e. the Digit Symbol Subtest from the WAIS-R) by the 6th month post-injury. Thus, this finding suggests that there is an absence of residual effects from earlier mild head injuries and that there is no evidence of cumulative effects from repeated mild head injury (as suggested by Gronwall & Wrightson, 1975). They also found that age and greater exposure to the game increased the likelihood of mild head injury. However, this study had a number of methodological flaws, including a reliance on players' accurate reporting of their mild head injury histories, the absence of a control group, and the use of a single neuropsychological test.

In another more recent Australian study, Hinton-Bayre et al. (1997) investigated the sensitivity of certain tests of speed of information processing to the acute effects of mild head injury in professional rugby league football players. 10 players were assessed within 24-48 hours following their mild head injury using a neuropsychological test battery (comprising the Digit Symbol Substitution Test, the Speed of Comprehension Test, and the Symbol Digit Modalities Test). When compared to baseline measures, the authors found that measures of speed of information processing (Digit Symbol Substitution Test and Symbol Digit Modalities Test) and speed of comprehension (Speed of Comprehension Test) were impaired in the post-acute phase of mild head injury, whereas an untimed word recognition task was not. The poorer performance post-injury on the Digit Symbol and Symbol Digit Modalities tests as compared to baseline maximum pre-injury scores is consistent with previous studies (e.g. Barth et al., 1989; Maddocks & Saling, 1991). While both the Maddocks & Saling (1991) and the Hinton-Bayre et al. (1997) studies had methodological strength in that they were prospective studies which utilised pre-injury data and control groups to account for practice effects, a few shared weaknesses were also noted. Both studies employed a limited test battery which does not enable testing across the entire range of cognitive functions that might be compromised in closed mild head injury. Furthermore, in attempting to examine the

effect of a single mild head injury (acute/sub-acute phase), this study failed to consider that the majority of contact sport players (particularly those at an elite/professional level) tend to have a history of previous mild or sub-concussive head injury in their sporting careers.

2.4.5.3. Rugby

Incidence of Mild Head Injury in Rugby. Nathan, Goedeke, & Noakes (1983) found that the incidence of schoolboy rugby injuries rose with increasing age and level of competence of players, with the most common injuries being mild head injury and muscle injuries, each accounting for 21.5% of all injuries. In addition, these researchers found that the forwards on average sustained more injuries than the backs. Roux, Goedeke, Visser, Van Zyl, & Noakes (1987) also found the incidence of mild head injury to be high (constituting 29% of all injuries) in schoolboy rugby players. They found that the top team players (of all age groups) suffered the greatest number of injuries (20%), that mild head injury (74%) occurred most frequently during tackling and loose scrummaging, and that mild head injury was the most common injury among eighthmen (forwards) and fullbacks (backs). An Irish study by McQuillan (1992) investigated the incidence of rugby injuries attending an accident and emergency department in a hospital in Dublin, Ireland. He found that head injuries accounted for 24% of all injuries (although most of these were minor head injuries), and that 54.3% of injuries were sustained by forwards and 45.9% by backs.

Neuropsychological Sequelae Following Mild Head Injury in Rugby. A South African study conducted by Shuttleworth-Jordan, Balarin, & Putchert (1993) assessed 60 university rugby players and 25 matched non-contact sport controls on a fairly comprehensive neuropsychological test battery, including tests of short-term verbal memory (Digit Span forwards), verbal new learning (Digit Supraspan), working memory (Trail Making Test parts A & B and Digit Span backwards), and hand-motor dexterity (Denckla Finger Tapping Test and Purdue Pegboard Test). The researchers found that the rugby players were impaired in the areas of working memory, verbal new learning ability, and hand motor dexterity - a pattern of impairment typically associated with closed head injury caused by diffuse brain damage effects. The rugby group scored significantly faster Finger Tapping Test scores (which was inconsistent with the general trend of the results), but the differences were essentially points of a second and the authors felt that it was impossible to score this test rigorously enough to ensure reliable differences with respect to points of a second. Additional analysis revealed greater impairment among the forwards when compared to the backs, which was explained in terms of the forward players participating in scrumming thereby predisposing these players in particular to cumulative brain damage effects. The prospective analysis of the mild head injured group of rugby players revealed significant

impairment in immediate auditory attention, verbal new learning ability, working memory, and hand motor dexterity at 3 days post-injury. Substantial recovery was indicated at 1 month and further recovery at 2 months post-injury. However, at the 3 month interval, the mild head injured rugby group was still not demonstrating the same degree of practice effects as the controls on Digits Backward, Digits Difference, Digit Supraspan A and B, and the Finger Tapping Test (Preferred and non-Preferred Hands), indicating that recovery on these measures was not complete. This study's methodological strengths lie in its utilisation of baseline date as well as the repeated differences between rugby players and non-contact sport controls, and the inclusion of a wider variety of tests particularly sensitive to the presence of diffuse brain damage.

Most recently, in order to build on the Shuttleworth-Jordan et al. (1993) study, a large project investigating head injury in rugby was initiated in 1997 by Rhodes University in collaboration with the South African Rugby and Football Union and the South African Sports Science Institute in Cape Town. The initial phase of the research compared the cognitive performance of 26 elite professional rugby players (the Springboks) with a matched control group of 21 elite professional cricket players (the Proteas) on a wide variety of neuropsychological measures. These data from the first phase of the research were analysed in three different research projects: (1) A direct comparison of group mean scores between Springbok rugby players and Protea cricket controls (Ancer, 1999); (2) A comparison of the cognitive profiles of the Springbok rugby players relative to the Protea cricket controls against available normative data (Reid, 1998); (3) A comparison of percentage cognitive deficit across each test modality for Springbok rugby players relative to Protea cricket controls, as well as a comparison of the frequency of reported postconcussive symptomatology in rugby players and controls (Dickinson, 1998). The latter two projects by Reid (1998) and Dickinson (1998) are complete and the results will be discussed below, while that of Ancer (1999) is still in the process of being completed.

In Reid's (1998) study, no significant differences in mean scores between the rugby and cricket players relative to the norms were found. However, an increased variability in the rugby playing group relative to the control group on tasks sensitive to diffuse brain damage was noted. Further subgroup analysis revealed significant impairment in the subgroup of forward players on the more challenging neuropsychological tests which was not demonstrated in the subgroup of back-line players. With respect to these tasks, strong indicators of reduced variability for forward players compared with the full rugby group were evidenced, indicating that most forward players have suffered some neuropsychological impairment, probably due to their increased positional exposure to repeated head injures.

Utilising a different method of data analysis, Dickinson (1998) reported similar findings to Reid and found cognitive deficit to be particularly evident in the subgroup of forward players relative to the back players on certain neuropsychological tests. In addition, Dickinson (1998) found that rugby players demonstrated impairment in the areas of visuoperceptual tracking (Digit Symbol Subtest), speed of information processing and attention (Trail Making Test) and, further, that there were tendencies towards impairment in the areas of verbal and/or visual memory (Digits Forward and Digit Symbol Incidental Recall). Thus, in general, positive results on tests particularly sensitive to brain damage were found, supporting the hypothesis that rugby players' cognitive functioning is impaired due to cumulative concussive and sub-concussive mild head injuries.

However, these studies did have some methodological weakness, including a relatively small sample size, and a problematic control group – cricket players were fatigued/unmotivated because they were tested post-season (whereas the rugby players were tested pre-season) and it was hypothesised that they were also depressed (they had lost the season). Furthermore, many of the cricket players participated in rugby during their off-season and consequently might have sustained cumulative mild head injuries, which may have resulted in an under-estimation of the extent of deficit in the rugby playing group. Although the third project by Ancer has not been completed, the methodological limitations pertain. With these limitations in mind, Reid and Dickinson recommended that their research be replicated using larger numbers of participants and a less confounded control group.

2.5. BRAIN RESERVE CAPACITY (BRC) THEORY

In order to locate the present research in a theoretical context, the theory of brain reserve capacity (BRC) as formulated by Satz (1993) is utilised. BRC theory is concerned with underlying neural processes, and the concept of BRC is associated with the idea of a threshold factor, which is present before the occurrence of symptoms due to disease in the central nervous system. BRC (which corresponds to the amount of functional brain tissue) represents physiological brain advantages or disadvantages, and general intelligence and educational level serve as the two psychosocial factors that represent indirect measures of BRC. Satz's theory is based on the hypothesis that the greater the BRC, the less the likelihood of an individual demonstrating symptoms of neuropsychological impairment, as greater BRC tends to serve as a *protective factor* and tends to decrease the risk of functional impairment. Thus, even in the presence of brain-damage, greater BRC will be reflected in higher premorbid IQ scores and higher levels of cognitive functioning. In addition, Satz hypothesises that the less

the BRC, the greater the likelihood of an individual demonstrating symptoms of neuropsychological impairment, as less BRC tends to serve as a *vulnerability factor* causing greater risk of functional impairment. In this case, the threshold (the critical amount of brain tissue at which normal functioning can be sustained) will be lower. Furthermore, based on Satz's theory, any reduction in BRC due to neurological pathology is likely to increase an individual's vulnerability to functional impairment. In this regard, research has indicated that mild head injury may be a predisposing (risk) factor to Alzheimer's disease (Rasmusson, Brandt, Martin, & Folstein, 1995) and it may further be argued that this dementing illness occurs as a result of a lowered brain reserve capacity.

In his review, Satz mentions a number of risk factors (including lower levels of education and, by implication, IQ, gender effects, and the effects of what he calls 'challenge') which may serve to reduce BRC. With regard to these risk factors, low education and IQ, as well as appropriately high task challenge, serve to lower the threshold and increase vulnerability to symptom onset. In addition, male gender membership may serve to enhance vulnerability to symptom onset. As noted earlier, a study conducted by Ewing et al. (1980) provides support for the notion that high task challenge may lead to a reduced BRC. Ewing et al. demonstrated that young mild head injured patients exhibited cognitive deficits during the stress of mild hypoxia, although no evidence of such impairment was evident when these subjects were initially tested under non-hypoxic conditions.

Both Satz (1993) and Jordan (1997) describe normal aging as a phenomenon which causes neuronal attrition, and which serves to reduce BRC and to increase the risk of functional impairment. Thus, advancing age is associated with a process of progressive neural attrition which causes a reduction in cerebral reserves resulting in the onset of deteriorated function. In addition, the normal aging process may be conceptualised as involving cumulative mild brain insults. Shuttleworth-Jordan (1999) extrapolates from aging as a form of progressive mild neurological insults to cumulative mild head injury in rugby, and using the concepts of BRC theory, proposes a number of specific hypothetical indications for a mild head injury study in rugby. In essence, she hypothesises that cumulative mild head injuries in rugby players are likely to reduce BRC and to increase the risk of functional impairment. Therefore, in a comparison of rugby versus non-contact sport playing groups, if certain risk factors are controlled for (i.e. given that gender is constant, that the groups are matched for levels of education and IQ, and that individuals with a history of neurological or psychiatric disorder are excluded), then it is possible to take this theme further and argue the following. Firstly, it may be argued that the rugby players' exposure to cumulative mild concussive and sub-concussive head injuries relative to the controls is likely to cause a reduction in BRC and to

increase the players' vulnerability to neuropsychological deficit. Secondly, it may be argued that the subgroup of forward rugby players who incur greater involvement in repetitive physical collisions (and hence increased exposure to cumulative mild head insults) relative to the subgroup of rugby back-line players, is likely to cause a reduction in BRC and to increase the forward players' vulnerability to neuropsychological deficit.

2.6. RATIONALE AND HYPOTHETICAL INDICATIONS FOR THE PRESENT STUDY

In the light of the previously discussed methodological limitations (including small sample numbers) of the first phase of the larger research study, it was decided by the researchers of the second phase (including the present study) to extend the existing sample numbers of top-level rugby players to include the group of Under 21 national rugby players. Further, because the Protea cricket group was viewed as a somewhat confounded control group (they played rugby and were tested post-season), a new control group comprising national hockey players, who were assessed pre-season, was created. The rationale for including the hockey group was that hockey is considered to be a non-contact sport and, because hockey is played in the same season as rugby, a decreased likelihood of players being involved in both sports was assumed.

Taking into consideration the empirical research findings of the literature review, the theoretical indications of Satz's (1993) brain reserve capacity (BRC) theory, and the hypothetical indications for a mild head injury study in rugby proposed by Shuttleworth-Jordan (1999), the following hypotheses were posed:

(1) Rugby players (comprising Springbok and Under 21 rugby players) are likely to show greater impairment on cognitive tasks known to be sensitive to diffuse brain damage compared with hockey players, due to the rugby players' increased exposure to long-term cumulative mild concussive and sub-concussive head injuries. The cumulative mild head injuries suffered by the rugby players are likely to cause neural change, which serves to lower the critical threshold at which functional symptomatology will manifest, which in turn is likely to lead to less BRC, thereby increasing the players' vulnerability to neuropsychological deficit.

(2) The subgroup of rugby forward players are likely to show greater impairment on cognitive tasks known to be sensitive to diffuse brain damage compared with the subgroup of rugby back-line players, due to the forward players' increased involvement in repetitive physical collisions and hence their increased exposure to head insults. The

cumulative mild head injuries suffered by the rugby forward players are likely to cause neural change, which serves to lower the critical threshold at which functional symptomatology will manifest, which in turn is likely to lead to less BRC, thereby increasing the players' vulnerability to neuropsychological deficit.

CHAPTER THREE: METHODOLOGY

This project forms part of a larger and ongoing research study investigating the brain damage effects of cumulative mild head injury in rugby. The study was initiated in 1997 by Rhodes University in collaboration with the South African Rugby and Football Union (SARFU) and the South African Sports Science Institute in Cape Town. The initial phase of the research compared the cognitive performance of Springbok professional rugby players on a wide variety of neuropsychological measures with the cognitive performance of a matched non-contact sport control group consisting of Protea professional cricket players. The present study, which comprises the second phase of the larger research study, aims to replicate Ancer's (1999) study (which involved a direct comparison of group mean scores between Springbok rugby players and Protea cricket controls) using a larger sample size and a new control group. To this end, the present study compared the cognitive performance of Springbok professional rugby players and Under 21 national rugby players with the performance of a matched non-contact sport control group consisting of national hockey players.

3.1. PARTICIPANTS

The sample was selected from a South African population of elite sportsmen. The sample for analysis comprised Springbok professional rugby players (n = 26) and Under 21 rugby players (n = 19), and a non-contact sport control of national hockey players (n = 21). The Springbok rugby players and the Under 21 rugby players were tested in February 1997 and February 1998 respectively during their pre-season medical, physical, and psychological assessment at the South African Sports Science Institute in Cape Town. The hockey players (matched non-contact sport controls) were tested individually pre-season at varying times from December 1998 to March 1999 in their home towns, as the researchers were denied access to them at their group pre-season medical evaluation.

The majority of sportsmen in the study (including Springbok and Under 21 rugby players, and hockey players) reported the incidence of at least one mild head injury in their history. As recommended by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (Satz et al., 1997), mild head injury was defined as the presence of at least one of the

following criteria: a duration of loss of consciousness of 30 minutes or less, with a GCS of 13-15 following the loss of consciousness; any loss of memory for events immediately preceding or proceeding the accident with a PTA for less than 24 hours; any change in the mental state at the time of the accident (e.g. dazed, disoriented, or confused); and focal neurological deficits that may or may not be transient. No participants were excluded on the basis of reports of isolated incidents of previous mild head injury, as this would have effectively excluded the majority of participants and furthermore, the purpose of this study is to investigate the effects of *cumulative* mild head injury expected to occur in rugby players and not isolated mild head injury in a participant's history.

In order to control for the effects of potentially confounding variables on cognitive performance, the following exclusion criteria were adhered to: a reported history of substance abuse; a neurological or psychiatric disorder known to negatively impact on cognitive functioning; and a reported history of moderate to severe non-sport related head injury (defined as any head injury which was too severe to classify as a mild head injury). No participants were excluded on the basis of these criteria.

Further, in order to control as far as possible for levels of IQ, it was necessary to establish an e*stimated premorbid IQ* for each of the rugby and control group players, which was accomplished in one of two ways. Lezak (1995, p. 106-108) refers to "The best performance method" which states that it is possible to use the single highest score of tests which are known to be good indicators of premorbid IQ and tend to hold up well in most brain damaged persons. The SAWAIS Comprehension Subtest and SAWAIS Picture Completion Subtest are two such tests. Thus, in this study, the estimated premorbid level of intellectual functioning was calculated for each participant using both these tests, except in instances where one of the two subtest scores was defective (subscale score < 8.5) and it fell three or more scalepoints below the subscale score of the other subtest. In the above-mentioned exceptional instance, the premorbid IQ was prorated using the single highest score in isolation. In such instances it was considered that the single score was a more valid indicator of premorbid IQ, as per Lezak (1995). Use of only one subtest score was applicable for three participants (see Appendix IV for the complete list of years of education and prorated IQ scores for all participants). On the basis of these IQ estimates, participants with IQ scores falling in the lower and upper extremes were excluded from the study. Since there were some extreme low IQ scores among the Under 21 rugby players and some extreme high IQ scores among the hockey players, it was considered that these extreme scores could bias the data in the direction of poorer performance for the Under 21 players due to lower IQ, and confound evidence for specific brain damage effects. Thus, any IQ falling in the defective range (IQ < 85) and any IQ equal

to or greater than the exceptionally superior range (IQ > 140) were excluded. On the basis of this exclusion criterion, two Under 21 rugby players (IQ < 85) and two national hockey players (IQ > 140) were excluded. No participants from the Springbok rugby group were excluded, since there were no players in the low or high extremes of IQ in this group (i.e. all these players fell in the IQ range 85 – 140).

The final sample for the study consisted of a total of 66 elite sportsmen, including a Total Rugby group (Springbok and Under 21 rugby players) (n = 45) and a Hockey Control group (n = 21). The Total Rugby group was divided into Springbok Rugby (n = 26) and Under 21 Rugby (n = 19), and into Total Rugby Forwards (n = 26) and Total Rugby Backs (n = 19). The Total Rugby Forwards were further divided into Springbok Rugby Forwards (n = 15) and Under 21 Rugby Forwards (n = 11) and the Total Rugby Backs into Springbok Rugby Backs (n = 11) and Under 21 Rugby Backs (n = 8). See Tables 3-1 and 3-2 below for demographic data of the participants.

Table 3-1: Rugby Group Comparison of Means with respect to Demographic Data

Group	n	Age			Education			Estimated Premorbid IQ		
		Mean	SD	p-value	Mean	SD	p-value	Mean	SD	p-value
Total Rugby	45	24.20	4.40	0.3674	13.40	1.74	0.0307 *	115.42	12.17	0.0306 *
Hockey Control	21	23.24	2.98		14.30	1.24		122.00	8.91	
Springbok Rugby	26	27.46	2.73	0.0000 **	14.19	1.41	0.7213	119.19	11.96	0.3763
Hockey Control	21	23.24	2.98		14.30	1.24		122.00	8.91	
Under 21 Rugby	19	19.74	0.73	0.0000 **	12.32	1.57	0.0001 **	110.26	10.72	0.0005 **
Hockey Control	21	23.24	2.98		14.30	1.24		122.00	8.91	

Significant Difference (* p<0.05; ** p<0.01)

Table 3-2: Within Rugby Group Comparisons of Means with respect to Demographic Data

Group		n	Age			Education			Estimated Premorbid IQ		
			Mean	SD	p-value	Mean	SD	p-value	Mean	SD	p-value
Total Rugby:	Forwards	26	23.96	4.40	0.6755	13.27	1.73	0.5608	114.81	12.21	0.6967
	Backs	19	24.53	4.50		13.58	1.77		116.26	12.40	
Springbok Rugby:	Forwards	15	27.20	2.78	0.5791	13.87	1.41	0.1754	117.20	12.27	0.3312
	Backs	11	27.82	2.75		14.64	1.36		121.91	11.52	
Under 21 Rugby:	Forwards	11	19.55	0.52	0.1900	12.45	1.86	0.6635	111.55	11.92	0.5562
	Backs	8	20.00	0.93		12.13	1.13		108.50	9.30	

Significant Difference (* p<0.05; ** p<0.01)

The demographic data (age, education, and estimated premorbid IQ) (see Tables 3-1 and 3-2 above) were analysed using a pooled independent two sample t-test, and the comparison of means across all the rugby groups and the control group were conducted. These analyses were carried out to determine whether or not the rugby sample and the control group were matched on certain demographic data.

With respect to *age*, there was a statistically significant difference ($p < 0.01$) between Springbok Rugby and Hockey Control (with Springbok Rugby players being older than Hockey Control players by an average of 4 years), and a significant difference ($p < 0.01$) between Under 21 Rugby and Hockey Control (with Under 21 Rugby players being younger than Hockey Control players by an average of 3½ years). Considering that age norms tend to span a decade and that age only begins to make a difference in neuropsychological performance from approximately age 60 (Lezak, 1995), the practical implications of this statistical difference is probably negligible. There was no significant difference between Total Rugby and Hockey Control for age, and no significant difference between the Forwards and Backs for any of the Rugby groups.

With respect to *education*, there was a statistically significant difference ($p < 0.05$) between Total Rugby and Hockey Control (with Total Rugby players having a lower education level than Hockey Control players by an average of 9 months), and a significant difference ($p < 0.01$) between Under 21 Rugby and Hockey Control (with Under 21 Rugby players having a lower education level than Hockey Control players by an average of 2 years). Thus, the

difference between the education of Total Rugby and Hockey Control in essence amounted to less than 1 year, and further there is a strong possibility that the Under 21 Rugby players had not completed their tertiary education due to their younger age. This, no doubt, would most likely have resulted in an underestimate of the potential educational level of the Under 21 group. There was no significant difference between Springbok Rugby and Hockey Control for education, and no significant difference between the Forwards and Backs for any of the Rugby groups.

With respect to *estimated premorbid IQ*, the identical pattern as with education was noted. There was a statistically significant difference ($p < 0.05$) between Total Rugby and Hockey Control (with Total Rugby players having a lower estimated premorbid IQ than Hockey Control players by an average of 6.5 points), and a significant difference ($p < 0.01$) between Under 21 Rugby and Hockey Control (with Under 21 Rugby players having a lower estimated premorbid IQ than Hockey Control players by an average of 12 points). Examination of the differences reveals that the estimated premorbid IQ of Total Rugby and Hockey Control is broadly in a similar range and that the difference between these two groups is marginal. However, the difference between Under 21 Rugby and Hockey Control is possibly more meaningful in that Under 21 Rugby is on the borderline average/above-average range of intellectual functioning, whereas Hockey Control is in the superior range. There was no significant difference between Springbok Rugby and Hockey Control for estimated premorbid IQ, and no significant difference between the Forwards and Backs for any of the Rugby groups.

In sum, with respect to the demographic data, there are some differences between groups, particularly between Under 21 Rugby and Hockey Control in terms of age, education, and estimated premorbid IQ. It can be argued, however, that these differences (with the exception of estimated premorbid IQ for Under 21 Rugby versus Hockey Control) fall within relatively close ranges which probably are not of clinical significance. Positional comparisons (Forwards versus Backs) within the rugby groups revealed no significant differences between them with respect to all demographic variables.

3.2. PROCEDURE

The Springbok rugby payers were tested at the South African Sports Science Institute between the 2nd and 5th of February 1997 during their pre-season medical, physical, and psychological assessment. The testing was carried out by the research team involved in the

initial phase of the research, comprising a core research team (research coordinator and three Intern Clinical Psychologists) and one research assistant. The Under 21 rugby players were tested at the South African Sports Science Institute on the 24th and 25th of February 1998 during their pre-season medical, physical, and psychological assessment. For this second phase of the research, a core research team (comprised of three Intern Clinical Psychologists) as well as a team of assistant researchers (comprised of two qualified Clinical Psychologists and one Research Psychology Master's student) were involved in the administration of the neuropsychological test battery. The national hockey players were tested individually pre-season over the course of four months (December 1998 to March 1999) in their home towns. The three members of the core research team shared the responsibility of locating, contacting, and testing individual hockey players.

The researchers and their assistants all received their training at the same university and were experienced in the administration of the neuropsychological tests included in the battery. In order to ensure uniformity across test administration, all the materials (including the consent form, questionnaires, and test protocols) and the administration of the tests, in particular, were discussed with the research assistants prior to the testing. Further, each test protocol was furnished with standardised written test instructions either from the original manual or from Lezak (1995).

Each participant was tested individually for approximately a two-hour period. While the rugby players were assessed in private offices at the South African Sports Science Institute, the hockey players were either tested in a quiet room at their homes or in an office at their place of employment. Most of the participants were English-speaking, although those who were better versed in Afrikaans were interviewed and tested in their first language so as to put them at their ease and give them the opportunity to perform to the best of their ability. Prior to the official assessment, each participant was provided with a thorough explanation as to the nature and purpose of the testing procedure, as well as with an opportunity to clarify any concerns. Besides providing information, this explanation aimed to reduce any anxiety, which may otherwise have negatively impacted on test performance.

3.3. CONSENT FORM AND QUESTIONNAIRES

Before testing began, each participant was requested to sign a written consent form (see Appendix I). The participant was then required to complete a demographic questionnaire (see Appendix II) which provided information on biographical details (including age, highest level

of education, occupational history, current level of functioning), sporting history, previous head injuries (both sports and non-sports related), and alcohol/substance usage. This questionnaire was used as a tool for gathering information about participants to be used in determining their suitability for inclusion in the study. A self-report postconcussive symptomatology questionnaire was then administered to the participant. It consisted of 31 items and was designed to assess the occurrence of a range of residual postconcussive symptoms. This questionnaire, however, did not form part of the focus of this study and hence is not included in the appendices.

3.4. NEUROPSYCHOLOGICAL TEST BATTERY

A comprehensive neuropsychological test battery was designed to include tests which have been shown to be sensitive to the effects of diffuse brain damage, which are typically associated with a closed head injury. The battery included tests measuring abilities and current functioning across a variety of cognitive modalities (e.g. attention and concentration, memory and learning, verbal fluency, visuoperceptual tracking, and hand-motor dexterity) which tend to be compromised when a closed head injury is sustained (see Lezak, 1995 and literature review). The tests employed also enabled the researchers to calculate an estimated premorbid level of cognitive functioning for each of the participants. The test battery included the following tests listed in the order of administration: South African Wechsler Adult Intelligence Scale (SAWAIS) Digit Symbol Subtest; Digit Symbol 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; Finger Tapping Test (Trial 1); Digit Symbol Incidental Recall (Delayed); Wechsler Memory Scale (WMS) Visual Reproduction Subtest (Immediate Recall); SAWAIS Picture Completion Subtest; SAWAIS Comprehension Subtest; WMS Visual Reproduction (Delayed Recall); WMS Associate Learning Subtest (Immediate Recall); SAWAIS Digit Span Subtest; Digit Supraspan; Digit Supraspan Sustained Learning; Finger Tapping Test (Trial 2); WMS Associate Learning Subtest (Delayed Recall). These tests measure functioning across major cognitive modalities including general intellectual functioning, verbal memory, visual memory, verbal fluency, visuoperceptual tracking, and hand-motor dexterity. Each test will be discussed in more detail under each modality. See Appendix III for the assessment schedule, including the above-mentioned neuropsychological tests.

3.4.1. GENERAL INTELLECTUAL FUNCTIONING

In this research, estimates of premorbid levels of cognitive functioning were deemed necessary for each of the participants in order to provide a comparison standard for that player, thereby assisting in estimating more accurately the level of individual deficit (Lezak, 1995). For this purpose, two subtests from the South African Wechsler Adult Intelligence Scale (SAWAIS), namely SAWAIS Picture Completion Subtest and SAWAIS Comprehension Subtest, were utilised. The method of calculating premorbid levels of IQ is discussed in section 3.1.

3.4.1.1. South African Wechsler Adult Intelligence Scale (SAWAIS)

Picture Completion Subtest. This test consists of 15 incomplete pictures, arranged in order of difficulty. As per SAWAIS manual (1969) instructions, the tester presented the cards in numerical order to the participant and requested that the testee indicate the most important missing part. There was a 20-second time limit per card. Picture Completion tests visual recognition, as well as visual organisation and reasoning abilities. It also tests remote memory, general information, and judgements concerning the relevance of both practical and conceptual issues. Picture Completion is a good indicator of premorbid ability and tends to hold in individuals with diffuse brain damage (Lezak, 1995).

Comprehension Subtest. This test consists of 10 open-ended questions involving common-sense judgement and practical reasoning, arranged in order of difficulty. As per SAWAIS manual (1969) instructions, the participant was instructed to respond to each question by telling the tester what he thought in each case. The testee was also informed that there were no fixed answers to the questions. If any answer was unclear or inadequate, the tester asked the participant for elaboration. Comprehension tests verbal reasoning and social judgement, conventionality, or common sense. Similarly to Picture Completion, Comprehension is a very good indicator of premorbid ability and serves well as a hold test (Crosson, Greene, et al., 1990, in Lezak, 1995).

3.4.2. VERBAL MEMORY

3.4.2.1. SAWAIS Digit Span Subtest

This test consists of two separate tests, namely Digits Forwards and Digits Backwards. As these two tests involve different mental activities and are differentially affected by brain damage (Lezak, 1995), they are separated out and examined individually. It is generally accepted that Digits Backwards is more sensitive to the effects of diffuse brain damage than Digits Forwards (Lezak, 1995).

Digits Forwards. This test consists of a series of different number sequences, arranged in order of difficulty. As per SAWAIS manual (1969) instructions, the tester presented the series of numbers to the participant at a rate of one number per second and then asked the testee to repeat that series in the same order. For each span, there is a second span of equal length (different numbers) and the test is failed after incorrect repetition of both trials of a span. If the testee correctly repeated (either trial of) a span, then the next span (comprising a new sequence with one extra number) was attempted. The score is the best span number achieved. Digits Forwards tests immediate memory span, but is primarily a measure related to the efficiency of attention (i.e. freedom from distractibility) (Kaufman, McLean, & Reynolds, 1991). This test is not as sensitive to the effects of diffuse brain damage as Digits Backwards and tends to hold in instances of such damage (Barth et al., 1989).

Digit Supraspan. This test is an extension of Digits Forwards and utilises the method proposed by McFie (1975). Following the second consecutive failure of a digit span on Digits Forward, the last failed sequence was repeated by the tester until the testee was able to recall and repeat the span correctly. The score is the number of trials the participant takes to learn the sequence correctly (with the first repetition of the previously failed span counting as learning trial 1). Digit Supraspan tests verbal new learning, a task which is more sensitive than Digits Forwards to the effects of memory impairment (Lezak, 1995).

Digit Supraspan Sustained Learning. The requirement of this test is that the participant repeats the Supraspan correctly twice in a row. The testee's score is based on the number of trials necessary to fulfil this criterion. This test measures sustained learning.

Digits Backwards. As with Digits Forwards, this test consists of a series of different number sequences, arranged in order of difficulty. The instructions were similar to those used for Digits Forwards, with one important exception - the participant was asked to repeat the span in reverse order. The score was the longest sequence of digits correctly recalled in reverse order. Digits Backwards involves mental double-tracking (both the memory and the reversing operations need to proceed simultaneously) and working memory. This test is particularly sensitive to many kinds of brain damage, including diffuse damage (Lezak, 1995).

3.4.2.2. Wechsler Memory Scale (WMS)

Associate Learning Subtest (Immediate Recall). The original Wechsler format (Form 1 version) and manual (Wechsler, 1945) were employed for the administration and scoring of this test. WMS Associate Learning consists of ten word pairs, comprising six "easy" pairs that

are readily associated and four "hard" pairs that are not easily associated. The tester read the list of ten word pairs to the participant and then read out the first word only of each pair and instructed the testee to recall the associated word. This procedure was repeated three times in total. An Afrikaans translation (Burbach, 1987) was utilised for the Afrikaans-speaking participants. This test measures two different activities, namely the recall of well-learned verbal associations ("easy" pairs) and the retention of new, unfamiliar verbal material ("hard" pairs). Because "hard" pairs require new learning, they tend to be more sensitive to the effects of brain damage (Lezak, 1995). This study maintained the separateness of activity by examining the results as separate scores, as opposed to a single combined one.

Associate Learning Subtest (Delayed Recall). This test was administered after a 20-minute delay, as delayed memory tends to be more sensitive than immediate memory to the effects of diffuse brain damage (Lezak, 1995). For the delayed version of Associate Learning, the first word of each word pair was read out to the participant who was required to recall the associated word.

3.4.3. VISUAL MEMORY

3.4.3.1. Digit Symbol Incidental Recall

Digit Symbol Incidental Recall (Immediate). This study made use of a short form of Incidental Recall (Shuttleworth-Jordan & Bode, 1995) as originally described by Kaplan et al. (1991, in Lezak, 1995 p. 463). On completion of the Digit Symbol Subtest, the tester marked the last symbol drawn by the testee at 90 seconds. Any participant who had failed to complete the digit symbol substitutions to the end of the second last row, was then requested to do so. This was to ensure exposure to all of the digit pairs up to 9. The testee was then given a separate sheet of paper marked with the numbers 1 – 9 and was requested to fill in the symbols he could recall which corresponded to each number. Digit Symbol Incidental Recall is a test of recent memory which has been shown to be sensitive to the effects of diffuse brain damage (Walsh, 1985).

Digit Symbol Incidental Recall (Delayed). After a 20-minute delay, the participant was again handed a fresh sheet of paper marked with the numbers 1 – 9 and was requested to fill in as many symbols (corresponding to each number) as he could recall. This delayed version was included in the neuropsychological battery because, as stated above, delayed memory tends to be more sensitive than immediate memory to the effects of diffuse brain damage (Lezak, 1995).

3.4.3.2. WMS Visual Reproduction

WMS Visual Reproduction (Immediate Recall). The original Wechsler format (Form 1 version) and manual (Wechsler, 1945) were employed for the administration and scoring of this test. WMS Visual Reproduction consists of three cards - Cards I and II are each furnished with a single design, while Card III has two designs. Each card was shown to the participant for 10-seconds, following which the testee was required to draw what he could remember of that design. This procedure was repeated for all 3 cards. This test taps into visual memory function and visuospatial problem solving ability (Lezak, 1995), and has been shown to be particularly sensitive to the effects of head injury (Stuss et al., 1985, in Lezak, 1995).

WMS Visual Reproduction (Delayed Recall). After a 20-minute delay, the participant was handed a blank sheet of paper and requested to draw the designs again (without being shown them for a second time). The delayed version was included, as delayed memory tends to be more sensitive than immediate memory to the effects of diffuse brain damage (Lezak, 1995).

3.4.4. VERBAL FLUENCY

3.4.4.1. Words in One Minute Unstructured Verbal Fluency Test (Terman & Merrill, 1973)

The participant was instructed to say as many different, unconnected words as he could think of in 1-minute, with the exception of: proper nouns (examples were given); use of the same word with a different suffix (examples were given); and counting or speaking in sentences. The tester then gave the participant examples of different, unconnected words. If necessary, the instructions were repeated until the participant clearly understood what was required. This unstructured test of verbal fluency is a sensitive indicator of brain dysfunction and it has been found that frontal lesions, in particular, tend to depress fluency scores (Miceli et al., 1981, in Lezak, 1995).

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

The same instructions as in 3.4.4.1 above were given to the participant, except that for this test the testee was only permitted to use unconnected words beginning with the letter "S". Again, a 1-minute time limit was enforced. As a verbal fluency test, this test is also sensitive to the effects of brain damage (Lezak, 1995).

3.4.5. VISUOPERCEPTUAL TRACKING

3.4.5.1. Digit Symbol Subtest

This test consists of three rows containing, in total, 75 blank squares, each paired with a randomly assigned number (1 to 9). Of the 75 squares, the first 8 serve as sample blank squares. Above these rows is a printed key in which the digits 1 to 9 are each paired with a symbol. As per SAWAIS manual (1969) instructions, the tester instructed the participant to fill in the blank spaces with the symbol paired with the number above each space as quickly and accurately as possible. By means of demonstration, the tester completed the sample section consisting of 8 blocks. The participant was then instructed to fill in as many symbols as possible, in order and without omitting any blank squares. The testee was urged to continue if he paused to correct an item during the test. The number of filled in blank squares was noted at 90-seconds for the purposes of the incidental recall task described in 3.4.3.1. This is predominantly a test of complex visuoperceptual tracking, although it does tap into many other functions including motor persistence, sustained attention, response speed, and visuomotor coordination (Lezak, 1995). This test is very sensitive to brain damage and tends to be affected regardless of the locus of the lesion and even when damage is minimal (Joy et al., 1992a, in Shuttleworth-Jordan & Bode, 1995). It is a good indicator of diffuse brain damage, commonly associated with a closed head injury.

3.4.5.2. Trail Making Test (Reitan, 1956)

The Trail Making Test is a test of complex visual scanning and involves motor speed and attention functions, and consequently is very sensitive to the effects of brain injury (Spreen & Benton, 1965). It is presented in two parts, Part A and Part B. It is generally accepted that Part B is more sensitive to the effects of diffuse brain damage than Part A (Lezak, 1995).

Trail Making Test (Part A). The participant was instructed to draw lines to connect consecutively numbered circles on a sheet of paper as quickly as possible, and without lifting the pencil from the paper. The testee first completed a sample trial (numbers from 1 to 8), before proceeding onto the test which consisted of 25 numbered circles. If the participant made an error during the test, this was pointed out to him, and he was required to make the correction. The score is the time taken to complete the trial.

Trail Making Test (Part B). The administration of Part B was similar to that of Part A, with the exception that the participant was instructed to alternately connect numbered and lettered circles (i.e. 1 – A, 2 – B etc. ending with the number 13). Part B, which involves complex conceptual tracking, working memory, and the ability to shift a response set, is particularly sensitive to the effects of diffuse brain damage (Lezak, 1995).

3.4.6. HAND-MOTOR DEXTERITY

3.4.6.1. Finger Tapping Test (Denckla, 1973)

The participant was instructed to place both elbows on the table with his hands in the air (the tester demonstrated) and then to touch each finger to his thumb beginning with the index finger (the tester demonstrated), as quickly as he could manage. The participant was given the opportunity to practice the sequence before attempting the timed trial. The score is the time taken to perform five sets of four taps with each hand. Two trials (each including taps with preferred and non-preferred hand) of this test were administered, with a delay between them, in order to obtain a measure of the participant's best performance. Tests of hand-motor dexterity are reportedly sensitive to the effects of brain damage (Shuttleworth-Jordan et al., 1993).

3.5. DATA PROCESSING

The test protocols for the Springbok Rugby players were scored by the original research team (comprising three Intern Clinical Psychologists), while those of the Under 21 Rugby and Hockey players were scored by the present research team (comprising three Intern Clinical Psychologists), who strictly adhered to the agreed scoring procedure so as to ensure test scoring uniformity. There was a bridging scorer to ensure consistency of scoring and all scoring was counterchecked (including the original protocols). Three separate research projects emerged from the data:

(1) A direct comparison of group mean scores across each neuropsychological test for Total Rugby players (Springbok Rugby and Under 21 Rugby) versus Hockey Control. In addition, analyses were conducted for the subgroups of Springbok Rugby versus Hockey Control, Under 21 Rugby versus Hockey Control, Total Rugby Forwards versus Total Rugby Backs, Springbok Rugby Forwards versus Springbok Rugby Backs, and Under 21 Rugby Forwards versus Under 21 Rugby Backs;

(2) A comparison of group mean scores across each neuropsychological test for Total Rugby players (Springbok Rugby and Under 21 Rugby) and Hockey Control with available normative data. In addition, subgroup analyses were conducted as outlined in (1) above.

(3) A comparison of the percentage of players with cognitive deficits on each neuropsychological test for rugby players relative to a norm established on the basis of the means for the non-contact sport control (Hockey Control), as well as a comparison of the frequency of cognitive deficit and postconcussive symptomatology in rugby players

and non-contact sport control (Hockey Control). In addition, subgroup analyses were conducted as outlined in (1) on p. 49.

The present project focused on the first level of analysis, namely a direct comparison of group mean scores for Total Rugby players (Springbok Rugby and Under 21 Rugby) and Hockey Control, as well as the additional analyses for the subgroup comparisons as outlined in number (1) on p. 49. This comparative study employed a matched control group of high-level hockey players, which is considered preferential to comparing the sample to available norms. Although head injury studies without control groups have tended to use available normative data, this norm comparison may not be ideal, as the norms tend to be derived from a more general population of individuals and not specifically top-level athletes.

3.6. STATISTICAL ANALYSES

The means and standard deviations were calculated for each group (i.e. Total Rugby (Springbok Rugby and Under 21 Rugby), Springbok Rugby, Under 21 Rugby, Hockey Control, Total Rugby Forwards, Total Rugby Backs, Springbok Rugby Forwards, Springbok Rugby Backs, Under 21 Rugby Forwards, and Under 21 Rugby Backs) on all administered neuropsychological tests.

The neuropsychological data were analysed using a pooled independent two sample t-test and the Mann-Whitney U-test. The t-test assumes normality of distribution of variables, while the latter is a non-parametric statistical test which makes no such assumption of normality. The Mann-Whitney U-test was run as an additional statistical test. Between the rugby playing groups and the non-contact sport control, the following comparisons were made: Total Rugby players (Springbok Rugby and Under 21 Rugby) versus Hockey Control; Springbok Rugby versus Hockey Control; Under 21 Rugby versus Hockey Control. Within the rugby playing groups, the following comparisons were made: Total Rugby Forwards versus Total Rugby Backs; Springbok Rugby Forwards versus Springbok Rugby Backs; and Under 21 Rugby Forwards versus Under 21 Rugby Backs.

CHAPTER FOUR: RESULTS

The comparative results for the group mean comparisons of neuropsychological test performance will be reported in the following order:

(i) Total Rugby (including Springbok Rugby and Under 21 Rugby) versus Hockey Control;

(ii) Springbok Rugby versus Hockey Control;

(iii) Under 21 Rugby versus Hockey Control;

(iv) Total Rugby Forwards versus Total Rugby Backs;

(v) Springbok Rugby Forwards versus Springbok Rugby Backs;

(vi) Under 21 Rugby Forwards versus Under 21 Rugby Backs.

4.1. COMPARISON OF MEANS ACROSS ALL GROUPS (i – vi) ON NEUROPSYCHOLOGICAL DATA

As described above (see methodology chapter), the neuropsychological data were analysed using a pooled independent two-sample t-test and Mann-Whitney U-test, resulting in a t-statistic and a U-statistic respectively. The U-statistic was run as an additional statistic and in most instances it revealed a similar result to the t-statistic (i.e. showed significance in the same areas). However, there were rare instances where t was significant and U missed significance and vice versa. The results will be discussed from the perspective of the t-statistic and the U-statistic will only be commented on if it adds value to the discussion. All the results (see Tables 4-1 to 4-6) are grouped together at the end of the chapter (pp. 56-61).

4.1.1. TOTAL RUGBY (SPRINGBOK RUGBY AND UNDER 21 RUGBY) VERSUS HOCKEY CONTROL (see Table 4-1, p. 56)

In the comparison of Total Rugby and Hockey Control, a significant difference was found between these two groups on Digit Symbol Subtest (t-and U-statistic: $p < 0.01$), Trail Making Test (Part B) (t-statistic: $p < 0.05$; U-statistic: $p < 0.01$), and Words in One Minute Unstructured Verbal Fluency Test (t-and U-statistic: $p < 0.05$) all in the direction of better performance of the control group. Digits Backward was found to be approaching significance (t-statistic: $p = 0.0687$; U-statistic: $p = 0.0771$) in the direction of better performance of the control group. On all other tests, there were no significant differences.

4.1.2. SPRINGBOK RUGBY VERSUS HOCKEY CONTROL (see Table 4-2, p. 57)

In the comparison of Springbok Rugby and Hockey Control, a similar picture as in 4.1.1 was seen. There was a significant difference between these two groups on Digit Symbol Subtest (t-statistic: $p < 0.05$) in the direction of better performance of the control group, which was supported by a strong trend in the same direction using the U-statistic which narrowly missed significance (U-statistic: $p = 0.0700$). There was a significant difference between the groups on Trail Making Test (Part B) (t-and U-statistic: $p < 0.05$) in the direction of better performance of the control group. Even though Words in One Minute Unstructured Verbal Fluency Test was not statistically significant (as in 4.1.1), there was some evidence of a trend in the same direction in that it approached significance (t-statistic: $p = 0.1526$), although this was not reflected in the U-statistic (U-statistic: $p = 0.4032$). In addition, WMS Associate Learning Subtest – Hard (Immediate and Delayed Recall) taken as a whole, strongly approached significance in the direction of better performance of the control group using the t-statistic ($p = 0.0526$ and $p = 0.0941$ respectively) and the U-statistic ($p = 0.1063$ and $p = 0.1811$). In contrast to the previously mentioned results in 4.1.1, there was a significant difference between Springbok Rugby and Hockey Control on the first and second trial of Finger Tapping Test using non-preferred hand (t-and U-statistic: $p < 0.01$ and t-statistic: $p < 0.05$ respectively) in the direction of better performance of the Springbok Rugby group. The result for the second finger tapping trial was supported by a strong trend in the same direction using the U-statistic which narrowly missed significance (U-statistic: $p = 0.0689$). Thus, taken together, the t-statistic and the U-statistic indicated significant differences and/or trends between Springbok Rugby and Hockey Control on Digit Symbol Subtest, Trail Making Test (Part B), Words in One Minute Unstructured Verbal Fluency Test, WMS Associate Learning Subtest – Hard (Immediate and Delayed Recall), all in the direction of better performance of the control group. In contrast, a significant difference was found between the two groups on both trials of Finger Tapping Test, in the direction of better performance of the Springbok Rugby group.

4.1.3. UNDER 21 RUGBY VERSUS HOCKEY CONTROL (see Table 4-3, p. 58)

In the comparison of Under 21 Rugby and Hockey Control, the same picture was replicated as in 4.1.1 and 4.1.2 in that there was a significant difference between the two groups on Digit Symbol Subtest (t-and U-statistic: $p < 0.01$), Trail Making Test (Part B) (t-and U-statistic: $p < 0.01$), and Words in One Minute Unstructured Verbal Fluency Test (t-and U-statistic: $p < 0.01$), in the direction of better performance of the control group. In addition, there was a significant difference between these two groups on Digits Forward (t-statistic: $p < 0.05$) in the direction of better performance of the control group, which was supported by a strong trend in the same direction using the U-statistic which narrowly missed significance (U-statistic: p =

0.0643). There was a significant difference between the groups on Digits Backward (t-and U-statistic: $p < 0.01$), and WMS Visual Reproduction (Immediate Recall) (t-and U-statistic: $p < 0.05$), again with Under 21 Rugby performing significantly worse than Hockey Control. The U-statistic indicated a significant difference on the first trial of Finger Tapping Test using the preferred hand ($p < 0.05$) in the direction of better performance of the control group, where the t-statistic narrowly missed significance in the same direction ($p = 0.0590$). Trail Making Test (Part A) just reached significance using the U-statistic ($p < 0.05$) in the direction of better performance of the control, although there was no significant difference using the t-statistic ($p = 0.1301$). Thus, taken together, the t-statistic and the U-statistic indicated significant differences and/or trends between Under 21 Rugby and Hockey Control on Digit Symbol Subtest, Trail Making Test (Part B), Words in One Minute Unstructured Verbal Fluency Test, Digits Forward and Digits Backward, WMS Visual Reproduction (Immediate Recall), the first trial of the Finger Tapping Test (preferred hand), and Trail Making Test (Part A), all in the direction of better performance of the control group. While there is a clear difference between the groups in the direction of better performance of the control group on Trail Making Test (Part B), this difference is not so strongly in evidence on Trail Making Test (Part A).

4.1.4. SUMMARY OF THREE GROUP ANALYSES

Across all three comparisons, a highly consistent pattern emerged for Digit Symbol Subtest, Trail Making Test (Part B), and Words in One Minute Unstructured Verbal Fluency Test, which all revealed a significant difference between the rugby groups and the control group in the direction of better performance of the control group. In contrast, across comparisons for Springbok Rugby and Hockey Control, there were inconsistent results occurring for the Finger Tapping Test in that when comparing Springbok Rugby and Hockey Control, a significant difference on Finger Tapping Test (non-preferred hand) in the direction of better performance of Springbok Rugby emerged. However, when comparing Under 21 Rugby and Hockey Control, a significant difference on Finger Tapping Test (preferred hand) and Trail Making Test (Part A) in the direction of better performance of the control group was indicated. In addition to the consistently occurring poorer performance across tests, Under 21 Rugby group performed significantly worse than Hockey Control on three additional neuropsychological tests (excluding Finger Tapping Test and Trail Making Test (Part A)), namely Digits Forward and Backward, and WMS Visual Reproduction (Immediate Recall).

4.1.5. TOTAL RUGBY FORWARDS VERSUS TOTAL RUGBY BACKS (see Table 4-4, p. 59)

In the comparison of Total Rugby Forwards and Total Rugby Backs, a significant difference was found on Digits Backward (t- and U-statistic: $p < 0.01$), WMS Associate Learning Subtest – Hard (Delayed Recall) (t- and U-statistic: $p < 0.05$), Digit Symbol Subtest (t- and U-statistic: $p < 0.001$), Digit Symbol Incidental Recall (Delayed) (t- and U-statistic: $p < 0.05$), in the direction of better performance of the Backs. In addition, the U-statistic found significant differences on Trail Making Test (Part B) (U-statistic: $p < 0.05$) and the first trial of Finger Tapping Test (non-preferred hand) (U-statistic: $p < 0.05$), in the direction of better performance of the Backs, which was supported by a strong trend in the same direction using the t-statistic which narrowly missed significance on both tests (t-statistic: $p = 0.0629$ and $p = 0.0515$ respectively). Further, Trail Making Test (Part A) and Finger Tapping Test (preferred hand) – Trial 2 were found to be approaching significance (t-statistic: $p = 0.1136$, U-statistic: $p = 0.1003$ and t-statistic: $p = 0.0616$, U-statistic: $p = 0.0606$ respectively) again in the direction of better performance of the Backs. Thus, taken together, the t-statistic and the U-statistic indicated significant differences and/or trends between Total Rugby Forwards and Total Rugby Backs on Digits Backward, WMS Associate Learning Subtest – Hard (Delayed Recall), Digit Symbol Subtest, Digit Symbol Incidental Recall (Delayed), Trail Making Test (Part B), and the first trial of the Finger Tapping Test (non-preferred hand), all in the direction of better performance of the Backs.

4.1.6. SPRINGBOK RUGBY FORWARDS VERSUS SPRINGBOK RUGBY BACKS (see Table 4-5, p. 60)

In the comparison of Springbok Rugby Forwards and Springbok Rugby Backs, a significant difference was found on Digits Backward (t- and U-statistic: $p < 0.01$), Digit Symbol Subtest (t- and U-statistic: $p < 0.001$), Digit Symbol Incidental Recall (Delayed) (t- and U-statistic: $p < 0.05$), Trail Making Test (Part A) (Trail A) (t- and U-statistic: $p < 0.05$), Trail Making Test (Part B) (t- and U-statistic: $p < 0.01$), Finger Tapping Test (non-preferred hand) – Trial 1 (t-statistic: $p < 0.01$; U-statistic: $p < 0.05$) and Trial 2 (t-statistic: $p < 0.05$), in the direction of better performance of the Backs, with the U-statistic marginally missing significance on the second trial of Finger Tapping (non-preferred hand) (U-statistic: $p = 0.0654$) in the same direction.

4.1.7. UNDER 21 RUGBY FORWARDS VERSUS UNDER 21 RUGBY BACKS (see Table 4-6, p. 61)

In the comparison of Under 21 Rugby Forwards and Under 21 Rugby Backs, a significant difference was found on one test only, namely WMS Associate Learning Subtest – Hard

(Delayed Recall) (t- and U-statistic: $p < 0.05$), in the direction of better performance of the Backs. However, there was evidence of trends in the direction of better performance of Under 21 Rugby Backs on those tests which showed significant differences between Total/ Springbok Rugby Forwards and Total/Springbok Rugby Backs in the direction of better performance of the Backs (including Digit Symbol Subtest, Trail Making Test (Part B), Digit Symbol Incidental Recall (Delayed), Digits Backward, and Finger Tapping Test (non-preferred hand)).

4.1.8. SUMMARY OF THREE GROUP ANALYSES COMPARING RUGBY FORWARDS TO RUGBY BACKS

Across the three comparisons, there was a general trend towards Rugby Forwards performing worse on neuropsychological tests than Rugby Backs. The tests which tended to show a significant difference when comparing Total/Springbok Rugby Forwards and Total/Springbok Rugby Backs (in favour of the Backs) were Digits Backward, Digit Symbol Subtest, Digit Symbol Incidental Recall (Delayed), Trail Making Test (Part B), and both trials of Finger Tapping Test (non-preferred hand). Comparison of Under 21 Rugby Forwards and Under 21 Rugby Backs did not reach significance on these tests, but strong trends in the same direction were indicated.

Table 4-1: Comparison of Means across Total Rugby (Springbok Rugby + Under 21 Rugby) and Hockey Control

Test	Total Rugby			Hockey Control			t-statistic	p-value	U-statistic	p-value
	n	Mean	SD	n	Mean	SD				
Digits Forward	45	6.91	1.24	21	7.05	1.16	-0.42	0.6724	461.0	0.8701
Digits Backward	45	5.67	1.52	21	6.43	1.63	-1.85	0.0687	346.5	0.0771
Digit Supraspan	45	2.84	2.31	21	2.19	1.47	1.19	0.2388	545.5	0.2972
Supra. Sust. L	45	4.04	2.80	21	3.52	1.50	0.80	0.4285	483.0	0.8817
ALE easy - Imm	45	8.74	0.44	21	8.86	0.28	-1.09	0.2819	414.0	0.3247
ALE hard - Imm	45	8.36	2.66	21	9.43	1.50	-1.72	0.0897	372.0	0.1607
ALE easy - Del.	45	5.91	0.29	21	6.00	0.00	-1.41	0.1635	430.5	0.1618
ALE hard - Del.	45	3.33	1.02	21	3.71	0.56	-1.59	0.1157	398.0	0.2130
Vis. Rep. Imm.	45	11.56	1.71	21	12.19	1.54	-1.45	0.1527	365.0	0.1284
Vis. Rep. Del.	45	11.22	1.86	21	11.76	1.55	-1.16	0.2518	403.5	0.3324
Dig. Sym. copy	45	49.59	10.20	21	57.55	7.36	-3.20 **	0.0021	261.5 **	0.0036
D.S inc. recall - Imm.	45	7.14	2.05	21	7.50	1.71	-0.69	0.4931	446.5	0.7138
D.S inc. recall - D	45	7.12	2.10	21	7.24	1.64	-0.22	0.8242	479.0	0.9273
Trail A	45	28.02	8.47	21	24.59	8.25	1.55	0.1272	612.0	0.0548
Trail B	45	62.41	24.25	21	47.58	13.43	2.61 *	0.0111	682.0 **	0.0039
Verbal Fl. uns.	45	36.73	8.65	21	42.67	9.87	-2.48 *	0.0157	326.5 *	0.0442
Verbal Fl. str.	45	16.24	4.70	21	16.62	4.35	-0.31	0.7587	445.0	0.7038
Finger t 1P	45	5.50	1.10	21	5.42	0.68	0.31	0.7554	486.5	0.7310
Finger t 1np	45	5.47	1.16	21	5.67	0.57	-0.74	0.4604	382.5	0.2646
Finger t 2p	45	4.95	0.83	21	5.01	0.73	-0.31	0.7599	444.5	0.6997
Finger t 2np	45	4.95	0.86	21	5.20	0.69	-1.14	0.2584	386.0	0.2336

Significant Difference (* p<0.05; ** p<0.01)

Table 4-2: Comparison of Means across Springbok Rugby and Hockey Control

Test	Springbok Rugby			Hockey Control			t-statistic	p-value	U-statistic	p-value
	n	Mean	SD	n	Mean	SD				
Digits Forward	26	7.42	0.99	21	7.05	1.16	1.20	0.2370	327.5	0.2261
Digits Backward	26	6.15	1.38	21	6.43	1.63	-0.63	0.5342	246.5	0.5610
Digit Supraspan	26	2.30	1.32	21	2.19	1.47	0.29	0.7749	295.5	0.6145
Supra. Sust. L	26	3.39	1.58	21	3.52	1.50	-0.31	0.7602	254.5	0.6821
ALE easy - Imm	26	8.81	0.43	21	8.86	0.28	-0.46	0.6496	265.0	0.8218
ALE hard - Imm	26	8.08	2.80	21	9.43	1.50	-1.99	0.0526	199.0	0.1063
ALE easy - Del.	26	5.92	0.27	21	6.00	0.00	-1.29	0.2021	252.0	0.1988
ALE hard - Del.	26	3.26	1.07	21	3.71	0.56	-1.71	0.0941	221.5	0.1811
Vis. Rep. Imm.	26	11.96	1.40	21	12.19	1.54	-0.53	0.5962	241.5	0.4852
Vis. Rep. Del.	26	11.30	1.89	21	11.76	1.55	-0.89	0.3803	239.5	0.4652
Dig. Sym. copy	26	52.60	9.06	21	57.55	7.36	-2.02 *	0.0493	188.5	0.0700
D.S inc. recall - Imm.	26	6.73	2.35	21	7.50	1.71	-1.26	0.2156	235.5	0.4118
D.S inc. recall - D	26	6.90	2.26	21	7.24	1.64	-0.57	0.5732	259.5	0.7683
Trail A	26	27.66	9.12	21	24.59	8.25	1.20	0.2370	338.0	0.1642
Trail B	26	58.64	18.60	21	47.58	13.43	2.28 *	0.0271	379.5 *	0.0227
Verbal Fl. uns.	26	38.92	7.78	21	42.67	9.87	-1.46	0.1526	234.0	0.4032
Verbal Fl. str.	26	17.50	4.58	21	16.62	4.35	0.67	0.5062	296.5	0.6134
Finger t 1P	26	5.14	0.95	21	5.42	0.68	-1.14	0.2614	208.0	0.2293
Finger t 1np	26	4.90	0.85	21	5.67	0.57	-3.50 **	0.0011	118.0 **	0.0014
Finger t 2p	26	4.73	0.71	21	5.01	0.73	-1.31	0.1971	223.5	0.2894
Finger t 2np	26	4.70	0.85	21	5.20	0.69	-2.14 *	0.0376	188.0	0.0689

Significant Difference (* p<0.05; ** p<0.01)

Table 4-3: Comparison of Means across Under 21 Rugby and Hockey Control

Test	Under 21 Rugby			Hockey Control			t-statistic	p-value	U-statistic	p-value
	n	Mean	SD	n	Mean	SD				
Digits Forward	19	6.21	1.23	21	7.05	1.16	-2.22 *	0.0328	133.5	0.0643
Digits Backward	19	5.00	1.49	21	6.43	1.63	-2.88 **	0.0065	100.0 **	0.0061
Digit Supraspan	19	3.58	3.10	21	2.19	1.47	1.84	0.0736	250.0	0.1568
Supra. Sust. L	19	4.95	3.78	21	3.52	1.50	1.59	0.1191	228.5	0.4211
ALE easy - Imm	19	8.66	0.44	21	8.86	0.28	-1.72	0.0939	149.0	0.1045
ALE hard - Imm	19	8.74	2.47	21	9.43	1.50	-1.08	0.2859	173.0	0.4652
ALE easy - Del.	19	5.90	0.32	21	6.00	0.00	-1.53	0.1338	178.5	0.1320
ALE hard - Del.	19	3.42	0.96	21	3.71	0.56	-1.19	0.2404	176.5	0.4270
Vis. Rep. Imm.	19	11.00	1.97	21	12.19	1.54	-2.14 *	0.0388	123.5 *	0.0354
Vis. Rep. Del.	19	11.11	1.85	21	11.76	1.55	-1.22	0.2295	164.0	0.3261
Dig. Sym. copy	19	45.47	10.45	21	57.55	7.36	-4.26 **	0.0001	73.0 **	0.0006
D.S inc. recall - Imm.	19	7.71	1.44	21	7.50	1.71	0.42	0.6775	211.0	0.7486
D.S inc. recall - D	19	7.42	1.87	21	7.24	1.64	0.33	0.7437	219.5	0.5804
Trail A	19	28.50	7.69	21	24.59	8.25	1.55	0.1301	274.0 *	0.0436
Trail B	19	67.56	30.14	21	47.58	13.43	2.75 **	0.0090	302.5 **	0.0053
Verbal Fl. uns.	19	33.74	9.09	21	42.67	9.87	-2.97 **	0.0052	92.5 **	0.0037
Verbal Fl. str.	19	14.53	4.41	21	16.62	4.35	-1.51	0.1397	148.5	0.1636
Finger t 1P	19	5.98	1.11	21	5.42	0.68	1.95	0.0590	278.5 *	0.0324
Finger t 1np	19	6.22	1.11	21	5.67	0.57	1.98	0.0554	264.5	0.0783
Finger t 2p	19	5.24	0.90	21	5.01	0.73	0.87	0.3892	221.0	0.5601
Finger t 2np	19	5.29	0.78	21	5.20	0.69	0.40	0.6912	198.0	0.9676

Significant Difference (* p<0.05; ** p<0.01)

Table 4-4: Comparison of Means across Total Rugby (Springbok + Under 21) Forwards and Backs

Test	Forwards			Backs			t-statistic	p-value	U-statistic	p-value
	n	Mean	SD	n	Mean	SD				
Digits Forward	26	6.89	1.34	19	6.95	1.13	-0.17	0.8691	247.0	1.0000
Digits Backward	26	5.08	1.35	19	6.47	1.39	-3.38 **	0.0015	119.5 **	0.0027
Digit Supraspan	26	2.85	2.28	19	2.84	2.41	0.01	0.9954	261.0	0.7395
Supra. Sust. L	26	4.04	2.93	19	4.05	2.70	-0.02	0.9869	252.0	0.9056
ALE easy - Imm	26	8.67	0.53	19	8.84	0.24	-1.30	0.2010	218.0	0.4302
ALE hard - Imm	26	7.96	2.81	19	8.90	2.40	-1.17	0.2488	201.5	0.2918
ALE easy - Del.	26	5.89	0.33	19	5.95	0.23	-0.72	0.4762	231.5	0.4700
ALE hard - Del.	26	3.08	1.09	19	3.68	0.82	-2.04 *	0.0478	164.5 *	0.0262
Vis. Rep. Imm.	26	11.58	1.27	19	11.53	2.22	0.10	0.9234	214.0	0.4357
Vis. Rep. Del.	26	11.39	1.63	19	11.00	2.16	0.68	0.4589	270.5	0.5799
Dig. Sym. copy	26	45.87	8.80	19	54.68	9.97	-3.14 **	0.0031	123.5 **	0.0045
D.S inc. recall - Imm.	26	6.85	2.15	19	7.55	1.89	-1.14	0.2587	196.5	0.2345
D.S inc. recall - D	26	6.52	2.31	19	7.95	1.44	-2.37 *	0.0223	152.0 *	0.0259
Trail A	26	29.73	8.18	19	25.67	8.50	1.62	0.1136	318.5	0.1003
Trail B	26	68.14	25.73	19	54.56	20.15	1.91	0.0629	343.0 *	0.0274
Verbal Fl. uns.	26	35.42	8.45	19	38.53	8.83	-1.19	0.2389	196.5	0.2449
Verbal Fl. str.	26	15.62	4.96	19	17.11	4.31	-1.05	0.2990	212.0	0.4192
Finger t 1P	26	5.72	1.11	19	5.22	1.04	1.52	0.1372	288.5	0.2267
Finger t 1np	26	5.77	1.00	19	5.08	1.27	2.00	0.0515	328.5 *	0.0309
Finger t 2p	26	5.14	0.89	19	4.68	0.65	1.92	0.0616	328.5	0.0606
Finger t 2np	26	5.14	0.81	19	4.70	0.90	1.71	0.0936	310.0	0.1474

Significant Difference (* $p<0.05$; ** $p<0.01$)

Table 4-5: Comparison of Means across Springbok Rugby Forwards and Backs

Test	Forwards			Backs			t-statistic	p-value	U-statistic	p-value
	n	Mean	SD	n	Mean	SD				
Digits Forward	15	7.40	1.05	11	7.45	0.93	-0.14	0.8926	80.5	0.9128
Digits Backward	15	5.47	1.19	11	7.09	1.04	-3.62 **	0.0014	24.5 **	0.0019
Digit Supraspan	15	2.30	1.16	11	2.36	1.56	-0.18	0.8575	84.5	0.9134
Supra. Sust. L	15	3.40	1.64	11	3.36	1.57	0.06	0.9551	83.5	0.9568
ALE easy - Imm	15	8.73	0.53	11	8.91	0.20	-1.04	0.3083	69.0	0.3650
ALE hard - Imm	15	7.87	2.88	11	8.36	2.80	-0.44	0.6639	75.5	0.7137
ALE easy - Del.	15	5.93	0.25	11	5.90	0.30	0.22	0.8274	84.5	0.8222
ALE hard - Del.	15	3.13	1.13	11	3.45	1.04	-0.74	0.4647	67.5	0.3723
Vis. Rep. Imm.	15	11.73	1.33	11	12.27	1.49	-0.97	0.3418	59.5	0.2155
Vis. Rep. Del.	15	11.53	1.73	11	11.00	2.14	0.70	0.4890	95.0	0.5068
Dig. Sym. copy	15	47.97	7.86	11	58.91	6.56	-3.75 **	0.0010	26.5 **	0.0036
D.S inc. recall - Imm.	15	6.00	2.30	11	7.72	2.10	-1.96	0.0621	44.5 *	0.0437
D.S inc. recall - D	15	5.97	2.40	11	8.18	1.25	-2.78 *	0.0103	35.0 *	0.0117
Trail A	15	30.69	9.24	11	23.54	7.50	2.11 *	0.0459	120.5 *	0.0485
Trail B	15	67.42	16.69	11	46.66	14.19	3.33 **	0.0028	137.5 **	0.0043
Verbal Fl. uns.	15	37.00	8.58	11	41.55	5.92	-1.51	0.1442	56.0	0.1679
Verbal Fl. str.	15	16.67	4.84	11	18.64	4.15	-1.09	0.2877	70.0	0.5144
Finger t 1P	15	5.40	0.74	11	4.79	1.12	1.65	0.1119	106.5	0.1062
Finger t 1np	15	5.30	0.65	11	4.41	0.84	2.98 **	0.0068	122.0 *	0.0136
Finger t 2p	15	4.94	0.65	11	4.45	0.72	1.82	0.0812	122.0 *	0.0402
Finger t 2np	15	4.99	0.73	11	4.30	0.87	2.19 *	0.0384	118.0	0.0654

Significant Difference (* $p<0.05$; ** $p<0.01$)

Table 4-6: Comparison of Means across Under 21 Rugby Forwards and Backs

Test	Forwards			Backs			t-statistic	p-value	U-statistic	p-value
	n	Mean	SD	n	Mean	SD				
Digits Forward	11	6.18	1.40	8	6.25	1.04	-0.12	0.9089	46.5	0.8299
Digits Backward	11	4.55	1.44	8	5.63	1.41	-1.63	0.1218	27.5	0.1604
Digit Supraspan	11	3.64	3.14	8	3.50	3.25	0.09	0.9277	47.5	0.7679
Supra. Sust. L	11	4.91	4.04	8	5.00	3.67	-0.05	0.9604	45.0	0.9329
ALE easy - Imm	11	8.59	0.54	8	8.75	0.27	-0.76	0.4550	40.0	0.7159
ALE hard - Imm	11	8.09	2.84	8	9.63	1.60	-1.37	0.1886	30.5	0.2603
ALE easy - Del.	11	5.82	0.41	8	6.00	0.00	-1.26	0.2243	36.0	0.2146
ALE hard - Del.	11	3.00	1.10	8	4.00	0.00	-2.56 *	0.0202	20.0 *	0.0160
Vis. Rep. Imm.	11	11.36	1.21	8	10.50	2.73	0.94	0.3607	48.0	0.7351
Vis. Rep. Del.	11	11.18	1.54	8	11.00	2.33	0.21	0.8396	44.5	0.9660
Dig. Sym. copy	11	43.00	9.56	8	48.88	11.29	-1.23	0.2366	32.0	0.3215
D.S inc. recall - Imm.	11	8.00	1.25	8	7.31	1.67	1.03	0.3167	55.0	0.3494
D.S inc. recall - D	11	7.27	2.05	8	7.63	1.71	-0.40	0.6976	40.0	0.7363
Trail A	11	28.42	6.67	8	28.61	9.40	-0.05	0.9585	48.5	0.7100
Trail B	11	69.12	35.53	8	65.43	22.88	0.26	0.8004	44.5	0.9671
Verbal Fl. uns.	11	33.27	8.16	8	34.38	10.78	-0.25	0.8024	41.5	0.8360
Verbal Fl. str.	11	14.18	4.98	8	15.00	3.78	-0.39	0.7018	40.5	0.7667
Finger t 1P	11	6.11	1.40	8	5.80	0.59	0.59	0.5645	53.0	0.4572
Finger t 1np	11	6.37	1.06	8	6.01	1.22	0.69	0.4988	59.5	0.2004
Finger t 2p	11	5.42	1.12	8	4.99	0.41	1.02	0.3235	55.0	0.3612
Finger t 2np	11	5.33	0.90	8	5.24	0.64	0.24	0.8144	47.0	0.8035

Significant Difference (* p<0.05; ** p<0.01)

CHAPTER FIVE: DISCUSSION

The present study forms part of a larger and ongoing research study investigating the brain damage effects of cumulative mild head injury in rugby. This study compared the cognitive performance of Springbok professional rugby players and Under 21 national rugby players with the performance of a matched non-contact sport control group comprising national hockey players. Participants were selected on the basis of a number of exclusion criteria, including a reported history of substance abuse, a neurological or psychiatric disorder known to negatively impact on cognitive functioning, and a reported history of moderate to severe non-sport related head injury. Hence, any differences between the groups could not be accounted for by these factors. Furthermore, in relation to the demographic data (see methodology chapter), although significant differences were noted between Springbok Rugby/Under 21 Rugby and Hockey Control with respect to age, these differences were within one decade of each other. Considering that age norms tend to span a decade, the practical implications of these differences were not anticipated to be meaningful. In addition, although significant differences were found between Total Rugby and Hockey Control with respect to education and estimated premorbid IQ, and between Under 21 Rugby and Hockey Control with respect to education, these differences fell within relatively close ranges and hence probably were not of clinical significance. However, the significant difference between Under 21 Rugby and Hockey Control with respect to estimated premorbid IQ is possibly more meaningful in that this difference encompassed an entire IQ range which needs to be taken into account when interpreting the results. Finally, in terms of positional comparisons (Forwards versus Backs) within the rugby groups, no significant differences were noted between the groups with respect to age, education, and estimated premorbid IQ. Consequently, any observed differences between the Forwards and the Backs on neuropsychological tests cannot be explained in terms of these variables.

Broadly, it was hypothesised that the performance of the rugby playing groups would be poorer on certain cognitive tasks known to be sensitive to diffuse brain damage compared with the performance of the hockey control group. This hypothesis was based on the premise that rugby players are more likely to be exposed to long-term cumulative mild concussive and sub-concussive head injuries than hockey players, and that these head injuries are likely to cause neural change, which serves to lower the critical threshold at which functional symptomatology will manifest, which in turn is likely to lead to less BRC, thereby increasing

the players' vulnerability to neuropsychological deficit. In addition, it was hypothesised that the performance of the subgroup of rugby forward players would be poorer on certain cognitive tasks known to be sensitive to diffuse brain damage compared with the performance of the subgroup of rugby back-line players. This hypothesis was based on the assertion that rugby forward players (who tend to be involved in more repetitive physical collisions) are likely to be exposed to more head insults than rugby back-line players. These cumulative mild head injuries suffered by the rugby forward players are likely to cause neural change, which serves to lower the critical threshold at which functional symptomatology will manifest, and which in turn is likely to lead to less BRC, thereby increasing the players' vulnerability to neuropsychological deficit.

In relation to these hypotheses, the findings of the present study will be discussed first in terms of Rugby versus Hockey Control comparisons and then in terms of within Rugby group comparisons (Rugby Forwards versus Rugby Backs comparisons). Where appropriate, referral to Satz's (1993) theory of brain reserve capacity (BRC) as it relates to these findings will be made, in order to provide a theoretical context for the argument.

5.1. RUGBY VERSUS HOCKEY CONTROL COMPARISONS

5.1.1. CONSISTENT FINDINGS ACROSS ALL THREE RUGBY GROUPS

A number of tests showed significantly poorer performance, or a strong trend towards poorer performance, for all three rugby groups (i.e. Total Rugby, Springbok Rugby, Under 21 Rugby) relative to hockey controls. These tests were Digit Symbol Subtest, Trail Making Test (Part B), and Words in One Minute Unstructured Verbal Fluency Test. The first two tests involve visuoperceptual tracking at speed and working memory, while the third test is a measure of verbal fluency. Specifically, Digit Symbol Subtest and Trail Making Test (Part B) have been isolated in the literature as being highly sensitive to the effects of diffuse brain damage, commonly associated with closed mild head injury. In this regard, neuropsychological studies into mild head injury in rugby/football-related sports have consistently found impaired performance in mild head injured players relative to controls on Digit Symbol Subtest (or similar tests of speed of information processing) (e.g. Barth et al., 1989; Dickinson, 1998; Hinton-Bayre et al., 1997; Macciocchi et al., 1996; Maddocks & Saling, 1991; Reid, 1998), and on Trail Making Test (Part B relative to Part A) (e.g. Dickinson, 1998; Reid, 1998; Shuttleworth-Jordan et al., 1993). Furthermore, other more general studies into mild head injury have found neuropsychological deficit in mild head

injured patients relative to controls on Digit Symbol Subtest (or similar tests of speed of information processing) (e.g. Gronwall & Wrightson, 1974, 1975; Gronwall, 1989; Leininger et al., 1990; Levin et al., 1987) and on Trail Making Test (Part B relative to Part A) (e.g. Leininger et al., 1990). Thus, the present findings indicate that the performance of the rugby players is poorer on those tests known to be sensitive to diffuse brain damage compared with the performance of the hockey controls, due to the rugby players' increased exposure to cumulative mild concussive and sub-concussive head injuries. In sum, aside from indicating the presence of brain damage, the findings also corroborate the sensitivity of Digit Symbol Subtest and Trail Making Test (Part B relative to Part A) to detecting diffuse brain damage.

5.1.2. FINDINGS SPECIFIC TO UNDER 21 RUGBY GROUP

In addition to the tests discussed under 5.1.1 (namely, Digit Symbol Subtest, Trail Making Test (Part B), and Words in One Minute Unstructured Verbal Fluency Test) which were in the direction of poorer performance for all three rugby groups relative to hockey controls, a further series of tests showed significantly poorer performance for Under 21 Rugby relative to Hockey Control, which did not come up for Total Rugby and Springbok Rugby. These tests were Digits Forward, Digits Backwards, WMS Visual Reproduction (Immediate Recall), first trial of Finger Tapping Test using the preferred hand, and Trail Making Test (Part A). Although some research studies have found an association between mild closed head injury and impaired performance on Digits Forward (e.g. Rimel et al., 1981), Digits Backwards (e.g. Reid, 1998; Shuttleworth-Jordan et al., 1993), and Finger Tapping Test (e.g. Drew et al., 1986), these tests are not regarded in the literature to be as sensitive indicators of diffuse brain damage as Digit Symbol Subtest and Trail Making Test (Part B). However, these tests will still detect deficit if there is more severe brain damage and, in addition, will still tap into the important cognitive functions of attention (Digits Forward and Trail Making Test (Part A)), working memory (Digits Backwards), visuospatial problem solving ability (WMS Visual Reproduction (Immediate Recall)), and hand-motor dexterity (Finger Tapping Test).

There are two plausible explanations for the greater number of tests showing impaired performance for Under 21 Rugby relative to Hockey Control. Firstly, because the Under 21 Rugby group has a lower level of education than the Total Rugby group and the Springbok Rugby group, it is possible that that the findings are a reflection of lower premorbid IQ. However, in contrast to what might be expected from a lower IQ group, the Under 21 Rugby group does not perform significantly poorer than Hockey Control across all tests in that there are no significant differences between these two groups on the majority of the tests (namely, Digit Supraspan (including Sustained Learning), WMS Associate Learning Subtest (Immediate and Delayed Recall), WMS Visual Reproduction (Delayed Recall), Digit Symbol

Incidental Recall (Immediate and Delayed), "S" Words in One Minute Structured Verbal Fluency Test, and Finger Tapping Test (first trial - non-preferred hand, second trial – both hands). An alternative explanation for the greater number of tests showing impaired performance for Under 21 Rugby relative to Hockey Control is related to the fact that the Under 21 Rugby players have a significantly lower education level and estimated premorbid IQ relative to Hockey Control. In light of this, it is possible that these risk factors of lower education level and lower IQ have lowered the brain reserve capacity (BRC) threshold of the Under 21 Rugby players, resulting in less BRC and hence increasing the players' vulnerability to neuropsychological impairment. Thus, it is suggested that the effects of diffuse brain damage may be showing up on tests not as sensitive to brain damage, because of the lower BRC in the Under 21 Rugby group.

5.1.3. ISOLATED FINDINGS

There were two isolated test findings, with the first being that Finger Tapping Test (non-preferred hand) showed significantly better performance for Springbok Rugby relative to Hockey Control. This result appears inconsistent with the results obtained for other tests discussed in this section of rugby versus hockey control comparisons. Further, in terms of expectations, theoretically one would expect hand-motor function to be depressed with diffuse brain damage. However, this was not found to be the case, as the performance of the Springbok Rugby players is actually better on this test compared with the performance of the Hockey Control. But, in terms of the rugby literature, this opposite tendency has been noticed (e.g. Reid, 1998; Shuttleworth-Jordan et al., 1993). In this regard, Shuttleworth-Jordan et al. (1993) argued that the very nature of rugby play, involving handling and controlling of the ball, may necessitate the development of superior hand-motor dexterity in rugby players. Based on this argument, one would expect players participating at an elite (Springbok) level of play to have highly developed skills. Thus, the present results support the hypothesis of Shuttleworth-Jordan et al. in that they demonstrate that the Springbok Rugby group has superior hand-motor dexterity. The fact that the Under 21 Rugby group show impaired performance on Finger Tapping Test, supports the notion that this group may be more vulnerable to brain damage due to the fact that, as a group, they have significantly lower levels of education and IQ (relative to Hockey Control) which in turn has lowered their threshold resulting in less BRC. Hence, while the Springbok Rugby group with higher levels of education and IQ and therefore higher BRC show superior performance on Finger Tapping Test, this protective factor seems to break down for the Under 21 Rugby group due to lower levels of education and IQ and therefore lower BRC.

The only other isolated finding was for WMS Associate Learning Subtest – Hard (Immediate and Delayed Recall), which showed a strong trend towards poorer performance for Springbok Rugby relative to Hockey Control. According to Lezak (1995), both the "hard" pairs task (which requires new learning) and delayed memory tend to be more sensitive than the "easy" pairs task and immediate memory to the effects of diffuse brain damage. Thus, the Springbok Rugby group's impaired performance on these tasks may be indicative of diffuse mild head injury. This impairment did not show up consistently across all rugby group comparisons and furthermore was only found to be *approaching* significance. On the one hand, this anomalous result may suggest that the poor performance of the Springbok Rugby group on this test is not necessarily due to head injury. On the other hand, the players from the Springbok Rugby group have had longer playing careers at high levels of the sport and consequently have had increased exposure to the cumulative effects of mild head injury which may be showing up as the beginning signs of mild impairment on this test.

5.1.4. SYNTHESIS OF FINDINGS: RUGBY VERSUS HOCKEY CONTROL COMPARISONS

With respect to all three rugby groups, a consistent pattern of significantly poorer performance, or a strong trend towards poorer performance, relative to the Hockey Control is demonstrated on Digit Symbol Subtest and Trail Making Test (Part B), both of which have been isolated in the literature as being tests that are highly sensitive to the effects of diffuse brain damage. The main functions impaired are visuoperceptual tracking at speed, as well as working memory, and verbal fluency. The present findings indicate that the performance of the rugby players is poorer on those tests known to be sensitive to diffuse brain damage compared with the performance of the hockey controls, as a result of the rugby players' increased exposure to cumulative mild concussive and sub-concussive head injuries.

With respect to the Under 21 Rugby group, significantly poorer performance relative to Hockey Control is demonstrated on a further series of tests which did not come up for Total Rugby and Springbok Rugby. These tests, although not as sensitive indicators of diffuse brain damage as Digit Symbol Subtest and Trail Making Test (Part B), will still detect deficit if there is more severe brain damage. The cognitive functions impaired are attention, working memory, visuospatial problem-solving ability, and hand-motor dexterity. As explicated above, it is possible that lower levels of education and IQ of this Under 21 rugby group may have lowered the threshold of these players, leading to reduced BRC and an increased vulnerability to neuropsychological impairment, which is demonstrated in the greater number of tests with impaired performance for this particular group.

With respect to the two isolated test findings, Springbok Rugby demonstrated significantly better performance relative to Hockey Control on Finger Tapping Test (non-preferred hand). Although seemingly opposite to expectations, this result of superior hand-motor dexterity in the Springbok Rugby group has been noted in the rugby literature (e.g. Shuttleworth-Jordan et al., 1993). However, the Under 21 Rugby group showed impaired performance on Finger Tapping Test, which supports the notion that this group may be more brain damaged due to lower BRC. In addition, Springbok Rugby also demonstrated a strong trend towards poorer performance relative to Hockey Control on WMS Associate Learning Subtest – Hard (Immediate and Delayed Recall). In this instance, new learning and recall are the functions which are impaired. It is suggested that the beginning signs of mild impairment are showing on this test, as a result of the Springbok Rugby players' longer playing careers at high levels of the sport and the consequent increased exposure to the cumulative effects of mild concussive and sub-concussive head injuries.

5.2. WITHIN RUGBY GROUP (FORWARDS VERSUS BACKS) COMPARISONS

5.2.1. CONSISTENT FINDINGS ACROSS TOTAL RUGBY AND SPRINGBOK RUGBY GROUPS (FORWARDS VERSUS BACKS)

As with the findings for all three rugby groups relative to hockey controls (see section 5.1.1), Digit Symbol Subtest and Trail Making Test (Part B) showed significantly poorer performance for Total Rugby Forwards and Springbok Rugby Forwards relative to the respective Total Rugby Backs and Springbok Rugby Backs. As previously mentioned (see section 5.1.1), Digit Symbol Subtest and Trail Making Test (Part B), which involve the functions of visuoperceptual tracking at speed and working memory, have been isolated in the literature as being highly sensitive to the effects of diffuse brain damage, commonly associated with closed mild head injury. More specifically, neuropsychological studies into mild head injury in rugby have found impaired performance in rugby forward players relative to rugby back-line players on both Digit Symbol Subtest (e.g. Dickinson, 1998; Reid, 1998) and Trail Making Test (Part B) (e.g. Dickinson, 1998; Reid, 1998; Shuttleworth-Jordan et al., 1993).

In addition to these two highly sensitive tests, Digit Symbol-Incidental Recall (Delayed), Digits Backward in comparison to Digits Forwards, and Trail Making Test (Part A) showed significantly poorer performance, or a strong trend towards poorer performance, for Total

Rugby Forwards and Springbok Rugby Forwards relative to the respective Total Rugby Backs and Springbok Rugby Backs. These tests tap into the functions of recent memory (Digit Symbol Incidental Recall (Delayed)), working memory (Digits Backwards), and attention (Trail Making Test (Part A)). The delayed version (compared with the immediate version) of Digit Symbol Incidental Recall has been shown to be particularly sensitive to the effects of diffuse brain damage (Lezak, 1995). However, this test, along with Digits Backwards and Trail Making Test (Part A), is not regarded in the literature to be as sensitive an indicator of diffuse brain damage as Digit Symbol Subtest and Trail Making Test (Part B). Regardless, these three tests will still detect deficit if there is more severe brain damage. Furthermore, consistent with the present findings, other neuropsychological studies into mild head injury in rugby have found impaired performance in rugby forward players relative to rugby back-line players on both Digit Symbol Incidental Recall (e.g. Dickinson, 1998; Reid, 1998) and Digits Backwards (e.g. Reid, 1998).

Finally, Finger Tapping Test (non-preferred hand) showed significantly poorer performance for Total Rugby Forwards and Springbok Rugby Forwards relative to the respective Total Rugby Backs and Springbok Rugby Backs. In the Springbok Rugby versus Hockey Control comparison (see section 5.1.3), the former group's performance was found to be significantly superior to that of the Hockey Control. Although this finding is consistent with the rugby literature (e.g. Shuttleworth-Jordan et al., 1993), it is *not* consistent with expectations of depressed hand-motor function due to diffuse brain damage. Interestingly, when the Rugby group was separated into Forwards and Backs, the performance of the Forwards was significantly impaired relative to the Backs. This finding, which is consistent with that of Reid (1998), indicates evidence of more damage in the rugby forward players as a result of their increased exposure to multiple concussive and sub-concussive head injury relative to the back-line players.

BRC theory provides an explanation for understanding why the cognitive performance of Total Rugby Forwards and Springbok Rugby Forwards is impaired relative to the respective Total Rugby Backs and Springbok Rugby Backs on certain neuropsychological tests. In this regard, the involvement of these forward players in more repetitive physical collisions and consequently their exposure to more cumulative concussive and sub-concussive head injuries relative to the back-line players, may be lowering the critical threshold at which functional symptomatology will manifest. This in turn, is possibly resulting in lower BRC, thereby increasing the players' vulnerability to neuropsychological deficit and predisposing them to functional impairment.

Thus, in sum, the above findings support the hypothesis that the performance of the subgroup of rugby forward players is poorer on those tests known to be sensitive to diffuse brain damage compared with the performance of the subgroup of rugby back-line players. Furthermore, it is of note that with a new control group (Hockey Control replaced the control group of cricket players used in the initial phase of the larger research project), the tests that showed up with deficit are the same tests that showed up in the studies by Dickinson (1998) and Reid (1998) using cricket players as the control group. However, the results from Ancer's study (1999) are not yet available and as the present study replicates her methodology, the decision as to whether or not hockey is a better control group in the sense of showing up greater neuropsychological deficit cannot be made at this time.

5.2.2. FINDINGS SPECIFIC TO UNDER 21 RUGBY GROUP (FORWARDS VERSUS BACKS)

On all the tests which showed significant differences for Total Rugby and Springbok Rugby (Forwards versus Backs) in the direction of poorer performance of Forwards, trends (as opposed to significant differences) were noted in the same direction for Under 21 Rugby (Forwards versus Backs). These tests included Digit Symbol Subtest, Trail Making Test (Part B), Digit Symbol Incidental Recall (Delayed), Digits Backward, and Finger Tapping Test (non-preferred hand). There are three possible explanations for this seeming lack of differentiation in performance on these tests between the Under 21 Rugby Forwards and the Under 21 Rugby Backs. Firstly, lower BRC due to lower levels of education and IQ, is possibly causing deficit to be shown in both the forward players as well as the back-line players, thereby preventing the detection of significant differences between these two subgroupings. Secondly, the small numbers in the Under 21 group may account for these differences not showing up. Thirdly, the shorter playing careers of the Under 21 players relative to the Springbok players may have resulted in less exposure to cumulative mild concussive and sub-concussive head injuries and hence the differentiation between the Under 21 Forwards and Under 21 Backs is not yet evident. Regardless, these trends in the Under 21 Rugby group (Forwards versus Backs) are showing up in the Total Rugby group (Forwards versus Backs) and hence have obviously influenced the results of Total Rugby (Forwards versus Backs). If, however, there had been no trends in the Under 21 Rugby group, then it is likely that the significant differences would not have shown up for the Total Rugby group.

In addition to the trends, there was a significant difference for WMS Associate Learning Subtest – Hard (Delayed Recall) for Under 21 Rugby (Forwards versus Backs). This test showed significantly poorer performance for Under 21 Rugby Forwards and Total Rugby Forwards relative to the respective Under 21 Rugby Backs and Total Rugby Backs. However,

this test did not show a significant difference for Springbok Rugby Forwards relative to Springbok Rugby Backs. As previously mentioned (see section 5.1.3), both the "hard" pairs task (which requires new learning) and delayed memory tend to be more sensitive than the "easy" pairs task and immediate memory to the effects of diffuse brain damage (Lezak, 1995). With this in mind, it seems plausible to infer that the impaired performance of the Rugby Forward groups relative to the Rugby Back groups on this test are indicative of diffuse mild head injury as a result of the Forwards increased exposure to the cumulative effects of mild head injuries which has lowered the threshold of these groups, resulting in less BRC and consequently increasing the forward players' vulnerability to neuropsychological impairment. Furthermore, with respect to the Under 21 Rugby group which, as previously discussed, has lowered BRC due to lower levels of education and IQ, it is possible that this result reflects the additional damage sustained by the Under 21 Rugby Forwards. It is difficult to explain the reason for this particular test showing up for the Under 21 Rugby group before the other tests. However, because WMS Associate Learning Subtest – Hard (Delayed Recall) is approaching significance in the comparison of Springbok Rugby versus Hockey Control (see section 5.1.3), it is a test which is showing up as a likely indicator of brain damage in players of contact sport relative to those participants of non-contact sport.

5.2.3. SYNTHESIS OF FINDINGS: WITHIN RUGBY GROUP (FORWARDS VERSUS BACKS) COMPARISONS

Positional comparisons (Rugby Forward players versus Rugby Back players for each of the Total, Springbok, and Under 21 Rugby groups) revealed no significant differences in terms of the two variables of education and IQ (see methodology, section 3.1), thereby leading to the assumption that the rugby players within these rugby subgroups are equivalent in terms of intellectual functioning and in terms of premorbid BRC. Thus, any differences noted between Rugby Forward players and Rugby Back players would strongly confirm the possibility for brain damage. This fact is taken into account when discussing the findings of the within rugby group comparisons.

With respect to Total Rugby Forwards and Springbok Rugby Forwards, a consistent pattern of significantly poorer performance, or a strong trend towards poorer performance, relative to the respective Total Rugby Backs and Springbok Rugby Backs is demonstrated on the highly sensitive Digit Symbol Subtest and Trail Making Test (Part B) (which are tests that have consistently differentiated between rugby and control groups in all aspects of this study), as well as on the following tests of Digit Symbol Incidental Recall (Delayed), Digits Backward, Trail Making Test (Part A), and Finger Tapping Test (non-preferred hand). The main functions impaired are visuoperceptual tracking at speed and working memory, as well as

recent memory, attention, and hand-motor dexterity. As explicated above, it is possible that the increased exposure of the Springbok Rugby Forwards and Total Rugby Forwards to cumulative mild head injuries may have lowered their threshold, leading to reduced BRC and an increased vulnerability to neuropsychological impairment, which is demonstrated in this group's impaired performance on a number of tests.

With respect to Under 21 Rugby (Forwards versus Backs), trends on the same tests and in the same direction as those noted for Total Rugby (Forwards versus Backs) and Springbok Rugby (Forwards versus Backs) are demonstrated (i.e. Forwards perform more poorly than Backs). In order to understand the seeming lack of differentiation in performance on these tests between the Under 21 Rugby Forwards and the Under 21 Rugby Backs, three possible explanations have been provided. As explicated previously, these explanations include lower BRC as a result of lower levels of education and IQ, small sample numbers, and shorter playing careers resulting in less exposure to the cumulative effects of mild head injury. In addition to these trends, there was a significant difference for WMS Associate Learning Subtest – Hard (Delayed Recall) for Under 21 Rugby (Forwards versus Backs) as well as Total Rugby (Forwards versus Backs) in the direction of poorer performance for the Forwards. The cognitive function impaired is new learning ability. Generally, this result is reflective of the fact that the increased exposure of the Forwards to cumulative mild head injuries has lowered the threshold of these groups resulting in less BRC and consequently increasing the forward players' vulnerability to neuropsychological impairment. In relation to the Under 21 group, this result possibly reflects the additional damage sustained by the Forwards as a result of lowered BRC.

5.3. CONCLUSION

The findings of the present study suggest that the rugby players are evidencing chronic or persisting signs of cognitive deficit associated with diffuse cerebral damage, due to their exposure to cumulative mild concussive and sub-concussive head injuries. The present findings are consistent with those of other rugby/football-related sports studies into both chronic cumulative sequelae of mild head injury (e.g. Dickinson, 1998; Reid, 1998; Shuttleworth-Jordan et al., 1993) as well as acute/subacute deficit post-single mild head injury (e.g. Barth et al., 1989; Macciocchi et al., 1996; Maddocks & Saling, 1991; Hinton-Bayre et al., 1997). Furthermore, the findings of this study suggest that the rugby forward players are most susceptible to cognitive impairment, due to their increased involvement in repetitive

physical collisions and consequently increased exposure to head insults. This finding was consistent, in particular, with the findings of those researchers involved in the first phase of the broader research project investigating the brain damage effects of cumulative mild head injury in rugby (i.e. Dickinson, 1998; Reid, 1998).

Satz's (1993) theory of brain reserve capacity (BRC) provided a conceptual basis for understanding the cumulative effects of mild head injury. In this regard, the poorer performance of the Under 21 Rugby players relative to Hockey Control on tests not as sensitive to diffuse brain damage may be understood in terms of the lower BRC of the Under 21 Rugby group. These players have a significantly lower education level and estimated premorbid IQ relative to Hockey Control which may have lowered their BRC threshold, resulting in less BRC and consequently increasing their vulnerability to neuropsychological impairment. In addition, the overriding lack of differentiation between the Under 21 Rugby Forwards and the Under 21 Rugby Backs may also be attributed to lower BRC which is causing deficit to be shown in both the forward players as well as the back-line players, thereby in the main leading to the lack of significant differences between these two subgroupings. The tendency for Total Rugby Forwards and Springbok Rugby Forwards to evidence impairment in certain areas of cognitive functioning relative to the respective Total Rugby Backs and Springbok Rugby Backs can also be understood in terms of BRC theory. In this regard, the involvement of these forward players in more repetitive physical collisions and consequently their exposure to more head insults relative to the back-line players, may have lowered the critical threshold, thereby resulting in lower BRC and an increased vulnerability to the risk of functional impairment.

Thus, the present study found definitive signs of mild cognitive deficit in rugby players, particularly those involved in forward positions. Although the practical consequences of such deficits are unknown, the risk of rugby players sustaining permanent neuropsychological deficit should be seriously considered by all concerned with the game of rugby.

5.4. EVALUATION OF PRESENT RESEARCH

In general, this study is considered to be methodologically rigorous. Although there are some limitations, these do not detract from the strength of the consistent finding of cognitive deficit across neuropsychological tests known to be sensitive to cerebral damage. Both the methodological strengths and limitations will be addressed in this section.

The methodological strengths were identified as follows:

- The present study expanded the existing rugby sample of Springbok rugby players (n = 26) by including the Under 21 Rugby group (n = 19) to form a total rugby sample of 45 top national rugby players. The larger numbers served to strengthen the indications provided by similar studies in the area of cumulative mild head injury in rugby players (e.g. Shuttleworth-Jordan et al., 1993; Dickinson, 1998; Reid, 1998).

- This study involved the comparison of group means across rugby groups relative to the group mean of the hockey control group on neuropsychological tests. This comparative study employed a matched control group of high-level hockey players, which is considered preferential to comparing the sample to available norms. Although head injury studies without control groups have tended to use available normative data, this norm comparison may not be ideal, as the norms tend to be derived from a more general population of individuals. Thus, the comparison of the rugby sample with a matched control group of top-level non-contact sport athletes as accomplished within this study is preferable.

- The Protea cricket group, which was employed in the first phase of the larger research project, was viewed as a somewhat confounded control group for the following reasons: they were assessed post-season (Springbok rugby players were tested pre-season) and consequently were fatigued and unmotivated; they were depressed as they had lost the cricket season; and many of them participated in rugby during their off-season and hence might have sustained cumulative mild head injuries resulting in a possible underestimation of the extent of deficit in the rugby playing group. Because this control group was not considered ideal, the cricket players were excluded from the second phase of the research, and a new control group comprising national hockey players was created. The rationale for including the hockey group was that hockey is considered to be a non-contact sport, the hockey players were assessed pre-season and furthermore, because hockey is played in the same season as rugby, a decreased likelihood of players being involved in both sports was assumed. Although the findings of the present study replicate the results of Dickinson (1998) and Reid (1998) using a new control group, the results from Ancer's study (1999) are not yet available. Since the present study replicates Ancer's methodology, it is not possible to discern whether or not Hockey Control was a more discriminating control group or not.

- A comprehensive neuropsychological test battery was employed which included tests known to be sensitive to the effects of diffuse brain damage, which is typically associated with a closed head injury. The battery included tests measuring abilities and current functioning across a variety of cognitive modalities which tend to be compromised when a closed head injury is sustained (Lezak, 1995). Delayed versions of tests were also included in the neuropsychological battery, because delayed memory tends to be more sensitive to the effects of diffuse brain damage than immediate recall (Lezak, 1995). This comprehensive battery allowed for greater clarity in determining the full extent of deficits present, which would have not been possible if a smaller battery had been employed. The tests employed also enabled the researchers to calculate an estimated premorbid level of cognitive functioning for each of the participants, allowing for comparisons of groups on different levels of intellectual functioning and the monitoring of possible effects.

- Exclusion factors (including a reported history of substance abuse, a neurological/ psychiatric disorder known to negatively impact on cognitive functioning, and a reported history of moderate to severe non-sport related head injury) were controlled for.

The methodological limitations were identified as follows:

- This study focused solely on group comparisons, and thus missed out on possible individual variations which could enrich interpretation of results.

- These results apply only to top-level national rugby players and consequently may not be representative of the total rugby playing population. If this is the case, then the generalisability of these results may be somewhat limited in terms of their applicability to rugby players participating at lower levels or even at school-levels of competition. Thus, whether or not the findings of the present study can be extrapolated to less experienced or lower exposure rugby players remains to be determined in future investigations.

- Some differences were noted between Under 21 Rugby and Hockey Control with respect to the demographic data (age, education, and estimated premorbid IQ). However, positional comparisons (Forwards versus Backs) within the rugby groups revealed no significant differences between them on all demographic variables.

- Estimated premorbid IQ was based on two tests (and in rare instances on only one test), namely SAWAIS Picture Completion Subtest and/or SAWAIS Comprehension Subtest. Although it would have been preferable to include more tests to gauge estimated

premorbid IQ, the two tests used are good indicators of premorbid intellectual ability and tend to hold in individuals with diffuse brain damage (Lezak, 1995).

5.5. RECOMMENDATIONS FOR FUTURE RESEARCH

It is recommended that a prospective study be undertaken into the cumulative effects of mild head injury in rugby players. This study should be initiated at schoolboy rugby level and should involve the follow-up of these boys through their rugby playing careers. The present study found a clear differentiation between the Springbok rugby forwards and backs (with the forwards demonstrating significant cognitive deficit). However, this differentiation was not as clear when examining the cognitive performance of the Under 21 rugby forwards relative to the backs, which was explained as being due to the lower BRC of this group. It is expected that little differentiation in terms of positional play would be evident among the schoolboy players, due to their being exposed to fewer and probably less severe (schoolboy rugby is not as rough as national level rugby) concussive and sub-concussive mild head injuries. However, as was noted in comparison of the Under 21 group with the hockey control, it is expected that cognitive deficit will begin to show up in these young players on tests known to be sensitive to diffuse brain damage when compared with a matched non-contact sport control group. As these boys are followed up over the years and they begin to play more consistently in a particular position, it is expected that this deficit will become more evident on certain of the tests and that some differentiation will begin to show between the forward and back players. Such a study would provide important baseline data for future studies and, in addition, might identify risk factors that might lower BRC and increase the players' vulnerability to risk of functional impairment. In this way, those rugby players at increasing risk of cognitive deficit due to their continued participation in this sport can be identified and cautioned against further involvement.

In line with the idea that secondary stressors, such as hypoxia (Ewing et al., 1980) or fatigue (Jordan, 1997), may temporarily lower an individual's critical threshold thereby eliciting underlying symptomatology, it is recommended that future research studies include such a stressor. Thus, the above-mentioned prospective study of schoolboy rugby players could be extended to include such a stressor which would add substantial value to the findings.

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Appendix I

Consent Form

Appendix II

Pre-assessment Questionnaire

RHODES UNIVERSITY
Grahamstown • 6140 • South Africa

PSYCHOLOGY CLINIC • Tel: (0461) 31 1296/7 • Fax: (0461) 31 1296

NEUROPSYCHOLOGICAL ASSESSMENT RESEARCH: CONSENT FORM

I hereby consent to undergo a neuropsychological assessment. I understand the following: (i) that the assessment takes 1½ to 2 hours per person, and will be conducted by a skilled clinician trained at Rhodes University; (ii) that the assessment involves a series of questions and a variety of intellectual tests which will not be harmful and are usually quite enjoyable for the testee; (iii) that the results will serve as a group data base for comparative purposes between sportsmen who are intensively involved in a contact sport and those who are not; (iv) that individual results will be *totally confidential and remain anonymous*

I further understand that the information gained in my assessment will not be divulged to anyone other than myself on request, and will have no implications with respect to my ability to play sport at the national level.

Name:_____

Signed:_____ Date:_____

RHODES UNIVERSITY PSYCHOLOGY DEPARTMENT

Pre-assessment Questionnaire

NAME:_____DATE OF BIRTH:_____

ADDRESS:_____

PHONE:_____HIGHEST QUALIFICATION:_____

FIRST LANGUAGE:_____

- GENERAL HISTORY

Question 1

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

If Yes, when?_____For what reason?_____

Question 2

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

If qualification lower than matric, please state average mark attained_____

Question 3

What was your final result at University?

Undergraduate:_____

Postgraduate:_____

Question 4

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

If Yes, please specify_____

_____

Question 5

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

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

Question 6

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

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

Question 7

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

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

Question 8

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

If **Yes**, please specify_____

Question 9

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

If **Yes**, how much?_____

Question 10

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

Question 11

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

Question 12

What other forms of substances do you take?_____

How often?_____

Question 13

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

[] Yes [] No

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

Injury 1

- What caused the injury/concussion?_____

_____

_____

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

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

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

  If Yes, for how long?_____

Injury 2

- What caused the injury/concussion?_____

_____

_____

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

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

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

  If Yes, for how long?_____

- **SPORTS HISTORY**

Question 14

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

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

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

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

_____

e) In which position do you play now?_____

Question 15

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

[] Yes **[] No**

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

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

Injury 1

- What caused the injury/concussion?_____

_____

_____

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

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

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

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

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

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

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

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

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

If Yes, please specify_____

_____

Injury 2

- What caused the injury/concussion?_____

_____

_____

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

  If Yes, for how long?_____

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

  If Yes, for how long?_____

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

  If Yes, for how long?_____

- Were you hospitalised? [] Yes [] No

  If Yes, for how long?_____

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

  If Yes, please specify_____

_____

Injury 3

- What caused the injury/concussion?_____

_____

_____

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

  If Yes, for how long?_____

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

  If Yes, for how long?_____

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

  If Yes, for how long?_____

- Were you hospitalised? [] Yes [] No

  If Yes, for how long?_____

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

  If Yes, please specify_____

_____

Injury 4

- What caused the injury/concussion?_____

_____

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

  If Yes, for how long?_____

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

  If Yes, for how long?_____

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

  If Yes, for how long?_____

- Were you hospitalised? [] Yes [] No

  If Yes, for how long?_____

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

  If Yes, please specify_____

_____

Injury 5

- What caused the injury/concussion?_____

_____

_____

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

  If Yes, for how long?_____

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

  If Yes, for how long?_____

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

  If Yes, for how long?_____

- Were you hospitalised? [] Yes [] No

  If Yes, for how long?_____

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

  If Yes, please specify_____

_____

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

_____

_____

Question 16

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

_____

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

[] Yes [] No

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

Injury 1

- What caused the injury/concussion?_____

  _____

  _____

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

  If Yes, for how long?_____

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

  If Yes, for how long?_____

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

If Yes, for how long?_____

- Were you hospitalised? [] Yes [] No

If Yes, for how long?_____

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

If Yes, please specify_____

_____

Injury 2

- What caused the injury/concussion?_____

_____

_____

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

If Yes, for how long?_____

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

If Yes, for how long?_____

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

If Yes, for how long?_____

- Were you hospitalised? [] Yes [] No

If Yes, for how long?_____

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

If Yes, please specify_____

_____

Injury 3

- What caused the injury/concussion?_____

_____

_____

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

If Yes, for how long?_____

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

  If Yes, for how long?_____

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

  If Yes, for how long?_____

- Were you hospitalised? [] Yes [] No

  If Yes, for how long?_____

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

  If Yes, please specify_____

Appendix III

Assessment Schedule and Neuropsychological Test Battery

NEUROPSYCHOLOGICAL TESTING

ASSESSMENT SCHEDULE

Testee:_____ **Date:**_____

Time	Test
	1. Consent form
	2. Pre-assessment questionnaire
	3. Symptom checklist
	4. Digit Symbol *including* INCIDENTAL RECALL
	5. Trail Making A and B
	6. Words-in-a-Minute
	7. "S" Words-in-a-Minute
	8. Finger Tapping Test A
	9. Digit Symbol DELAYED RECALL (20mins)
	10. WMS - Designs - IMMEDIATE RECALL
	11. Picture Completion
	12. Comprehension
	13. WMS - Designs - DELAYED RECALL (20mins)
	14. WMS - Paired Associate Learning - IMMEDIATE RECALL
	15. Digit Span
	16. Digit Supraspan A and B
	17. Finger Tapping Test B
	18. WMS - Paired Associate Learning - DELAYED RECALL (20mins)

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.

NAAM
NAME .....

Datum
Date .....

SLEUTEL
KEY

1	2	3	4	5	6	7	8	9
–	И	⊐	L	∪	O	∧	X	=

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

Aantal korrek 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)

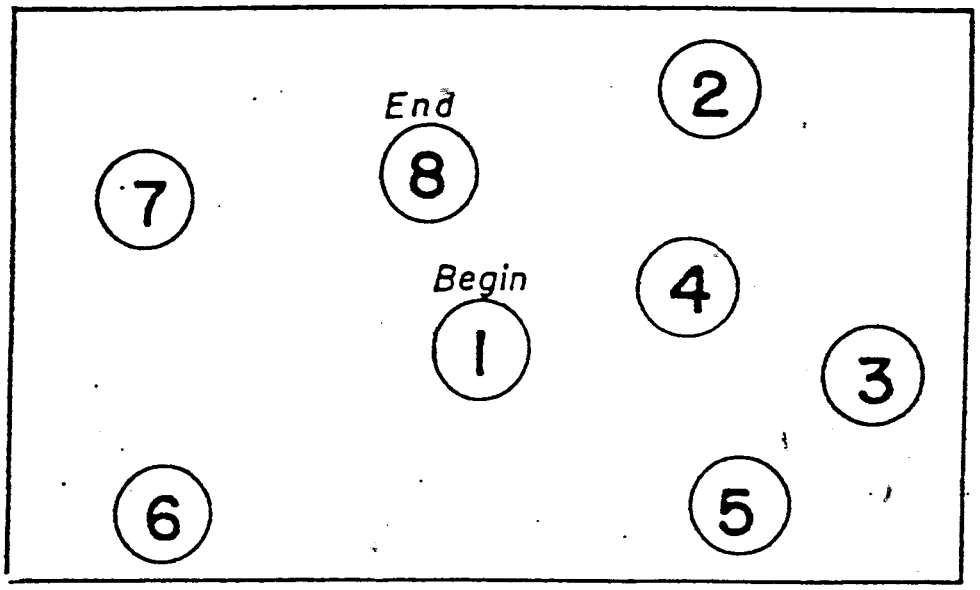
Draw a line to join the circles consecutively by alternating between 1 and A, as fast as you can.

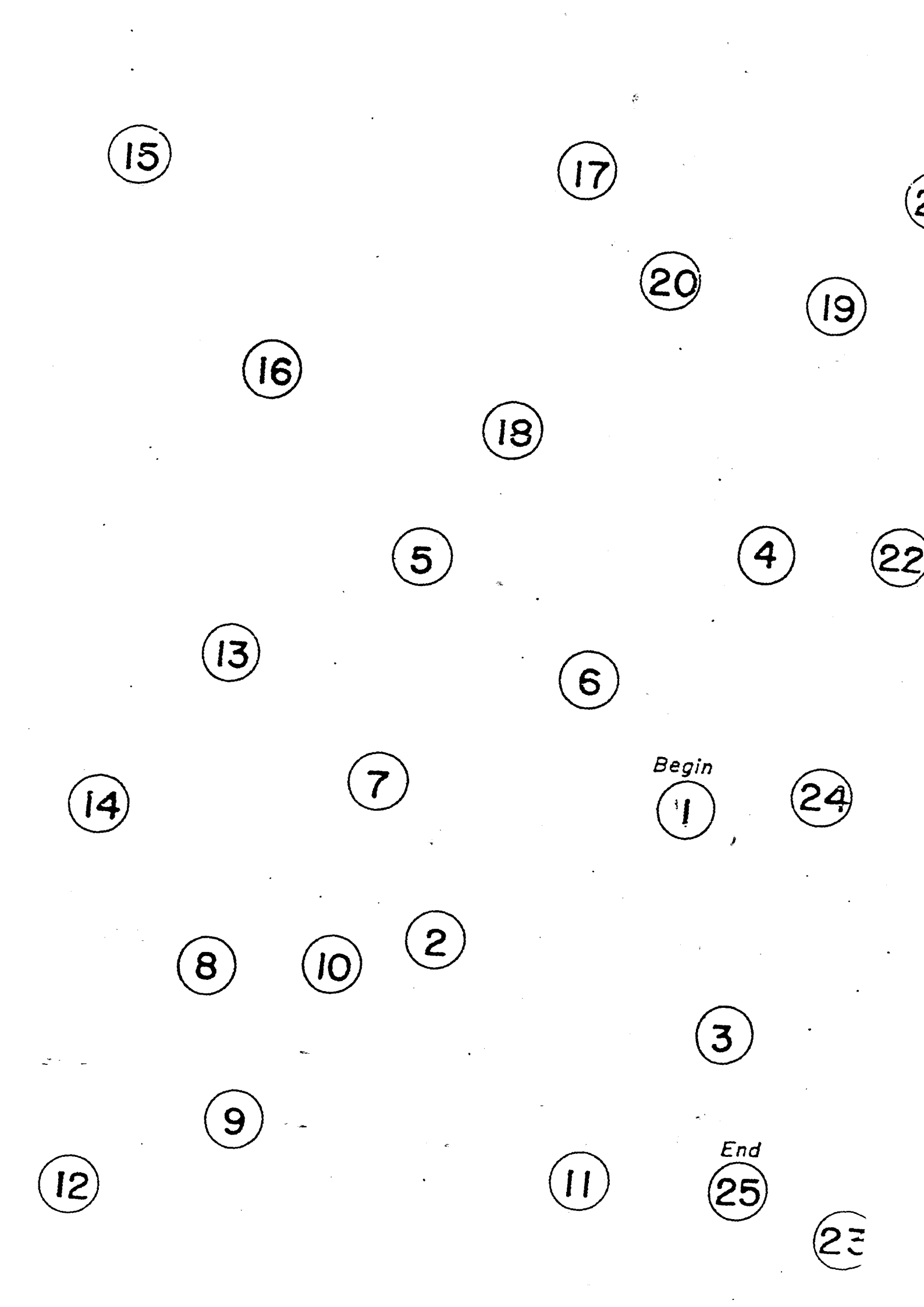
(Note: If subject makes mistake, **don't stop timing**; point out mistake and subject carries on).

TRAIL MAKING

Part A

SAMPLE

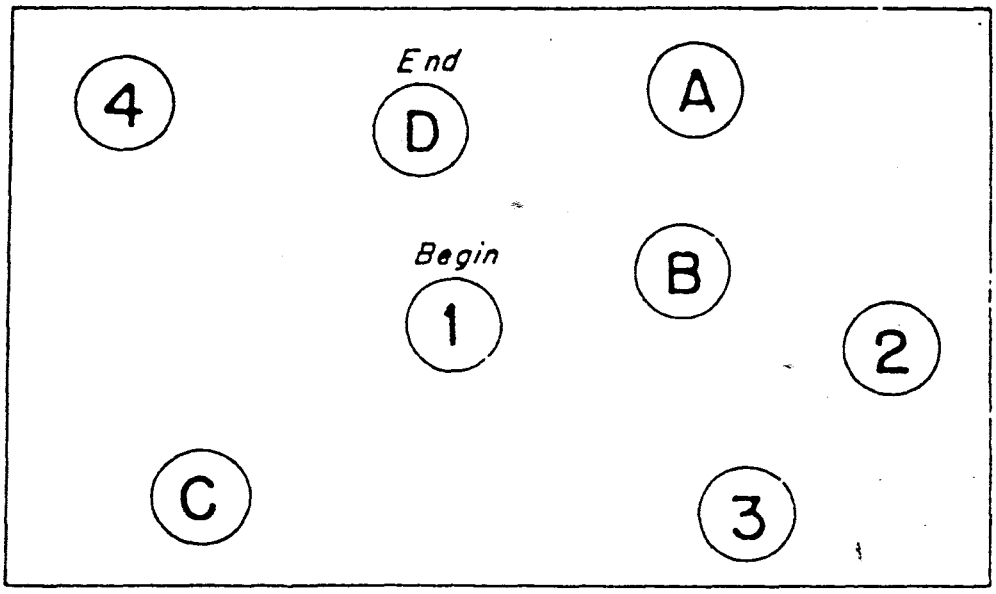


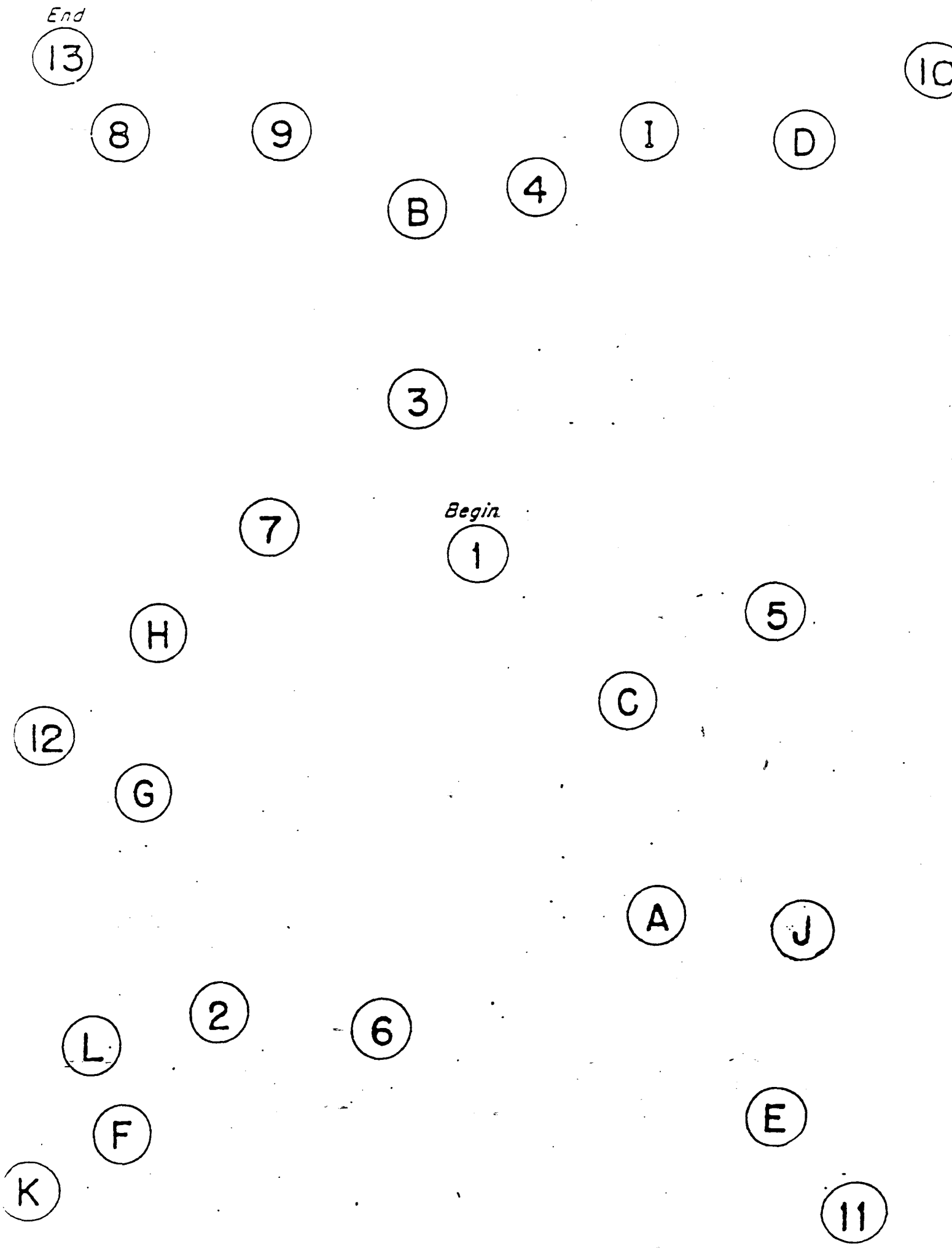

15
17
20
19
16
18
5
4
22
13
6
7
Begin
1
24
14
2
8
10
3
9
End
12
11
25
23

TRAIL MAKING

Part B

SAMPLE




End
13
10
8
9
I
D
4
B
3
7
Begin
1
5
H
C
12
G
A
J
2
6
L
E
F
K
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:

FINGER TAPPING TEST A

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 - 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

DIGITS BACKWARD

"I am going to say some more numbers. This time I want you to say them to me backwards. For example, if I say 6 - 2 - 9, you say .....(wait for them to say 9 - 2 - 6)."

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

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

SCORE:

Digits Forwards: _____

Supraspan A: _____

Supraspan B: _____

Digits Backwards: _____

Digits Difference: _____ (Forwards minus Backwards)

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 = ____

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:

Test 7

PICTURE COMPLETION

Directions

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

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

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

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

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

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

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

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

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

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

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

"Yes, and what else?"

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

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

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

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

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

Test 7

PICTURE COMPLETION

Scoring

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

Maximum Score: 15

PICTURE COMPLETION
VOLTOOIING VAN PRENTE

RESPONSE/ANTWOORD

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

SCORE
TELLING .....

Test 2

GENERAL COMPREHENSION

Directions

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

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

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

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

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

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

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

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

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

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

Test 2

GENERAL COMPREHENSION

Scoring

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

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

Total Score: The sum of marks on the 10 questions

Maximum : 20

Test 2

GENERAL COMPREHENSION

Questions

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

Toets 2

ALGEMENE BEGRIP

Aanwysings

Sorg dat die toetspersoon luister wanneer u die vrae stel. Jong toetslinge en kliniese pasiënte vind dit soms moeilik om die hele vraag te onthou wanneer dit slegs eenmaal gestel word. Dit is derhalwe wenslik om die **vraag te herhaal** indien geen antwoord binne tien tot vyftien sekondes verkry word nie, **maar moenie die bewoording verkort of verander nie.**

Sê: "Nou gaan ek aan u 'n paar vrae stel en ek wil hê dat u my moet vertel wat u in elkeen van die gevalle dink. Daar is geen vasgestelde antwoord nie. Sê net wat u dink. Hier is die eerste een....."

Skryf die toetsling se antwoorde woordeliks neer. As die antwoord baie breedvoerig is en hy so vinnig praat dat sy volle antwoord nie neergeskryf kan word nie, stip die belangrikste punte aan en probeer om soveel as moontlik van die antwoord te benou.

Dit is somtyds nodig om die toetsling aan te moedig. Dit kan gedoen word deur middel van aanmerkings soos: "Ja?", "Gaan voort", ens. As 'n antwoord nie duidelik is nie, sê dan: "Verduidelik asb. verder", of "Kan jy dit vir my 'n bietjie duideliker maak?" Moenie enige vraag vra wat 'n aanduiding kan gee van die soort antwoord wat verlang word nie.

L.W.: Moet nooit oorgaan na die volgende vraag voordat seker gemaak is dat die betekenis van elke antwoord duidelik is nie. Toetsafnemers word aangeraai om die Gids vir Toekenning van Punte voor hulle te hou gedurende toepassing van die toets, veral aangesien bepaalde antwoorde wat verduideliking vereis hier aangegee word.

bv. Vraag 2 "Gaan vertel dit", "Die bestuurder in kennis stel".

Hier moet die toetsafnemer vasstel wat die toetsling in gedagte het en mag volle punte gee slegs waar die toetsling dit duidelik maak dat van die bestuur verwag word om in te gryp om paniek te voorkom en om te sorg dat die vuur geblus word.

Dit is belangrik om **sulke verduidelikings neer te skryf.** Moenie net "Verduidelik" aanteken nie.

L.W.: Ingeval meer as een antwoord gegee word, moet die toetspersoon gevra word watter een hy as die belangrikste beskou en punte moet hiervolgens toegeken word.

Stel al die vrae, behalwe vir persone met baie lae intelligensie.

Toets 2

ALGEMENE BEGRIP

Toekenning van Punte

Toekenning van punte in hierdie toets is 2, 1 of 0, na gelang van die veralgemening en gehalte van die antwoorde. Dit word derhalwe weer beklemtoon dat die toetsafnemer **moet volhou ten einde presies vas te stel wat bedoel word wanneer antwoorde nie duidelik is nie. Dit is veral belangrik in die geval van eenvoudiger persone wat hulself swak uitdruk,** of van persone wat ontwykend antwoord, maar wat skynbaar die korrekte beginsel in gedagte het. Tensy twyfel-

Toets 2

ALGEMENE BEGRIP

Vrae

1. Wat behoort mens te doen as jy in die straat 'n koevert optel wat toegeplak, geadresseer en van 'n nuwe seël voorsien is?
2. Wat sal u doen as u die eerste persoon is wat 'n brand ontdek (of rook en vlamme sien) terwyl u in 'n bioskoop (of teater) sit?
3. Hoekom behoort 'n mens slegte geselskap te vermy?
4. Hoekom moet 'n mens belasting betaal?
5. Waarom word skoene van leer gemaak?
6. Waarom is grond duurder in die stad as op die platteland?
7. Waarom moet 'n motorvoertuig gelisensieer wees voordat dit gebruik mag word?
8. Hoekom is wette nodig?
9. Waarom moet 'n persoon wat buite sy eie land wil reis 'n paspoort besit?
10. Waarom kan mense wat doof gebore is gewoonlik nie praat nie?

GENERAL COMPREHENSION
ALGEMENE BEGRIP

RESPONSE/ANTWOORD

1		
2		
3		
4		
5		
6		
7		
8		
9		
10		

REMARKS
OPMERKINGS .....

.....

.....

.....

.....

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 - *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 = ____

WMS : ASSOCIATE LEARNING - *IMMEDIATE RECALL* **AFRIKAANS**

Testee's Name:_____

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

NOT TIMED

Instruction: "Ek sal nou vir u 'n lys woorde lees, twee op 'n slag. Luister goed want as ek klaar is will ek dat u die woorde onthou wat saamhoort. Byvoorbeeld, as die woorde OOS-WES, GOUD-SILWER is, wanneer ek die woord OOS sê, moet u antwoord (pause) WES. En as ek GOUD sê sal u natuurlik antwoord (pause) SILWER. Verstaan u?"

If the subject is clear as to the directions:

"Nou luister goed na die lys woorde." *P.T.O. for list of words.*

SCORE:

First Recall			Second Recall			Third Recall		
TOTAL	___	___	**TOTAL**	___	___	**TOTAL**	___	___
Easy: 1.		___	Hard: 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		
Metaal	-	Yster	Roos	-	Blom	Baba	-	Huil
Baba	-	Huil	Luister	-	Duim	Luister	-	Duim
Breek	-	Donker	Noord	-	Suid	Noord	-	Suid
Noord	-	Suid	Kool	-	Pen	Skool	-	Winkel
Skool	-	Winkel	Op	-	Af	Roos	-	Blom
Roos	-	Blom	Vrugte	-	Appel	Kool	-	Pen
Op	-	Af	Skool	-	Winkel	Op	-	Af
Luister	-	Duim	Metaal	-	Yster	Vrugte	-	Appel
Vrugte	-	Appel	Breek	-	Donker	Breek	-	Donker
Kool	-	Pen	Baba	-	Huil	Metaal	-	Yster

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
Noord	____		Kool		____	Luister	____	
Vrugte	____		Baba	____		Vrugte		____
Luister		____	Metaal	____		Baba	____	
Roos	____		Skool		____	Metaal	____	
Baba	____		Op	____		Breek		____
Op	____		Roos	____		Skool	____	
Kool		____	Luister		____	Roos	____	
Metaal	____		Vrugte	____		Noord	____	
Skool		____	Breek		____	Kool	____	
Breek		____	Noord	____		Op		____
TOTAL	____	____	**TOTAL**	____	____	**TOTAL**	____	____

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

Score: A/2 + B = ____

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.

DIGIT SUPRASPAN A (*Learning*):
After the second consecutive failure of a digit span on Digits Forward, say:
"I will repeat that one again and see if you can get it this time."

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

SCORE: SUPRASPAN A and B:

TRIAL	1	2	3	4	5	6	7	8	9	10

DIGIT SUPRASPAN B (*Sustained Learning*):
After they have the Supraspan A score you get a Supraspan B score. This is the score for the amount of time it takes them to get the supraspan correct TWICE IN A ROW.
"Let's see if you can get that right again."

If they have a supraspan A score of 4 trials and they are able to repeat the span on the 5th trial - they receive a supraspan B score of 5. If they get the 5th trial wrong - they would need to get the 6th and 7th trials correct to get a supraspan B score of7. Continue until the 10th trial if necessary. If they are still unable to get the span correct twice in a row they receive a score of 10+.

Score above

P.T.O. for Digits Backwards

DIGITS BACKWARD

"I am going to say some more numbers. This time I want you to say them to me backwards. For example, if I say 6 - 2 - 9, you say .....(wait for them to say 9 - 2 - 6)."

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

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

SCORE:

Digits Forwards: _____

Supraspan A: _____

Supraspan B: _____

Digits Backwards: _____

Digits Difference: _____ (Forwards minus Backwards)

FINGER TAPPING TEST B

Testee's Name:_____

Requirements: stop watch

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

Time Limit: **No**

Instruction: "I would now like to repeat the finger tapping test that we did earlier. To refresh your memory, place both your elbows on the table *(examiner models what is required)* and touch each finger to your thumb in turn starting with your index finger *(examiner can again model what is required)*. 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:

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 = ____

WMS ASSOCIATE LEARNING *DELAYED RECALL* AFRIKAANS

Testee's Name:_____

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

NOT TIMED

Instruction: "Onthou u die woorde wat ek vroe vir u gelees het. Ek will sien hoeveel van dir pare u kan onthou."

First Recall	Easy	Hard
Noord	____	
Vrugte	____	
Luister		____
Roos	____	
Baba	____	
Op	____	
Kool		____
Metaal	____	
Skool		____
Breek		____
TOTAL	____	____

SCORE:

Delayed recall = ____

Appendix IV

Years of Education and Prorated IQ Scores for Springbok Rugby, Under 21 Rugby, and Hockey Control

Springbok Rugby: Years of Education and Prorated IQ Scores

PLAYER NO.	POSITION	EDUCATION	COMP.	PIC. COMP.	IQ
1	Forward	13	12.5	11.0	115
2	Forward	12	15.0	11.0	125
3	Forward	12	11.0	14.5	123
4	Forward	13	11.5	11.0	113
5	Forward	13	8.5	12.5	104
6	Forward	15	10.5	13.0	115
7	Forward	15	12.5	14.5	129
8	Forward	15	13.0	15.0	133
9	Forward	15	12.5	15.0	132
10	Forward	14	11.5	11.0	111
11	Back	16	11.0	12.5	115
12	Back	15	10.5	15.0	123
13	Back	15	12.5	15.0	132
14	Back	16	15.5	12.5	133
15	Back	15	11.0	12.5	115
16	Back	12	10.5	12.5	113
17	Back	15	12.5	12.5	121
18	Forward	16	11.5	14.5	125
19	Forward	14	12.0	12.5	119
20	Forward	15	12.5	14.5	129
21	Forward	12	9.0	9.5	94
22	Forward	15	9.5	6.5 *	96
23	Back	15	12.5	15.0	132
24	Forward	15	14.0	14.0	133
25	Forward	16	13.5	12.5	125
26	Back	12	10.5	8.5	96

KEY

EDUCATION	- Total number of years of education
IQ	- Estimated Premorbid IQ
COMP.	- SAWAIS Comprehension Subtest
PIC. COMP.	- SAWAIS Picture Completion Subtest
*	- Premorbid IQ was calculated using the single highest score in isolation

Under 21 Rugby: Years of Education and Prorated IQ Scores

PLAYER NO.	POSITION	EDUCATION	COMP.	PIC. COMP.	IQ
27	Back	12	12.0	8.5	103
28	Back	12	10.0	10.0	100
29	Forward	14	10.0	12.5	111
30	Back	12	8.5	6.0	77 **
31	Forward	14	14.0	14.5	136
32	Forward	13	10.5	9.5	100
33	Back	10	9.5	10.0	98
34	Forward	15	9.5	14.5	117
35	Forward	12	11.0	12.5	115
36	Forward	12	11.5	11.0	111
37	Back	13	13.0	12.5	123
38	Forward	11	8.5	10.5	96
39	Forward	13	12.0	9.5	107
40	Forward	13	13.0	10.5	115
41	Back	14	10.0	12.5	111
42	Back	12	8.0 *	12.5	121
43	Forward	8	10.5	8.5	96
44	Forward	12	11.0	14.5	123
45	Back	12	11.0	11.0	108
46	Back	12	8.5	12.5	104
47	Forward	11	7.5	7.0	76 **

KEY

EDUCATION	- Total number of years of education
IQ	- Estimated Premorbid IQ
COMP.	- SAWAIS Comprehension Subtest
PIC. COMP.	- SAWAIS Picture Completion Subtest
*	- Premorbid IQ was calculated using the single highest score in isolation
**	- IQ < 85, Player excluded from final sample

Hockey Control: Years of Education and Prorated IQ Scores

PLAYER NO.	EDUCATION	COMP.	PIC. COMP.	IQ
48	13	14.0	12.5	128
49	12	7.5 *	12.5	121
50	16	12.0	12.5	119
51	16	15.5	15.0	144 **
52	15	14.0	14.5	136
53	15	10.0	15.0	121
54	14	13.5	14.5	133
55	15	16.0	14.5	144 **
56	12	14.0	14.5	136
57	16	15.5	11.0	128
58	14	11.5	14.5	125
59	12	13.5	10.0	115
60	15	13.0	12.5	123
61	16	10.0	12.5	111
62	15	11.5	14.5	125
63	14	13.5	12.5	125
64	15	11.5	11.0	111
65	15	13.0	15.0	133
66	15	11.5	14.5	125
67	14	11.5	10.0	107
68	14	9.5	12.5	108
69	15	12.5	12.5	121
70	14	10	12.5	111

KEY

EDUCATION	- Total number of years of education
IQ	- Estimated Premorbid IQ
COMP.	- SAWAIS Comprehension Subtest
PIC. COMP.	- SAWAIS Picture Completion Subtest
*	- Premorbid IQ was calculated using the single highest score in isolation
**	- IQ > 140, Player excluded from final sample

Appendix V

Similarities and Differences Between the Various Forms of Rugby/Football-Related Sports (i.e. Rugby Union, Rugby League, Australian Rules Football, and American Football)

Similarities and differences between the various forms of rugby/football-related sports (i.e. Rugby Union, Rugby League, Australian Rules Football, and American Football)

Within the related sports of rugby/football, tackling forms a central common feature. Tackling occurs when a player, frequently running at speed, is held by one or more opponents and brought to the ground, resulting in sudden deceleration. In all forms of rugby/football-related sports, the forward players tend to be involved in more body contact throughout a game and hence tend to be physically bigger and stronger individuals relative to the back-line players who tend to be smaller and faster.

Rugby Union is the form of rugby/football played in South Africa and is the focus of the present research study. Hence, the other three related sports (namely Rugby League, Australian Rules Football, and American Football) will be described in relation to Rugby Union. The main difference between Rugby Union and the other forms of rugby/football lies in the inclusion of 'rucks' and 'mauls'. Rucks and mauls, which form a unique and prominent part of the game in Rugby Union, involve those instances in which the ball-carrier is held by the opposition while the forward and back players of both teams attempt to gain possession of the ball. Players joining the ruck or maul tend to run headfirst into this situation, thereby placing themselves at risk for mild head injury. Rucks and mauls do not occur in Rugby League and Australian Rules Football, although a similar type of activity forms part of the game in American Football.

A scrum involves the positioning of the ball between two opposing teams consisting of forward players, with each team attempting to push the opposing team off the ball so as to gain possession for their team. While scrums commonly occur in both Rugby Union and Rugby League, the two sports differ in terms of the number of players involved in the scrum, with Rugby Union and Rugby League requiring eight and six forward players respectively per team. Scrums, however, do not occur in Australian Rules Football nor in American Football.

Scrum caps, made of foam rubber, are occasionally worn by players participating in Rugby Union, as well as in the related contact sports of Rugby League and Australian Rules Football. While these caps do provide some protection from mild head injury, their main purpose is to protect the players' ears from being damaged during a scrummage. American Football differs from the other three related sports in that protective clothing (e.g. helmets, shoulder pads etc.) is compulsory for all players at all levels of this game.

Thus, because the various forms of rugby/football-related sports clearly share a number of commonalities (e.g. tackling), the extent to which the differences between the sports will affect cognitive performance is unknown. However, the injuries of these related sports are similar and hence the findings of research in the field of rugby/football have relevance for the present study which focuses on the cumulative effects of mild head injury in Rugby Union.