- HEADS AND TALES -

THE EFFECT OF MILD HEAD INJURIES ON RUGBY PLAYERS: COGNITIVE DEFICIT AND POSTCONCUSSIVE SYMPTOMS

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To

Ursula Constance Border

Thank you

for the sense of history you provide,

for unconditional love

and

for countless cups of tea.

Your life is an inspiration.

ABSTRACT

This study investigated the cumulative effect of mild head injuries on rugby players. A comprehensive battery of neuropsychological tests was administered and subjects completed a self-report postconcussive symptom questionnaire. Data were collected for the two rugby groups, Springbok rugby players (n = 26) and Under 21 rugby players (n = 19), and for the control group, national hockey players (n = 21). Group comparisons of the percentage of individuals with deficit or self-reported symptomatology were made between: (i) the contact sport groups and the control group; (ii) the forwards and the backs within each rugby group and the rugby forwards and the control group; and (iii) the Springbok and Under 21 rugby players. Broadly speaking, comparative results on the neuropsychological tests and the self-reported postconcussive symptoms clearly distinguished between contact sport players and non-contact sport players and indicated the presence of diffuse brain damage in the contact sport players. There was also clear evidence of positional variation within the rugby groups, with the forwards (more full contact positions) most susceptible to impairment. Neuropsychological test results revealed deficit in information processing speed, attention and concentration, mental flexibility, visual memory and verbal new learning. The most significant neuropsychiatric complaints were reported in the areas of memory, social contact, sensitivity to noise, lowered frustration tolerance, anxiety and worry, and depression. The most sensitive neuropsychological test used in the present study was the Digit Symbol Substitution test. This test clearly distinguished contact sport players from non-contact sport players, and forwards from backs.

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CHAPTER 1 : INTRODUCTION

Many myths exist about the nature and consequences of head injuries sustained playing contact sport and there is a serious lack of knowledge about sporting injuries which needs to be addressed. A graphic case in point occurred on Saturday 16 May 1998. Morné Prince, a young flyhalf, collapsed, fell into a coma and subsequently died after apparently landing on his head while being tackled during a rugby match. The editorial in the Eastern Province Herald stated that "...we hope that this tragedy will not send out the wrong signals to parents of young rugby players. At this stage there is every indication that this was a freak occurrence which happened in open play, and not in a ruck or a loose maul... areas which the game's international authorities have made much safer through new laws" ("A Salute to Morné", 1998). While Morné's death may indeed have been a "freak occurrence", the fact is that concussion in rugby is a common occurrence (Jordan, 1998). As the age and competence of rugby players increases so does the incidence of injury (Nathan, Goedeke, Noakes, 1983). In South Africa there has been an increase in rugby injuries, of which concussion is common, and this trend shows no sign of abating (Jacobson & Speechley, 1988).

Only recently has it been realised that minor or trivial head injuries can have long-term and even permanent neurocognitive effects (Anderson, 1996) and although concern about mild head injuries in sport has increased over the last ten years, few well-controlled studies exist (Macciocchi, Barth & Littlefield, 1998). It is difficult to accurately establish the prevalence of sports-related concussion or mild head injury as many cases are unreported. Boll (1983) described mild head injury as:

> "...a quiet disorder. It is common, typically bloodless and without call for significant medical intervention. It seems even more quiet because the noise it does make (its symptoms) is often attributed to other causes." (Boll, 1983, p. 74).

Because of the apparent lack of symptomatology, players are reluctant to leave the field during a match or to miss a match because of such an injury. There is, therefore, a tendency amongst players to deny the presence (or seriousness) of head injuries, and in this they are often supported by sporting authorities. During the 1999 Rugby Union World Cup the researcher witnessed an incident in a televised match where a player received a heavy tackle and was obviously extremely dazed and confused as a result. The player, however, was not removed from the field immediately as this would have resulted in a compulsory three-week absence from the game. Instead the player continued with the game for about 10 minutes before being brought off for some 'other' injury, thus a key player remained eligible to play the following weekend. The 'tales' players tell about such head injuries put them at risk of further head injury which can have catastrophic consequences.

Players of contact sports are, by the very nature of the game, at risk for head injuries. Research into these sports injuries can aid in understanding the mechanisms and effects of mild head injuries which occur in more common situations (Ruchinskas, Francis & Barth, 1997). Professional athletes tend to make good research participants as they are generally not as prone to spontaneous cognitive decline from confounding factors such as age, poor health, substance abuse etc (Ruchinskas et al., 1997). Professional athletes provide a laboratory in which mild head injuries can be studied (Ruchinskas et al., 1997). However, care must be taken when applying knowledge gained in this manner to single head injuries as there is very little published data comparing those with multiple head injuries (such as sportsmen) with those with single head injuries (Binder, 1997).

There is a growing body of neuropsychological research into mild head injuries in the various contact sports of American football (Barth et al., 1989), Australian rules football (Maddocks & Saling, 1991), Rugby League (Hinton-Bayre, Geffen & McFarland, 1997) and Rugby Union (Shuttleworth-Jordan, Balarin & Puchert, 1993). However, most research involving Rugby Union has been in the form of epidemiological studies, with very little of the current neuropsychological research focussing on Rugby Union, apart from the original study by Shuttleworth-Jordan et al. (1993) and the recent research by Ancer (1999), Dickinson (1998) and Reid (1998).

Although American football, Australian rules football, Rugby League and Rugby Union[1] are different sports, due to their nature they all have similar injury profiles and thus there are sufficient parallels between them that research based on the other sports may be applied to Rugby Union. All four sports involve physical collisions between players (and often the ground), resulting in sudden acceleration and deceleration which can place stress on the brain and cause impacts on the head and neck. An example is tackling the opponent. In a tackle the player holding the ball is prevented from moving forward and/or brought to the ground by members of the opposing team. Players who are tackled and/or the player making the tackle are often running at speed when this occurs, resulting in sudden deceleration. In Rugby Union and Rugby League only the player holding the ball may be tackled while in the other two sports players may also be 'blocked' while not carrying the ball. Players of American Football gain some protection through the use of protective equipment such as helmets and shoulder pads while in Rugby Union and Rugby League protective clothing is only allowed provided it contains nothing rigid.

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[1] Although Rugby Union and Rugby League are the most similar amongst these sports, Rugby Union does seem to present more opportunity for head injury in terms of the numbers of players involved in each game, the nature of its scrum (involving more players and more contact) and, unlike Rugby League, its use of rucks and mauls.

Recently research in South Africa was begun into the effect of mild head injuries in top-level Rugby Union players using top-level cricketers as a non-contact sport control group (Ancer, 1999; Dickinson, 1998; Reid, 1998). Participants completed two questionnaires, one providing demographic and historical information and the other a symptom checklist, and then underwent a neuropsychological assessment. Three separate analyses were conducted and the results of this research strongly implied the presence of subtle brain damage following mild head injuries. However, the cricket players did not prove to be an ideal non-contact sport control group. While the rugby players were assessed pre-season, the cricket players were assessed post-season, after returning from a long (and unsuccessful) tour of England. In addition, some cricketers played rugby as their winter sport while at school and university with some continuing on to play at a high level, thereby putting themselves at risk for similar minor head injuries as the rugby players. This may have led to an underestimation of the deficit present amongst the contact sport players. Thus it was decided to expand this research, not only by including additional rugby players, but also by making use of a less confounded control group in order to further highlight any deficit amongst the contact sport players. To boost the number of rugby players it was decided to include the top-level Under 21 rugby players. The Springbok hockey players were used as a non-contact sport control group since most hockey players have played very little rugby, if any, as both are winter sports. Participants completed two questionnaires and underwent the same neuropsychological assessment as mentioned above. The same three levels of analyses used for the first phase were replicated for the second phase of this research, namely, (i) a comparison of mean scores and standard deviations of the contact sport group and controls (Ancer, 1999); (ii) a comparison of mean scores and standard deviations of the contact sport group and controls to normative data (Reid, 1998); and (iii) a comparison of the percentage of contact sport players and controls with deficit relative to normative data and a comparison of the percentage of contact sport players and controls with postconcussive symptoms (Dickinson, 1998). This study replicates Dickinson's methodology which proved to be a powerful method.

This research will begin by first reviewing the literature surrounding mild head injuries and then describing the methods and results of this study. Finally, the discussion will tie together the literature review and the results of this study, comparing the results of this research to existing published studies.

CHAPTER 2 : LITERATURE REVIEW

The effects of head injuries have been noted and recorded for hundreds of years. The ancient Egyptians were aware that head injuries caused diverse disturbances of function and described cases of paraplegia and speechlessness following left temporal depressed skull fractures (Levin, Benton & Grossman, 1982). Similarly, Hippocrates said that no head injury is too trivial to ignore (Galbraith, 1985). Since then much has been learnt about head injuries but there is often confusion over the language and terminology of head injuries. In order to aid this discussion it is first necessary to develop a common language of head injuries. This chapter will provide a context for an understanding of mild head injuries in general and, more specifically, mild head injuries in sport. Research into the effect of mild head injuries in boxing, soccer, American football, Australian rules football, Rugby League and Rugby Union will then be discussed in detail.

2.1. OPEN AND CLOSED HEAD INJURIES

When discussing head injuries, an important distinction must be drawn between an open head injury and a closed head injury. In an open head injury, also known as a penetrating head injury (Richardson, 1990) or a missile injury (Levin et al., 1982), the dura mater is torn and the contents of the skull are exposed (Richardson, 1990). This kind of injury, where the scalp is lacerated, the skull perforated or fractured, and the brain tissue in the path of the foreign body is lacerated, is often caused by explosively propelled objects such as a gunshot or fragments from an exploding shell (Levin et al., 1982; Richardson, 1990), or by sharp instruments such as knives or umbrellas (Richardson, 1990). The result of open head injury is determined to a large degree by the energy of the penetrating impact. With a lower energy impact, damage tends to be concentrated in the path of the penetrating object. A circumscribed focal lesion results which produces fairly limited and predictable results (Lezak, 1995). Higher energy impacts can cause further damage due to shockwaves and pressure effects, possibly causing haemorrhages, ischemia or edema which may also leave permanent damage (Lezak, 1995).

Closed head injuries are more common, and account for over 90% of all civilian head trauma (Lezak, 1995; Lishman, 1987). A closed head injury is generally caused by blunt trauma to the head, either as a result of acceleration of the head due to the impact of a faster moving object upon the slower moving or stationary head, or due to the sudden deceleration of the head when it comes into contact with a slower moving or stationary object (Levin, 1982; Graham, Adams, Path & Gennarelli, 1987; Richardson, 1990). Closed head injuries can range in severity from mild to moderate to severe (Dacey & Dikmen, 1987; Levin et al., 1982). Closed head injuries will be the focus of this research.

Many terms have been used in reference to closed head injuries, with 'concussion' the most commonly used term. Gasquoine (1997) writes that the term concussion has generally been replaced by 'closed head injury' or 'traumatic brain injury', with the less severe of these injuries generally referred to as 'mild head injury' or 'minor traumatic brain injury'. However, authors sometimes refer to the terms 'mild head injury' and 'concussion' interchangeably (Bohnen & Jolles, 1992). In addition, not only do some authors still appear to use the term 'concussion', but older research studies regularly refer to 'concussion' and not 'mild head injury'. As Gennarelli (1987) states, 'cerebral concussion' is a term that is so well established that it would serve little purpose to attempt to discontinue its use. In addition, although Gasquoine (1997) states that there has been a move away from using the term 'concussion' synonymously with head injury, the regularly reported physical, emotional and cognitive sequelae following mild head injury are still referred to as 'postconcussive symptoms' or 'postconcussive syndrome'.

For the purposes of this research the trend noted by Gasquoine (1997) will be followed, and the term 'mild head injury' will be used exclusively, except when reporting work where authors have used terms such as 'minor head injury', 'concussion', or 'cerebral concussion'. When discussing their work, terms will be referred to as originally used by the author(s). The term 'postconcussive symptoms' will be used with specific reference to the subjective symptoms that may be reported following a mild head injury. Mild head injuries in general will be discussed in more detail below, looking specifically at defining mild head injuries, their epidemiology and demographics, and the pathophysiology of mild head injuries.

2.2. MILD HEAD INJURIES

As stated above, closed head injuries can range in severity from mild to moderate to severe (Levin et al., 1982). However, it is not an easy task to define the severity of closed head injuries. Numerous differing classifications abound in the literature (Binder, 1986) with greater consensus on the definition of more severe head injuries than the more minor head injuries (Satz et al., 1997). Kibby and Long (1996) state that the term 'minor head injury' is commonly used to indicate those suffering from traumatic brain injury of mild to moderate severity. Binder (1986) reports that 'mild' has traditionally referred to those head injuries where the period of posttraumatic amnesia is relatively short, there is no structural damage to the skull or brain, and there is a Glasgow Coma Scale (GCS) score of 13 or more. Some studies have used one or more of these criteria while others have added other criteria. Dacey and Dikmen (1987) used the GCS score alone to determine the severity of a head injury. A GCS score of less than 8 indicates severe head injury, a score of 9 to 12 indicates moderate head injury and a score of 13 to 15 indicates mild head injury (Dacey & Dikmen, 1987). However, while the GCS is effective in evaluating severe head injury it was never intended as a means of distinguishing between different types of mild injury and lacks the sensitivity for this task (Jennett, 1989; Kraus & Nourjah, 1989; Schoenhuber & Gentilini, 1989). For Rimel, Giordani, Barth, Boll and Jane

(1981), minor head injury refers to a cranial trauma resulting in a period of unconsciousness of 20 minutes or less, a Glasgow Coma Scale score of 13 or more, and hospitalisation of less than 48 hours. McLean, Temkin, Dikmen and Wyler (1983) used a similar definition but excluded patients with chronic alcoholism and previous head injury.

The Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (in Kibby and Long, 1996) took the above mentioned definitions of minor head injury further and developed a more formal definition that attempts to delineate both the upper and the lower limits of mild head injury as follows:

> "A patient with mild traumatic brain injury is a person who has had a traumatically induced physiological disruption of brain function as manifested by at least one of the following:
>
> 1. any period of loss of consciousness;
> 2. any loss of memory for events immediately before or after the accident;
> 3. any alteration in mental state at the time of the accident (e.g. feeling dazed, disorientated, or confused); and
> 4. focal neurological deficit(s) that may or may not be transient;
>
> but where the severity of the injury does not exceed the following:
>
> loss of consciousness of approximately 30 minutes or less;
> after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15; and
> post-traumatic amnesia (PTA) not greater than 24 hours."
>
> (Kibby and Long, 1996, p. 161).

Although this definition takes an important step by delineating both the upper and the lower limits of mild head injury, some difficulties remain. Kibby and Long (1996) consider that the lower limits proposed above are not sufficient as it does not necessarily imply structural damage to the brain and now includes any impact to the head, however mild, regardless of whether or not it has any consequences. Further, these authors point out that in some respects this definition also combines mild and moderate traumatic brain injury as several studies have defined mild traumatic brain injury as PTA under one hour and moderate traumatic brain injury as PTA ranging from one to 24 hours. They draw attention to the work of Rutherford, Merrett and McDonald (1977) who demonstrated differences on outcome measures depending on length of PTA. The result is that this definition now covers a wide range of severity, making comparison with existing research difficult (Kibby & Long, 1996).

Commensurate with the concerns expressed by Kibby and Long, Evans (1992) states that strict criteria used in recent studies should be used when studying similar injuries in order to avoid further confounding variables. This author suggests that for further study the criteria for mild head injury should be: loss of consciousness for 30 minutes or less or being dazed and confused without loss of consciousness; an initial

GCS of 13 to 15 without further deterioration; an absence of focal neurological deficits and further neurological complications. This definition is not as broad as that of the Mild Traumatic Brain Injury Committee of the American Congress of Rehabilitation Medicine stated above from Kibby and Long. In particular, it excludes the presence of any focal neurological deficit which has the problem of moving into the realm of moderate head injury and thus it more clearly separates mild head injury from moderate head injury. Further, it was Evans' (1992) definition of mild head injury which was used for phase one of this research (Reid, 1998). It was, therefore, deemed appropriate to adopt Evans' more restricted definition for the present research in order to maintain continuity between phases.

2.2.1. EPIDEMIOLOGY AND DEMOGRAPHICS

Estimates of epidemiology of mild head injuries in the general populations vary with Evans (1992) and Kraus (Kraus & Nourjah, 1989) reporting that mild head injuries account for more than 75% of all brain injuries while Lezak (1995) and Lishman (1987) report that this figure is over 90% of all brain injuries. Kraus and Nourjah (1989) estimate that between 66% and 75% of all hospital admissions for head trauma are for mild head injuries. Jennett (1989) states that the percentage of mild head injury could be higher as only 20% of those who attend the emergency room are admitted. Jennett (1976, in Richardson, 1990) also observed that approximately a third of all patients with head injuries had other injuries, and that it might have been these other injuries which resulted in admission. It is estimated that in the United States of America, between 20% and 40% of all patients with mild head injuries do not even seek medical care (Evans, 1992), and the numbers of patients rendered unconscious briefly who do not seek medical assistance are unknown (Rimel et al., 1981). More than two million Americans suffer closed head injuries annually (Weight, 1998) which, given the varying epidemiological percentages for mild head injuries quoted above (between 66% and 90% of all closed head injuries), means this affects at least 1,32 million and possibly more than 1,8 million Americans annually.

About half the mild head injuries suffered in the United States occur in persons between the ages of 15 and 34, with males at about twice the risk of females, although this rate diminishes in the very young and those over the age of 45 (Evans, 1992; Kraus & Nourjah, 1989). Other risk factors include substance abuse (including alcohol consumption), a pre-existing psychiatric disorder, a previous head injury, lower socioeconomic status, living in congested urban areas and divorce (Levin et al., 1982; Richardson, 1990; Weight, 1998). The frequency of head injury has also been shown to have seasonal and temporal variation. In England, peak frequency is reached in the spring, and while children are more likely to suffer head injuries between the end of school classes and early evening, in adults most head injuries occur between 10pm and 4am (Levin et al., 1982).

2.2.2 PATHOPHYSIOLOGY

Gama, in 1835 (in Evans, 1992, p. 821) wrote:

> "Fibres as delicate as those of which the organ of mind is composed are liable to break as a result of violence to the head."

Neuropathologists of Gama's period concentrated on more obvious focal contusions owing to coup and contrecoup injuries and haematomas (Evans, 1992) and it was only in 1943 that Holbourn (in Barth et al., 1989) postulated the existence of 'shear strain' of axonal and dendritic tissue as a result of what is now known as rotational, acceleration/deceleration head trauma (Barth et al., 1989). In 1956 Strich (in Strich, 1961) described five cases of patients who had suffered uncomplicated head injuries but had remained in a state of extreme dementia, with severe neurological abnormalities, until they died. Strich (1961) described a further 15 cases with the same condition. Her findings were that following a closed and apparently uncomplicated head injury there may be diffuse severe degeneration of the white matter of the brain resulting in permanent incapacitation and dementia (Strich, 1961). Evidence showed that the extensive white matter lesions were as a result of secondary degeneration of nerve-fibres stretched and torn by the shear stresses and strains present during rotational acceleration of the head during the accident (Strich, 1961).

The view that damage was caused by diffuse axonal injury was challenged with some believing that damage to the white matter is more often as a result of hypoxia, oedema or secondary brainstem damage resulting from an intracranial expanding lesion (Graham et al., 1987). However, experiments on non-human primates subjected to non-impact controlled angular acceleration of the head supported Strich's views (Graham et al., 1987).

Lezak (1995) reports that brain damage typically occurs in two stages: the primary injury, where damage is caused at the time of the impact; and the second injury, where damage is caused as a result of the physiological processes begun by the primary injury (Graham et al., 1987). Lesions may occur on the brain at the point of impact (coup) and where the brain subsequently rebounds off the opposite side of the skull's bony protuberances (contrecoup) which account for localisable behavioural changes that accompany closed head injuries (Lezak, 1995; Walsh, 1987). Even in mild head injuries, acceleration or deceleration results in rapid acceleration/deceleration expanding and contracting wave form movements of the brain matter, usually accompanied by the fast rotational propulsion of the brain within the skull (Lezak, 1995). The swirling movements of the brain, and the resulting rotational and linear stresses, can stretch delicate nerve fibres, causing some to shear and resulting in microscopic lesions throughout the brain (Gentilini et al., 1985; Lezak, 1995; Lishman, 1987). This can result in widespread interruption and degeneration of nerve fibres, with breakdown and re-absorption of myelin and the formation of retraction balls (Lishman, 1987), with changes mainly confined to the central white matter of the hemispheres, the corpus callosum, and the long

tracts of the brain stem (Lezak, 1995; Lishman, 1987). Lezak (1995) refers to the resultant damage to axons in the cerebral and brain stem white matter as Diffuse Axonal Injury (DAI). Tiny haemorrhages from ruptured blood vessels scattered throughout the cerebral white matter and lower structures accompanies the neuronal damage (Boström & Helander, 1986 in Lezak, 1995).

> "The tremendous clinical significance of these microscopic lesions is easily understood if one realizes that myriad microscopic shearing injuries occur simultaneously within a rapidly rotating brain, resulting in myriad axonal and neuronal disruptions within the deep white matter of both cerebral hemispheres, which in essence disconnect the cortex from subcortical structures in widespread regions of the brain."
>
> (Pang, 1989, in Lezak, 1995, p. 178).

Lezak (1995) sees the combination of translatory force and rotational acceleration of the brain within the skull as a prominent mechanism of injury in mild head trauma. This sort of injury can occur without any direct impact on the head, solely through exposure to acceleration/deceleration forces which cause rapid flexion-extension movement of the neck, such as in whiplash injuries (Anderson, 1996; Lezak, 1995) although Lezak (1995) points out that this is viewed with some skepticism by some clinicians.

Diffuse cerebral damage, arising at the moment of impact, is regarded as the primary mechanism of brain damage in closed head injuries and the severity of this damage is a more important prognostic indicator than the presence of focal lesions (Adams, Mitchell, Graham & Doyle, 1977; Levin et al., 1982). Secondary damage generally happens as a result of haemorrhages and their sequelae such as cerebral haematoma, cerebral oedema or cerebral anoxia (Levin et al., 1982; Lezak, 1995; Lishman, 1987). Bleeding in the brain may create a haematoma which exerts pressure on the surrounding structures. As the skull does not give way there is therefore no outlet for any swelling. This intracranial pressure (ICP) produces swelling as a result of oedema which compounds any damage which has already occurred (Lezak, 1995). The excess blood flow in the brain also tends to cut off blood flow, and thus oxygen, to damaged areas of the brain at a time when these areas need it most (Lezak,1995).

When the head is at rest at the time of injury, lesions will be maximal at the site of impact. When the head is in motion at the time of injury the contrecoup effect is likely to be more pronounced (Lishman, 1987). Contrecoup effects are often particularly marked in the temporal and orbital regions and the resulting lesions initially lead to a loss of neurons locally, and ultimately to areas of subcortical demyelination (Lishman, 1987).

2.2.3. SECTION SUMMARY

In summary, a distinction can be made between open and closed head wounds, with closed head wounds ranging in severity from mild to moderate to severe. A broad definition of a mild head injury is one where loss of consciousness is less than 30 minutes, posttraumatic amnesia is of relatively short duration, and there is no structural pathology of the skull. Mild head injuries, most commonly caused by acceleration/deceleration forces, may account for as much as 90% of all brain injuries. The result of a mild head injury is generally diffuse type cerebral damage.

For this research the terms 'mild head injury', 'postconcussive symptoms', and 'postconcussive syndrome' will be used. The term 'concussion', as a synonym for head injury, will only be used when reference is made to authors who use this term and not 'mild head injury'.

2.3. MILD HEAD INJURY : SEQUELAE AND RECOVERY

This section will describe the research that has been done into the sequelae of mild head injuries and the process of recovery from these sequelae. The sequelae of mild head injuries may be divided into two categories: (i) neurocognitive or neuropsychological deficit and (ii) postconcussive symptoms (PCS). There is some confusion in the literature as to whether the term 'postconcussive symptoms' includes both the neurocognitive deficits and the postconcussive symptoms that may follow a mild head injury or merely refers to the self-reported or subjective symptoms, separate from the objective, measurable cognitive deficits. Bohnen, Jolles and Twijnstra (1992), Erlanger, Kutner, Barth and Barnes (1999), Kibby and Long (1996) and King (1997) all differentiate between cognitive (or neuropsychological deficits) *and* postconcussive symptoms. King (1997) states that neuropsychological deficits are measured objectively using psychometric testing whilst Bohnen & Jolles (1992) state that postconcussive symptoms are based on a patient's self-reports. Kibby and Long (1996) report that research into the sequelae of mild head injury must separate the cognitive deficit from postconcussive symptoms because PCS can occur in the absence of cognitive deficit.

For the purposes of this research, therefore, differentiation will be made between *neuropsychological deficits*, i.e. cognitive impairment determined by objective measurement following a mild head injury, and *postconcussive symptoms*, i.e. subjective or self-reported symptoms present after a mild head injury. While postconcussive symptoms may include difficulty with memory and concentration, these are self-reported or subjective complaints, as opposed to the objectively measured neuropsychological deficits in memory and concentration. The neuropsychological deficits and postconcussive symptoms which may follow a mild head injury will be discussed separately below.

2.3.1. NEUROPSYCHOLOGICAL DEFICITS

Although severe head trauma often produces explicit cognitive and behavioural deficits, mild head injury is frequently overlooked as a condition that may cause neurological dysfunction requiring intensive assessment and treatment (Barth et al., 1983). However, Sir Charles Symonds (in Rimel et al., 1981) states that:

> "It is questionable whether the effects of concussion, however slight, are ever completely reversible". (in Rimel et al., 1981, p. 227)

Similarly, Oppenheimer (1968) reports that:

> "...permanent damage, in the form of microscopic destructive foci, can be inflicted on the brain by what are regarded as trivial head injuries."
> (Oppenheimer, 1968, p. 306)

It is to be expected that axonal degeneration would have a disruptive effect on cortical arousal and therefore on cognitive performances (Gentilini et al., 1985; Lezak, 1995). Thus it is not uncommon that cognitive impairment may follow a head injury, even where the injury has been minor and does not require assessment or management (Bohnen & Jolles, 1992). The subtle after-effects which may accompany mild head injuries can only be determined by careful neuropsychological assessment (Segalowitz & Lawson, 1995). The importance of recognising these after-effects, and understanding that they may be even more long-lasting than current clinical capacity is able to identify, must be emphasised (Boll, 1985). Boll (1985) states that:

> "As with justice, treatment delayed may well, effectively, turn out to be treatment denied." (Boll, 1985, p. 483)

Mild head injury is a multifactorial disorder and the sequelae following damage caused by such an injury will vary from person to person depending on age, education level, premorbid neuropsychological integrity, injury characteristics and psychological reaction to the injury (Barth et al., 1983). In the first few days following a minor head trauma, subacute disturbance in attention, memory and information-processing efficiency is common (Barth et al., 1983; Erlanger et al., 1999; Evans, 1992; Levin et al., 1987; Rimel et al., 1981; Szymanski & Linn, 1992). Deficits in reasoning and visuospatial processing are also reported (Erlanger et al., 1999). Bohnen and Jolles (1992) report that whereas they found no evidence of gross deficits in intelligence or memory, subtle deficits were found which appear to selectively impair functions of attention and information processing. However, not all patients demonstrate significant problems in all these areas (Szymanski & Linn, 1992). Levin et al. (1987) found that although most patients exhibit cognitive recovery by one to three months, a residue of isolated neurobehavioural defects may occasionally persist for a longer duration. In contrast, Rimel et al. (1981) found that a large number of patients with minor head injuries (34%) were experiencing difficulty with their lives three months after injury.

Thus neuropsychological deficits in (1) memory, (2) information processing, (3) attention, and (4) vigilance and reaction time have all been reported following mild head injury. These neuropsychological deficits will be discussed in these categories in more detail below.

Memory

Luria (in Ruff et al., 1989) states that memory is commonly considered to encompass a number of separate but interacting systems that allow specific facts or information to become conscious through recall or recognition. These memory systems are often described using terms such as long-term and short-term, episodic and semantic, working or reference, but despite the bipolar terminology there is overlap between the memory systems as these distinctions are based more on experimental paradigms than on any generally accepted theory (Ruff et al., 1989). Historically, loss of memory has been explained by the 'first in, last out' hypothesis such as is found in patients with Korsakoff's syndrome who demonstrate the greater susceptibility of more recent recollections to loss (Ruff et al., 1989). However, conflicting findings have been reported in closed head injury patients (Levin et al., 1985 in Ruff et al., 1989; Warrington and Sanders, 1971, in Ruff et al., 1989). Differences between head injury patients and other clinical populations can partly be explained by the difference in onset of the memory problems - in Korsakoff's syndrome, for example, there is a gradual onset of memory problems which differs from the immediate onset of memory problems in young head injured patients who are presumed to have normal long-term memory prior to injury (Ruff et al., 1989).

There is strong evidence of specific neuropsychological deficits in memory and attention within the early stages after a mild head injury (Gasquoine, 1997). The first systematic follow-up study of memory function in patients with a minor closed head injury was done by Conkey (in Richardson, 1990) in 1938. The results of this study indicated that patients had a persistent and pronounced decrement in performance on tests of learning and remembering, associated with a specific deficit in the acquisition of new memories (Richardson, 1990). The results of the more recent three centre study conducted by Ruff et al. (1989) found that generally, patients who had sustained a single uncomplicated mild head injury, showed compromised memory functioning when tested within one week of the injury. The patients' memory for visual and verbal information was significantly below that of the controls' but, within a period of one month following the injuries, this situation improved until they were no longer significantly lower than the control group (Ruff et al., 1989). While Levin et al. (1987) admitted that the improvement in neurobehavioural performance could be as a result of practice effect, this would assume a preserved capacity for retaining detailed information over the first month after injury. However, their baseline data indicated a subacute long-term memory deficit in the patients, a finding that is incompatible with a view of potent retention and positive transfer to the one-month follow-up examination (Levin et al., 1987).

Barth et al. (1983) hypothesise that the memory deficits are secondary to problems with information processing. Memory impairment results in a need to process greater amounts of information and attentional impairment results in more forgetfulness as less information is encoded and subsequently retained (King, 1997).

Information Processing

Information processing capacity can broadly be described as the number of operations the brain can carry out at the same time (Gronwall, 1989). This capacity increases as we develop, with an adult able to perform more operations at one time than a child. If the demands on processing space become too much the person will have to switch processing space to deal with each task (Gronwall, 1989). Most studies on the cognitive deficits of mild head injuries have used rather global measures of neuropsychological functioning, but these indices are not sensitive to subtle changes in information processing capacity (Barth et al., 1983). Gronwall and Wrightson (1975) assessed patients using the Paced Auditory Serial Addition Task (PASAT), hypothesized to be sensitive to an individual's rate of information processing. For Gronwall and her colleagues (in Parasuraman, Mutter & Malloy, 1991) the PASAT also reflects aspects of attention such as concentration and sustained attention. The patients chosen for the research had been concussed, had a PTA of less than 24 hours, had no skull fracture nor any evidence of intracranial haematoma, cerebral contusion or other complication, had no history of a previous head injury or psychiatric illness requiring treatment, and were not taking any sedative medication (Gronwall &Wrightson, 1975). Patients were able to process a limited amount of information as quickly as the controls but as the number of items increased, their performance deteriorated. Their results suggested that minor head trauma significantly reduces the capacity to process information rapidly and that successive injuries produced deficits in information storage and retrieval capacity (Gronwall &Wrightson, 1975). This deficit in information processing ability can also be found elsewhere in the literature (for example, Leininger, Gramling, Farrell, Kreutzer and Peck, 1990; Levin et al., 1987; Rimel et al., 1981).

Several researchers have suggested that the deficit in information processing speed may be the underlying factor involved in the decline of cognitive functioning following a mild head injury. Thus the use of complex tasks requiring the integration of multiple systems should be most reliably sensitive to any deficit (Hinton-Bayre et al., 1997). According to Gronwall (1989), patients who have suffered a mild head injury will have difficulty in all tasks requiring them to simultaneously analyse more items of information than they can handle. Patients may present as 'slow' because of the extra time required to process chunks of information, or they may appear to be distractible as they are unable to focus on irrelevant stimuli while attending to the relevant stimulus (Gronwall, 1989). Gronwall (1989) also found that these patients, when given more information than their capacities can cope with, present as inattentive, or they sometimes present as forgetful,

because when they are focussed on point A they do not have the processing capacity to simultaneously think about point B. These are all aspects of attention and the relationship between attention and information processing has been well documented elsewhere (Gronwall & Wrightson, 1974; Levin et al., 1987).

Attention

It is generally accepted that 'attention' is not a unitary aspect of cognition but is instead made up of a variety of interacting processes: selective/divided attention involves selecting a stimulus source in the presence of competing information; sustained attention/vigilance involves the ability to maintain attention for infrequent critical events over a sustained period of time; attentional capacity allocation involves the ability to vary the amount of attention paid to a stimulus in response to information processing requirements (Parasuraman et al., 1991).

Controlled studies of neuropsychological functioning following mild head injury have consistently demonstrated that impairment often exists on tasks requiring divided and/or sustained attention, tasks which are sensitive to the brain's speed of information processing (King, 1997). Everyday life is full of tasks which require divided attention, sustained attention and intact speed of processing and it is in these areas that patients with mild head injury have the most difficulty while recovering (King, 1997). The findings of Binder, Rohling and Larrabee (1997) suggest that measures of attention may be the most sensitive indicators of dysfunction associated with mild head trauma.

Vigilance and Reaction Time

An area of attention which has not been widely researched is vigilance and reaction time. Vigilance tasks require the ability to focus on an uninteresting task for an extended period of time (Gronwall, 1989) and are designed to assess a decline in performance efficiency over time (Parasuraman et al., 1991). With a normal performance there is a high level of accuracy in detecting critical targets at the beginning of the test but thereafter there is a decline in the detection rate over time, known as the vigilance decrement (Parasuraman et al., 1991). Buchtel (in Parasuraman et al., 1991) has suggested that vigilance tasks might be ideal in determining attentional deficits in patients who have suffered even minor trauma, but evidence on this has been mixed.

Parasuraman et al. (1991) found, one month post injury, vigilance performance under normal task conditions was unimpaired, but that it fell short on tasks requiring sustained effortful processing. Practically, this implies that vigilance decrement is only a problem if performance falls below the level required for a specific job. After having suffered a mild head injury, performance was functionally lower than the controls', especially on tasks requiring substantial amounts of effort, resulting in their falling below the required level

of performance before individuals without head injuries (Parasuraman et al., 1991). MacFlynn, Montgomery, Fenton and Rutherford (1984) found that reaction time was slowed, both immediately and six weeks after a mild head injury, but that it improved between six weeks and six months after the injury. Selective attention and reaction time deficits following mild head injury have been found on tests demanding attention and concentration (Gentilini, Nichelli & Schoenburger, 1987).

2.3.1.1. Recovery From Neuropsychological Deficits

The evidence in the literature concerning the recovery of patients from neuropsychological deficits due to a mild head injury is inconsistent. In a review of literature pertaining to mild head injuries in children and adolescents, Satz et al. (1997) reported that researchers have found more variable outcomes following mild head injuries than the clear pattern of adverse outcomes associated with severe head injury. Binder (1986) reported that although there is clear evidence of cognitive deficit in the first few days after mild injury, there is inconsistent evidence of long-term cognitive impairment following a mild head injury.

It has generally been found that full recovery from neuropsychological deficits which may follow a mild head injury has occurred by about three months post-injury (for example, Alves et al., 1986; Binder, 1986; Dikmen, Temkin & Armsden, 1989; Evans, 1992; Levin et al., 1987). Gentilini et al. (1985) and Levin et al. (1987) both reported impressive neurobehavioural recovery after one month. Scores on the PASAT normalised in one group by five weeks after the trauma (Gronwall & Wrightson, 1974). However, practice effect could have led to an underestimation of deficit (Binder, 1986). MacFlynn et al. (1984) reported that although reaction time of concussed patients was abnormal after six weeks, patients had recovered by six months post-injury. Several weeks after injury, Ruff et al. (1989) and Barth et al. (1983) found striking impairment compared to test norms. However, these were uncontrolled studies and the results need to be confirmed by controlled observations (Binder, 1986).

Levin et al. (1987) reported that, in the majority of cases, when a patient is free of preexisting neuropsychiatric disorder or substance abuse, a single uncomplicated minor head injury produces no permanent disabling neurobehavioural impairment. However, the authors do state that although most patients exhibit cognitive recovery by one to three months, a residue of isolated neurobehavioural defects may occasionally persist for a longer duration. Similarly, Binder, Rohling and Larrabee (1997), in their meta-analytical review of residual deficits present at least three months post-injury, which may follow mild head injury, concluded that a weak association was suggested between mild head trauma and persistent neuropsychological deficits. Binder (1997) states that not only is there little evidence for neurological causation of most persisting complaints, but that there is also little empirical evidence that prolonged neuropsychological deficits typically are caused by mild head trauma. The author does concede, however,

that it is possible that persisting deficit following a mild head injury may occur is a small number of cases. In addition, Binder (1997) states that the persistence of impairment beyond one month after a mild head injury may only be apparent under conditions of stress. Ewing et al. (1981 in Binder, 1986) found deficits in cognitive functioning one to three years after concussion only under conditions of hypoxia. While Gasquoine (1997) agrees that impairment may only be apparent under conditions of stress, he also suggests the possibility that this might only reflect the limitations of current neuropsychological methods of assessment techniques.

While the argument that persisting, measurable cognitive deficits *typically* occur after a mild head injury is unsupportable, there is the possibility of selective vulnerability in patients (Binder, 1986). Recovery over time from the neuropsychological consequences of traumatic head injury is dependent on a number of factors, including the severity of the injury, characteristics of the sample, measures utilised, and the time frame in which the observations are made (McLean et al., 1983). Older patients (over 40 years old) are more likely to have prolonged disability than younger patients (under 30 years old). Socioeconomic status has also been identified as being related to the period of disability, with those of a lower socioeconomic status taking longer to return to work than those of a higher socioeconomic status (Binder, 1986). While the length of posttraumatic amnesia has been shown to be problematic as an predictor of outcome, the use of GCS may show more promise (Binder, 1986). Rimel et al. (1981) found that patients with a GCS of 9-12 had only a 38% chance of making a good recovery after three months while this figure rose to 75% in those with a GCS of 13 or more. In addition, Binder (1986) states that most studies have failed to take into account the effects of previous head injuries, a serious omission given Gronwall and Wrightson's (1975) finding that neuropsychological recovery is slower in patients with a previous concussion. As Shuttleworth-Jordan (1999) points out, mild head injury may cause permanent (albeit subclinical) brain injury, thus becoming a risk factor in itself for future functional impairment.

Gronwall and Wrightson (1975) reported slower neuropsychological recovery in patients with a history of previous concussion than in patients with a single mild head injury. Moreover, Binder (1986) and King (1997) pointed out that these authors did not take into account practice effects in their study which could have resulted in an underestimation of deficits. This finding is extremely significant given that a significant proportion (20 - 30 percent) of the population who sustain a head injury sustain more than one (King, 1997). Given the repetitive nature of mild head injuries suffered by rugby players, this last point has important implications for this research.

2.3.2. POSTCONCUSSIVE SYMPTOMS

2.3.2.1. Postconcussive Symptoms/Syndrome/Disorder

It has been shown that a number of subjective, self-reported symptoms may develop following a mild head injury, commonly referred to as 'postconcussive symptoms'. This section will examine the postconcussive symptoms described in the literature that may follow a mild head injury, and its pathogenesis and the process of recovery from these symptoms. The manner in which the terminology around postconcussive symptoms is delineated will also be discussed by including postconcussive syndrome and the proposed postconcussional disorder.

Postconcussive Symptoms

Although most individuals are reported to recover completely after a head injury there are a minority of patients who continue to report symptoms for an extended period after injury (Macciocchi et al., 1998). Even when a patient appears to have made a good recovery from a clinical point of view, the period following a mild head injury may be marked by a particular set of subjective complaints unique to the patient (Richardson, 1990), with some patients complaining of these symptoms for weeks, months, or years after the accident (Rutherford, Merrett & McDonald, 1977). These postconcussive symptoms include reported difficulties in the three broad areas of (i) cognitive deficits, (ii) physical symptoms and (iii) emotional sequelae (Anderson, 1996) although not all patients with mild head injury demonstrate problems in all of these areas (Macciocchi et al., 1998; Szymanski & Linn, 1992). More specifically, while the criteria for defining a symptom as postconcussive are loose, a series of authors have described many postconcussive symptoms that may follow a mild head injury. Although not all authors describe the identical set of symptoms, taken together the following symptoms have been reported: irritability; fatigue; headaches; difficulty concentrating; dizziness; anxiety; blurred vision; insomnia; slowed information processing; memory problems; depression; tinnitus; decreased libido; intolerance to alcohol; reduced tolerance to light, sound and bustle (for example, Barth et al., 1983; Barth et al., 1987; Binder, 1986; Bohnen & Jolles, 1992; Erlanger, et al., 1999; Evans, 1992; Gasquoine, 1997; Macciocchi et al., 1998; Meyer, 1904; Segalowitz & Lawson, 1995; Szymanski & Linn, 1992). It has been shown that, in some cases, the presence of these postconcussive symptoms can lead to vocational and relationship difficulties (Bohnen & Jolles, 1992).

Rutherford et al. (1977) studied 145 patients admitted to hospital with concussion from minor head injuries over a period of one year. At six weeks after the accident, 49% had no symptoms, 38,9% had between one and six symptoms, and 2,1% had more than six symptoms. 51% of the patients had at least one symptom six weeks after the accident, with the following symptoms being reported: headache (24,8%); anxiety (19,3%); insomnia (15,2%); dizziness (14,5%); irritability (9%); fatigue (9%); loss of concentration (8,3%); loss of memory (8,3%); hearing defect (6,9%); sensitivity to alcohol (6,2%); depression (5,5%); visual defect

(4,8%); anosmia (2,8%); epilepsy (2,1%); diplopia (1,4%); other (11%). After one year 15% of these patients were still symptomatic (Rutherford et al., 1977). Other authors have also found headaches to be the most commonly reported symptom (for example, Lezak, 1995; Lishman,1987; Rimel et al., 1981), occurring in between 30% and 90% of all patients who are symptomatic after a head injury (Evans, 1992). Levin et al. (1987) also reported headaches (71%), fatigability (60%) and dizziness (53%) as the most common subacute symptoms. By the end of three months the frequency of these symptoms had declined (47%, 22% and 22% respectively). In addition, a factor analysis of all the symptoms identified a cognitive-depressive factor which included complaints of depression, impaired recent and remote memory, poor concentration and impaired thinking (Levin et al., 1987). These results were supported by those reported by Dikmen, Temkin and Armsden (1989). McLean et al. (1983) examined 20 patients with mild head injuries at three days and one month. Although some symptoms decreased or remained constant over this time: headaches (65% to 35%); fatigue (70% to 65%); dizziness (45% to 35%); insomnia (31,6% to 30%); blurred vision (20% to 20%); and anxiety (35% to 35%), other symptoms increased during this period: difficulty concentrating (40% to 45%); bothered by noise (25% to 30%); bothered by light (15% to 25%); irritability (30% to 35%); easy loss of temper (5% to 25%); and memory difficulties (35% to 40%) (McLean et al., 1983).

Rutherford (1989) divides symptoms into early and late symptoms (See Table 2-1, p. 18). The early symptoms are what the patient complains of immediately upon regaining full consciousness following a mild head injury. These may also be reported the following morning. The late symptoms are those that the patient complains of a few weeks later (Rutherford, 1989).

Table 2-1 : Rutherford's Early and Late Concussion Symptoms

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

(Rutherford, 1989, p. 218)

Postconcussive Syndrome

Reference is sometimes made in the literature to a 'postconcussive syndrome' as opposed to merely 'postconcussive symptoms'. This subsection will attempt to show how authors differentiate between the two terms. In 1934 Strauss and Savitsky (in Anderson, 1996) coined the term postconcussion syndrome. Gronwall and Wrightson (1974, p. 607) wrote that:

> "If we exclude the patients whose symptoms are due to readily identifiable conditions...we are left with a group whose complaints are remarkably uniform. They cannot concentrate, their memory is poor, they tire easily, and they are irritable. Attempts to work bring on a headache. These will be recognised as the symptoms of post-concussion syndrome..."

Gronwall and Wrightson (1974) suggest that it is when symptoms persist beyond the normal period of recovery that they emerge as the postconcussive syndrome. These authors have identified the presence of postconcussive syndrome as early as a week after injury. Similarly, Richardson (1990) states that while it is normal for a patient to suffer some postconcussional symptoms in the posttraumatic period following a closed head injury, these subside within a matter of days or, at most, weeks. There are, however, a substantial proportion of head-injured patients who continue to complain of postconcussional symptoms beyond this initial posttraumatic period, and it is this condition of persistent postconcussional symptoms which is referred to as 'postconcussional syndrome' (Richardson, 1990). For Jacobson (1995) postconcussional syndrome refers to the emergence and variable persistence of a group of symptoms following a mild head injury. These symptoms can include somatic symptoms (headaches, dizziness etc) accompanied by psychological symptoms both cognitive (poor memory and concentration) and affective (depression, anxiety etc) (Jacobson, 1995). Dacey and Dikmen (1987) agree that while a large proportion of mild head injury patients who complain of postconcussive symptoms recover, there are some patients whose symptoms persist and evolve into postconcussion syndrome. Binder (1986) reports that postconcussive syndrome is a term reserved for persisting subjective symptomatology following a cerebral concussion.

King (1997) argues that by calling it a syndrome treats all postconcussive symptoms as a single entity whereas recent literature considers them a multifactorial grouping of symptoms which can form distinct symptom clusters. Gasquoine (1997) rebuts this, stating that although the reference to a postconcussive syndrome appears to imply a consistent symptom complex, in practice, the persistence of any one self-reported symptom has been deemed significant. Evans (1992) also states that postconcussion syndrome refers to a large number of signs and symptoms that may occur alone or in combination following what is usually seen as a mild head injury. However, the term postconcussive syndrome is rarely clearly defined and different authors include different symptoms under this heading (Lishman, 1987). Lishman (1987) states that while headaches and dizziness are central to most definitions, to this could also be added fatigue, noise

intolerance, irritability, emotional instability, insomnia, memory difficulty, concentration problems or simply 'mental symptoms'.

Postconcussional Disorder

Finally, for completeness it is necessary to report on the most recent DSM-IV classification system (DSM-IV, 1994) which is a major complicating factor from a terminology point of view. The new DSM-IV contains a proposal for 'Postconcussional Disorder' (see Table 2-2, p. 20) as a *possible* future category which still requires further study and refinement. The aim is to provide a common language for researchers and clinicians working in this field in the hope that research will help determine the utility of the proposed category and, by providing tentative thresholds and durations, will refine the criteria sets (DSM-IV, 1994). At present this definition collapses both objectively measured deficits (neuropsychological deficits) *and* subjectively reported symptoms (postconcussive symptoms) into one disorder (see Table 2-2, p. 20). Importantly, however, this is *not* the way the terminology is used by the present researcher (see earlier argument on p. 10). Rather, objectively-measured neuropsychological deficits are treated as separate from subjective self-reported postconcussive symptoms.

Table 2-2 : DSM-IV Research Criteria for Postconcussional Disorder

Research criteria for postconcussional disorder

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

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

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

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

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

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

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

2.3.2.2 Pathogenesis of Postconcussive Symptoms

The presence of postconcussive symptoms is often conceptualised as a psychological disturbance rather than as trauma-induced damage to brain tissue (Barth et al., 1983). While Miller (in Levin et al., 1982) emphasised litigation as the predominant etiological factor, Lishman (in Jacobson, 1995) and Levin et al. (1982) argued that postconcussive symptoms may begin on an organic basis but that they persist on a psychological basis. Binder (1986), however, contends that there is little basis for assuming that the organic cause disappears and that it would appear more reasonable that psychological factors play a contributing role to sequelae begun by organic injury. In some cases the psychological symptoms might completely replace the organic symptoms but it would appear more common that the psychological and organic symptoms coexist (Binder, 1986). Barth et al. (1983) report that while the argument for psychogenic causes of these symptoms has merit, recent histological, neurophysiological and neuropsychological data point to the possibility of a specific neuropathological contribution to these symptoms in the cases of minor head injuries. This view is supported by Dikmen et al. (1989) who state that, contrary to the view that emotional disturbance and/or motivation for compensation are the primary causes of postconcussion symptoms following a minor head injury, there is evidence of neuropathological and neurophysiological alterations after these injuries. Thus postconcussive symptoms which persist over time should not be viewed as only physiological or only psychological (Jacobson, 1995), but rather as a combination of both processes.

2.3.2.3. Recovery From Postconcussive Symptoms

As for the recovery from neuropsychological deficits, the evidence of recovery from the postconcussive symptoms which may arise following a mild head injury is inconsistent and sometimes contradictory. Many authors report that full recovery from postconcussive symptoms usually occurs within three months of a mild head injury (for example, Alves et al., 1986; Binder, 1986; Dikmen et al., 1989; Evans, 1992; Levin et al., 1987). However, Rimel et al. (1981) reported that a large number of patients with minor head injuries (34%) were experiencing difficulty with their lives three months after injury. Szymanski and Linn (1992) report that postconcussive syndrome is not necessarily a short-term phenomena as almost 50 percent of those affected demonstrate symptoms for at least three months. Macciocchi et al. (1998) state that although most individuals are reported to recover completely after a head injury there are a minority of patients who continue to report symptoms for an extended period after injury. Dikmen et al. (1989) found that, at one month post-injury, patients endorsed many postconcussive symptoms which were in excess of what would be anticipated on the basis of the neuropsychological impairments discovered earlier on the same patients. At one month post-injury most patients were limited in both employment and recreational activities, but one year post-injury most patients had resumed activities in these areas (Dikmen et al., 1989).

Ruff et al. (1989) state that, in order to prevent secondary psychological reaction to the trauma, it is vital that within the first month or so following the injury patients are cautioned against participating in activities that may be too complex for them to undertake. The authors suggest that failure in complex activities may result in aggravation of the postconcussive symptoms resulting in confusion, depression and self-doubt.

Recovery over time from the psychosocial consequences of traumatic head injury are dependent on a number of factors, including the severity of the injury, characteristics of the sample, measures utilised, and the time frame in which the observations are made (McLean et al., 1983). King (1997) states that there are four variables which appear to be predictive of patients who will report persistent postconcussive symptoms: (1) older age is linked with poorer recovery; (2) women appear to recover more slowly than men; (3) alcohol and substance abuse are associated with delayed recovery; (4) a history of previous head injury (both mild and severe) appears to increase the likelihood of postconcussive symptoms persisting. As stated earlier, this last finding is significant given that a significant proportion (20 - 30 percent) of the population who sustain a head injury sustain more than one (King, 1997). Again this factor has important implications for the present research, given the repetitive nature of mild head injuries suffered by rugby players.

2.3.3. THE RELATIONSHIP BETWEEN NEUROCOGNITIVE DEFICIT AND POSTCONCUSSIVE SYMPTOMS

Contrasting results have been reported when postconcussion symptoms are compared to neuropsychological test results. The relationship between some neuropsychological impairment and postconcussive symptoms is obvious: poor concentration and fatigue would be expected problems with a reduction in speed of information processing; forgetfulness would be expected with measurable memory impairment (King, 1997). Gronwall and Wrightson (1974) found that the subjective elements were accompanied by objective changes in intellectual function, and that as intellectual function returns to normal so the other symptoms regress. McLean et al. (1983) found that patients had recovered from cognitive deficit after one month although they continued to report more postconcussive symptoms than the controls. For Gasquoine (1997), a striking clinical phenomenon, especially true within the first year after injury, was the under-reporting of symptoms when compared to neuropsychological test results or the ratings of relatives or therapists. However, although Ruff et al. (1989) also reported the lack of correspondence between the patients' subjective complaints and the objective test results, they found that after one month the patients' subjective complaints remained virtually unchanged (and in two of the three centres, had actually increased) despite significant recovery on the neuropsychological tests. Levin et al. (1987) also reported that subjective distress was present despite improvements in cognitive functioning. Their study found that although subjective complaints were frequently present at baseline and at both one- and three-month follow-up examinations, even in patients

whose cognitive functioning had improved relative to their control group, subjective distress was greatly reduced by the third month after injury (Levin et al., 1987).

2.3.4. SECTION SUMMARY

Permanent damage can result from what are considered to be minor head injuries. Objectively measurable neuropsychological deficits and subjective reports of postconcussive symptoms may both be present following a minor head injury. The neuropsychological deficits following a mild head injury vary from person to person but, in the first few days following a minor head injury, subacute disturbances in attention, memory, information processing, reasoning and visuospatial processing have been reported. It would appear that, in the majority of cases, a single mild head injury produces no permanent disabling neuropsychological impairment. However, although most patients exhibit cognitive recovery by one to three months, a residue of isolated neurobehavioural defects may persist for longer in some patients. Recovery is slower and not as complete in patients who have previously suffered a mild head injury.

The most commonly reported postconcussive symptoms are generally headaches, fatigue, dizziness and memory problems. Although most patients recover fully from a minor head injury within about three months, there are some patients who continue to report symptoms for much longer periods of time despite having made a good clinical recovery. A history of previous head injury appears to increase the likelihood of postconcussive symptoms persisting, a significant finding as between 20% and 30% of those who sustain a minor head injury sustain more than one. While it was originally believed that the cause of persistent symptoms was psychological, it now seems generally accepted that persistent symptoms are the result of both physiogenic and psychogenic causes. If the postconcussive symptoms persist beyond the expected recovery period, then the patient is said to have postconcussive syndrome.

Evidence is divided on the relationship between neuropsychological deficits and postconcussive symptoms. Some research has indicated that as intellectual functioning returns, so the self-reported symptoms decrease. However, research has also found a lack of correspondence between neuropsychological deficits and postconcussive symptoms, with some patients continuing to report postconcussive symptoms despite apparent recovery from neuropsychological deficits.

2.4. CUMULATIVE HEAD INJURIES

As stated earlier, although the long-range or delayed effects of minor head injuries are as yet unknown, the inference can be made that a single uncomplicated minor head injury rarely produces immediate evidence on neuropsychological measurement of chronic disability or permanent cognitive impairment (Binder, 1997; Levin et al., 1987; Satz et al., 1997). While this might be true following a single head injury it would appear

that this statement does not hold true in the case of multiple head injuries. This is an important aspect of the study of mild head injury, especially in light of Gronwall and Wrightson's (1975) findings which were mentioned previously (see p. 16) and are discussed below. It is, therefore, necessary that the neuropsychological deficits and postconcussive symptoms which may follow after multiple head injuries are investigated.

2.4.1. NEUROPSYCHOLOGICAL DEFICITS

Improvement in cognitive functioning after a minor head injury does not exclude the presence of microscopic or otherwise subtle brain lesions which may reduce the patient's cerebral reserve in response to later insults (Levin et al., 1987). As mentioned earlier, Oppenheimer states that:

> "...permanent damage, in the form of microscopic destructive foci, can be inflicted on the brain by what are regarded as trivial head injuries. If such injuries are repeated (as they may be, for instance, in an unsuccessful boxer), one would anticipate that a cumulative loss of tissue, and of nervous function, would occur."
> (Oppenheimer, 1968, p. 306)

Wrightson and Gronwall (1980) report that it has been shown that intellectual functioning is impaired for some weeks following a single concussion. With each subsequent injury impairment occurs, and although each time it appears that functioning eventually appears to return to normal, there may be permanent loss of reserve which can later become evident under stress (Wrightson & Gronwall, 1980). Gronwall (1989) also states that the fact that cognitive deficits may be temporary does not mean that mild head injury is reversible, as this would entail regeneration of central nervous system tissue. The cumulative effect of mild head injury is evidence of a residual effect that is only noticeable when a second head injury is imposed upon persistent cognitive 'fragility' (Gronwall, 1989).

Other research has also shown that the disturbing features of mild concussive and sub-concussive head injury are: that the effects are cumulative (Anderson, 1996; Gronwall & Wrightson, 1975; Shuttleworth-Jordan et al., 1993), a second concussion before recovery from an initial concussion could be potentially catastrophic (Hinton-Bayre et al., 1997), and multiple injuries may have long-term irreversible consequences (Macciocchi et al., 1998). One concussion reduces intellectual performance temporarily, while a second reduces it further and for longer - the effects of repeated concussions are cumulative and for each person there is a point beyond which recovery is not complete (Gronwall & Wrightson, 1975). Recent research supporting this has discovered that repeated minor injuries have an additive effect on the cognitive abilities of athletes (Warren & Bailes, 1998).

2.4.2. POSTCONCUSSIVE SYMPTOMS

Dencker (in Szymanski & Linn, 1992) found that at an average of ten years after mild head injury, those patients who still showed postconcussive symptoms were likely to have had previous head injuries and other accidents. It is, therefore, to be expected that those subjects with more than one head injury would be more likely to show symptoms of postconcussive syndrome. It would appear that some symptoms, such as depression, sleep disturbance and social difficulties, are more prevalent after multiple head injuries and it may be possible that some symptoms only manifest themselves after the cumulative effect of several mild head injuries (Segalowitz & Lawson, 1995).

Players of contact sports are particularly at risk for repetitive head injuries during their playing careers. The cumulative effect of multiple mild head injuries is, therefore, an important factor when investigating the effect of mild head injuries in contact sport.

2.5. MILD HEAD INJURIES IN SPORT

This section will describe the research into mild head injuries generally in the sports arena and specifically for the following sports: boxing, soccer, American football, Australian rules football, Rugby League and Rugby Union. Although soccer has historically been considered a non-contact sport, it has recently been re-classified as a contact/collision sport by the American Academy of Pediatrics (Green & Jordan, 1998; Matser, Kessels, Jordan, Lezak & Troost, 1998) and researchers have investigated the effect of head injuries amongst soccer players. The aspects of these sports which are relevant to this research will be discussed and, where appropriate, the differences and similarities between certain of these sports will be mentioned.

The first recorded instances of head injuries occurring in sport happened on the plains of Troy at the funeral games of Patrocles (Homer). The first recorded injury occurred in a horse race due to equipment failure, the second in a boxing match (Gleave, 1986). Minor head injuries occur across a wide range of sporting and recreational activities but until recently these were not treated in a very serious light. However, it is now believed that even minor or trivial head injuries can have long-term, and possibly even permanent, neurocognitive consequences, even without direct impact or loss of consciousness (Anderson, 1996). Neurologists are treating more and more athletes for head injuries ranging from the frequent uncomplicated mild head trauma to the less frequent cerebrovascular compromise, oedema and Dementia Pugilistica (Erlanger et al., 1999). The neuropsychology of sports-related head injuries is, however, still a new and developing field which is characterised by the diagnosis and treatment of the cognitive and emotional sequelae secondary to central nervous system injuries caused by sporting activities (Erlanger et al., 1999). Mild head injury is a challenging area as no two athletes are alike and no two brain injuries identical (Sturmi, Smith & Lombardo, 1990).

Contact sports have an inherent risk of injuries, one of which is the risk of sustaining a head injury (Lehman & Ravich, 1990; Warren & Bailes, 1998). Head injuries are seen as a relatively frequent occurrence in sport (Macciocchi et al., 1998) and occur in a wide range of sports from contact sports, such as boxing and martial arts, where contact with the head is an integral part of the sport, to football, rugby and hockey, where contact with the head is incidental, to non-contact sports usually not associated with head injury, such as basketball, soccer and baseball (Erlanger et al., 1999). Head injuries suffered in sport tend to be as a result of relatively low velocity impact when compared to those occurring in non-sport situations such as a motor vehicle accident. The resulting rotation and shear strain is thus less severe with the majority of sports related concussions falling in the mild range of severity. Episodes of confusion and disorientation are more common than the loss of consciousness that often accompanies more severe head injuries. However, if the blow or impact is not anticipated, acceleration forces can be greatly increased (Erlanger et al., 1999). The majority of head injuries in sport occur when the moving head hits the ground or some other relatively large and relatively stationary object, for example, being tackled or carrying out a tackle at rugby, or a collision of heads at soccer. If the velocity of the head is great, a shock wave is produced that travels through the brain. When the head comes to a sudden halt relative movement of the brain continues with translational and rotational acceleration (Gleave, 1986). The athlete's equipment, baseline neck strength and ability to tense their neck muscles may reduce the potential for serious injury by absorption or dissipation of forces involved in the sport. The force not absorbed is transmitted to the brain and may result in concussive injury (Sturmi et al., 1990). Any athlete who sustains a mild head injury, or any athlete who receives a blow to the head, or a sudden jolt to the body resulting in a sudden acceleration/deceleration force to the head should, therefore, be removed from the game and carefully evaluated (Sturmi et al., 1990; Vegso & Lehman, 1987).

The prevalence of mild head injuries in sport is difficult to determine as most cases go unreported (Anderson, 1996; Ruchinskas et al., 1997). This is often because to report the injury would be seen as a sign of weakness and could result in possible elimination from certain competitions, which could have ramifications for both the prestige and pocket of the player (Barth et al., 1989; Ruchinskas et al., 1997). It is not uncommon for players, parents, coaches and other 'interested parties' to minimise players' symptoms in order for them to continue competing (Sturmi et al., 1990). Watson (1993) also reports that elite athletes have a tendency to tolerate discomfort and continue to exercise under circumstances that would discourage the recreational participant. Wrightson and Gronwall (1980) conducted research into the attitudes of young New Zealand men towards concussion with very interesting results. While the majority were concerned about the unpleasant symptoms at the time or possible consequences still to follow, one group, sometimes quite flippantly, denied any concern. Another group would not admit to having been affected by concussion but upon being questioned admitted to definite symptoms. Those injured playing sport showed greater concern for the late effects than those injured on the road, in falls or in assaults (Wrightson and Gronwall,

1980). Wrightson and Gronwall (1980) hypothesise that this is possibly because the sportsmen know that they will be at risk again. Despite this, a third of football players do not favour any restriction of play after a concussion, a third are seriously concerned about the consequences, while a third remain undecided. Wrightson and Gronwall (1980) argue that authorities and sportsmen need to be persuaded that players should not be allowed to suffer repeated concussion, and that amongst the players themselves the nucleus of a receptive audience is to be found.

Although head injuries are an almost inevitable and unavoidable sequelae of most contact sports, much can and should be done to reduce the serious neurologic complications inherent in these activities (Lehman & Ravich, 1990). Although some attempts have been made to lower the incidence of potentially devastating head injuries in sport primarily through rule and equipment changes (Warren & Bailes, 1998), the major focus in this area has been in researching ways of eliminating significant or severe head trauma, while little has been done with respect to mild head injuries in sport (Barth et al., 1987). At present, therefore, professional contact sports provide an ideal opportunity for measuring cognitive functioning pre- and post-injury (Hinton-Bayre et al., 1997). Professional athletes tend to make good research participants as they are generally not as prone to spontaneous cognitive decline from confounding factors such as age, poor health, substance abuse etc (Ruchinskas et al., 1997). In addition, athletes tend to have above normal neurobehavioural skills, are highly motivated and receive very limited reinforcement or gain for persistent symptoms. As a group athletes also tend to be homogenous in education, achievement, intellectual skills and physical functioning (Macciocchi, Barth, Alves, Rimel & Jane, 1996).

Some authors believe that research into these sports injuries can aid in understanding the mechanisms and effects of mild head injuries which occur in more common situations (Ruchinskas et al., 1997). Specifically, Hinton-Bayre et al. (1997) makes a separation between boxing and other sports. Whereas boxing involves multiple head insults, the *acute effect* of a single concussive blow may be assessed in contact sports such as Australian rules football, Rugby League, Rugby Union, soccer and American football. However, Binder (1997), in terms of *permanent effects*, makes a different observation and does not separate between boxing and American football. The author argues that studies of boxers and American football players have little relevance to most patients with two or three head injuries because these sportsmen may receive thousands of blows to the head. Although Binder doubts the relevance of these types of studies to most patients with two or three head injuries, the number of head injuries received in these sports indicates that this is an important area of study, especially given the paucity of data concerning the long-term effects of multiple mild head injuries. Research into the neurocognitive sequelae and postconcussive symptoms arising from boxing, soccer, American football, Australian rules football, Rugby League and Rugby Union will be discussed below. Although research has primarily focussed on the neurological and neuropsychological consequences

of these sports, where research has investigated the presence of postconcussive symptoms this aspect will be discussed separately.

2.5.1. BOXING

In 1928 Martland wrote "For some time now fight fans and promoters have recognised a peculiar condition occurring among prize fighters which, in ring parlance, they speak of as 'punch drunk'" (p. 1103). This generally seemed to happen to second rate boxers who either took considerable head punishment or were used as training partners and were knocked down several times a day. Martland (1928) identified early symptoms such as unsteady gait or balance and sometimes some mental confusion. In some cases this would be followed by a more distinct gait disturbance, muscular slowness and mental hesitancy (especially verbal hesitancy) progressing to a movement disorder very similar to Parkinson's Disease. There could also be a marked mental deterioration which could result in the boxer being placed in an asylum (Martland, 1928).

The inherent objective of boxing is to disable your opponent (Ryan, 1987), to render your opponent unconscious (Macciocchi et al., 1998), and it remains the only sport in which the goal is to induce a cerebral concussion (Lehman & Ravich, 1990) in order to reduce your opponent to a state of total and complete helplessness (Council on Scientific Affairs, 1983). In order to win when boxing you must either punch your opponent more times than he can hit you, disable him or knock him out. A knockout is achieved when one boxer manages to hit another and render him unconscious or at least unable to function effectively (a technical knockout). This occurs in 1% to 4% of all matches (McCunney & Russo, 1984). As the most effective manner of doing this is to attack the brain by punching the head (Ryan, 1987), the result is that the head is the preferred target and the most injured part of a boxer's body (Ross, Casson, Siegel, & Cole, 1987), with minor head injuries being the most common form of injury in boxing (Wilberger, 1988).

It is important to differentiate between professional and amateur boxers as studies on these two groups have reported differing results. Although the mechanisms of injury (linear acceleration, rotational and deceleration forces, and carotid injuries due to blows) remain the same for both professional and amateur boxers, the latter are afforded more protection by stricter rules (Lampert & Hardman, in Ruchinskas et al., 1997). In addition, amateur boxers, unlike professionals, have fewer and shorter fights, engage in less sparring, and wear protective headgear (Brooks, Kupshik, Wilson, Galbraith, Ward, 1987; Ruchinskas et al., 1997). The minor head injury rate amongst amateur boxers has been set at 5% while for professionals this rises to 6,3% (Wilberger, 1988). Professional boxers can land punches whose force may exceed 100 gravity (Ross et al., 1987). Not only does this produce trauma but further trauma may result if the boxer's head hits the ring mat (Ross et al., 1987). The cumulative effect of multiple blows to the head is an important factor which contributes to the severity of head trauma in boxing (for example, Barth et al., 1989; Butler, Forsythe,

Beverly & Adams, 1993; McCunney & Russo, 1984). It is for these reasons that studies on head injuries have frequently looked to boxers for their subjects, with amateur boxers and young professional boxers, who have had fewer fights, being the main focus of research into mild head injuries (Barth et al., 1989).

Neuropsychological Deficits

The evidence suggests that professional boxers may suffer brain damage as a result of the sport, and the greater the number of fights, the greater the likelihood of damage (Brooks et al., 1987). Ross et al. (1987) assessed 15 former and active professional boxers using a battery of tests which included the Trail Making Test, the Digit Symbol test, the Weschler Memory Scale and the Bender-Gestalt test. The results were then compared with normative data established for the general population. Every boxer had more than one abnormal neuropsychologic test score, with ninety percent of the memory test scores (Weschler Memory Scale) and fifty percent of the nonmemory test scores (Trail Making Test, Digit Symbol test and Bender-Gestalt test) falling within the abnormal range. A significant correlation between poor test performance and both the number of fights and increasing age was also reported. The authors suggest that the development of abnormal neuropsychological test scores might be the earliest and first signs of subtle chronic brain injury (Ross et al., 1987). Casson et al. (1984) studied 18 professional and amateur boxers, some of whom were still actively boxing. The authors found that 87% of their subjects had definite evidence of brain damage, while the other boxers had suggestive evidence of subtle brain injury. All the boxers had abnormal results on at least one of the neuropsychological tests, with subjects performing particularly poorly on the tests of short-term memory. A neurological study by Casson, Sham, Campbell, Tarlau and DiDomenico (1982), which studied 10 professional boxers shortly after being knocked out, showed the presence of cerebral atrophy in half their subjects. Each boxer had been knocked out once only, and thus the authors concluded that the damage was not due to the number of knockouts but rather to multiple sub-concussive blows to the head.

Kaste et al. (1982) studied 14 boxers, eight amateurs and six professionals. Neuropsychological assessments of boxers have found that 86% showed mild impairment on the Trail Making Test, while two of the professional boxers showed more severe neuropsychological difficulties. Although brain damage in the amateur boxers was both less frequent and less advanced than in the professional boxers, it was nonetheless present, supporting the concept of cumulative effects of repeated brain injuries. Although none of the amateur boxers reported subjective symptoms, objective evidence of damage was present, and these boxers are thus still at risk for subsequent symptoms and signs of boxer's encephalopathy. Kaste et al. (1982) further reported that their findings refuted the statement that the subjects' boxing careers had not adversely affected them even though their subjects had achieved more in both education and occupation than either their parents or siblings.

While evidence of brain damage in professional boxers is suggested as discussed above, conflicting results have been reported in studies on amateur boxers. McLatchie et al. (1987) studied 20 active amateur boxers seeking evidence of neurological dysfunction. The test battery was designed on the assumption that boxing may cause the same kind of damage as found after minor head injuries. The authors reported that there was clinical, electroencephalographic and neuropsychological evidence of abnormal brain function. The boxers performed significantly more poorly than controls on the Inglis Word Learning Test and on the copy and immediate recall of the Rey Figure but there were no differences between the boxers and controls on the Weschler Memory Word Learning Test, Digit Span and Story Recall. The authors emphasised that they were unable to conclude that, on the basis of the data, the abnormalities found were as a result of boxing, and that it was possible that any group of young men examined in the same manner would produce the same results (McLatchie et al., 1987). Their results were supported by Heilbronner, Henry and Carson-Brewer (1991), who assessed the cognitive functioning of 23 amateur boxers immediately before and after an amateur boxing event. Boxers demonstrated impairments in verbal and incidental memory compared to their prefight performance. These authors also stated that, like McLatchie et al. (1987), it was not possible to conclude that the abnormalities observed were as a direct result of boxing. Heilbronner et al. (1991) also state that while it is unlikely that a single bout would lead to irreversible and permanent cognitive deficits, this question remained unanswered by their study.

However, the study by Brooks et al. (1987) on 29 amateur boxers and 19 controls matched for age, ethnicity and education had different results. The cognitive tests aimed at assessing verbal and visuospatial memory, attention, information processing and motor function, and intellectual functioning. The results of this neuropsychological examination provided no evidence of significant impairment in the boxers, nor any possible predictors of lower cognitive performance. While the authors identify the possibility that the tests used may have been inappropriate, they remained confident that, based on the assumption that any brain damage found would generally be in information processing, attention and memory, the battery was adequately chosen. While the authors do admit that the subjects may have been inappropriately chosen or the match between subjects and controls may have been inaccurate, they were unable to find any consistent pattern of cognitive deficit in the participants (Brooks et al., 1987). Similar results were reported by Murelius and Haglund (1991) and Haglund and Eriksson (1993) who studied 50 former amateur boxers, 25 soccer players and 25 track and field athletes. Results of standardised neuropsychological tests indicated that none of the boxers were considered to have definite signs of intellectual impairment. The only significant difference was that the 25 high match boxers, i.e. boxers who had fought a large number of bouts, had inferior finger-tapping performance. While this could indicate slight brain dysfunction, it could also be due to peripheral nervous and/or motor functioning rather than central. Butler et al. (1993) also found that amateur boxing showed no evidence of causing neuropsychological dysfunction, either from one bout or

following a series of bouts. The authors assessed 86 amateur boxers, and as controls used 31 water polo players and 47 rugby union players. The tests used were selected to examine those cognitive functions (memory and speed of functioning) which, in previous studies, had proved vulnerable amongst boxers. The boxers were assessed on three occasions: pre-bout, immediately post-bout, and follow-up within two years. The amateur boxers used in this study had a mean age of only 16,7 years. It is, therefore, difficult to extrapolate these results to amateur boxing at a more experienced and elite level.

Postconcussive Symptoms

Very few studies have focussed on the postconcussive symptoms reported by boxers. In 1957 Critchley (in Jordan, 1987) reported that following a bout, a boxer may experience transient nonspecific symptoms such as headache, dizziness, imbalance, irritability, fatigue, poor memory and dysarthria. These usually pass and the boxer returns to his normal state. Jordan (1987), however, states that the true frequency of postconcussive syndrome among boxers following a bout is unknown. In their study on amateur and professional boxers, Kaste et al. (1982) questioned the participants about possible subjective symptoms related to their boxing careers such as clumsiness of speech or movements, loss of memory, changes in personality, or any other subjective symptoms. None of the amateur boxers reported any subjective symptoms despite some objective evidence of damage. However, as the subjects were still quite young (average age: 26 years) even those without current subjective symptoms or neurological deficits were still at risk of subsequent symptoms and signs.

2.5.2. SOCCER

Soccer is the most widely played team sport in the world (Abreau, Templer, Schuyler & Hutchison, 1990) with at least 200 million registered participants (Matser et al., 1998). Recently its popularity has spread from Europe to the United States, thus becoming a major factor on the American sport scene (Abreau et al., 1990). Although soccer is considered safe by the general public, the American Academy of Pediatrics has classified soccer as a contact/collision sport (Green & Jordan, 1998; Matser et al., 1998). Head injury in soccer can occur in a number of ways: one's head hitting the ground, or being struck by an opponent's head, elbow, boot or hand, or from heading the ball (Abreau et al., 1990). Concussions account for about 2% of all soccer injuries (Baroff, 1998), more common in soccer than anticipated, and acute head injuries may have the potential for long-term neuropsychologic changes (Boden, Kirkendall & Garrett, 1998).

Head injuries in soccer generally occur in one of two ways: either (1) through major impact with another object (e.g. foot, head, elbow, ground or goalpost) resulting in an acute injury; or (2) though repetitive minor head injuries caused by heading the ball resulting in chronic injury (Jordan, Green, Galanty, Mandelbaum & Jabour, 1996). The use of the head to propel or direct the ball is relatively unique to soccer (Abreau et

al., 1990; Barnes et al., 1998; Boden et al., 1998; Spear, 1995). A famous English soccer player once remarked:

> "If you catch the ball wrongly, it makes your eyes water and your head ache."
> (Tysvaer & Storli, 1981, p. 164).

This is not surprising as a modern synthetic football weighs about 400g and can travel up to 120km an hour which can create a significant impact when heading the ball. The old leather ball, replaced in the 1960s, weighed considerably more when it was wet and would, therefore, have exerted even greater force upon the head (Spear, 1995). The modern ball is water resistant and does not become heavier in wet and muddy conditions (Jordan et al., 1996). Tysvaer and Storli (1981) have estimated that if a soccer player plays 300 games during his soccer career, he will receive about 2000 blows to the head from heading. While it is often claimed that proper heading avoids all ill effects, five out of ten professionals demonstrating proper heading developed headaches after a 10 to 15 minute demonstration (Matthews, in Abreau et al., 1990). It is suggested that footballers, as a result of repeatedly heading the ball and the clash of heads, are at much greater risk of recurrent minor head injuries than the general population (Spear, 1995). Barnes et al. (1998) report that head injuries account for between 4% and 22% of all soccer injuries and that about 2% to 3% of all soccer injuries are concussions. While the authors report that within a 10-year period the odds are 50% that a male soccer player will sustain a concussion, their findings indicated that most concussions were caused by head-to-head contact, i.e. the act of heading when another player's head is struck rather than, or in addition to, the ball. Only 18% of concussions were as a result of heading the ball itself (Barnes et al., 1998).

Neuropsychological Deficits

Abreau et al. (1990) conducted a neuropsychological assessment of the attention and concentration of soccer players, using 31 soccer players with 31 tennis players as a control group. The Raven Progressive Matrices, Symbol Digit Modalities, Perceptual Speed Test and Paced Auditory Serial Addition Task (PASAT) were administered with no significant differences found between the groups on these tests. However, within the soccer-playing group there was a significant negative correlation between the number of games played and performance on the PASAT. The authors report that while soccer players do not warrant a clean bill of neuropsychological health based on these findings, nor do they lead to the inference that soccer seriously harms the brain. Rather the findings suggest, while not conclusively, that soccer provides minor brain damage or dysfunction (Abreau et al., 1990).

A study of soccer players by Matser et al. (1998) found more definite evidence of impaired performance in memory, planning and visuoperceptual processing compared with control subjects. Performance in these areas was inversely related to the number of concussions incurred and the frequency of heading the ball.

Forwards and defensive players tend to be more vulnerable to cognitive impairment because they are more likely to head the ball and have a higher frequency of soccer-related concussions. The authors concluded that some aspects of cognitive functioning (i.e. memory, planning and visuoperceptual processing) may be adversely affected through playing soccer (Matser et al., 1998). Their findings agreed with those of Tysvaer (1992) and Tysvaer and Løchen (1991) who showed a higher degree of neuropsychological impairment in headers than non-headers. Of the players studied, 81% demonstrated mild to severe deficits in the areas of attention, concentration, memory and judgement. Tysvaer and Løchen (1991) state that this may indicate permanent organic brain damage and hypothesise that it's probably due to repeated traumas from heading the ball. Tysvaer, Storli and Bachen (1989) conducted a neurological and EEG examination of 37 former football players (aged 34 to 64 years) to investigate the incidence of head injuries due to heading the ball. An increased incidence of EEG abnormalities amongst the former players when compared with matched controls was noted and the authors concluded that it was probably as a result of a cumulative effect due to repeated head traumas.

The findings of Jordan et al. (1996) were, however, that there was no association between heading the ball and neurologic symptoms, including MRI-detected abnormalities. The authors state that any evidence of encephalopathy in soccer players related more to acute head injuries received while playing soccer than from repeated heading of the ball. While no relationship between repetitive heading of the ball and brain injury could be shown, the authors did find that soccer players were exposed to a substantial risk of acute head injury. These findings led to speculation that repetitive heading may exacerbate the effects of acute head injury. In addition, the population of this study was much younger than that used by Tysvaer and Storli and it has been suggested that the cumulative effects of heading may not become apparent until a later age (Baroff, 1998). The findings by Jordan et al. (1996) were supported by the results found by Barnes et al. (1998) who reported that concussions from player-to-player contact may have more of an influence on findings of physiologic and psychologic deficiencies than heading the ball.

The neuropsychological study of Witol and Webbe (in Baroff, 1998) looked systematically at heading itself, dividing players into groups according to estimated frequency of heading. Data were analysed in terms of two variables: current heading frequency and a cumulative measure of estimated number of lifetime headers. The authors reported neuropsychological impairments in attention, concentration, cognitive flexibility and general intellectual functioning in players who were frequent headers and had a history of frequent heading (Baroff, 1998). However, several methodological limitations have been identified with this study: the small sample size (n=60) and the use of a very small control group (n=12); the use of tests which are not very sensitive to the effects of brain injury; and the study did not control for a history of acute head injury (Green & Jordan, 1998). Green and Jordan (1998) report that data appears to indicate that heading the ball is a

relatively safe activity with regard to the brain. Where it does lead to problems it would appear to happen to players who have suffered one or more acute concussions.

Postconcussive Symptoms

In the study by Abreau et al. (1990), participants were also asked whether they had ever suffered a headache after practice or a game and whether they ever suffered blurred vision, dizziness or had been knocked out. The authors found that a significantly greater number of soccer than tennis players reported experiencing headaches, blurred vision, dizziness and passing out after a game. However, questions referred to symptoms occurring after the game and it is not known, therefore, whether these symptoms occurred at other times, nor is it known whether these symptoms were long-lasting or permanent.

Jordan et al. (1996) administered a 10-question survey to the soccer players and the controls (track athletes) regarding common head and neck symptoms reported by soccer players, i.e. headache, attention deficit, dizziness, memory deficit, depression, irritability, lack of energy, sleep disturbance, hearing impairment and neck pain. The only significant correlation found was between reported symptoms and the number of prior acute head injuries amongst the soccer players. This was not statistically significant amongst the track athletes. The authors concluded that reported symptoms were related more to acute head injuries received playing soccer than from heading the ball. While this study did raise the possibility that repetitive heading may exacerbate the effects of an acute head injury, the subjects of this study were fairly young (average age: 24,9 years) so the possibility of long-term problems cannot be excluded.

In their study, Barnes et al. (1998) asked specific questions about sequelae following head injury including headaches and dizziness, difficulty with sleep, hearing, or vision, or other symptoms. In addition players were asked whether the symptoms had begun after a head injury. All concussions reported were graded as per the Colorado Medical Society guidelines: grade I, confusion without amnesia (68%); grade II, amnesia without loss of responsiveness (30%); and grade III, complete loss of consciousness (3%). Of these concussions, 65% were as a result of collisions with other players. This study did not include being 'dazed' as a specific head injury in order to avoid overestimating the number of concussions. The authors found that headaches (54%), being dazed (31%) and dizziness (18,1%) were the most common symptoms reported by players after heading the ball, with frontal being the most common location for headaches. Only 6,9% of players reported long-term sequelae such as recurrent headaches or vertigo. Limitations of this study were its retrospective nature and the small sample size, made up of American College soccer players who play far fewer matches than their South American or European colleagues.

2.5.3. AMERICAN FOOTBALL

In 1904 President Theodore Roosevelt threatened to ban American Football because 19 athletes were killed or paralysed playing the game. Between 1931 and 1986 at least 819 deaths were directly attributable to the game, mostly from head injuries. Over the past 20 years, however, there has been a dramatic reduction in the most serious head injuries (Cantu, 1996). Although vigorous body contact has always occurred in this game, the increasing emphasis on speed has resulted in it becoming a collision sport (Reid, Tarkington, Epstein & O'Dea, 1971). Originally, American football was played with no protective clothing, football helmets only being introduced in 1896. Rule changes have been made since 1969 in order to help prevent injuries and it is now mandatory for all student athletes, at both school and college, to wear certified helmets (Meuller & Blyth, 1987). Ironically, the development of a protective helmet-face mask system which protected the head, also allowed it to be used as a battering ram when tackling and blocking thus increasing the risk of cervical spine injuries (Torg et al., 1978) until rule changes were adopted to control the 'head first' techniques (Torg, Vegso & Sennett, 1987). In spite of wearing helmets, which provide both padding and a suspension system, the athlete is not completely protected as the mechanism of most minor head injuries is sudden deceleration combined with rotation of the head (Wilberger, 1988). The mechanism of injury in American football is similar to that of boxing, and severity of injury often appears to be directly related to the number and recency of previous blows to the head or acceleration/deceleration injuries (Barth et al., 1989). Players were at slightly more risk during a game as compared to a practice session and tackling and blocking were the primary activities players were involved in when injured (Barth et al., 1989).

The risk of injury in American football increases with age because at young ages the weight and speed, and therefore the force of impact, is low compared with skeletally mature participants (Cantu, 1995). Approximately 10% of all college football players will sustain a mild head injury over any given season with most football players reporting one or more mild concussions during their careers (Barth et al., 1989). One in five American football players will suffer a concussion annually, and the risk of sustaining a concussion in football is four to six times greater for the player who has sustained a previous concussion (Cantu, 1996). While recognition of a head injury is easy if there is a loss of consciousness, over 90% of all head injuries fall into the mild category - no loss of consciousness, only a transient loss of alertness - and are therefore more difficult to recognise (Cantu, 1996). While the incidence of severe neural trauma is low there may be a much larger, unmeasured number of minor head injuries often referred to as 'dings' (Alves, Rimel & Nelson, 1987). Dave Meggyesy (in Yarnell & Lynch, 1973, p. 196), a professional footballer turned author, defined being dinged as:

> "Getting hit in the head so hard that your memory is affected, although you can still walk around and sometimes even continue playing. You don't feel pain, and the only way other players or the coaches know you've been dinged is when they realize you can't remember the plays."

The discovery that athletes may develop significant long-term neuropsychological problems from repeated minor head injuries has resulted in the initiation of interest in the detection of minor head injury and in rule changes affecting which and when athletes may return to competition after head injury (Warren & Bailes, 1998). Mild head injuries are often characterised by a change in (but not loss of) consciousness, as well as confusion, retrograde amnesia, or immediate memory loss, and yet these players usually continue to play (Barth et al., 1989). All too often players who do not display gross external signs of injury or neurological deficit, or who quickly recover consciousness are rushed back into play without proper evaluation (Warren & Bailes, 1998) and it is estimated that about 70% of American football players who are 'knocked out' return to play the same day (Alves et al., 1987; Gerberich, Priest, Boen, Straub & Maxwell, 1983; Warren & Bailes, 1998). Players are also reluctant to bring attention to minor head injuries for fear they will be removed from the game and miss subsequent competitions (Wilberger, 1993). While cognitive and physical disabilities following more severe head injuries are usually obvious and easy to identify, the long term effects of mild head injuries on players are more difficult to assess (Warren & Bailes, 1998).

Neuropsychological Deficits

In Barth et al.'s (1989) preliminary study of American college football, head injured players completed a neuropsychological test battery 24 hours, 5 days and 10 days post-injury as well as post-season. Their results were compared with two control groups, one consisting of players who sustained a mild orthopaedic injury, and the other made up of male college students. Results indicated that players reporting mild head injuries had deficits in global cognitive functioning and impaired information processing abilities (deficit on the Symbol Digit and PASAT tests). In addition, there was a pattern of rapid but possibly incomplete recovery up to 10 days. These results were similar to those found by Levin et al. (in Barth et al., 1989) and McLean et al. (in Barth et al., 1989). However, this study failed to deal with the long term effects of concussive head injury or to consider the possible effects of unreported sub-concussive head injury. Questions regarding the full extent of recovery and compensation, the short- and long-term effects of multiple head injuries, and factors predisposing a player to the risk of mild head injury remain unanswered (Barth et al., 1989). Despite these shortcomings, it was the first study to emphasise the usefulness of baseline testing of athletes, the most effective way of measuring cognitive change after a suspected concussion (Lovell & Collins, 1998).

In the follow-up to the preliminary study above, Macciocchi et al. (1996) excluded all players with multiple head injuries. The authors found that neuropsychological dysfunction does occur following a single mild head injury but that it is relatively circumscribed. The duration of cognitive dysfunction is brief, with the test performance of head-injured players and controls essentially equivalent by the fifth day post-injury. Impairment was present in both sustained auditory attention (PASAT) and visuomotor speed (Trail Making Test, Digit Symbol Test) but the deficits were primarily evident in the failure of head-injured subjects to

show improved performance over time as was present amongst the controls. While the authors' findings indicate the definite but transitory effects of mild head injury, they cannot be generalised to players who suffer multiple head injuries.

Postconcussive Symptoms

Gerberich et al. (1983) conducted a retrospective study of 103 secondary school football teams using a sample of 3802 players. While this study did define concussion in terms of severity, it did not differentiate between symptoms reported by players suffering mild, moderate or severe concussions. It is, therefore, not possible to determine the symptoms, and the duration of these symptoms, reported by players suffering from mild head injuries only. While it is important to note that the authors reported persistent postconcussive symptoms as long as six to nine months following the end of the football season, it is not clear which symptoms were reported following mild, moderate and severe head injuries.

In a preliminary study, Barth et al. (1989) investigated the presence of the following postconcussive symptoms in the head-injured football players and controls (orthopaedic patients and male college students): headache, memory, nausea, dizziness and weakness. Their findings indicated that, compared to pre-season symptom reporting rates, there was a considerable increase in reported symptoms 24 hours post-injury. These symptoms diminished over time to return to the pre-season rate 10 days post-injury. This pattern was not present for the control subjects, indicating that the sequelae of mild head injury were unique to this form of injury and not a consequence of general trauma or population reporting rates (Barth et al., 1989). In the follow-up to this study, Macciocchi et al. (1996) found that, in comparison with the controls, there was a clinically and statistically significant increase in headaches, dizziness and memory problems amongst head injured players. Although these self-reported symptoms appeared to resolve by 10 days post-injury (somewhat more slowly than the neuropsychological dysfunction), there was a slight increase in self-reported memory problems (6,5%) and dizziness (7,1%).

2.5.4. AUSTRALIAN RULES FOOTBALL

Australian rules football is one of the most popular sports in Australia, with over 15 000 teams registered with the National Australian Football Council in 1991 (Maddocks, Saling & Dicker, 1995). It is played by 18 players and uses a bigger field than Rugby League and Rugby Union. It involves more running, kicking and jumping, with less significant body contact than Rugby League (Gibbs, 1993). It is a contact sport, with frequent injuries. At a professional level it has been found that nearly one quarter of all injuries are to the head and neck region and that 5% of the total injuries are concussive (Maddocks et al., 1995).

Neuropsychological Deficits

Cremona-Meteyard and Geffen (1994) studied persistent visuospatial attention deficits following mild head injury in Australian rules footballers. Their results indicated that the inability to act quickly in response to expected spatial events may be a persistent consequence of mild head injury. Maddocks & Saling (1991) studied concussive injury in Australian rules football players using baseline premorbid data established pre-season and a matched control group. The premorbid assessment included details of age, concussive history and the administration of the Digit Symbol Substitution Test, the PASAT and a Four Choice Reaction Time Test. Tests were selected on the basis of their advantage in brevity of admission, face validity and sensitivity to mild head injury. Players with subsequent concussion, diagnosed by a medical practitioner, were assessed at five days post-injury and members of the control group were assessed on corresponding occasions. Impaired information processing and reduced measures of decision time and reaction time were noted. Neuropsychological deficits were noted after the resolution of neurological/neurobehavioural symptoms (Maddocks & Saling, 1991). However, this study failed to deal with the long term effects of concussive head injury or to consider the possible effects of unreported sub-concussive head injury.

A study by Maddocks et al. (1995) chose to investigate the effectiveness of the Digit Symbol Subtest for determining deficits in information processing speed following a concussion and the time course of recovery. This test was chosen as it is easy to administer and has been shown to be sensitive to the effects of concussion in both American footballers (Barth et al., 1989) and Australian rules footballers (Maddocks & Saling, 1991). While previous research had indicated that performance on this test was likely to be affected in the first few weeks following a concussion, the results of this study indicated normal levels of performance six months or longer post-injury. There were no residual effects from earlier concussions, thus disagreeing with the notion of cumulative effects from repeated concussive injury. However, although Maddocks et al. (1995) showed that, six months post-trauma, Digit Symbol Subtest performance does not differentiate between concussed and non-concussed players, the researchers did not make use of a non-contact sport control group in this study. By merely comparing concussed and non-concussed Australian rules footballers the authors were ignoring any possible long-term or permanent effects that extended exposure to mild head injuries might have had on their subjects.

Postconcussive Symptoms

To the author's knowledge no research has been conducted amongst Australian rules football players into the presence or absence of postconcussive symptoms following mild head injury. While the study by Maddocks and Saling (1991) did not study postconcussive symptomology, the authors do state that when the concussed players were assessed five days post-injury all neurological signs had clearly resolved and that

neuropsychological deficits were noted after the resolution of all neurological/neurobehavioural symptoms (Maddocks & Saling, 1991).

2.5.5. RUGBY LEAGUE

Rugby League is an extremely physical game in which players need to use speed, stamina, strength and agility (Gibbs, 1993; Stephenson, Gissane & Jennings, 1996). It involves a player in 20 to 40 physical 'confrontations' per game and has been likened to being mugged 30 times in 80 minutes (Stephenson et al., 1996). Each team consists of 13 players (6 forwards and 7 backs) who have six tackles or 'downs' in which time to move the ball, either by carrying or kicking it, as far up field as possible. The same players are used as both offensive and defensive players depending on which team has the ball (Gibbs, 1992). On attack, the role of the forwards is to gain ground quickly and keep the opposition on the back foot, while the backs attempt to move the ball wide and exploit open space. On defence, the forwards do the majority of the tackling as they attempt to stop the opposition from gaining ground, so denying them space to exploit (Gissane, Jennings, Cumine, Stephenson & White, 1997). Play continues in this nonstop fashion for two 40-minute halves during which time the aim is to carry the ball over the goal line and score a try (Gibbs, 1992). It is a fast moving contact sport which results in some spectacular injuries (Alexander, Kennedy & Kennedy, 1979). As the players wear minimal protective clothing, injuries from direct trauma are common (Gibbs, 1993; Stephenson, 1996), and while some research indicates that Rugby League has more injuries than both Australian rules football and Rugby Union (Seward, Orchard, Hazard & Collinson, 1993; Stephenson et al., 1996), the similarities in injury profile between Australian rules football, Rugby League and Rugby Union are greater than the differences (Seward et al., 1993).

Injury rates have been shown to be higher at the highest level of the game (Stephenson et al., 1996) with the head and neck region the most frequently injured area of the body (Gibbs, 1993; Seward et al., 1993; Stephenson et al., 1996), particularly among the forwards, and it is estimated that concussion accounts for 8% of all injuries (Seward et al., 1993). Players who have already sustained a concussion are at greater risk of impaired playing performance, further injury and possible catastrophic consequences due to second impact syndrome (Hinton-Bayre et al., 1997). The high rates of injury are undoubtedly due to the high amount of bodily contact in the game. Being tackled has the highest risk of injury, due to being hit with force by the opposition (Gissane et al., 1997; Stephenson, 1996). The tackle is a prominent part of the game and carries inherent dangers, such as whiplash, being knocked over backwards and the clashing of heads. The authors report that 46,3% of injuries were suffered when a player was being tackled and 21,3% of injuries were suffered while tackling. The remaining injuries were suffered during activities such as running and scrummaging (Stephenson et al., 1996).

Forwards are involved in a larger number of collisions than the backs and therefore suffer more injuries (Gissane et al., 1997; Stephenson et al., 1996). Forwards receive more injuries in absolute terms, and when the rate is standardised for the number of players (6 forwards and 7 backs) the difference in injury rate becomes even larger (Stephenson et al., 1996). Forwards perform on average over twice as many tackles per game as backs and had significantly higher injury rates both when they were attacking, i.e. being tackled, and when they were defending, i.e. the tackler (Gissane et al., 1997). Alexander et al. (1979) report that the rate of concussion between forwards and backs is 12:1. In Rugby League, concussion is graded by severity (see Table 2-3, p. 40) which determines when the player is allowed to return to play (Stephenson et al., 1996).

Table 2-3: The Rugby Football League's Classification of Concussions

Severity of Concussions	**Action**
Mild: no loss of consciousness (LOC)	
i. Full memory of the event	Can usually continue playing (after being checked)
ii. Memory deficit of the event	Must cease playing: no training or playing for 48 hours, and only after medical check by the club doctor
Moderate: LOC of up to 2 minutes	Must cease playing: no training or playing for 15 days, and only after medical check by the club doctor
Severe:	
i. LOC of up to 3 minutes	Must cease playing: no training or playing for 22 days, and only after medical check by the club doctor
ii. LOC of over 3 minutes	Must cease playing and be admitted to hospital for observation: no training or playing for 29 days, and only after medical check by the club doctor

(The Rugby Football League, in Stephenson et al., 1996, p. 333)

Neuropsychological Deficits

Hinton-Bayre et al. (1997) studied the sensitivity of several short tests of speed of information processing to the effects of mild head injury in Rugby League. The tests used were the Symbol Digit Modalities Test, the Digit Symbol Substitution Test and the Speed of Comprehension Test. Players of Rugby League were assessed pre-season and within 24-48 hours after a concussion. This study showed that the speed of both information processing and comprehension were impaired in the postacute phase of mild head injury, but that an untimed task of word recognition (Spot-the-Word) was not (Hinton-Bayre et al., 1997). However,

this study failed to deal with the long term effects of concussive head injury or to consider the possible effects of unreported sub-concussive head injury.

Postconcussive Symptoms

To the author's knowledge no research has been conducted amongst Rugby League players into the presence or absence of postconcussive symptoms following mild head injury.

2.5.6. RUGBY UNION

Rugby Football, or Rugby Union, is a contact sport which caters for a wide range of players with varying physical and psychological characteristics by identifying an appropriate position among the forwards or backs. While the numbers of players worldwide have never been accurately determined, estimates include over 200 000 players in England, New Zealand, France and South Africa, 100 000 players in Japan and the USA, 35 000 players in Wales and 14 000 players in Scotland (MacLeod, 1993).

Rugby Union and Rugby League, while very similar, are two separate games which developed from a common rugby origin. The original split was over the payment of players - Rugby Union remained an amateur sport while Rugby League became professional. While they are now both professional sports they have evolved so far apart that they are best regarded as different sports, although some players do switch between the two versions of the game (http://www.uidaho.edu/clubs/womens_rugby/RugbyRoot/rugby/FAQ/faq.html). Rugby League teams consist of 13 players (6 forwards and 7 backs) while Rugby Union teams have 15 players (8 forwards and 7 backs). Another important difference between the two sports occurs after a tackle has been made. In Rugby League, the attacking side retains possession of the ball while the defending side must retire 10 metres further into their own territory until the attacking side put the ball into play again. The attacking side now have 10 metres in which to perform various manoeuvres in order to outwit the opposition's defenders. In Rugby Union, however, both teams try to retrieve the ball in a ruck (if the ball is on the ground) or a maul (if the ball is carried). Both teams try and push the opposition backwards while trying to gain possession of the ball. Players often join a ruck or maul at speed in order to do this. The side that gains possession then attempts to move the ball forward either using their forwards, who try and run through (or over) the opposition, or by using their backs, who are more agile and attempt to run around or past the opposition. Play may continue in this manner for an unlimited number of tackles until one side either scores or there is an infringement of the rules (http://www.personal.u-net.com/~interzone/faq.htm).

The stresses and impacts on the head and neck from tackling, scrumming and collisions between players can result in mild head injuries (Shuttleworth-Jordan et al., 1993) and rugby players, by the very nature of the

game, are therefore at particular risk for multiple head injuries. Research has shown that as the age of the participants and their level of competence increases, so does the incidence of injury. The reason for this increase could be that not only are higher level players likely to be faster, bigger and more competitive than those at lower levels but the greater emphasis placed on winning means players are more 'psyched-up' before a match (Nathan et al., 1983).

Rugby players accept the ordinary risks of injury that occur in the game as a result of collisions between players or falling to the ground. While minimal protective clothing is allowed, all players should use individually fitted mouth-guards, which can help reduce the incidence of, amongst other things, concussion. Laws also permit the use of a 'scrum cap' to help protect the player's scalp or ears (MacLeod, 1993). Some research indicates that Rugby League has more injuries than both Australian Rules Football and Rugby Union (Seward et al., 1993; Stephenson et al., 1996), however, Walker (1985) suggests that while the overall injury pattern in Rugby League may be higher, the level of serious injury is lower than in Rugby Union. Either way, the similarities in injury profile between Australian rules football, Rugby League and Rugby Union are greater than the differences (Seward et al., 1993).

Rugby League and Rugby Union deal with concussion in different ways. As stated earlier, the severity of concussion in Rugby League (see Table 2.2, p. 40) determines when a player may return to play or training (Stephenson, 1996). Rugby Union, however, is guided by Resolution 5.7 of the International Rugby Football Board which states that:

> "A player who has suffered definite concussion should not participate in any match or training session for a period of at least three weeks from the time of injury, and then only subject to being cleared by a proper neurological examination." (MacLeod, 1993, p. 373).

The accepted recommendations are that, should a player receive a second concussion in a single season, the player should avoid all contact sport for three months; a third concussion in the same season and the player should avoid all contact sport for six months (MacLeod, 1993).

Epidemiology

Seward et al. (1993) reports that injuries to the head and neck are most common, with concussion accounting for about 5% of these injuries. A survey of rugby players attending an accident and emergency department in Dublin showed that head injuries accounted for 24% of all injuries (McQuillan, 1992). While this study does not state what percentage of these injuries were concussive injuries, it is also a study of those injured who attended the hospital, therefore not including those injuries considered too minor to require medical attention. A similar study conducted in Stellenbosch, South Africa reported that 20,5% of injuries were to the head and neck, with 10% of these patients either unconscious or suffering from concussion (Roy, 1974).

A second study conducted in Stellenbosch, South Africa over the following two years reported very similar results, with 20,9% of all injuries to the head and neck and 13,8% of these injuries involving concussion (van Heerden, 1976).

Research indicates that the incidence of concussion amongst schoolboys may be higher than among adults (Seward, 1993). Two studies of schoolboy rugby injuries found that concussion accounted for 21,5% (Nathan et al., 1983) and 12% (Roux, Goedeke, Visser, van Zyl & Noakes, 1987) of all injuries. The discrepancy between these two figures is explained by the difference in reporting of concussion figures between schools monitored by correspondence (8,7%) and those monitored by personal contact (18,4%), possibly due to ignorance about the nature of the injury (Roux et al., 1987).

Discrepancies in figures between older and more recent studies could reflect the influence of rule changes which have occurred between these studies, as well as an increasing tendency to under-report concussive injuries as a result of some of these rule changes. There is often a lot of pressure exerted upon the players to continue playing despite suffering from mild concussion (Roy, 1974; Van Heerden, 1976) and it would appear likely that the incidence of concussion in rugby may be 'poorly recorded' (Sturmi et al., 1990). For MacLeod (1993), there is no doubt that the incidence of mild concussion is under-reported because of a degree of collusion between players, coaches and medical attendants. Should a concussion be diagnosed the player is required to refrain from playing rugby for three weeks. As mentioned earlier (see Chapter 1, p. 1), players sometimes risk possible serious consequences by remaining on the field for some time after a concussion in order to avoid being sidelined by this regulation.

In Rugby Union, as for Rugby League, tackling is the manoeuvre most strongly associated with injury, accounting for 49% of injuries suffered (Garraway & MacLeod, 1995). Roy, Nathan et al. and Roux et al. reported similar results, with their findings showing that 49% (Roy, 1974), 47% (Nathan et al., 1983) and 55% (Roux et al., 1987) of all injuries occurred during the tackle. This is followed by the ruck (15%), lineout (12%), scrum (8%), gathering the ball (8%) and the maul (6%) (Garraway & MacLeod, 1995). In this version of the game it is also the forwards (as in Rugby League) who are injured more frequently than the backs - 54,1% versus 45,9% (McQuillan, 1992).

Neuropsychological Deficits

To the author's knowledge there has been no research into the cognitive sequelae of mild head injuries in Rugby Union outside of South Africa, even though MacLeod (1993) reports that Rugby Union is played in over 100 countries around the world. The pioneering research in this field was by Shuttleworth-Jordan et al. (1993) at Rhodes University in South Africa on university rugby players. This led to the studies

conducted as part of phase one of the present research, under the coordination of Professor Shuttleworth-Jordan, on professional rugby players (Ancer, 1999; Dickinson, 1998; Reid, 1998).

First, Shuttleworth-Jordan et al. (1993) investigated (1) pre- and post-season test differences between non-concussed top level university rugby players and matched controls and (2) repeated test differences between those rugby players reporting mild head injury during the season and matched controls assessed pre-season, three days, one month, two months and three months post-injury. By making use of repeated differences between rugby players and non-contact controls and a wider variety of tests, some of the methodological shortcomings of previous research into sports with similar injury profiles were overcome.

The pre- and post-season comparisons of non-concussed rugby players and controls showed a pattern of impairment similar to that associated with diffuse brain damage, namely deficits in working memory, verbal new learning ability, hand-motor dexterity and less capacity for practice effects than the controls. Since any players who reported more than one concussion in the previous three years were excluded from the study it is, therefore, likely that the results are an estimate of permanent deficits in a rugby playing group, either as a result of previous concussions, as a result of unreported concussions during the season they were assessed, or as a combination of both of these possibilities.

Five players (out of 60) did report sustaining a mild head injury during the season and, together with the matched controls, were followed up by repeat testing three days, one month, two months and three months post-injury. Relative to the controls these players showed the presence of significant impairment to attention, verbal new learning, working memory and hand-motor dexterity at three days post-injury. At one month post-injury substantial recovery had occurred, with further recovery indicated at two months. At three months post-injury, however, the concussed group still did not exhibit a practice effect to the same degree as the controls on Digits Backwards, Digit Difference, Digit Supraspan and Finger Tapping, thereby suggesting that recovery was not yet complete. The pattern of deficit found in the concussed players was highly comparable to that found in the non-concussed players with regard to deficit in working memory, verbal new learning ability and hand- motor dexterity. While some of the methodological limitations of previous research were overcome, this study had a small sample of concussed players and, although the tests were chosen on the basis of their sensitivity to the presence of diffuse brain damage, the limited test battery only permitted testing across a few cognitive modalities.

Following this, a research project was initiated on professional rugby players. Ancer (1999), Dickinson (1998) and Reid (1998) assessed 26 professional rugby players and a control group of 21 professional cricket players. A comprehensive test battery comprising five modalities (Verbal Memory, Visual Memory, Verbal

Fluency, Visuoperceptual Tracking and Hand-Motor Dexterity) was administered and participants completed a self-report postconcussive symptomology questionnaire. Results of Dickinson's (1998) research indicated the presence of impairment in the areas of speed of information processing, reduced mental flexibility, attention and concentration, sustained attention, verbal and/or visual memory and new learning. These studies also indicated that the performance of the rugby forwards, as opposed to the rugby backs, was disproportionately poor on tests sensitive to mild head injury (specifically the SAWAIS Digit Symbol Substitution Subtest, the Trail Making Test, the Digits Forwards, and the Digit Symbol Incidental Recall). Methodological limitations of these studies were the fairly small number of participants, the assessment of the rugby players pre-season and the cricket players only post-season, and the high number of head injuries reported among the cricket players.

Postconcussive Symptoms

The study by Shuttleworth-Jordan et al. (1993) also investigated the presence of postconcussive symptoms in top level university rugby players who had received a concussion. Three days post-concussion players reported the following symptoms (in decreasing order of frequency): headaches (mild to severe), nausea, visual disturbance, poor attention and concentration, anxiety, insomnia, severe fatigue, vomiting, weakness of limbs, loss of appetite, sensitivity to noise, restlessness, clumsiness and speech problems. There had been a marked reduction in symptoms one month post-concussion with only the following symptoms present: mild headaches, mild fatigue, mild problems with attention and concentration, and blurred vision. By two months post-concussion there had been a further slight reduction in symptoms: occasional mild headaches, fatigue, problems with attention and concentration, and restlessness. At three months post-concussion a marked reduction had taken place with no symptoms being reported which had not been part of the subject's premorbid presentation. For the first two months post-injury the postconcussive symptoms followed a similar pattern to the cognitive deficits measured: severe symptoms three days post-injury, significant recovery at the one month interval and the continued presence of some symptoms at two months post-injury. However, at three months post-concussion the presence of cognitive deficits indicated that recovery was not necessarily complete despite the absence of any reported postconcussive symptoms.

A second aspect of Dickinson's (1998) study investigated the percentage of rugby and cricket players with postconcussive symptoms. During the assessment mentioned above, the 26 professional rugby players and 21 professional cricket players completed a self-report questionnaire listing 31 possible post-concussive symptoms which the participants were asked to rate whether they 'never', 'sometimes', or 'often' suffered from these symptoms. Significant positional variation was found with the forwards reporting higher proportion of deficit than the backs. The most significant postconcussive symptoms present for the rugby players were in the areas of anxiety, depression, irritability and lowered frustration tolerance

(argumentativeness). The results of this aspect of the study indicated that while the presence of postconcussive symptoms amongst the rugby players was corroborated by the cognitive deficit measured, amongst the cricket players, some symptoms were reported which were not supported by the cognitive deficit measured (specifically headaches and eyesight). It was assumed that this was due to the cricket players being assessed post-season (the rugby players were assessed pre-season) after a long, tiring and unsuccessful overseas tour and that they were thus less motivated and suffering from fatigue and depression.

2.5.7 SECTION SUMMARY

Head injuries, due to the very nature of the game, are a frequent occurrence in contact sports. Thus research into these injuries can aid, to a limited degree, our understanding of mild head injuries which occur in more common situations, and more specifically, our understanding of the cumulative effects of multiple head injuries. Head injuries have been shown to occur to varying degrees in sports such as boxing, soccer, American football, Australian rules football, Rugby League and Rugby Union. It is hypothesised that head injuries in sport occur through acceleration/ deceleration injuries to the head, not necessarily followed by loss of consciousness, or through whiplash-type injuries as a result of manoeuvres such as tackling.

Research into the neuropsychological consequences of professional boxing supports the presence of cognitive deficit and the concept of cumulative damage following multiple mild head injuries. Results of research into amateur boxing have been mixed. In soccer, it is hypothesised that mild head injuries, such as caused by heading the ball or by collisions between players, can lead to cognitive deficit amongst players. While American football, Australian rules football, Rugby League and Rugby Union are different sports, they have been shown to have similar injury profiles, yet findings across these sports have been inconsistent and variable. In American football, while some research indicated the presence of cognitive impairment at three months post-injury in the areas of memory and visuospatial skills, other research found no impairment in general neuropsychological functioning but did find some evidence of a specific deficit in selective attention. Research into Australian rules footballers found permanent visuospatial attention deficits. In addition there are indications of impairment in information processing speed, decision time and reaction time in the acute stages following a mild head injury. Further research indicated no residual effects from earlier concussions on the basis of Digit Symbol Subtest performance. Rugby League researchers found impairment in speed of information processing and comprehension in the post-acute phase of mild head injury. Research into the effect of mild head injuries in Rugby Union found permanent deficits in rugby players similar to that associated with diffuse brain damage. In addition, while there was significant recovery of functioning in players who received a mild head injury during the season, there was evidence that suggested that at three months post-injury recovery was not yet complete. The most recent research in this area produced evidence of impairment in visuoperceptual tracking, speed of information processing and attention, and suggested a

tendency towards visual and/or verbal memory impairment, which was supported by the presence of postconcussive symptoms. Rugby forwards also showed greater levels of impairment than the rugby backs.

Less research has focussed on postconcussive symptomatology than on neurocognitive deficits. Following a bout, boxers report transient nonspecific symptoms such as: headache, dizziness, imbalance, fatigue, poor memory and dysarthria. While professional boxers report postconcussive symptoms related to their boxing careers, amateur boxers do not. Evidence suggests that they are still at risk for developing such symptoms as they grow older. Symptoms reported by soccer players appear related to the number of prior head injuries received. There is conflicting evidence as to the main cause of these injuries. While some researchers believe it is due to repeated heading of the ball, other researchers report that it is mainly as a result of collisions between players. Headaches and dizziness appear to be the most common symptoms reported amongst soccer players. Some research into American football found that postconcussive symptoms were present as long as six to nine months following the end of the season. Other researchers report that while most symptoms return to pre-season levels within about 10 days of the injury, there is a slight increase in the incidence of self-reported memory problems and dizziness. In Rugby Union, evidence suggests that symptoms present at three days post-injury take about three months to resolve. For the first two months following the injury, the resolution of postconcussive symptoms follows a similar pattern to the improvement in cognitive deficit. However, at three months players were symptom free despite the presence of measurable cognitive deficit. Further research indicated that the most significant postconcussive symptoms were in the areas of anxiety, depression, irritability and lowered frustration tolerance (argumentativeness). Amongst the rugby players the postconcussive symptoms provided cross-validation for the cognitive deficits which were apparent.

2.6. RATIONALE FOR THE PRESENT RESEARCH STUDY

This project forms part of ongoing research by Rhodes University, the South African Rugby Football Union (SARFU) and the Sports Science Institute, begun in 1996 to study the effect of head injuries on professional sportsmen (Ancer, 1999; Dickinson, 1998; Reid, 1998). The first phase of the research involved the assessment of the National Rugby squad, the Springboks (n=26) and the National Cricket squad, the Proteas (n=21). These data were analysed in three ways:

1. A direct comparison of mean scores and standard deviations of rugby and cricket players (Ancer, 1999);
2. A direct comparison of mean scores and standard deviations of both rugby and cricket players to existing university norms (Reid, 1998);
3. A comparison of the percentage of rugby and cricket players with deficit relative to the norms for each test and a comparison of the percentage of rugby and cricket players with postconcussive symptoms (Dickinson, 1998).

Results of the first phase of the research support the presence of significant deficit on those tests known to be sensitive to mild head injuries and corroboration was obtained by the presence of postconcussive symptoms. The methodological limitations of these studies were the fairly small number of participants, the assessment of the rugby players pre-season and the cricket players only post-season, and the high number of head injuries reported among the cricket players. The cricket players were assessed at the end of an unusually long cricket season which had culminated in an unsuccessful tour of England and appeared, as a result, to be suffering from fatigue, a lack of motivation and depression. Some of the cricketers had also played rugby as their winter sport and were, therefore, as at risk for similar mild head injuries as the rugby group (Ancer, 1999; Dickinson, 1998; Reid, 1998). As a result they were not an ideal control group to use for this research.

In the current phase of the research it was decided to include the National Under 21 Rugby squad (n=21) and the National Hockey squad (n=21) to increase the number of contact sport participants and to provide a better non-contact sport control group which offers fewer confounding variables. Hockey is generally considered to be a non-contact sport and as both rugby and hockey are winter sports very few top class hockey players played both these sports. Those hockey players who did play rugby usually gave it up at an early age.

As with the first phase of this study it was decided to analyse this data in three different ways:

1. A direct comparison of mean scores and standard deviation of all rugby players and controls. Additional sub-group comparisons between forwards and backline players, Springbok and Under 21 rugby players;
2. A comparison of mean scores and standard deviations of rugby players and existing university norms with additional sub-group comparisons as noted above;
3. A comparison of the percentage of rugby players with cognitive deficit on each neuropsychological test relative to established normative data with additional sub-group comparisons as noted above, and a comparison of the percentage of individuals with post-concussive symptoms in the rugby players with the non-contact sport controls.

This research focussed on the third level of analysis to add to the limited but growing body of literature dealing with mild head injuries and their sequelae. It was decided to replicate Dickinson's (1998) basic methodology which compares the percentage of participants who are impaired between groups. It was considered that this methodology is effective as it provides more information than merely comparing group means by investigating the proportion of players who influence the mean. In order to improve on the methodological limitations of this research as mentioned above, the present research made use of a less

confounded control group (hockey players instead of cricket players) and made comparisons using a larger sample size for contact sport players.

The following hypotheses were posed:

1. Since rugby players, compared with hockey players, are exposed to more mild head injuries due to the nature of the game, either as a result of blunt trauma to the head (e.g. from knees or elbows, or from contact with the ground during play) or as a result of whiplash-like injuries (e.g. acceleration/deceleration resulting from tackling), it is expected that rugby players, relative to hockey players, will show higher proportions of deficit on tests sensitive to diffuse brain damage and will report higher proportions of postconcussive symptoms.
2. Since rugby forwards, compared with rugby backs, are exposed to more collisions and impacts which can result in mild head injuries due to the nature of their role in the game, it is expected that the forwards at both Springbok and Under 21 level will, relative to the hockey players and the backs of both rugby groups, show higher proportions of deficit on tests sensitive to diffuse brain damage and will report higher proportions of postconcussive symptoms.
3. Since the Springbok rugby players, compared with the Under 21 rugby players, have had more exposure to collisions and impacts which can cause mild head injury and are also generally heavier, faster, and stronger such that their collisions involve greater forces, it is expected that the Springbok rugby players, relative to the Under 21 rugby players, will show higher proportions of deficit on tests sensitive to diffuse brain damage and will report higher proportions of postconcussive symptoms.

CHAPTER 3 : METHODOLOGY

This study forms part of ongoing research into the effects of mild concussive and sub-concussive head injuries being conducted by Rhodes University, the South African Rugby Football Union (SARFU) and the South African Sports Science Institute. The research has been conducted in two phases: phase one involved a comparison of Springbok rugby players with Proteas cricket players (cricket's equivalent of the Springboks); phase two, an extension of this research, has expanded the sample of rugby players by including players from the National Under 21 rugby squad, and comparing their results with Springbok hockey players, the non-contact sport control group.

3.1. PARTICIPANTS

The subjects for this study consisted of senior (open age group) and junior (under 21 age group) rugby players and hockey players who had been selected for their respective national squads. The senior national rugby squad (26 players) is referred to here as 'Springbok rugby', and the junior national rugby squad (19 players) is referred to here as 'Under 21 rugby'. The hockey players (referred to here as hockey) were all those players selected as members of the national hockey squad who were available to participate in the research during the assessment period (21 players).

Although the national cricket team (the Proteas) was also assessed for the first phase of this research, the results of the previous research indicated that they were not an ideal control group as they were assessed post-season and were suffering from fatigue, a lack of motivation and depression. Some of the Proteas had also played rugby as their winter sport and were, therefore, at risk for similar mild head injuries as the rugby group (Ancer, 1999; Dickinson, 1998; Reid, 1998). The assessments of the Proteas cricket team were therefore not used in this study. The inclusion of the national hockey players into the research attempted to provide a more ideal control group. The hockey squad was assessed pre-season (as were all the rugby players), and because hockey and rugby are both winter sports none of the hockey players played rugby beyond primary school level.

The Springbok rugby players were assessed as part of the first phase of this research conducted in February 1997 (Ancer, 1999; Dickinson, 1998; Reid, 1998). The Under 21 rugby players received a neuropsychological assessment as part of a broader assessment conducted by the Sports Science Institute in February 1998. The hockey players were assessed between February and March 1999 and assessments were conducted either at their place of abode or at their place of work. An attempt was made to assess as many squad members as possible although there were some members of the squad who were not assessed due to private overseas playing commitments. All groups were assessed pre-season.

The players' estimated premorbid IQ was calculated using two South African Wechsler Adult Intelligence Scale (SAWAIS) Subtests: Comprehension and Picture Completion, and then prorated (see Appendix A). The estimated premorbid IQ was calculated from these two subtests as both are considered to be relatively unaffected in the presence of diffuse damage and are therefore good indicators of premorbid ability (Lezak, 1983). Although the Digit Span and Digit Symbol Substitution Subtests were also administered they were

not used to calculate the estimated premorbid IQ as they are susceptible to diffuse damage and are therefore not reliable premorbid indicators (Lezak, 1995). The premorbid IQ score estimated on this basis will be referred to as 'IQ'.

Although use of a cluster of subtest scores provides a more accurate premorbid IQ, where one of the two subtest scores was more than 1 SD below the norm (less than 8,5) and there was a significant difference of three or more between the two subtest scores, the premorbid IQ was calculated using the single highest score only. The 'best performance method', the use of a single high subtest score to estimate premorbid IQ (with the exception of the vocabulary, general information or arithmetic subtests), is permissible according to Lezak (1995). This method was necessary for four players (see Appendix A). Given the time and data constraints it was deemed the most appropriate measure to use in order to estimate an approximate premorbid level of functioning.

In order to reduce the significant difference on IQ between the two main groups of subjects (rugby players and non-contact sport controls) any subjects with an estimated premorbid IQ falling more than 1 SD below the norm (less than 85) were discarded. In addition, any subjects with an IQ score greater or equal to 140 were excluded. Only the results of those participants whose IQ scores were within the range of 85 to 140 were analysed. As a result of these exclusion criteria the following subjects were excluded: Springbok rugby players - no exclusions; Under 21 rugby players - 2 exclusions (estimated premorbid IQ below 85); Springbok hockey players - 2 exclusions (estimated premorbid IQ above 140). Only the IQ scores and test results of the remaining players were used for this research (see Appendix A for the estimated premorbid IQ scores of the remaining players).

The following exclusion criteria were also applied to the participants in order to prevent further confounding variables: history of substance abuse; neurological or psychiatric disorder; previous moderate to severe non-sport related head injury. No participants were excluded on these grounds.

The demographic data of the three groups (Springbok Rugby, Under 21 Rugby and Springbok Hockey), less those excluded, appears in Table 3-1 below (see Table 3-1, p. 51).

Table 3-1. Demographic Data of Participants

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	
Springbok Rugby	26	27.46	2.73	0.0000**	14.19	1.41	0.0001**	119.19	11.96	0.0127**
Under 21 Rugby	19	19.74	0.73		12.32	1.57		110.26	10.72	

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

3.2. DATA COLLECTION PROCEDURE

3.2.1. CONTINUITY BETWEEN PHASES OF THE RESEARCH

3.2.1.1. Protocols

Protocols were developed for the assessments in phase one of this research by the three Intern Clinical Psychologists who were conducting the research together with their supervisor. These instructions were based on the original test manuals and/or from Lezak (1995). On verbal tests, Afrikaans versions of the instructions and tests were used when appropriate. Protocols were developed in order to ensure that each participant was assessed in accordance with standard instructions. An assessment schedule (Appendix B) was drawn up to ensure that tests were administered in the appropriate order, allowing for interference tasks and time delays between recall elements of tests where necessary. In order to ensure standardisation between the two phases of the research this researcher held several meetings with the researchers involved in phase one to ensure that the instructions and the procedures for administering the assessments were unambiguous and consistent (see Appendix C).

3.2.1.2. Administration of Protocols

All the Intern Clinical Psychologists involved in Phases One and Two of the research, and two additional Clinical Psychologists and a research masters student who assisted with the administration of the assessments to the Under 21 rugby players, all received their training at Rhodes University. All assessors were provided with the standardised protocols, and were briefed thoroughly on the instructions and procedures to be followed during assessments.

3.2.1.3. Scoring

All protocols were scored by the three Intern Clinical Psychologists involved in phase two of this research. Instructions for the scoring of the protocols were drafted in consultation with phase one researchers based upon the methods they used when scoring their assessments. In addition to these instructions, examples of those tests with a subjective scoring component were given to the phase two researchers in order to provide a guideline as to the degree with which scoring criteria had been applied in phase one. Phase one researchers also checked several protocols at random to ensure continuity of scoring standards between the two phases of the research. Phase two researchers scored all protocols in consultation with each other to ensure inter-rater reliability.

3.2.2. CONSENT OF PARTICIPANTS

The nature and purpose of the procedure was explained to all participants immediately prior to testing. Once any questions had been answered the participants were required to sign a consent form before the assessment was begun. Different consent forms (Appendix D) were used for the two phases of the research. Phase one was initiated to provide clinical baseline data on the Springbok rugby players, and it was agreed that this data could be used for research purposes. It was, therefore, necessary to gain consent from the players as brief reports containing individual results were written for the sports physician of the Sports Science Institute and formed part of a comprehensive report for SARFU. As these reports did not form part of the brief of phase two, the consent form was altered accordingly. For phase two the form stated that: the participant understood that the assessment would take approximately two hours to administer and would be administered by a

clinician trained at Rhodes University; the assessment consisted of a series of questions and a variety of intellectual tests; the results would form a group database for comparative purposes between players of contact sport and non-contact sport; the results would remain totally confidential and anonymous. The assessment only commenced once this consent was received.

The parents of those hockey players under the age of 21 were asked to sign the consent form on behalf of their minor child. The under 21 rugby players were deemed able to sign the consent form on their own behalf as their parents had already consented to the broad assessment at the Sports Science Institute, and the neuropsychological assessment was a part of this assessment. All the Springbok rugby players were older than 21 and were able to sign in their own capacity.

3.2.3. QUESTIONNAIRES

All participants were required to complete two questionnaires developed as part of phase one of the research (Ancer, 1999; Dickinson, 1998; Reid, 1998). The first questionnaire provided important demographic data while the second was a self-report questionnaire concerning the presence or absence of possible postconcussive symptoms.

3.2.3.1. Demographic Questionnaire

This questionnaire (see Appendix E) was completed by the participant and provided important demographic information regarding four main areas: personal history (age, level of education, first language, learning disability, occupation, etc); sporting history (what sports played, at what age begun, positions played, etc); previous head injuries (both sport-related and non-sports related); and exclusion criteria (neurological or psychiatric disorders, substance abuse).

3.2.3.2. Postconcussive Questionnaire

The self-report postconcussive questionnaire (see Appendix F) provided information on the frequency that players suffered from 31 possible symptoms. This questionnaire was developed as part of phase one of the research. The 31 questions were designed around 14 content areas and were derived from the following sources: Burbach, 1987; Levin et al., 1987; Lezak, 1995; Lishman, 1978; and Walsh, 1985 (as per Dickinson, 1998). The 14 content areas from which these symptoms were drawn were: 1. Physical/neurological symptoms; 2. Perceptual disturbances; 3. Sexual problems; 4. Speech and language; 5. Memory; 6. Attention and concentration; 7. Emotional lability; 8. Frustration tolerance; 9. Depression; 10. Social withdrawal; 11. Restlessness; 12. Vegetative symptoms; 13. Anxiety; 14. Aggression.

3.2.4. NEUROPSYCHOLOGICAL TEST BATTERY

The neuropsychological test battery was designed to provide both an estimated pre-morbid level of general intellectual functioning as well as current functioning in the following cognitive modalities: verbal fluency; visual memory; verbal memory; visuoperceptual scanning speed; and fine hand-motor dexterity. Normative data exists for all the tests used in the neuropsychological battery. Scaled scores for the SAWAIS Subtests, using the age appropriate standardisation, were used to determine the estimated premorbid IQ for each participant (SAWAIS Manual, 1969). Normative data for the other tests, including the SAWAIS Digit

Symbol Substitution (raw score), were taken from Shuttleworth-Jordan and Bode (1995). The only exception was for the "S" Words Fluency Test where the normative data were taken from Yeudal (1986). Shuttleworth-Jordan and Bode's (1995) normative data was acquired by assessing university students, 18-25 years old. This group closely matches both the rugby and hockey groups in terms of important variables such as age, as well as having a relatively high level of education and intellectual functioning.

The following tests were used for each of these areas (these are discussed in more detail below):

- **General intellectual functioning:**
  South African Wechsler Adult Intelligence Scale (SAWAIS):
    Picture Completion Subtest;
    Comprehension Subtest;
- **Verbal memory:**
  SAWAIS Digit Span Subtest including Supraspan A & B;
  Wechsler Memory Scale (WMS) Associate Learning Subtest;
- **Visual memory:**
  Digit-Symbol Incidental recall: Immediate;
  Digit-Symbol Incidental recall: Delayed;
  Wechsler Memory Scale (WMS) Visual Reproduction Subtest;
- **Verbal fluency:**
  Words in One Minute Unstructured Verbal Fluency Test;
  Structured Verbal Fluency Test - 'S' Words;
- **Visuoperceptual scanning speed:**
  SAWAIS Digit-Symbol Substitution Subtest;
  Trail Making Tests A & B;
- **Fine hand motor dexterity:**
  Sequential Finger Tapping Test.

All tests used in this battery are regularly used in neuropsychological assessment.

3.2.4.1. General intellectual functioning

As noted earlier in Chapter Two, it is important to estimate the premorbid level of functioning of the players as this allows a more accurate estimate of deficit among individual players. It is also an important variable in the ability of individuals to adjust to cognitive deficit following brain injury and research has found a consistent relationship between premorbid ability and the level of impairment suffered (Lezak, 1995). An estimated IQ was calculated using two Subtests of the South African Wechsler Adult Intelligence Scale (SAWAIS) and then prorating the scores (as described earlier in this Chapter).

SAWAIS Comprehension Subtest:

Instructions for this test were taken from the SAWAIS Manual (1969). This test consists of 10 open-ended questions of common-sense judgement and practical reasoning (Lezak, 1995). Participants are given the instruction that there are no right or wrong answers, just to say what they thought in each case. Where

answers were brief, amplification was gained by questioning participants further (SAWAIS Manual, 1969). This is a test of verbal reasoning, an ability which holds well in cases of diffuse brain damage. It is, therefore, a good test of previous ability (Lezak, 1995).

SAWAIS Picture Completion Subtest:

Instructions for this test were taken from the SAWAIS Manual (1969). This test consists of 15 drawings, each of which is missing a key detail. The cards are presented in numerical order and the participant is asked to name the missing detail. There is a time limit of 20 seconds per picture. This is primarily a test of visual reasoning but does involve both visuoperceptual and verbal abilities. Picture completion has consistently demonstrated resilience to the effects of brain damage and is relatively unaffected by diffuse brain damage. It is, therefore, a good test of previous ability (Lezak, 1995).

3.2.4.2. Verbal memory

SAWAIS Digit Span Subtest:

This is a test of verbal memory and the version used comes from the SAWAIS Manual (1969). Digits Forwards and Backwards were reported and analysed as separate tests as they involve different mental processes and are, therefore, affected differently by brain damage (Lezak, 1995).

Digits Forwards:

A sequence of random numbers (between 1 and 9) was read which the participants were asked to repeat in the same order. Each trial consisted of a pair of sequences containing the same amount of numbers but in a different random order. If the participant correctly repeated at least one of the pairs in each trial then the researcher continued to the next trial. Each new trial included one more number than the previous trial.

While Digits Forwards does test immediate verbal memory, it is primarily a test of efficiency of attention or "freedom from distraction". It is not as sensitive to the effects of diffuse brain damage as Digits Backwards and would, therefore, be expected to maintain relative to Digits Backwards in the presence of diffuse brain damage (Lezak, 1995).

Digit Supraspan - New Learning:

The method of McFie (1975) was used, after a participant failed both sequences on a trial. The researcher repeated the last failed sequence until the participant repeated it correctly. The score is the number of attempts required to learn the sequence correctly. This tests verbal new learning ability, and as an extended version of the Digits Forwards test, is more sensitive to memory function (Shuttleworth-Jordan, 1992).

Digits Backwards:

This test is similar to the Digits Forwards, except that the participant must repeat the sequence of numbers which the researcher reads in reverse order. The test is discontinued after failure of both sequences of a trial. The score is the longest sequence of numbers correctly repeated (SAWAIS Manual, 1969). This test involves storing data while manipulating it mentally, tapping working memory function, and is particularly sensitive to diffuse brain damage (Lezak, 1995) which might be expected after a closed head injury.

WMS Associate Learning Subtest (Immediate Recall):

The version used was taken from Form I of the WMS manual (Wechsler, 1945). The test consists of a series of 10 paired words which are divided into six easy pairs and four hard pairs. The easy pairs are words that are usually associated with each other, while the hard pairs are more difficult to learn because they are words that are not normally associated with each other. The participant is instructed to remember the pairs of words which the researcher reads out. The researcher then provides the first word and the participant has to recall the paired associated word. This procedure is repeated three times. The Afrikaans translation (Burbach, 1987) was used for Afrikaans first-language participants.

Because the ability to remember the easy pairs relies primarily on old associate learning, while the hard pairs rely more on new learning ability (Lezak, 1995), the hard pairs are more susceptible to the effects of brain damage. This distinction is lost if the results are reported as a single score, therefore, for purposes of this research, the easy and hard scores are reported and analysed separately.

WMS Associate Learning Subtest (Delayed Recall):

After a 20 minute delay the delayed version of this memory test was administered. The list of paired words is not repeated, the participant is given the first word and instructed to try and recall the paired associated word from the list of paired words read earlier. Delayed memory is typically more sensitive to the effects of diffuse brain damage than immediate memory (Lezak, 1995). Stuss et al. (1985) were able to distinguish between patients who had apparently recovered from mild head injury and normal controls by slightly, but fairly consistent, lower scores on the delayed versions of the WMS.

3.2.4.3. Visual memory

Digit Symbol Incidental Recall (Immediate):

The short form method of the incidental Recall test was used (Shuttleworth-Jordan & Bode, 1995). The researcher notes how far the subject has managed to get after 90 seconds but allows the participant to continue to the end of the second last row. The participant was then given a sheet on which the numbers were written and was asked to write as many of the matching symbols as could be remembered. This is a test which taps recent memory, which has been shown to be susceptible to the effects of diffuse brain damage (Shuttleworth-Jordan & Bode, 1995).

Digit-Symbol Incidental Recall (Delayed):

After a 20 minute delay, participants were again handed a sheet with the numbers written on it and asked to write as many of the matching pairs as they could remember. Most patients remember as many or almost as many digit-symbol pairs on delayed as on immediate recall. Patients with significant retention problems recall fewer, while some patients recall more, possibly because of a slowed processing problem (Lezak, 1995).

WMS Visual Reproduction (Immediate Recall):

The version used was taken from Form I of the WMS manual (Wechsler, 1945). The test consists of three cards, Cards I and II have one design each while Card III has two designs on it. The participants are shown

each card for 10 seconds and then instructed to draw the design from memory. According to Lezak (1995) this test is sensitive to the effects of head trauma while Stuss et al. (1985) have shown that this test can be used to significantly differentiate patients with mild head trauma from uninjured controls.

WMS Visual Reproduction (Delayed Recall):

After a 20 minute interval a delayed version of this test was administered. Participants were asked to draw the designs, which they had been shown earlier, from memory. As stated above, this test is sensitive to the effects of head trauma (Lezak, 1995) while Stuss et al. (1985) have shown that this test can be used to significantly differentiate patients with mild head trauma from uninjured controls.

3.2.4.4. Verbal fluency

Unstructured Verbal Fluency Test - Words in One Minute: (Terman & Merrill, 1973)

This is a test of unstructured verbal fluency, and instructions were derived from Lezak (1995). The participants were instructed to say as many words as they could think of within one minute. They were not allowed to make sentences, count, use proper nouns or use variations of the same word. Instructions were repeated until they were clear. Many patients who have suffered brain injury experience changes in the speed and ease of verbal production. This test also indirectly employs short-term memory in order to keep track of words already used (Lezak, 1995).

Structured Verbal Fluency Test - 'S' Words:

The instructions were given as above but this time the participant was instructed to only use words starting with an 'S'. Structured word fluency tests provide the greatest scope for subjects seeking a strategy for guiding the search for words and are most difficult for subjects who cannot develop strategies of their own (Lezak, 1995).

3.2.4.5. Visuoperceptual scanning speed

SAWAIS Digit Symbol Substitution Subtest:

This test consists of three rows consisting of 67 digits with an open block below each digit. There is a key which matches each of the numbers from 1 to 9 with a symbol. Instructions used were from the SAWAIS Manual (1969). Participants are instructed to draw the matching symbol in the block below each number in the three rows as per the key. The examiner demonstrates how this is done by completing a sample at the beginning of the first row after which the participant is instructed to continue as quickly as possible and without leaving any symbols out. If a participant paused to correct an error they were instructed to leave it and carry on. The number of blocks completed at the end of 90 seconds was noted.

This is a test of complex visuoperceptual tracking (Lezak, 1995) and requires psychomotor problem solving and visual perceptual abilities (Barth et al., 1989). It is consistently sensitive to brain damage and its score is likely to be depressed even with minimal damage (Lezak, 1995; Russell, 1986). It is thus particularly useful in picking up the sort of diffuse brain damage expected in players of contact sports.

Trail Making Test A & B: (Reitan, 1956)

The Trail Making Test consists of two parts, A and B. It is particularly sensitive to the effects of brain injury (Lezak, 1995) as it is a measure of sustained attention and concentration which requires sequential problem solving and the ability to keep two things in mind simultaneously (Barth et al., 1989). Patients with mild head trauma are slower than control subjects and the slowing increases with the severity of the damage (Leininger et al., 1990).

Part A - The test consists of a series of numbers within circles on a sheet of paper. Participants are instructed to join the numbers sequentially without lifting their pencil from the page. They first practice by completing a mini-trial before proceeding onto the test proper. If they make a mistake it is pointed out immediately and they are required to correct the mistake immediately and continue. The score is the time taken to complete the trial.

Part B - The format for this test is similar to Part A, except that the participant is required to sequentially join both numbers and letters, alternating between them. The test is begun after a mini-trial has been completed and instructions and scoring are as for Part A. This part of the test involves complex visuoperceptual tracking, the ability to shift a response set, and taps working memory function. Part B requires more information processing ability than Part A (Spreen & Strauss, 1991). Because of its difficulty, scores on Part B are likely to be more markedly lowered than those on Part A in the presence of diffuse brain damage as it is more sensitive (Spreen & Strauss, 1991).

3.2.4.6. Fine hand motor dexterity

Sequential Finger Tapping Test: (Denckla, 1973)

The participants were instructed to place both elbows on the desk and, as fast as they can, to touch each finger in order to the thumb, starting with the index finger. The examiner demonstrated what was required and the participants practised until they were ready to begin. The score is the time taken to do five sets of the above. The test is repeated for the preferred and non-preferred hand. In order to obtain the participant's best score this test was administered twice during the assessment. As it is a timed test, bilateral slowing would be an indication of diffuse brain damage (Lezak, 1995).

3.3 DATA ANALYSIS

3.3.1. NEUROPSYCHOLOGICAL RESULTS

First the level of impairment or deficit shown by each player was calculated for each neuropsychological test. The level of deficit was determined relative to the degree with which their score deviated from the norms used. "Deficit" was defined in terms of Dickinson's (1998) criteria and reported in 'none', 'mild', and 'moderate/severe' terms relative to the deviation from the normative data as follows:

None - the test score is within 1 standard deviation from the norm;

Mild - the test score is equal to or greater than 1 standard deviation from the norm but less than 2 standard deviations in the direction indicating poor performance;

Moderate/Severe - the test score is equal to or greater than 2 standard deviations from the norm in the direction indicating poor performance.

In order to perform statistical analyses each level of deficit was coded as follows:

None = 0

Mild = 1

Moderate/Severe = 3

Once the level of deficit for each individual player had been calculated, the number (n) of players with each level of deficit within each group (eg rugby, hockey, forwards, backs etc) was determined. This number was then represented as a percentage (%), that is, the proportion of each level of deficit for each group. The chi-squared formula was used to compare the percentages of deficit between the various groups and sub-groups (see Table 4-1, p. 60 for the full list of comparisons and their results).

3.3.2. POSTCONCUSSIVE SYPTOMATOLOGY RESULTS

In order to perform statistical analyses each frequency level was coded as follows:

Never = 1

Sometimes = 2

Often = 3

The number (n) of players reporting each frequency level within each group (eg rugby, hockey, forwards, backs etc) was determined and then represented as a percentage (%), that is, the proportion of each level of frequency for each group. The chi-squared formula was used to compare the percentages of post-concussive symptomatology between the various groups and sub-groups (see Table 4-1, p. 60 for the full list of comparisons and their results).

3.3.3. CHI SQUARED ANALYSES

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

Results of the chi-square test were then interpreted in terms of two levels of significance: the difference between the two groups was taken to be **significant** if $p < 0.05$. The difference between the two groups was taken to be **approaching significance** if $p > 0.05$ but $p < 0.15$. Bonferroni adjustments to the level of significance to ensure that the overall error rate was at most 0.10 was not necessary because the analyses were performed on subsets of groups (ie forwards being a subset of the rugby players) and not on multiple pairwise comparisons (Miller, 1981).

The results of the comparisons were then tabulated in Chapter 4 (Tables 4-2 to 4-49, pp. 78 - 109) and are discussed in Chapter 5 (pp. 110 - 142).

CHAPTER 4 : RESULTS

The comparative results for (i) the neuropsychological assessment and (ii) the postconcussive symptomatology are presented below. In each case the following comparisons were made:

Table 4-1 : List of Comparisons and Tables

Comparison	Neuropsychological Assessment	Postconcussive Symptomatology
RUGBY versus HOCKEY	Table 4-2 to 4-6, pp. 78 - 79	Table 4-42, pp. 94 - 95
SPRINGBOK RUGBY versus HOCKEY	Table 4-7 to 4-11, pp. 80 - 81	Table 4-43, pp. 96 - 97
UNDER 21 RUGBY versus HOCKEY	Table 4-12 to 4-16, pp. 82 - 83	Table 4-44, pp. 98 - 99
RUGBY FORWARDS versus HOCKEY	Table 4-17 to 4-21, pp. 84 - 85	Table 4-45, pp. 100 - 101
RUGBY: FORWARDS versus BACKS	Table 4-22 to 4-26, pp. 86 - 87	Table 4-46, pp. 102 - 103
SPRINGBOKS: FORWARDS versus BACKS	Table 4-27 to 4-31, pp. 88 - 89	Table 4-47, pp. 104 - 105
UNDER 21: FORWARDS versus BACKS	Table 4-32 to 4-36, pp. 90 - 91	Table 4-48, pp. 106 - 107
SPRINGBOK RUGBY versus UNDER 21	Table 4-37 to 4-41, pp. 92 - 93	Table 4-49, pp. 108 - 109

Results were tabulated and all the tables appear at the end of Section 4.1. (pp. 78 - 109). In all the tables 'significance' and 'approaching significance' are represented as follows:

* significance ($p < 0,05$)

~ approaching significance ($0,05 < p < 0,15$)

Neuropsychological Assessment

The tables indicate the number (n) and percentage (%) of players across each level of deficit for all the cognitive tests together with the x^2 statistic. Where *'No Statistic'* is reported this indicates that for both groups no player showed any deficit and thus statistical comparison was not required.

Postconcussive Symptomatology

The tables indicate the number (n) and percentage (%) of players across three levels of frequency for all the symptoms together with the x^2 statistic. Where *'No Statistic'* is reported this indicates that for both groups no symptomatology was reported and thus statistical comparison was not required.

4.1. SUMMARY OF RESULTS

In this summary, the results of the comparison of the neuropsychological assessment and the results of the comparison of the postconcussive symptoms will be reported separately. Those tests or symptoms where

results were either significant ($p < 0,05$) or approaching significance ($0,05 < p < 0,15$) will be summarised and followed by a description indicating the nature and direction of the differences.

4.1.1. NEUROPSYCHOLOGICAL ASSESSMENT

Significant differences ($p < 0,05$) were found on the following neuropsychological tests: Digits Forwards; Unstructured Verbal Fluency; SAWAIS Digit Symbol Substitution; Finger Tapping Test: Non-Preferred Hand - Trial 1. Results approaching significance ($0,05 < p < 0,15$) were found on the following neuropsychological tests: WMS Paired Associate Learning - Hard (Immediate); Digit Symbol Incidental Recall (Immediate); WMS Memory for Designs (Delayed); Unstructured Verbal Fluency; SAWAIS Digit Symbol Substitution; Trail Making Test - Part B; Sequential Finger Tapping: Preferred hand - Trial 1 and 2; Non-Preferred Hand - Trial 1. No other results were of statistical significance. Significant results and results approaching significance will be discussed together for each of the tests mentioned above.

With regard to deficit, as stated earlier, 'None' indicates that the individuals' scores were within 1 standard deviation from the norm. 'Mild' indicates that the individuals' scores were equal to or greater than 1 standard deviation from the norm but less than 2 standard deviations from the norm in the direction indicating poor performance. 'Moderate/severe' indicates that the individuals' scores were equal to or greater than 2 standard deviations from the norm in the direction indicating poor performance.

4.1.1.1. Digits Forwards

For this test, a significant result was found for Springbok rugby versus Under 21 rugby only ($p = 0,0152$; Table 4-37, p. 92). For Springbok rugby, 76,9% showed no deficit compared with 47,4% of Under 21 rugby. Amongst Springbok rugby, 23,1% showed mild deficit compared with 26,3% of Under 21 rugby. Finally, for Springbok rugby, 0% showed moderate/severe deficit compared with 26,3% of Under 21 rugby.

4.1.1.2. WMS Associate Learning (Hard) Immediate Recall

For this test, results approaching significance were found on the following comparisons: Springbok rugby versus hockey; Under 21 rugby versus hockey; and rugby forwards versus hockey.

When Springbok rugby was compared with hockey (Table 4-7, p. 80) the result was approaching significance ($p = 0,1044$). For Springbok rugby, only 80,8% showed no deficit compared with 100% of hockey. Amongst Springbok rugby, 15,4% showed mild deficit compared with 0% of hockey. Finally, for Springbok rugby, 3,8% showed moderate/severe deficit compared with 0% of hockey.

When Under 21 rugby was compared with hockey (Table 4-12, p. 82) the result was approaching significance ($p = 0,1272$). For Under 21 rugby, 89,5% showed no deficit compared with 100% of hockey.

Amongst Under 21 rugby, 10,5% showed mild deficit compared with 0% of hockey. There was no moderate/severe deficit present in either group.

When the rugby forwards were compared with hockey (Table 4-17, p. 84) the result was approaching significance (p = 0,1044). For the rugby forwards, 80,8% showed no deficit compared with 100% of hockey. Amongst the rugby forwards, 15,4% showed mild deficit compared with 0% of hockey. Finally, for the rugby forwards, 3,8% showed moderate/severe deficit compared with 0% of hockey.

4.1.1.3. SAWAIS Digit Symbol Substitution Incidental Recall

For this test, results approaching significance were found on the following comparisons: Springbok rugby versus hockey; rugby forwards versus hockey; Springbok forwards versus Springbok backs; and Springbok rugby versus Under 21 rugby.

When Springbok rugby was compared with hockey (Table 4-8, p. 80) the result was approaching significance (p = 0,0707). For Springbok rugby, only 69,2% showed no deficit compared with 95,2% of hockey. Amongst Springbok rugby, 11,5% showed mild deficit compared with 0% of hockey. Finally, for Springbok rugby, 19,2% showed moderate/severe deficit compared with only 4,8% of hockey.

When the rugby forwards were compared with hockey (Table 4-18, p. 84) the result was approaching significance (p = 0,1140). For the rugby forwards, 73,1% showed no deficit compared with 95,2% of hockey. Amongst the rugby forwards, 11,5% showed mild deficit compared with 0% of hockey. Finally, for the rugby forwards, 15,4% showed moderate/severe deficit compared with only 4,8% of hockey.

When the Springbok forwards were compared with Springbok backs (Table 4-28, p. 88) the result was approaching significance (p = 0,1047). For the Springbok forwards, only 53,3% showed no deficit compared with 90,9% of the Springbok backs. Amongst the Springbok forwards, 20% showed mild deficit compared with 0% of the Springbok backs. Finally, for the Springbok forwards, 26,7% showed moderate/severe deficit compared with 9,1 % of the Springbok backs.

When Springbok rugby was compared with Under 21 rugby (Table 4-38, p. 92) the result was approaching significance (p = 0,0807). For Springbok rugby, only 69,2% showed no deficit compared with 94,7 % of Under 21 rugby. Amongst Springbok rugby, 11,5% showed mild deficit compared with 5,3% of Under 21 rugby. Finally, for Springbok rugby, 19,2% showed moderate/severe deficit compared with 0% of Under 21 rugby.

4.1.1.4. WMS Visual Reproduction Delayed Recall

For this test, a result approaching significance was found on the following comparisons: Springbok rugby versus hockey; and Springbok rugby versus Under 21 rugby.

When Springbok rugby was compared with hockey (Table 4-8, p. 80) the result was approaching significance (p = 0,0795). For Springbok rugby, only 76,9% showed no deficit compared with 95,2% of

hockey. Amongst Springbok rugby, 23,1% showed mild deficit compared with only 4,8% of hockey. There was no moderate/severe deficit present in either group.

When Springbok rugby was compared with Under 21 rugby (Table 4-38, p. 92) the result was approaching significance (p = 0,1482). For Springbok rugby, only 76,9% showed no deficit compared with 89,5% of Under 21 rugby. Amongst Springbok rugby, 23,1% showed mild deficit compared with only 5,3% of hockey. Finally, for Springbok rugby, 0% showed moderate/severe deficit compared with 5,3% of Under 21 rugby.

4.1.1.5. Unstructured Verbal Fluency

For this test, significant results were found on the following comparisons: Under 21 rugby versus hockey; and Springbok rugby versus Under 21 rugby. A result approaching significance was found on the following comparisons: rugby versus hockey; and rugby forwards versus hockey.

When Under 21 rugby was compared with hockey (Table 4-14, p. 82) the result was significant (p = 0,0075). For Under 21 rugby, 21,1% showed no deficit compared with 66,7% of hockey. Amongst Under 21 rugby, 63,2% showed mild deficit compared with 33,3% of hockey. Finally, for Under 21 rugby, 15,8% showed moderate/severe deficit compared with 0% of hockey.

When Springbok rugby was compared with Under 21 rugby (Table 4-39, p. 92) the result was significant (p = 0,0210). For Springbok rugby, 61,5% showed no deficit compared with 21,1% of Under 21 rugby. Amongst Springbok rugby, 34,6% showed mild deficit compared with 63,2% of Under 21 rugby. Finally, for Springbok rugby, only 3,8% showed moderate/severe deficit compared with 15,8% of Under 21 rugby.

When rugby was compared with hockey (Table 4-4, p. 78) the result was approaching significance (p = 0,1467). For rugby, 44,4% showed no deficit compared with 66,7% of hockey. Amongst rugby, 46,7% showed mild deficit compared with 33,3% of hockey. Finally, for rugby, 8,9% showed moderate/severe deficit compared with 0% of hockey.

When the rugby forwards were compared with hockey (Table 4-19, p. 84) the result was approaching significance (p = 0,0509). For the rugby forwards, 34,6% showed no deficit compared with 66,7% of hockey. Amongst the rugby forwards, 53,8% showed mild deficit compared with 33,3% of hockey. Finally, for the rugby forwards, 11,5% showed moderate/severe deficit compared with 0% of hockey.

4.1.1.6. SAWAIS Digit Symbol Substitution

For this test, significant results were found on the following comparisons: Under 21 rugby versus hockey; rugby forwards versus hockey; rugby forwards versus rugby backs; Springbok forwards versus Springbok backs; and Springbok rugby versus Under 21 rugby. Results approaching significance were found on the following comparisons: rugby versus hockey; and Springbok rugby versus hockey.

When Under 21 rugby was compared with hockey (Table 4-15, p. 83) the result was significant (p = 0,0155). For Under 21 rugby, only 57,9% showed no deficit compared with 95,2% of hockey. Amongst Under 21 rugby, 21,1% showed mild deficit compared with only 4,8% of hockey. Finally, for Under 21 rugby, 21,1% showed moderate/severe deficit compared with 0% of hockey.

When the rugby forwards were compared with hockey (Table 4-20, p. 85) the result was significant (p = 0,0066). For the rugby forwards, only 53,8% showed no deficit compared with 95,2% of hockey. Amongst the rugby forwards, 34,6% showed mild deficit compared with only 4,8% of hockey. Finally, for the rugby forwards, 11,5% showed moderate/severe deficit compared with 0% of hockey.

When the rugby forwards were compared with the rugby backs (Table 4-25, p. 87) the result was significant (p = 0,0340). For the rugby forwards, only 53,8% showed no deficit compared with 89,5% of the rugby backs. Amongst the rugby forwards, 34,6% showed mild deficit compared with only 5,3% of the rugby backs. Finally, for the rugby forwards, 11,5% showed moderate/severe deficit compared with 5,3% of the rugby backs.

When the Springbok forwards were compared with the Springbok backs (Table 4-30, p. 89) the result was significant (p = 0,0168). For the Springbok forwards, only 60% showed no deficit compared with 0% of the Springbok backs. Amongst the Springbok forwards, 40% showed mild deficit compared with 0% of the Springbok backs. There was no moderate/severe deficit present in either group.

When Springbok rugby was compared with Under 21 rugby (Table 4-40, p. 93) the result was significant (p = 0,0481). For Springbok rugby, 76,9% showed no deficit compared with 57,9% of Under 21 rugby. Amongst Springbok rugby, 23,1% showed mild deficit compared with 21,1% of Under 21 rugby. Finally, for Springbok rugby, 0% showed moderate/severe deficit compared with 21,1% of Under 21 rugby.

When rugby was compared with hockey (Table 4-5, p. 79) the result was approaching significance (p = 0,0558). For rugby, only 68,9% showed no deficit compared with 95,2% of hockey. Amongst rugby, 22,2% showed mild deficit compared with only 4,8% of hockey. Finally, for rugby, 8,9% showed moderate/severe deficit compared with 0% of hockey.

When Springbok rugby was compared with hockey (Table 4-10, p. 81) the result was approaching significance (p = 0,0795). For Springbok rugby, only 76,9% showed no deficit compared with 95,2% of hockey. Amongst Springbok rugby, 23,1% showed mild deficit compared with only 4,8% of hockey. There was no moderate/severe deficit present in either group.

4.1.1.7. Trail Making Test B

For this test, results approaching significance were found on the following comparisons: rugby versus hockey; Under 21 rugby versus hockey; rugby forwards versus hockey; and Springbok forwards versus Springbok backs.

When rugby was compared with hockey (Table 4-5, p. 79) the result was approaching significance (p = 0,1185). For rugby, only 75,6% showed no deficit compared with 95,2% of hockey. Amongst rugby, 8,9% showed mild deficit compared with only 4,8% of hockey. Finally, for rugby, 15,6% showed moderate/severe deficit compared with 0% of hockey.

When Under 21 rugby was compared with hockey (Table 4-15, p. 83) the result was approaching significance (p = 0,0569). For Under 21 rugby, only 68,4% showed no deficit compared with 95,2% of hockey. Amongst Under 21 rugby, 10,5% showed mild deficit compared with only 4,8% of hockey. Finally, for Under 21 rugby, 21,1% showed moderate/severe deficit compared with 0% of hockey.

When the rugby forwards were compared with hockey (Table 4-20, p. 85) the result was approaching significance (p = 0,0597). For the rugby forwards, 69,2% showed no deficit compared with 95,2% of hockey. Amongst the rugby forwards, 11,5% showed mild deficit compared with 4,8% of hockey. Finally, for the rugby forwards, 19,2% showed moderate/severe deficit compared with 0% of hockey.

When the Springbok forwards were compared with the Springbok backs (Table 4-30, p. 89) the result was approaching significance (p = 0,1033). For the Springbok forwards, only 66,7% showed no deficit compared with 100% of the Springbok backs. Amongst the Springbok forwards, 13,3% showed mild deficit compared with 0% of the Springbok backs. Finally, for the Springbok forwards, 20% showed moderate/severe deficit compared with 0% of the Springbok backs.

4.1.1.8. Finger Tapping Test:

Preferred hand - Trial 1

For this test, a result approaching significance was found for Under 21 forwards versus Under 21 backs only (p = 0,0848; Table 4-36, p. 91). For the Under 21 forwards, only 54,5% showed no deficit compared with 100% of the Under 21 backs. Amongst the Under 21 forwards, 36,4% showed mild deficit compared with 0% of the Under 21 backs. Finally, for the Under 21 forwards, 9,1% showed moderate/severe deficit compared with 0% of the Under 21 backs.

Preferred hand - Trial 2

For this test, results approaching significance were found on the following comparisons: Under 21 rugby versus hockey; and Springbok rugby versus Under 21 rugby.

When Under 21 rugby was compared with hockey (Table 4-16, p. 83) the result was approaching significance (p = 0,1272). For Under 21 rugby, 89,5% showed no deficit compared with 100% of hockey. Amongst Under 21 rugby, 10,5% showed mild deficit compared with 0% of hockey. There was no moderate/severe deficit present in either group.

When Springbok rugby was compared with Under 21 rugby (Table 4-41, p. 93) the result was approaching significance (p = 0,0906). For Springbok rugby, 100% showed no deficit compared with 89,5%

of Under 21 rugby. Amongst Springbok rugby, 0% showed mild deficit compared with 10,5% of Under 21 rugby. There was no moderate/severe deficit present in either group.

Non-Preferred Hand - Trial 1

For this test, significant results were found on the following comparisons: Under 21 rugby versus hockey; and Springbok rugby versus Under 21 rugby. Results approaching significance were found on the following comparisons: rugby forwards versus hockey; rugby forwards versus rugby backs; and Under 21 forwards versus Under 21 backs.

When Under 21 rugby was compared with hockey (Table 4-16, p. 83) the result was significant ($p = 0,0092$). For Under 21 rugby, only 63,2% showed no deficit compared with 100% of hockey. Amongst Under 21 rugby, 26,3% showed mild deficit compared with 0% of hockey. Finally, for Under 21 rugby forwards, 10,5% showed moderate/severe deficit compared with 0% of the hockey.

When Springbok rugby was compared with Under 21 rugby (Table 4-41, p. 93) the result was significant ($p = 0,0042$). For Springbok rugby, 100% showed no deficit compared with only 63,2% of Under 21 rugby. Amongst Springbok rugby, 0% showed mild deficit compared with 26,3% of Under 21 rugby. Finally, for Springbok rugby, 0% showed moderate/severe deficit compared with 10,5% of Under 21 rugby.

When the rugby forwards were compared with hockey (Table 4-21, p. 85) the result was approaching significance ($p = 0,0551$). For the rugby forwards, 76% showed no deficit compared with 100% of hockey. Amongst the rugby forwards, 20% showed mild deficit compared with 0% of hockey. Finally, for the rugby forwards, 4% showed moderate/severe deficit compared with 0% of hockey.

When the rugby forwards were compared with the rugby backs (Table 4-26, p. 87) the result was approaching significance ($p = 0,1172$). For the rugby forwards, only 76% showed no deficit compared with 94,7% of the rugby backs. Amongst the rugby forwards, 20% showed mild deficit compared with only 0% of the rugby backs. Finally, for the rugby forwards, 4,9% showed moderate/severe deficit compared with 5,3% of the rugby backs.

When the Under 21 forwards were compared with the Under 21 backs (Table 4-36, p. 91) the result was approaching significance ($p = 0,0827$). For the Under 21 forwards, only 45,5% showed no deficit compared with 87,5% of the Under 21 backs. Amongst the Under 21 forwards, 45,5% showed mild deficit compared with 0% of the Under 21 backs. Finally, for the Under 21 forwards, 9,1% showed moderate/severe deficit compared with 12,5% of the Under 21 backs.

4.1.2. POSTCONCUSSIVE SYMPTOMS

Significant differences ($p < 0,05$) were found on the following postconcussive symptoms: Headaches; Weakness in Limbs; Clumsiness; Fatigue; Hallucinations; Memory; Attention/Concentration; Easily Angered; Social Contact; Anxiety; Argumentative; and Aggression. Results approaching significance

(0,05 < p < 0,15) were found on the following postconcussive symptoms: Headaches; Weakness in Limbs; Fatigue; Sensitivity to Noise; Hallucinations; Clumsy Speech; Slurred Speech; Attention/Concentration; Sustained Attention; Irritability; Easily Angered; Depressed; Social Contact; Restlessness; Sleep Difficulties; Appetite Difficulties; Anxiety; Worry; Short-tempered; and Aggression. No other results were of statistical significance. Significant results and results approaching significance will be discussed together for each of the symptoms mentioned above.

As stated earlier, the frequency of each symptom was rated by the participants on three possible levels: 'Never', 'Sometimes', and 'Often'.

4.1.2.1. Headaches

For this symptom, a significant result was found on the following comparison: Under 21 forwards versus Under 21 backs. Results approaching significance were found on the following comparisons: rugby versus hockey; Springbok rugby versus hockey; rugby forwards versus rugby backs.

When the Under 21 forwards were compared with the Under 21 backs (Table 4-48, p. 106) the result was significant (p = 0,0397). For the Under 21 forwards, only 27,3% reported never suffering headaches compared with 75% of the Under 21 backs. Amongst the Under 21 forwards, 72,7% reported sometimes suffering headaches compared with only 25% of the Under 21 backs. There was no moderate/severe experience of the symptom reported in either group.

When rugby was compared with hockey (Table 4-42, p. 94) the result was approaching significance (p = 0,1424). For rugby, 57,8% reported never suffering headaches compared with only 38,1% of hockey. Amongst rugby, 42,2% reported sometimes suffering headaches compared with 57,1% of hockey. Finally, for rugby, 0% reported often suffering headaches compared with 4,8% of hockey.

When Springbok rugby was compared with hockey (Table 4-43, p. 96) the result was approaching significance (p = 0,1234). For Springbok rugby, 65,4% reported never suffering headaches compared with 38,1% of hockey. Amongst Springbok rugby, 34,6% reported sometimes suffering headaches compared with 57,1% of hockey. Finally, for Springbok rugby, 0% reported often suffering headaches compared with 4,8% of hockey.

When the rugby forwards were compared with the rugby backs (Table 4-46, p. 102) the result was approaching significance (p = 0,0648). For the rugby forwards, only 46,2% reported never suffering headaches compared with 73,7% of the rugby backs. Amongst the rugby forwards, 53,8% reported sometimes suffering headaches compared with only 26,3% of the rugby backs. There was no moderate/severe experience of the symptom reported in either group.

4.1.2.2. Weakness in Limbs

For this symptom, a significant result was found on the following comparison: Springbok rugby versus Under 21 rugby. A result approaching significance was found on the following comparisons: Springbok rugby versus hockey.

When Springbok rugby was compared with Under 21 rugby (Table 4-49, p. 108) the result was significant (p = 0,0384). For Springbok rugby, 92,3% reported never experiencing weakness in their limbs compared with only 68,4% of Under 21 rugby. Amongst Springbok rugby, only 7,7% reported sometimes experiencing weakness in their limbs compared with 31,6% of Under 21 rugby. There was no moderate/severe experience of the symptom reported in either group.

When Springbok rugby was compared with hockey (Table 4-43, p. 96) the result was approaching significance (p = 0,1228). For Springbok rugby, 92,3% reported never experiencing weakness in their limbs compared with only 76,2% of hockey. Amongst Springbok rugby, only 7,7% reported sometimes experiencing weakness in their limbs compared with 23,8% of hockey. There was no moderate/severe experience of the symptom reported in either group.

4.1.2.3. Clumsiness

For this symptom, significant results were found on the following comparisons: Under 21 rugby versus hockey; and Springbok rugby versus Under 21 rugby.

When Under 21 rugby was compared with hockey (Table 4-44, p. 98) the result was significant (p = 0,0175). For Under 21 rugby, 57,9% reported never feeling clumsy compared with 90,5% of hockey. Amongst Under 21 rugby, 42,1% reported sometimes feeling clumsy compared with only 9,5% of hockey. There was no moderate/severe experience of the symptom reported in either group.

When Springbok rugby was compared with Under 21 rugby (Table 4-49, p. 108) the result was significant (p = 0,0061). For Springbok rugby, 92,3% reported never feeling clumsy compared with only 57,9% of Under 21 rugby. Amongst Springbok rugby, only 7,7% reported sometimes feeling clumsy compared with 42,1% of Under 21 rugby. There was no moderate/severe experience of the symptom reported in either group.

4.1.2.4. Fatigue

For this symptom, significant results were found on the following comparison: Springbok rugby versus Under 21 rugby. A result approaching significance was found on the following comparison: Under 21 rugby versus hockey.

When Springbok rugby was compared with Under 21 rugby (Table 4-49, p. 108) the result was significant (p = 0,0009). For Springbok rugby, 84,6% reported never suffering fatigue compared with only 36,8% of Under 21 rugby. Amongst Springbok rugby, only 15,4% reported sometimes suffering fatigue

compared with 63,2% of Under 21 rugby. There was no moderate/severe experience of the symptom reported in either group.

When Under 21 rugby was compared with hockey (Table 4-44, p. 98) the result was approaching significance (p = 0,0726). For Under 21 rugby, only 36,8% reported never suffering fatigue compared with 66,7% of hockey. Amongst Under 21 rugby, 63,2% reported sometimes suffering fatigue compared with only 28,6% of hockey. Finally, for Under 21 rugby, 0% reported often suffering from sensitivity to noise compared with 4,8% of hockey.

4.1.2.5. Sensitivity to Noise

For this symptom, results approaching significance were found on the following comparisons: rugby versus hockey; Springbok rugby versus hockey; Under 21 rugby versus hockey; and rugby forwards versus hockey.

When rugby was compared with hockey (Table 4-42, p. 94) the result was approaching significance (p = 0,1158). For rugby, only 66,7% reported never suffering from sensitivity to noise compared with 90,5% of hockey. Amongst rugby, 31,1% reported suffering from sensitivity to noise compared with only 9,5% of hockey. Finally, for rugby, 2,2% reported often suffering from sensitivity to noise compared with 0% of hockey.

When Springbok rugby was compared with hockey (Table 4-43, p. 96) the result was approaching significance (p = 0,1318). For Springbok rugby, only 73,1% reported never suffering from sensitivity to noise compared with 90,5% of hockey. Amongst Springbok rugby, 26,9% reported sometimes suffering from sensitivity to noise compared with only 9,5% of hockey. There was no moderate/severe experience of the symptom reported in either group.

When Under 21 rugby was compared with hockey (Table 4-44, p. 98) the result was approaching significance (p = 0,0543). For Under 21 rugby, only 57,9% reported never suffering from sensitivity to noise compared with 90,5% of hockey. Amongst Under 21 rugby, 36,8% reported sometimes suffering from sensitivity to noise compared with only 9,5% of hockey. Finally, for the Under 21 rugby, 5,3% reported often suffering from sensitivity to noise compared with 0% of hockey.

When the rugby forwards were compared with hockey (Table 4-45, p. 100) the result was approaching significance (p = 0,1208). For the rugby forwards, only 65,4% reported never suffering from sensitivity to noise compared with 90,5% of hockey. Amongst the rugby forwards, 30,8% reported sometimes suffering from sensitivity to noise compared with only 9,5% of hockey. Finally, for the rugby forwards, 3,8% reported often suffering from sensitivity to noise compared with 0% of hockey.

4.1.2.6. Hallucinations

For this symptom, a significant result was found on the comparison between Under 21 rugby and hockey. A result approaching significance was found on the comparison between the rugby forwards and hockey.

When Under 21 rugby was compared with hockey (Table 4-44, p. 98) the result was significant (p = 0,0258). For Under 21 rugby, 68,4% reported never suffering hallucinations compared with 95,2% of hockey. Amongst Under 21 rugby, 31,6% reported sometimes suffering hallucinations compared with only 4,8% of hockey. There was no moderate/severe experience of the symptom reported in either group.

When the rugby forwards were compared with hockey (Table 4-45, p. 100) the result was approaching significance (p = 0,0795). For the rugby forwards, only 76,9% reported never suffering hallucinations compared with 95,2% of hockey. Amongst the rugby forwards, 23,1% reported sometimes suffering hallucinations compared with only 4,8% of hockey. There was no moderate/severe experience of the symptom reported in either group.

4.1.2.7. Clumsy Speech

For this symptom, results approaching significance were found on the following comparisons: rugby versus hockey; Under 21 rugby versus hockey; and rugby forwards versus hockey.

When rugby was compared with hockey (Table 4-42, p. 94) the result was approaching significance (p = 0,1294). For rugby, 46,7% reported never experiencing clumsy speech compared with 66,7% of hockey. Amongst rugby, 53,3% reported sometimes experiencing clumsy speech compared with 33,3% of hockey. There was no moderate/severe experience of the symptom reported in either group.

When Under 21 rugby was compared with hockey (Table 4-44, p. 98) the result was approaching significance (p = 0,1189). For Under 21 rugby, only 42,1% reported never experiencing clumsy speech compared with 66,7% of hockey. Amongst Under 21 rugby, 57,9% reported sometimes experiencing clumsy speech compared with only 33,3% of hockey. There was no moderate/severe experience of the symptom reported in either group.

When the rugby forwards were compared with hockey (Table 4-45, p. 100) the result was approaching significance (p = 0,0545). For the rugby forwards, only 38,5% reported never experiencing clumsy speech compared with 66,7% of hockey. Amongst the rugby forwards, 61,5% reported sometimes experiencing clumsy speech compared with only 33,3% of hockey. There was no moderate/severe experience of the symptom reported in either group.

4.1.2.8. Slurred Speech

For this symptom, a result approaching significance was found for Under 21 forwards versus Under 21 backs only (p = 0,1075; Table 4-48, p. 106). For the Under 21 forwards, only 72,7% reported never suffering from slurred speech compared with 100% of the Under 21 backs. Amongst the Under 21 forwards, 27,3% reported sometimes suffering from slurred speech compared with 0% of the Under 21 backs. There was no moderate/severe experience of the symptom reported in either group.

4.1.2.9. Memory

For this symptom, significant results were found on the following comparisons: rugby forwards versus rugby backs; and Springbok forwards versus Springbok backs.

When the rugby forwards were compared with the rugby backs (Table 4-46, p. 102) the result was significant (p = 0,0055). For the rugby forwards, only 50% reported never suffering memory problems compared with 89,5% of the rugby backs. Amongst the rugby forwards, 50% reported sometimes suffering memory problems compared with only 10,5% of the rugby backs. There was no moderate/severe experience of the symptom reported in either group.

When the Springbok forwards were compared with the Springbok backs (Table 4-47, p. 104) the result was significant (p = 0,0080). For the Springbok forwards, only 53,3% reported never suffering memory problems compared with 100% of the Springbok backs. Amongst the Springbok forwards 46,7% reported sometimes suffering memory problems compared with 0% of the Springbok backs. There was no moderate/severe experience of the symptom reported in either group.

4.1.2.10. Attention/Concentration

For this symptom, a significant result was found on the following comparison: Springbok rugby versus hockey. A result approaching significance was found on the following comparison: Springbok rugby versus Under 21 rugby.

When Springbok rugby was compared with hockey (Table 4-43, p. 97) the result was significant (p = 0,0380). For Springbok rugby, 69,2% reported never having problems with attention/concentration compared with only 42,9% of hockey. Amongst Springbok rugby, only 23,1% reported sometimes having problems with attention/concentration compared with 57,1% of hockey. Finally, for Springbok rugby, 7,7% reported often having problems with attention/concentration compared with 0% of hockey.

When Springbok rugby was compared with Under 21 rugby (Table 4-49, p. 109) the result was approaching significance (p = 0,1229). For Springbok rugby, 69,2% reported never having problems with attention/concentration compared with only 42,1% of Under 21 rugby. Amongst Springbok rugby, only 23,1% reported sometimes having problems with attention/concentration compared with 52,6% of Under 21 rugby. Finally, for Springbok rugby, 7,7% reported often having problems with attention/concentration compared with only 5,3% of Under 21 rugby.

4.1.2.11. Sustained Attention

For this symptom, a result approaching significance was found for the Under 21 forwards versus the Under 21 backs only (p = 0,1205; Table 4-48, p. 107). For the Under 21 forwards, only 18,2% reported never having problems with sustained attention compared with 50% of the Under 21 backs. Amongst the Under 21 forwards, 72,7% reported sometimes having problems with sustained attention compared with 25%

of the Under 21 backs. Finally, for the Under 21 forwards, 9,1% reported often having problems with sustained attention compared with 25% of the Under 21 backs.

4.1.2.12. Irritability

For this symptom, results approaching significance were found on the following comparisons: rugby forwards versus rugby backs; and Under 21 forwards versus Under 21 backs.

When the rugby forwards were compared with the rugby backs (Table 4-46, p. 103) the result was approaching significance (p = 0,0592). For the rugby forwards, only 11,5% reported never feeling irritable compared with 42,1% of the rugby backs. Amongst the rugby forwards, 76,9% reported sometimes feeling irritable compared with only 52,6% of the rugby backs. Finally, for the rugby forwards, 11,5% reported often feeling irritable compared with 5,3% of the rugby backs.

When the Under 21 forwards were compared with the Under 21 backs (Table 4-48, p. 107) the result was approaching significance (p = 0,0861). For the Under 21 forwards, 0% reported never feeling irritable compared with 37,5% of the Under 21 backs. Amongst the Under 21 forwards, 81,8% reported sometimes feeling irritable compared with only 50% of the Under 21 backs. Finally, for the Under 21 forwards, 18,2% reported often feeling irritable compared with 12,5% of the Under 21 backs.

4.1.2.13. Easily Angered

For this symptom, a significant result was found on the following comparison: Under 21 forwards versus Under 21 backs. A result approaching significance was found on the following comparison: rugby forwards versus rugby backs.

When the Under 21 forwards were compared with the Under 21 backs (Table 4-48, p. 107) the result was significant (p = 0,0331). For the Under 21 forwards, only 9,1% reported never being easily angered compared with 62,5% of the Under 21 backs. Amongst the Under 21 forwards, 81,8% reported sometimes being easily angered compared with only 25% of the Under 21 backs. Finally, for the Under 21 forwards, 9,1% reported often being easily angered compared with 12,5% of the Under 21 backs.

When the rugby forwards were compared with the rugby backs (Table 4-46, p. 103) the result was approaching significance (p = 0,0501). For the rugby forwards, 23,1% reported never being easily angered compared with 57,9% of the rugby backs. Amongst the rugby forwards, 65,4% reported sometimes being easily angered compared with 31,6% of the rugby backs. Finally, for the rugby forwards, 11,5% reported often being easily angered compared with 10,5% of the rugby backs.

4.1.2.14. Depressed

For this symptom, results approaching significance were found on the following comparisons: rugby forwards versus rugby backs; Springbok forwards versus Springbok backs; and Springbok rugby versus Under 21 rugby.

When the rugby forwards were compared with the rugby backs (Table 4-46, p. 103) the result was approaching significance ($p = 0,0628$). For the rugby forwards, 34,6% reported never feeling depressed compared with 68,4% of the rugby backs. Amongst the rugby forwards, 61,5% reported sometimes feeling depressed compared with only 26,3% of the rugby backs. Finally, for the rugby forwards, 3,8% reported often feeling depressed compared with 5,3% of the rugby backs.

When the Springbok forwards were compared with the Springbok backs (Table 4-47, p. 105) the result was approaching significance ($p = 0,0687$). For the Springbok forwards, only 46,7% reported never feeling depressed compared with 81,8% of the Springbok backs. Amongst the Springbok forwards, 53,3% reported sometimes feeling depressed compared with only 18,2% of the Springbok backs. There was no moderate/severe experience of the symptom reported in either group.

When Springbok rugby was compared with Under 21 rugby (Table 4-49, p. 109) the result was approaching significance ($p = 0,0596$). For Springbok rugby, 61,5% reported never feeling depressed compared with of only 31,6% Under 21 rugby. Amongst Springbok rugby, only 38,5% reported sometimes feeling depressed compared with 57,9% of Under 21 rugby. Finally, for Springbok rugby, 0% reported often feeling depressed compared with 10,5% of Under 21 rugby.

4.1.2.15. Social Contact

For this symptom, significant results were found on the following comparisons: rugby versus hockey; Springbok rugby versus hockey; rugby forwards versus hockey. A result approaching significance was found on the following comparison: Under 21 rugby versus hockey.

When rugby was compared with hockey (Table 4-42, p. 95) the result was significant ($p = 0,0158$). For rugby, 4,4% reported never enjoying social contact compared with 0% of hockey. Amongst rugby, 26,7% reported sometimes enjoying social contact compared with 0% of hockey. Finally, for rugby, only 68,9% reported often enjoying social contact compared with 100% of hockey.

When Springbok rugby was compared with hockey (Table 4-43, p. 97) the result was significant ($p = 0,0059$). For Springbok rugby, 3,8% reported never enjoying social contact compared with 0% of hockey. Amongst Springbok rugby, 34,6% reported sometimes enjoying social contact compared with 0% of hockey. Finally, for Springbok rugby, 61,5% reported often enjoying social contact compared with 100% of hockey.

When the rugby forwards were compared with hockey (Table 4-45, p. 101) the result was significant ($p = 0,0361$). For the rugby forwards, 3,8% reported never enjoying social contact compared with

0% of hockey. Amongst the rugby forwards, 23,1% reported sometimes enjoying social contact compared with 0% of hockey. Finally, for the rugby forwards, only 73,1% reported often enjoying social contact compared with 100% of hockey.

When Under 21 rugby was compared with hockey (Table 4-44, p. 99) the result was approaching significance (p = 0,0858). For Under 21 rugby, 5,3% reported never enjoying social contact compared with 0% of hockey. Amongst Under 21 rugby, 15,8% reported sometimes enjoying social contact compared with 0% of hockey. Finally, for Under 21 rugby, only 78,9% reported often enjoying social contact compared with 100% of hockey.

4.1.2.16. Restlessness

For this symptom, results approaching significance were found on the following comparisons: rugby forwards versus rugby backs; and Springbok forwards versus Springbok backs.

When the rugby forwards were compared with rugby backs (Table 4-46, p. 103) the result was approaching significance (p = 0,0591). For the rugby forwards, only 42,3% reported never feeling restless compared with 73,7% of the rugby backs. Amongst the rugby forwards, 42,3% reported sometimes feeling restless compared with 26,3% of the rugby backs. Finally, for the rugby forwards, 15,4% reported often feeling restless compared with 0% of the rugby backs.

When the Springbok forwards were compared to the Springbok backs (Table 4-47, p. 105) the result was approaching significance (p = 0,1343). For the Springbok forwards, 46,7% reported never feeling restless compared with 81,8% of the Springbok backs. Amongst the Springbok forwards, 33,3% reported sometimes feeling restless compared with 18,2% of the Springbok backs. Finally, for the Springbok forwards, 20% reported often feeling restless compared with 0% of the Springbok backs.

4.1.2.17. Sleep Difficulties

For this symptom, a result approaching significance was found for the rugby forwards versus the rugby backs only (p = 0,1093; Table 4-46, p. 103). For the rugby forwards, 57,7% reported never having difficulty sleeping compared with 68,4% of the rugby backs. Amongst the rugby forwards, 42,3% reported sometimes having difficulty sleeping compared with only 21,1% of the rugby backs. Finally, for the rugby forwards, 0% reported often having difficulty sleeping compared with 10,5% of the rugby backs.

4.1.2.18. Appetite Difficulties

For this symptom, results approaching significance were found on the following comparisons: rugby forwards versus rugby backs; and Springbok forwards versus Springbok backs.

When the rugby forwards were compared with the rugby backs (Table 4-46, p. 103) the result was approaching significance (p = 0,0605). For the rugby forwards, only 73,1% reported never having

appetite difficulties compared with 94,7% of the rugby backs. Amongst the rugby forwards, 26,9% reported sometimes having appetite difficulties compared with only 5,3% of the rugby backs. There was no moderate/severe experience of the symptom reported in either group.

When the Springbok forwards were compared with the Springbok backs (Table 4-47, p. 105) the result was approaching significance (p = 0,0626). For the Springbok forwards, 73,3% reported never having appetite difficulties compared with 100% of the Springbok backs. Amongst the Springbok forwards, 26,7% reported sometimes having appetite difficulties compared with 0% of the Springbok backs. There was no moderate/severe experience of the symptom reported in either group.

4.1.2.19. Anxiety

For this symptom, significant results were found on the following comparisons: rugby forwards versus rugby backs; and Springbok forwards versus the Springbok backs. A result approaching significance was found on the following comparison: rugby forwards versus hockey.

When the rugby forwards were compared with the rugby backs (Table 4-46, p. 103) the result was significant (p = 0,0220). For the rugby forwards, only 23,1% reported never feeling anxious compared with 63,2% of the rugby backs. Amongst the rugby forwards, 73,1% reported sometimes feeling anxious compared with only 36,8% of the rugby backs. Finally, for the rugby forwards, 3,8% reported often feeling anxious compared with 0% of the rugby backs.

When the Springbok forwards were compared with the Springbok backs (Table 4-47, p. 105) the result was significant (p = 0,0247). For the Springbok forwards, only 20% reported never feeling anxious compared with 72,7% of the Springbok backs. Amongst the Springbok forwards, 73,3% reported sometimes feeling anxious compared with only 27,3% of the Springbok backs. Finally, for the Springbok forwards, 6,7% reported often feeling anxious compared with 0% of the Springbok backs.

When the rugby forwards were compared with hockey (Table 4-45, p. 101) the result was approaching significance (p = 0,0914). For the rugby forwards, 23,1% reported never feeling anxious compared with 52,4% of hockey. Amongst the rugby forwards, 73,1% reported sometimes feeling anxious compared with 47,6% of hockey. Finally, for the rugby forwards, 3,8% reported often feeling anxious compared with 0% of hockey.

4.1.2.20. Worry

For this symptom, results approaching significance were found on the following comparisons: rugby forwards versus rugby backs; and Under 21 forwards and Under 21 backs.

When the rugby forwards were compared with the rugby backs (Table 4-46, p. 103) the result was approaching significance (p = 0,0756). For the rugby forwards, only 26,9% reported never feeling worried compared with 57,9% of the rugby backs. Amongst the rugby forwards, 65,4% reported feeling

worried compared with only 42,1% of the rugby backs. Finally, for the rugby forwards, 7,7% reported often feeling worried compared with 0% of the rugby backs.

When the Under 21 forwards were compared with the Under 21 backs (Table 4-48, p. 107) the result was approaching significance (p = 0,1407). For the Under 21 forwards, only 18,2% reported never feeling worried compared with 50% of the Under 21 backs. Amongst the Under 21 forwards, 81,8% reported sometimes feeling worried compared with only 50% of the Under 21 backs. There was no moderate/severe experience of the symptom reported in either group.

4.1.2.21. Argumentative

For this symptom, significant results were found on the following comparisons: rugby forwards versus rugby backs; and Springbok forwards versus Springbok backs.

When the rugby forwards were compared with the rugby backs (Table 4-46, p. 103) the result was significant (p = 0,0399). For the rugby forwards, only 23,1% reported never feeling argumentative compared with 57,9% of the rugby backs. Amongst the rugby forwards, 61,5% reported sometimes feeling argumentative compared with only 26,3% of the rugby backs. Finally, for the rugby forwards, 15,4% reported often feeling argumentative compared with 15,8% of the rugby backs.

When the Springbok forwards were compared with Springbok backs (Table 4-47, p. 105) the result was significant (p = 0,0393). For the Springbok forwards, only 20% reported never feeling argumentative compared with 63,6% of the Springbok backs. Amongst the Springbok forwards, 66,7% reported sometimes feeling argumentative compared with only 18,2% of the Springbok backs. Finally, for the Springbok forwards, 13,3% reported often feeling argumentative compared with 18,2% of the Springbok backs.

4.1.2.22. Short-tempered

For this symptom, results approaching significance were found on the following comparisons: Under 21 rugby versus hockey; rugby forwards versus rugby backs; Springbok rugby versus Under 21 rugby.

When Under 21 rugby was compared with hockey (Table 4-44, p. 99) the result was approaching significance (p = 0,1382). For Under 21 rugby, 52,6% reported never feeling short- tempered compared with 52,4% of hockey. Amongst Under 21 rugby, only 31,6% reported sometimes feeling short-tempered compared with 47,6% of hockey. Finally, for Under 21 rugby, 15,8% reported often feeling short-tempered compared with 0% of hockey.

When the rugby forwards were compared with rugby backs (Table 4-46, p. 103) the result was approaching significance (p = 0,1066). For the rugby forwards, only 42,3% reported never feeling short-tempered compared with 73,7% of the rugby backs. Amongst the rugby forwards, 50% reported sometimes

feeling short-tempered compared with 21,1% of the rugby backs. Finally, for the rugby forwards, 7,7% reported often feeling short-tempered compared with 5,3% of the rugby backs.

When Springbok rugby was compared with Under 21 rugby (Table 4-49, p. 109) the result was approaching significance (p = 0,1059). For Springbok rugby, 57,7% reported never feeling short-tempered compared with 52,6% of Under 21 rugby. Amongst Springbok rugby, 42,3% reported sometimes feeling short-tempered compared with 31,6% of Under 21 rugby. Finally, for Springbok rugby, 0% reported often feeling short-tempered compared with 15,8% of Under 21 rugby.

4.1.2.23. Aggression

For this symptom, significant results were found on the following comparisons: Under 21 rugby versus hockey ; and Springbok rugby versus Under 21 rugby. A result approaching significance was found on the following comparison: rugby forwards versus rugby backs.

When Under 21 rugby was compared with hockey players (Table 4-44, p. 99) the result was significant (p = 0,0491). For Under 21 rugby, only 57,9% reported never feeling aggressive compared with 85,7% of hockey. Amongst Under 21 rugby, 42,1% reported sometimes feeling aggressive compared with only 14,3% of hockey. There was no moderate/severe experience of the symptom reported in either group.

When Springbok rugby was compared to Under 21 rugby (Table 4-49, p. 109) the result was significant (p = 0,0061). For Springbok rugby, 92,3% reported never feeling aggressive compared with only 57,9% of Under 21 rugby. Amongst Springbok rugby, only 7,7% reported sometimes feeling aggressive compared with 42,1% of Under 21 rugby. There was no moderate/severe experience of the symptom reported in either group.

When the rugby forwards were compared with the rugby backs (Table 4-46, p. 103) the result was approaching significance (p = 0,1067). For the rugby forwards, only 69,2% reported never feeling aggressive compared with 89,5% of the rugby backs. Amongst the rugby forwards, 30,8% reported sometimes feeling aggressive compared with only 10,5% of the rugby backs. There was no moderate/severe experience of the symptom reported in either group.

RUGBY versus HOCKEY

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

TEST	RUGBY			HOCKEY			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forwards									
n	29	11	5	13	7	1			
%	64.4	24.4	11.1	61.9	33.3	4.8	1.064	2	0.5874
Digits Backwards									
n	36	5	4	18	2	1			
%	80.0	11.1	8.9	85.7	9.5	4.8	0.413	2	0.8134
Digit Supraspan									
n	37	7	4	17	3	1			
%	75.6	15.6	8.9	81.0	14.3	4.8	0.391	2	0.8224
WMS Associate Learning (Easy) Immed. Recall									
n	41	2	2	20	1	0			
%	91.1	4.4	4.4	95.2	4.8	0.0	0.963	2	0.6179
WMS Associate Learning (Hard) Immed. Recall									
n	38	6	1	21	0	0			
%	84.4	13.3	2.2	100.0	0.0	0.0	3.654	2	0.1609
WMS Associate Learning (Easy) Delayed Recall									
n	45	0	0	21	0	0			
%	100.0	0.0	0.0	0.0	0.0	0.0	*No Statistic*[1]		
WMS Associate Learning (Hard) Delayed Recal									
n	45	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		

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

TEST	RUGBY			HOCKEY			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall									
n	36	4	5	20	0	1			
%	80.0	8.9	11.1	95.2	0.0	4.8	2.893	2	0.2353
WMS Visual Reproduction Immed. Recall									
n	36	3	6	17	3	1			
%	80.0	6.7	13.3	81.0	14.3	4.8	1.908	2	0.3852
WMS Visual Reproduction Delayed Recall									
n	37	7	1	20	1	0			
%	82.2	15.6	2.2	95.2	4.8	0.0	2.124	2	0.3458

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

TEST	RUGBY			HOCKEY			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	20	21	4	14	7	0			
%	44.4	46.7	8.9	66.7	33.3	0.0	3.839	2	0.1467 ~
Structured Verbal Fluency									
n	40	4	1	17	4	0			
%	88.9	8.9	2.2	81.0	19.0	0.0	1.790	2	0.4086

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

Continued overleaf

RUGBY versus HOCKEY (Continued)

Table 4-5. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Deficit.

TEST	RUGBY			HOCKEY			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Digit Symbol Substitution										
n	31	10	4	20	1	0				
%	68.9	22.2	8.9	95.2	4.8	0.0	5.772	2	0.0558	~
Trail Making Test A										
n	38	4	3	17	3	1				
%	84.4	8.9	6.7	81.0	14.3	4.8	0.500	2	0.7789	
Trail Making Test B										
n	34	4	7	20	1	0				
%	75.6	8.9	15.6	95.2	4.8	0.0	4.267	2	0.1185	~

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

TEST	RUGBY			HOCKEY			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Finger Tapping Test I (Preferred Hand)									
n	37	6	1	20	1	0			
%	84.1	13.6	2.3	95.2	4.8	0.0	1.718	2	0.4235
Finger Tapping Test I (Non-preferred Hand)									
n	37	5	2	21	0	0			
%	84.1	11.4	4.5	100.0	0.0	0.0	3.744	2	0.1538
Finger Tapping Test II (Preferred Hand)									
n	43	2	0	21	0	0			
%	95.6	4.4	0.0	100.0	0.0	0.0	0.962	1	0.3266
Finger Tapping Test II (Non-preferred Hand)									
n	44	1	0	21	0	0			
%	97.8	2.2	0.0	100.0	0.0	0.0	0.474	1	0.4912

SPRINGBOK RUGBY versus HOCKEY

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

TEST	SPRINGBOKS			HOCKEY			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forwards									
n	20	6	0	13	7	1			
%	76.9	23.1	0.0	61.9	33.3	4.8	2.053	2	0.3582
Digits Backwards									
n	23	2	1	18	2	1			
%	88.5	7.7	3.8	85.7	9.5	4.8	0.079	2	0.9614
Digit Supraspan									
n	22	3	1	17	3	1			
%	84.6	11.5	3.8	81.0	14.3	4.8	0.110	2	0.9463
WMS Associate Learning (Easy) Immed. Recall									
n	25	0	1	20	1	0			
%	96.2	0.0	3.8	95.2	4.8	0.0	2.047	2	0.3594
WMS Associate Learning (Hard) Immed. Recall									
n	21	4	1	21	0	0			
%	80.8	15.4	3.8	100.0	0.0	0.0	4.519	2	0.1044 ~
WMS Associate Learning (Easy) Delayed Recall									
n	26	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
WMS Associate Learning (Hard) Delayed Recal									
n	26	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		

Table 4-8 . VISUAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	SPRINGBOKS			HOCKEY			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall									
n	18	3	5	20	0	1			
%	69.2	11.5	19.2	95.2	0.0	4.8	5.300	2	0.0707 ~
WMS Visual Reproduction Immed. Recall									
n	22	2	2	17	3	1			
%	84.6	7.7	7.7	81.0	14.3	4.8	0.650	2	0.7226
WMS Visual Reproduction Delayed Recall									
n	20	6	0	20	1	0			
%	76.9	23.1	0.0	95.2	4.8	0.0	3.074	1	0.0795 ~

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

TEST	SPRINGBOKS			HOCKEY			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	16	9	1	14	7	0			
%	61.5	34.6	3.8	66.7	33.3	0.0	0.861	2	0.6501
Structured Verbal Fluency									
n	24	1	1	17	4	0			
%	92.3	3.8	3.8	81.0	19.0	0.0	3.503	2	0.1735

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

Continued overleaf

SPRINGBOK RUGBY versus HOCKEY

Table 4-10. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Deficit.

TEST	SPRINGBOKS			HOCKEY			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Digit Symbol Substitution										
n	20	6	0	20	1	0				
%	76.9	23.1	0.0	95.2	4.8	0.0	3.074	1	0.0795	~
Trail Making Test A										
n	22	3	1	17	3	1				
%	84.6	11.5	3.8	81.0	14.3	4.8	0.110	2	0.9463	
Trail Making Test B										
n	21	2	3	20	1	0				
%	80.8	7.7	11.5	95.2	4.8	0.0	2.858	2	0.2395	

Table 4-11. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Deficit.

TEST	SPRINGBOKS			HOCKEY			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Finger Tapping Test I (Preferred Hand)									
n	23	2	0	20	1	0			
%	92.0	8.0	0.0	95.2	4.8	0.0	0.196	1	0.6577
Finger Tapping Test I (Non-preferred Hand)									
n	25	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
Finger Tapping Test II (Preferred Hand)									
n	26	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
Finger Tapping Test II (Non-preferred Hand)									
n	26	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		

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

UNDER 21 RUGBY versus HOCKEY

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

TEST	UNDER 21			HOCKEY			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Digits Forwards										
n	9	5	5	13	7	1				
%	47.4	26.3	26.3	61.9	33.3	4.8	3.636	2	0.1623	
Digits Backwards										
n	13	3	3	18	2	1				
%	68.4	15.8	15.8	85.7	9.5	4.8	1.911	2	0.3846	
Digit Supraspan										
n	12	4	3	17	3	1				
%	63.2	21.1	15.8	81.0	14.3	4.8	1.910	2	0.3849	
WMS Associate Learning (Easy) Immed. Recall										
n	16	2	1	20	1	0				
%	84.2	10.5	5.3	95.2	4.8	0.0	1.682	2	0.4313	
WMS Associate Learning (Hard) Immed. Recall										
n	17	2	0	21	0	0				
%	89.5	10.5	0.0	100.0	0.0	0.0	2.327	1	0.1272	~
WMS Associate Learning (Easy) Delayed Recall										
n	19	0	0	21	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
WMS Associate Learning (Hard) Delayed Recal										
n	19	0	0	21	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			

Table 4-13 . VISUAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	UNDER 21			HOCKEY			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Digit Symbol Substitution Incidental Recall										
n	18	1	0	20	0	1				
%	94.7	5.3	0.0	95.2	0.0	4.8	2.010	2	0.3660	
WMS Visual Reproduction Immed. Recall										
n	14	1	4	17	3	1				
%	73.7	5.3	21.1	81.0	14.3	4.8	2.998	2	0.2234	
WMS Visual Reproduction Delayed Recall										
n	17	1	1	20	1	0				
%	89.5	5.3	5.3	95.2	4.8	0.0	1.146	2	0.5638	

Table 4-14. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Deficit.

TEST	UNDER 21			HOCKEY			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Unstructured Verbal Fluency										
n	4	12	3	14	7	0				
%	21.1	63.2	15.8	66.7	33.3	0.0	9.796	2	0.0075	*
Structured Verbal Fluency										
n	16	3	0	17	4	0				
%	84.2	15.8	0.0	81.0	19.0	0.0	0.073	1	0.7865	

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

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UNDER 21 RUGBY versus HOCKEY

Table 4-15. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Deficit.

TEST	UNDER 21			HOCKEY			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Digit Symbol Substitution										
n	11	4	4	20	1	0				
%	57.9	21.1	21.1	95.2	4.8	0.0	8.334	2	0.0155	*
Trail Making Test A										
n	16	1	2	17	3	1				
%	84.2	5.3	10.5	81.0	14.3	4.8	1.267	2	0.5308	
Trail Making Test B										
n	13	2	4	20	1	0				
%	68.4	10.5	21.1	95.2	4.8	0.0	5.733	2	0.0569	~

Table 4-16. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Deficit.

TEST	UNDER 21			HOCKEY			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Finger Tapping Test I (Preferred Hand)										
n	14	4	1	20	1	0				
%	73.7	21.1	5.3	95.2	4.8	0.0	3.768	2	0.1520	
Finger Tapping Test I (Non-preferred Hand)										
n	12	5	2	21	0	0				
%	63.2	26.3	10.5	100.0	0.0	0.0	9.378	2	0.0092	*
Finger Tapping Test II (Preferred Hand)										
n	17	2	0	21	0	0				
%	89.5	10.5	0.0	100.0	0.0	0.0	2.327	1	0.1272	~
Finger Tapping Test II (Non-preferred Hand)										
n	18	1	0	21	0	0				
%	94.7	5.3	0.0	100.0	0.0	0.0	1.134	1	0.2870	

RUGBY FORWARDS versus HOCKEY

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

TEST	FORWARDS			HOCKEY			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forwards									
n	17	6	3	13	7	1			
%	65.4	23.1	11.5	61.9	33.3	4.8	1.091	2	0.5796
Digits Backwards									
n	19	3	4	18	2	1			
%	73.1	11.5	15.4	85.7	9.5	4.8	1.512	2	0.4695
Digit Supraspan									
n	20	5	1	17	3	1			
%	76.9	19.2	3.8	81.0	14.3	4.8	0.214	2	0.8986
WMS Associate Learning (Easy) Immed. Recall									
n	22	2	2	20	1	0			
%	84.6	7.7	7.7	95.2	4.8	0.0	1.918	2	0.3832
WMS Associate Learning (Hard) Immed. Recall									
n	21	4	1	21	0	0			
%	80.8	15.4	3.8	100.0	0.0	0.0	4.519	2	0.1044 ~
WMS Associate Learning (Easy) Delayed Recall									
n	26	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
WMS Associate Learning (Hard) Delayed Recal									
n	26	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		

Table 4-18 . VISUAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			HOCKEY			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall									
n	19	3	4	20	0	1			
%	73.1	11.5	15.4	95.2	0.0	4.8	4.343	2	0.1140 ~
WMS Visual Reproduction Immed. Recall									
n	22	2	2	17	3	1			
%	84.6	7.7	7.7	81.0	14.3	4.8	0.650	2	0.7226
WMS Visual Reproduction Delayed Recall									
n	23	3	0	20	1	0			
%	88.5	11.5	0.0	95.2	4.8	0.0	0.685	1	0.4078

Table 4-19. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			HOCKEY			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	9	14	3	14	7	0			
%	34.6	53.8	11.5	66.7	33.3	0.0	5.956	2	0.0509 ~
Structured Verbal Fluency									
n	23	2	1	17	4	0			
%	88.5	7.7	3.8	81.0	19.0	0.0	2.058	2	0.3574

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

Continued overleaf

RUGBY FORWARDS versus HOCKEY

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

TEST	FORWARDS			HOCKEY			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Digit Symbol Substitution										
n	14	9	3	20	1	0				
%	53.8	34.6	11.5	95.2	4.8	0.0	10.041	2	0.0066	*
Trail Making Test A										
n	22	2	2	17	3	1				
%	84.6	7.7	7.7	81.0	14.3	4.8	0.650	2	0.7226	
Trail Making Test B										
n	18	3	5	20	1	0				
%	69.2	11.5	19.2	95.2	4.8	0.0	5.637	2	0.0597	~

Table 4-21. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			HOCKEY			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Finger Tapping Test I (Preferred Hand)										
n	19	5	1	20	1	0				
%	76.0	20.0	4.0	95.2	4.8	0.0	3.370	2	0.1854	
Finger Tapping Test I (Non-preferred Hand)										
n	19	5	1	21	0	0				
%	76.0	20.0	4.0	100.0	0.0	0.0	5.796	2	0.0551	~
Finger Tapping Test II (Preferred Hand)										
n	24	2	0	21	0	0				
%	92.3	7.7	0.0	100.0	0.0	0.0	1.687	1	0.1940	
Finger Tapping Test II (Non-preferred Hand)										
n	25	1	0	21	0	0				
%	96.2	3.8	0.0	100.0	0.0	0.0	0.825	1	0.3636	

RUGBY: FORWARDS versus BACKS

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

TEST	FORWARDS			BACKS			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forwards									
n	17	6	3	12	5	2			
%	65.4	23.1	11.5	63.2	26.3	10.5	0.066	2	0.9677
Digits Backwards									
n	19	3	4	17	2	0			
%	73.1	11.5	15.4	89.5	10.5	0.0	3.302	2	0.1918
Digit Supraspan									
n	20	5	1	14	2	3			
%	76.9	19.2	3.8	73.7	10.5	15.8	2.312	2	0.3148
WMS Associate Learning (Easy) Immed. Recall									
n	22	2	2	19	0	0			
%	84.6	7.7	7.7	100.0	0.0	0.0	3.208	2	0.2011
WMS Associate Learning (Hard) Immed. Recall									
n	21	4	1	17	2	0			
%	80.8	15.4	3.8	89.5	10.5	0.0	1.024	2	0.5994
WMS Associate Learning (Easy) Delayed Recall									
n	26	0	0	19	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
WMS Associate Learning (Hard) Delayed Recal									
n	26	0	0	19	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		

Table 4-23. VISUAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall									
n	19	3	4	17	1	1			
%	73.1	11.5	15.4	89.5	5.3	5.3	1.867	2	0.3931
WMS Visual Reproduction Immed. Recall									
n	22	2	2	14	1	4			
%	84.6	7.7	7.7	73.7	5.3	21.1	1.731	2	0.4209
WMS Visual Reproduction Delayed Recall									
n	23	3	0	14	4	1			
%	88.5	11.5	0.0	73.7	21.1	5.3	2.299	2	0.3168

Table 4-24. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	9	14	3	11	7	1			
%	34.6	53.8	11.5	57.9	36.8	5.3	2.505	2	0.2858
Structured Verbal Fluency									
n	23	2	1	17	2	0			
%	88.5	7.7	3.8	89.5	10.5	0.0	0.831	2	0.6599

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

Continued overleaf

RUGBY: FORWARDS versus BACKS

Table 4-25. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Digit Symbol Substitution										
n	14	9	3	17	1	1				
%	53.8	34.6	11.5	89.5	5.3	5.3	6.765	2	0.0340	*
Trail Making Test A										
n	22	2	2	16	2	1				
%	84.6	7.7	7.7	84.2	10.5	5.3	0.197	2	0.9064	
Trail Making Test B										
n	18	3	5	16	1	2				
%	69.2	11.5	19.2	84.2	5.3	10.5	1.347	2	0.5099	

Table 4-26. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Finger Tapping Test I (Preferred Hand)										
n	19	5	1	18	1	0				
%	76.0	20.0	4.0	94.7	5.3	0.0	2.930	2	0.2311	
Finger Tapping Test I (Non-preferred Hand)										
n	19	5	1	18	0	1				
%	76.0	20.0	4.0	94.7	0.0	5.3	4.289	2	0.1172	~
Finger Tapping Test II (Preferred Hand)										
n	24	2	0	19	0	0				
%	92.3	7.7	0.0	100.0	0.0	0.0	1.530	1	0.2162	
Finger Tapping Test II (Non-preferred Hand)										
n	25	1	0	19	0	0				
%	96.2	3.8	0.0	100.0	0.0	0.0	0.747	1	0.3873	

SPRINGBOKS: FORWARDS versus BACKS

Table 4-27. VERBAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forwards									
n	11	4	0	9	2	0			
%	73.3	26.7	0.0	81.8	18.2	0.0	0.257	1	0.6119
Digits Backwards									
n	12	2	1	11	0	0			
%	80.0	13.3	6.7	100.0	0.0	0.0	2.487	2	0.2884
Digit Supraspan									
n	13	2	0	9	1	1			
%	86.7	13.3	0.0	81.8	9.1	9.1	1.480	2	0.4771
WMS Associate Learning (Easy) Immed. Recall									
n	14	0	1	11	0	0			
%	93.3	0.0	6.7	100.0	0.0	0.0	0.763	1	0.3825
WMS Associate Learning (Hard) Immed. Recall									
n	12	2	1	9	2	0			
%	80.0	13.3	6.7	81.8	18.2	0.0	0.833	2	0.6594
WMS Associate Learning (Easy) Delayed Recall									
n	15	0	0	11	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
WMS Associate Learning (Hard) Delayed Recal									
n	15	0	0	11	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		

Table 4-28. VISUAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Digit Symbol Substitution Incidental Recall										
n	8	3	4	10	0	1				
%	53.3	20.0	26.7	90.9	0.0	9.1	4.514	2	0.1047	~
WMS Visual Reproduction Immed. Recall										
n	13	1	1	9	1	1				
%	86.7	6.7	6.7	81.8	9.1	9.1	0.115	2	0.9443	
WMS Visual Reproduction Delayed Recall										
n	13	2	0	7	4	0				
%	86.7	13.3	0.0	63.6	36.4	0.0	1.896	1	0.1685	

Table 4-29. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Deficit.

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

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

Continued overleaf

SPRINGBOKS: FORWARDS versus BACKS

Table 4-30. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Digit Symbol Substitution										
n	9	6	0	11	0	0				
%	60.0	40.0	0.0	100.0	0.0	0.0	5.720	1	0.0168	*
Trail Making Test A										
n	12	2	1	10	1	0				
%	80.0	13.3	6.7	90.9	9.1	0.0	0.922	2	0.6308	
Trail Making Test B										
n	10	2	3	11	0	0				
%	66.7	13.3	20.0	100.0	0.0	0.0	4.450	2	0.1033	~

Table 4-31. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Deficit.

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

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

UNDER 21: FORWARDS versus BACKS

Table 4-32. VERBAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forward									
n	6	2	3	3	3	2			
%	54.5	18.2	27.3	37.5	37.5	25.0	0.950	2	0.6219
Digits Backward									
n	7	1	3	6	2	0			
%	63.6	9.1	27.3	75.0	25.0	0.0	3.012	2	0.2218
Digit Supraspan									
n	7	3	1	5	1	2			
%	63.6	27.3	9.1	62.5	12.5	25.0	1.223	2	0.5424
WMS Associate Learning (Easy) Immed. Recall									
n	8	2	1	8	0	0			
%	72.7	18.2	9.1	100.0	0.0	0.0	2.951	2	0.2738
WMS Associate Learning (Hard) Immed. Recall									
n	9	2	0	8	0	0			
%	81.8	18.2	0.0	100.0	0.0	0.0	1.626	1	0.2023
WMS Associate Learning (Easy) Delayed Recall									
n	11	0	0	8	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
WMS Associate Learning (Hard) Delayed Recal									
n	11	0	0	8	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		

Table 4-33. VISUAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall									
n	11	0	0	7	1	0			
%	100.0	0.0	0.0	87.5	12.5	0.0	1.451	1	0.2283
WMS Visual Reproduction Immed. Recall									
n	9	1	1	5	0	3			
%	81.8	9.1	9.1	62.5	0	37.5	2.737	2	0.2544
WMS Visual Reproduction Delayed Recall									
n	10	1	0	7	0	1			
%	90.9	9.1	0.0	87.5	0.0	12.5	2.108	2	0.3485

Table 4-34. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	2	7	2	2	5	1			
%	18.2	63.6	18.2	25.0	62.5	12.5	0.198	2	0.9058
Structured Verbal Fluency									
n	10	1	0	6	2	0			
%	90.9	9.1	0	75.0	25.0	0	0.882	1	0.3478

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

Continued overleaf

UNDER 21: FORWARDS versus BACKS

Table 4-35. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution									
n	5	3	3	6	1	1			
%	45.5	27.3	27.3	75.0	12.5	12.5	1.659	2	0.4364
Trail Making Test A									
n	10	0	1	6	1	1			
%	90.9	0	9.1	75.0	12.5	12.5	1.565	2	0.4572
Trail Making Test B									
n	8	1	2	5	1	2			
%	72.7	9.1	18.2	62.5	122.5	25.0	0.224	2	0.8939

Table 4-36. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Deficit.

TEST	FORWARDS			BACKS			x^2	df	p
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Finger Tapping Test I (Preferred Hand)									
n	6	4	1	8	0	0			
%	54.5	36.4	9.1	100.0	0.0	0.0	4.935	2	0.0848 ~
Finger Tapping Test I (Non-preferred Hand)									
n	5	5	1	7	0	1			
%	45.5	45.5	9.1	87.5	0.0	12.5	4.984	2	0.0827 ~
Finger Tapping Test II (Preferred Hand)									
n	9	2	0	8	0	0			
%	81.8	18.2	0.0	100.0	0.0	0.0	1.626	1	0.2023
Finger Tapping Test II (Non-preferred Hand)									
n	10	1	0	8	0	0			
%	90.9	9.1	0.0	100.0	0.0	0.0	0.768	1	0.3809

SPRINGBOK RUGBY versus UNDER 21 RUGBY

Table 4-37. VERBAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	SPRINGBOKS			UNDER 21			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digits Forward									
n	20	6	0	9	5	5			
%	76.9	23.1	0.0	47.4	26.3	26.3	8.377	2	0.0152 *
Digits Backward									
n	23	2	1	13	3	3			
%	88.5	7.7	3.8	68.4	15.8	15.8	2.961	2	0.2276
Digit Supraspan									
n	22	3	1	12	4	3			
%	84.6	11.5	3.8	63.2	21.1	15.8	3.069	2	0.2155
WMS Associate Learning (Easy) Immed. Recall									
n	25	0	1	16	2	1			
%	96.2	0.0	3.8	84.2	10.5	5.3	2.958	2	0.2278
WMS Associate Learning (Hard) Immed. Recall									
n	21	4	1	17	2	0			
%	80.8	15.4	3.8	89.5	10.5	0.0	1.024	2	0.5994
WMS Associate Learning (Easy) Delayed Recall									
n	26	0	0	19	0	0			
%	100.0	0	0	100.0	0	0	*No Statistic*[1]		
WMS Associate Learning (Hard) Delayed Recal									
n	26	0	0	19	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		

Table 4-38. VISUAL MEMORY: Comparison of the Percentage of Subjects with Deficit.

TEST	SPRINGBOKS			UNDER 21			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Digit Symbol Substitution Incidental Recall									
n	18	3	5	18	1	0			
%	69.2	11.5	19.2	94.7	5.3	0.0	5.033	2	0.0807 ~
WMS Visual Reproduction Immed. Recall									
n	22	2	2	14	1	4			
%	84.6	7.7	7.7	73.7	5.3	21.1	1.731	2	0.4209
WMS Visual Reproduction Delayed Recall									
n	20	6	0	17	1	1			
%	76.9	23.1	0.0	89.5	5.3	5.3	3.818	2	0.1482 ~

Table 4-39. VERBAL FLUENCY: Comparison of the Percentage of Subjects with Deficit.

TEST	SPRINGBOKS			UNDER 21			x^2	df	*p*
	None	Mild	Mod/Sev	None	Mild	Mod/Sev			
Unstructured Verbal Fluency									
n	16	9	1	4	12	3			
%	61.5	34.6	3.8	21.1	63.2	15.8	7.727	2	0.0210 *
Structured Verbal Fluency									
n	24	1	1	16	3	0			
%	92.3	3.8	3.8	84.2	15.8	0	2.573	2	0.2762

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

Continued overleaf

SPRINGBOK RUGBY versus UNDER 21 RUGBY

Table 4-40. VISUOPERCEPTUAL TRACKING: Comparison of the Percentage of Subjects with Deficit.

TEST	SPRINGBOKS			UNDER 21			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Digit Symbol Substitution										
n	20	6	0	11	4	4				
%	76.9	23.1	0.0	57.9	21.1	21.1	6.071	2	0.0481	*
Trail Making Test A										
n	22	3	1	16	1	2				
%	84.6	11.5	3.8	84.2	5.3	10.5	1.221	2	0.5430	
Trail Making Test B										
n	21	2	3	13	2	4				
%	80.8	7.7	11.5	68.4	10.5	21.1	0.960	2	0.6189	

Table 4-41. HAND MOTOR DEXTERITY: Comparison of the Percentage of Subjects with Deficit.

TEST	SPRINGBOKS			UNDER 21			x^2	df	p	
	None	Mild	Mod/Sev	None	Mild	Mod/Sev				
Finger Tapping Test I (Preferred Hand)										
n	23	2	0	14	4	1				
%	92.0	8.0	0.0	73.7	21.1	5.3	3.095	2	0.2128	
Finger Tapping Test I (Non-preferred Hand)										
n	25	0	0	12	5	2				
%	100.0	0.0	0.0	63.2	26.3	10.5	10.953	2	0.0042	*
Finger Tapping Test II (Preferred Hand)										
n	26	0	0	17	2	0				
%	100.0	0.0	0.0	89.5	10.5	0.0	2.864	1	0.0906	~
Finger Tapping Test II (Non-preferred Hand)										
n	26	0	0	18	1	0				
%	100.0	0.0	0.0	94.7	5.3	0.0	1.400	1	0.2368	

Postconcussive Symptomology: TOTAL RUGBY versus HOCKEY

Table 4-42. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	RUGBY			HOCKEY			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
1. Headaches										
n	26	19	0	8	12	1				
%	57.8	42.2	0.0	38.1	57.1	4.8	3.898	2	0.1424	~
2. Eyesight										
n	41	2	2	18	2	1				
%	91.1	4.4	4.4	85.7	9.5	4.8	0.659	2	0.7192	
3. Hearing										
n	40	4	1	16	4	1				
%	88.9	8.9	2.2	76.2	19.0	4.8	1.796	2	0.4074	
4. Weakness in Limbs										
n	37	8	0	16	5	0				
%	82.2	17.8	0.0	76.2	23.8	0.0	0.329	1	0.5660	
5. Clumsiness										
n	35	10	0	19	2	0				
%	77.8	22.2	0.0	90.5	9.5	0.0	1.552	1	0.2128	
6. Seizures										
n	45	0	0	21	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
7. Dizziness										
n	28	15	2	14	7	0				
%	62.2	33.3	4.4	66.7	33.3	0.0	0.978	2	0.6133	
8. Fatigue										
n	29	16	0	14	6	1				
%	64.4	35.6	0.0	66.7	28.6	4.8	2.363	2	0.3068	
9. Sensitivity to Noise										
n	30	14	1	19	2	0				
%	66.7	31.1	2.2	90.5	9.5	0.0	4.312	2	0.1158	~
10. Hallucinations										
n	35	9	1	20	1	0				
%	77.8	20.0	2.2	95.2	4.8	0.0	3.185	2	0.2034	
11. Sexual Difficulties										
n	45	0	0	21	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
12. Speech Difficulties										
n	39	6	0	19	2	0				
%	86.7	13.3	0.0	90.5	9.5	0.0	0.195	1	0.6587	
13. Clumsy Speech										
n	21	24	0	14	7	0				
%	46.7	53.3	0.0	66.7	33.3	0.0	2.299	1	0.1294	~
14. Stutter										
n	41	4	0	18	3	0				
%	91.1	8.9	0.0	85.7	14.3	0.0	0.440	1	0.5072	
15. Slurred Speech										
n	40	5	0	20	1	0				
%	88.9	11.1	0.0	95.2	4.8	0.0	0.698	1	0.4033	
16. Memory										
n	30	15	0	14	7	0				
%	66.7	33.3	0.0	66.7	33.3	0.0	0.000	1	1.0000	

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

Continued overleaf

Postconcussive Symptomology: TOTAL RUGBY versus HOCKEY (Continued)

Table 4-42. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire. (continued).

Question	RUGBY			HOCKEY			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
17. Attention/Concentration										
n	26	16	3	9	12	0				
%	57.8	35.6	6.7	42.9	57.1	0.0	3.574	2	0.1675	
18. Sustained Attention										
n	13	28	4	7	13	1				
%	28.9	62.2	8.9	33.3	61.9	4.8	0.415	2	0.8124	
19. Impatience										
n	10	25	10	4	13	4				
%	22.2	55.6	22.2	19.0	61.9	19.0	0.236	2	0.8886	
20. Irritability										
n	11	30	4	6	14	1				
%	24.4	66.7	8.9	28.6	66.7	4.8	0.417	2	0.8120	
21. Easily Angered										
n	17	23	5	8	11	2				
%	37.8	51.1	11.1	38.1	52.4	9.5	0.039	2	0.9807	
22. Depressed										
n	22	21	2	10	11	0				
%	48.9	46.7	4.4	47.6	52.4	0.0	1.035	2	0.5962	
23. Social Contact										
n	2	12	31	0	0	21				
%	4.4	26.7	68.9	0.0	0.0	100.0	8.292	2	0.0158	*
24. Restlessness										
n	25	16	4	9	11	1				
%	55.6	35.6	8.9	42.9	52.4	4.8	1.761	2	0.4146	
25. Sleep Difficulties										
n	28	15	2	11	8	2				
%	62.2	33.3	4.4	52.4	38.1	9.5	0.937	2	0.6258	
26. Appetite Difficulties										
n	37	8	0	18	3	0				
%	82.2	17.8	0.0	85.7	14.3	0.0	0.126	1	0.7229	
27. Anxiety										
n	18	26	1	11	10	0				
%	40.0	57.8	2.2	52.4	47.6	0.0	1.237	2	0.5387	
28. Worry										
n	18	25	2	10	11	0				
%	40.0	55.6	4.4	47.6	52.4	0.0	1.156	2	0.5611	
29. Argumentative										
n	17	21	7	9	10	2				
%	37.8	46.7	15.6	42.9	47.6	9.5	0.479	2	0.7872	
30. Short-tempered										
n	25	17	3	11	10	0				
%	55.6	37.8	6.7	52.4	47.6	0.0	1.765	2	0.4137	
31. Aggression										
n	35	10	0	18	3	0				
%	77.8	22.2	0.0	85.7	14.3	0.0	0.570	1	0.4502	

Postconcussive Symptomology: SPRINGBOK RUGBY versus HOCKEY

Table 4-43. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	SPRINGBOK RUGBY			HOCKEY			x2	df	*p*
	Never	Sometimes	Often	Never	Sometimes	Often			
1. Headaches									
n	17	9	0	8	12	1			
%	65.4	34.6	0.0	38.1	57.1	4.8	4.184	2	0.1234 ~
2. Eyesight									
n	25	0	1	18	2	1			
%	96.2	0.0	3.8	85.7	9.5	4.8	2.637	2	0.2675
3. Hearing									
n	24	2	0	16	4	1			
%	92.3	7.7	0.0	76.2	19.0	4.8	2.766	2	0.2508
4. Weakness in Limbs									
n	24	2	0	16	5	0			
%	92.3	7.7	0.0	76.2	23.8	0.0	2.381	1	0.1228 ~
5. Clumsiness									
n	24	2	0	19	2	0			
%	92.3	7.7	0.0	90.5	9.5	0.0	0.050	1	0.8230
6. Seizures									
n	26	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
7. Dizziness									
n	18	7	1	14	7	0			
%	69.2	26.9	3.8	66.7	33.3	0.0	0.979	2	0.6129
8. Fatigue									
n	22	4	0	14	6	1			
%	84.6	15.4	0.0	66.7	28.6	4.8	2.676	2	0.2624
9. Sensitivity to Noise									
n	19	7	0	19	2	0			
%	73.1	26.9	0.0	90.5	9.5	0.0	2.272	1	0.1318 ~
10. Hallucinations									
n	22	3	1	20	1	0			
%	84.6	11.5	3.8	95.2	4.8	0.0	1.581	2	0.4536
11. Sexual Difficulties									
n	26	0	0	21	0	0			
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]		
12. Speech Difficulties									
n	24	2	0	19	2	0			
%	92.3	7.7	0.0	90.5	9.5	0.0	0.050	1	0.8230
13. Clumsy Speech									
n	13	13	0	14	7	0			
%	50.0	50.0	0.0	66.7	33.3	0.0	1.320	1	0.2506
14. Stutter									
n	23	3	0	18	3	0			
%	88.5	11.5	0.0	85.7	14.3	0.0	0.079	1	0.7790
15. Slurred Speech									
n	24	2	0	20	1	0			
%	92.3	7.7	0.0	95.2	4.8	0.0	0.167	1	0.6828
16. Memory									
n	19	7	0	14	7	0			
%	73.1	26.9	0.0	66.7	33.3	0.0	0.228	1	0.6328

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

Continued overleaf

Postconcussive Symptomology: SPRINGBOK RUGBY versus HOCKEY (Continued)

Table 4-43. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire (continued).

Question	SPRINGBOK RUGBY			HOCKEY			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
17. Attention/Concentration										
n	18	6	2	9	12	0				
%	69.2	23.1	7.7	42.9	57.1	0.0	6.542	2	0.0380	*
18. Sustained Attention										
n	7	18	1	7	13	1				
%	26.9	69.2	3.8	33.3	61.9	4.8	0.278	2	0.8704	
19. Impatience										
n	4	15	7	4	13	4				
%	15.4	57.7	26.9	19.0	61.9	19.0	0.434	2	0.8049	
20. Irritability										
n	8	17	1	6	14	1				
ɔt Responses o	30.8	65.4	3.8	28.6	66.7	4.8	0.045	2	0.9779	
21. Easily Angered										
n	11	12	3	8	11	2				
%	42.3	46.2	11.5	38.1	52.4	9.5	0.187	2	0.9106	
22. Depressed										
n	16	10	0	10	11	0				
%	61.5	38.5	0.0	47.6	52.4	0.0	0.911	1	0.3399	
23. Social Contact										
n	1	9	16	0	0	21				
%	3.8	34.6	61.5	0.0	0.0	100.0	10.260	2	0.0059	*
24. Restlessness										
n	16	7	3	9	11	1				
%	61.5	26.9	11.5	42.9	52.4	4.8	3.355	2	0.1868	
25. Sleep Difficulties										
n	19	6	1	11	8	2				
%	73.1	23.1	3.8	52.4	38.1	9.5	2.246	2	0.3253	
26. Appetite Difficulties										
n	22	4	0	18	3	0				
%	84.6	15.4	0.0	85.7	14.3	0.0	0.011	1	0.9162	
27. Anxiety										
n	11	14	1	11	10	0				
%	42.3	53.8	3.8	52.4	47.6	0.0	1.148	2	0.5633	
28. Worry										
n	12	12	2	10	11	0				
%	46.2	46.2	7.7	47.6	52.4	0.0	1.713	2	0.4247	
29. Argumentative										
n	10	12	4	9	10	2				
%	38.5	46.2	15.4	42.9	47.6	9.5	0.373	2	0.8297	
30. Short-tempered										
n	15	11	0	11	10	0				
%	57.7	42.3	0.0	52.4	47.6	0.0	0.133	1	0.7158	
31. Aggression										
n	24	2	0	18	3	0				
%	92.3	7.7	0.0	85.7	14.3	0.0	0.531	1	0.4661	

Postconcussive Symptomology: UNDER 21 RUGBY versus HOCKEY

Table 4-44. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	UNDER 21 RUGBY			HOCKEY			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
1. Headaches										
n	9	10	0	8	12	1				
%	47.4	52.6	0.0	38.1	57.1	4.8	1.144	2	0.5645	
2. Eyesight										
n	16	2	1	18	2	1				
%	84.2	10.5	5.3	85.7	9.5	4.8	0.018	2	0.9912	
3. Hearing										
n	16	2	1	16	4	1				
%	84.2	10.5	5.3	76.2	19.0	4.8	0.568	2	0.7527	
4. Weakness in Limbs										
n	13	6	0	16	5	0				
%	68.4	31.6	0.0	76.2	23.8	0.0	0.302	1	0.5826	
5. Clumsiness										
n	11	8	0	19	2	0				
%	57.9	42.1	0.0	90.5	9.5	0.0	5.647	1	0.0175	*
6. Seizures										
n	19	0	0	21	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
7. Dizziness										
n	10	8	1	14	7	0				
%	52.6	42.1	5.3	66.7	33.3	0.0	1.637	2	0.4410	
8. Fatigue										
n	7	12	0	14	6	1				
%	36.8	63.2	0.0	66.7	28.6	4.8	5.246	2	0.0726	~
9. Sensitivity to Noise										
n	11	7	1	19	2	0				
%	57.9	36.8	5.3	90.5	9.5	0.0	5.826	2	0.0543	~
10. Hallucinations										
n	13	6	0	20	1	0				
%	68.4	31.6	0.0	95.2	4.8	0.0	4.969	1	0.0258	*
11. Sexual Difficulties										
n	19	0	0	21	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
12. Speech Difficulties										
n	15	4	0	19	2	0				
%	78.9	21.1	0.0	90.5	9.5	0.0	1.040	1	0.3079	
13. Clumsy Speech										
n	8	11	0	14	7	0				
%	42.1	57.9	0.0	66.7	33.3	0.0	2.431	1	0.1189	~
14. Stutter										
n	18	1	0	18	3	0				
%	94.7	5.3	0.0	85.7	14.3	0.0	0.902	1	0.3422	
15. Slurred Speech										
n	16	3	0	20	1	0				
%	84.2	15.8	0.0	95.2	4.8	0.0	1.348	1	0.2457	
16. Memory										
n	11	8	0	14	7	0				
%	57.9	42.1	0.0	66.7	33.3	0.0	0.327	1	0.5671	

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

Continued overleaf

Postconcussive Symptomology: UNDER 21 RUGBY versus HOCKEY (continued)

Table 4-44. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire. (continued).

Question	UNDER 21 RUGBY			HOCKEY			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
17. Attention/Concentration										
n	8	10	1	9	12	0				
%	42.1	52.6	5.3	42.9	57.1	0.0	1.144	2	0.5645	
18. Sustained Attention										
n	6	10	3	7	13	1				
%	31.6	52.6	15.8	33.3	61.9	4.8	1.372	2	0.5037	
19. Impatience										
n	6	10	3	4	13	4				
%	31.6	52.6	15.8	19.0	61.9	19.0	0.836	2	0.6583	
20. Irritability										
n	3	13	3	6	14	1				
%	15.8	68.4	15.8	28.6	66.7	4.8	1.942	2	0.3787	
21. Easily Angered										
n	6	11	2	8	11	2				
%	31.6	57.9	10.5	38.1	52.4	9.5	0.186	2	0.9111	
22. Depressed										
n	6	11	2	10	11	0				
%	31.6	57.9	10.5	47.6	52.4	0.0	2.907	2	0.2337	
23. Social Contact										
n	1	3	15	0	0	21				
%	5.3	15.8	78.9	0.0	0.0	100.0	4.912	2	0.0858	~
24. Restlessness										
n	9	9	1	9	11	1				
%	47.4	47.4	5.3	42.9	52.4	4.8	0.100	2	0.9511	
25. Sleep Difficulties										
n	9	9	1	11	8	2				
%	47.4	47.4	5.3	52.4	38.1	9.5	0.493	2	0.7814	
26. Appetite Difficulties										
n	15	4	0	18	3	0				
%	78.9	21.1	0.0	85.7	14.3	0.0	0.316	1	0.5738	
27. Anxiety										
n	7	12	0	11	10	0				
%	36.8	63.2	0.0	52.4	47.6	0.0	0.973	1	0.3239	
28. Worry										
n	6	13	0	10	11	0				
%	31.6	68.4	0.0	47.6	52.4	0.0	1.069	1	0.3011	
29. Argumentative										
n	7	9	3	9	10	2				
%	36.8	47.4	15.8	42.9	47.6	9.5	0.404	2	0.8172	
30. Short-tempered										
n	10	6	3	11	10	0				
%	52.6	31.6	15.8	52.4	47.6	0.0	3.958	2	0.1382	~
31. Aggression										
n	11	8	0	18	3	0				
%	57.9	42.1	0.0	85.7	14.3	0.0	3.872	1	0.0491	*

Postconcussive Symptomology: RUGBY FORWARDS versus HOCKEY

Table 4-45. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	FORWARDS			HOCKEY			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
1. Headaches										
n	12	14	0	8	12	1				
%	46.2	53.8	0.0	38.1	57.1	4.8	1.438	2	0.4872	
2. Eyesight										
n	24	1	1	18	2	1				
%	92.3	3.8	3.8	85.7	9.5	4.8	0.666	2	0.7167	
3. Hearing										
n	23	3	0	16	4	1				
%	88.5	11.5	0.0	76.2	19.0	4.8	1.889	2	0.3889	
4. Weakness in Limbs										
n	21	5	0	16	5	0				
%	80.8	19.2	0.0	76.2	23.8	0.0	0.145	1	0.703	
5. Clumsiness										
n	20	6	0	19	2	0				
%	76.9	23.1	0.0	90.5	9.5	0.0	1.511	1	0.219	
6. Seizures										
n	26	0	0	21	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
7. Dizziness										
n	14	10	2	14	7	0				
%	53.8	38.5	7.7	66.7	33.3	0.0	2.020	2	0.3642	
8. Fatigue										
n	15	11	0	14	6	1				
%	57.7	42.3	0.0	66.7	28.6	4.8	1.996	2	0.3687	
9. Sensitivity to Noise										
n	17	8	1	19	2	0				
%	65.4	30.8	3.8	90.5	9.5	0.0	4.227	2	0.1208	~
10. Hallucinations										
n	20	6	0	20	1	0				
%	76.9	23.1	0.0	95.2	4.8	0.0	3.074	1	0.0795	~
11. Sexual Difficulties										
n	26	0	0	21	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
12. Speech Difficulties										
n	22	4	0	19	2	0				
%	84.6	15.4	0.0	90.5	9.5	0.0	0.358	1	0.5494	
13. Clumsy Speech										
n	10	16	0	14	7	0				
%	38.5	61.5	0.0	66.7	33.3	0.0	3.698	1	0.0545	~
14. Stutter										
n	23	3	0	18	3	0				
%	88.5	11.5	0.0	85.7	14.3	0.0	0.079	1	0.7790	
15. Slurred Speech										
n	22	4	0	20	1	0				
%	84.6	15.4	0.0	95.2	4.8	0.0	1.379	1	0.2403	
16. Memory										
n	13	13	0	14	7	0				
%	50.0	50.0	0.0	66.7	33.3	0.0	1.320	1	0.2506	

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

Continued overleaf

Postconcussive Symptomology: RUGBY FORWARDS versus HOCKEY (Continued)

Table 4-45. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire. (continued).

Question	FORWARDS			HOCKEY			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
17. Attention/Concentration										
n	13	10	3	9	12	0				
%	50.0	38.5	11.5	42.9	57.1	0.0	3.416	2	0.1812	
18. Sustained Attention										
n	5	19	2	7	13	1				
%	19.2	73.1	7.7	33.3	61.9	4.8	1.274	2	0.5288	
19. Impatience										
n	5	16	5	4	13	4				
%	19.2	61.5	19.2	19.0	61.9	19.0	0.001	2	0.9997	
20. Irritability										
n	3	20	3	6	14	1				
%	11.5	76.9	11.5	28.6	66.7	4.8	2.556	2	0.2786	
21. Easily Angered										
n	6	17	3	8	11	2				
%	23.1	65.4	11.5	38.1	52.4	9.5	1.254	2	0.5343	
22. Depressed										
n	9	16	1	10	11	0				
%	34.6	61.5	3.8	47.6	52.4	0.0	1.463	2	0.4811	
23. Social Contact										
n	1	6	19	0	0	21				
%	3.8	23.1	73.1	0.0	0.0	100.0	6.643	2	0.0361	*
24. Restlessness										
n	11	11	4	9	11	1				
%	42.3	42.3	15.4	42.9	52.4	4.8	1.485	2	0.4759	
25. Sleep Difficulties										
n	15	11	0	11	8	2				
%	57.7	42.3	0.0	52.4	38.1	9.5	2.586	2	0.2744	
26. Appetite Difficulties										
n	19	7	0	18	3	0				
%	73.1	26.9	0.0	85.7	14.3	0.0	1.108	1	0.2926	
27. Anxiety										
n	6	19	1	11	10	0				
%	23.1	73.1	3.8	52.4	47.6	0.0	4.786	2	0.0914	~
28. Worry										
n	7	17	2	10	11	0				
%	26.9	65.4	7.7	47.6	52.4	0.0	3.321	2	0.1901	
29. Argumentative										
n	6	16	4	9	10	2				
%	23.1	61.5	15.4	42.9	47.6	9.5	2.144	2	0.3424	
30. Short-tempered										
n	11	13	2	11	10	0				
%	42.3	50.0	7.7	52.4	47.6	0.0	1.881	2	0.3905	
31. Aggression										
n	18	8	0	18	3	0				
%	69.2	30.8	0.0	85.7	14.3	0.0	1.761	1	0.1845	

Postconcussive Symptomology: RUGBY: FORWARDS versus BACKS

Table 4-46. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	FORWARDS			BACKS			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
1. Headaches										
n	12	14	0	14	5	0				
%	46.2	53.8	0.0	73.7	26.3	0.0	3.411	1	0.0648	~
2. Eyesight										
n	24	1	1	17	1	1				
%	92.3	3.8	3.8	89.5	5.3	5.3	0.109	2	0.9470	
3. Hearing										
n	23	3	0	17	1	1				
%	88.5	11.5	0.0	89.5	5.3	5.3	1.856	2	0.3953	
4. Weakness in Limbs										
n	21	5	0	16	3	0				
%	80.8	19.2	0.0	84.2	15.8	0.0	0.089	1	0.7655	
5. Clumsiness										
n	20	6	0	15	4	0				
%	76.9	23.1	0.0	78.9	21.1	0.0	0.026	1	0.8718	
6. Seizures										
n	26	0	0	19	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
7. Dizziness										
n	14	10	2	14	5	0				
%	53.8	38.5	7.7	73.7	26.3	0.0	2.642	2	0.2669	
8. Fatigue										
n	15	11	0	14	5	0				
%	57.7	42.3	0.0	73.7	26.3	0.0	1.225	1	0.2683	
9. Sensitivity to Noise										
n	17	8	1	13	6	0				
%	65.4	30.8	3.8	68.4	31.6	0.0	0.748	2	0.6879	
10. Hallucinations										
n	20	6	0	15	3	1				
%	76.9	23.1	0.0	78.9	15.8	5.3	1.666	2	0.4348	
11. Sexual Difficulties										
n	26	0	0	19	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
12. Speech Difficulties										
n	22	4	0	17	2	0				
%	84.6	15.4	0.0	89.5	10.5	0.0	0.224	1	0.6358	
13. Clumsy Speech										
n	10	16	0	11	8	0				
%	38.5	61.5	0.0	57.9	42.1	0.0	1.666	1	0.1968	
14. Stutter										
n	23	3	0	18	1	0				
%	88.5	11.5	0.0	94.7	5.3	0.0	0.534	1	0.4650	
15. Slurred Speech										
n	22	4	0	18	1	0				
%	84.6	15.4	0.0	94.7	5.3	0.0	1.139	1	0.2859	
16. Memory										
n	13	13	0	17	2	0				
%	50.0	50.0	0.0	89.5	10.5	0.0	7.697	1	0.0055	*

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

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Table 4-46. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire. (continued).

Question	FORWARDS			BACKS			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
17. Attention/Concentration										
n	13	10	3	13	6	0				
%	50.0	38.5	11.5	68.4	31.6	0.0	2.963	2	0.2250	
18. Sustained Attention										
n	5	19	2	8	9	2				
%	19.2	73.1	7.7	42.1	47.4	10.5	3.254	2	0.1966	
19. Impatience										
n	5	16	5	5	9	5				
%	19.2	61.5	19.2	26.3	47.4	26.3	0.893	2	0.6400	
20. Irritability										
n	3	20	3	8	10	1				
%	11.5	76.9	11.5	42.1	52.6	5.3	5.654	2	0.0592	~
21. Easily Angered										
n	6	17	3	11	6	2				
%	23.1	65.4	11.5	57.9	31.6	10.5	5.987	2	0.0501	~
22. Depressed										
n	9	16	1	13	5	1				
%	34.6	61.5	3.8	68.4	26.3	5.3	5.534	2	0.0628	~
23. Social Contact										
n	1	6	19	1	6	12				
%	3.8	23.1	73.1	5.3	31.6	63.2	0.504	2	0.7773	
24. Restlessness										
n	11	11	4	14	5	0				
%	42.3	42.3	15.4	73.7	26.3	0.0	5.658	2	0.0591	~
25. Sleep Difficulties										
n	15	11	0	13	4	2				
%	57.7	42.3	0.0	68.4	21.1	10.5	4.428	2	0.1093	~
26. Appetite Difficulties										
n	19	7	0	18	1	0				
%	73.1	26.9	0.0	94.7	5.3	0.0	3.523	1	0.0605	~
27. Anxiety										
n	6	19	1	12	7	0				
%	23.1	73.1	3.8	63.2	36.8	0.0	7.634	2	0.0220	*
28. Worry										
n	7	17	2	11	8	0				
%	26.9	65.4	7.7	57.9	42.1	0.0	5.165	2	0.0756	~
29. Argumentative										
n	6	16	4	11	5	3				
%	23.1	61.5	15.4	57.9	26.3	15.8	6.442	2	0.0399	*
30. Short-tempered										
n	11	13	2	14	4	1				
%	42.3	50.0	7.7	73.7	21.1	5.3	4.477	2	0.1066	~
31. Aggression										
n	18	8	0	17	2	0				
%	69.2	30.8	0.0	89.5	10.5	0.0	2.603	1	0.1067	~

Postconcussive Symptomology: SPRINGBOKS: FORWARDS versus BACKS

Table 4-47. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	FORWARDS			BACKS			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
1. Headaches										
n	9	6	0	8	3	0				
%	60.0	40.0	0.0	72.7	27.3	0.0	0.454	1	0.5004	
2. Eyesight										
n	14	0	1	11	0	0				
%	93.3	0.0	6.7	100.0	0.0	0.0	0.763	1	0.3825	
3. Hearing										
n	13	2	0	11	0	0				
:t Responses o	86.7	13.3	0.0	100.0	0.0	0.0	1.589	1	0.2075	
4. Weakness in Limbs										
n	14	1	0	10	1	0				
%	93.3	6.7	0.0	90.9	9.1	0.0	0.053	1	0.8187	
5. Clumsiness										
n	13	2	0	11	0	0				
%	86.7	13.3	0.0	100.0	0.0	0.0	1.589	1	0.2075	
6. Seizures										
n	15	0	0	11	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
7. Dizziness										
n	10	4	1	8	3	0				
%	66.7	26.7	6.7	72.7	27.3	0.0	0.768	2	0.6812	
8. Fatigue										
n	12	3	0	10	1	0				
%	80.0	20.0	0.0	90.9	9.1	0.0	0.580	1	0.4462	
9. Sensitivity to Noise										
n	11	4	0	8	3	0				
%	73.3	26.7	0.0	72.7	27.3	0.0	0.001	1	0.9725	
10. Hallucinations										
n	12	3	0	10	0	1				
%	80.0	20.0	0.0	90.9	0.0	9.1	3.653	2	0.1610	
11. Sexual Difficulties										
n	15	0	0	11	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
12. Speech Difficulties										
n	13	2	0	11	0	0				
%	86.7	13.3	0.0	100.0	0.0	0.0	1.589	1	0.2075	
13. Clumsy Speech										
n	6	9	0	7	4	0				
%	40.0	60.0	0.0	63.6	36.4	0.0	1.418	1	0.2337	
14. Stutter										
n	13	2	0	10	1	0				
%	86.7	13.3	0.0	90.9	9.1	0.0	0.112	1	0.7380	
15. Slurred Speech										
n	14	1	0	10	1	0				
%	93.3	6.7	0.0	90.9	9.1	0.0	0.053	1	0.8187	
16. Memory										
n	8	7	0	11	0	0				
%	53.3	46.7	0.0	100.0	0.0	0.0	7.025	1	0.0080	*

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

Continued overleaf

Table 4-47. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire. (continued).

Question	FORWARDS			BACKS			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
17. Attention/Concentration										
n	9	4	2	9	2	0				
%	60.0	26.7	13.3	81.8	18.2	0.0	2.101	2	0.3498	
18. Sustained Attention										
n	3	11	1	4	7	0				
%	20.0	73.3	6.7	36.4	63.6	0.0	1.451	2	0.4842	
19. Impatience										
n	2	10	3	2	5	4				
%	13.3	66.7	20.0	18.2	45.5	36.4	1.223	2	0.5425	
20. Irritability										
n	3	11	1	5	6	0				
%	20.0	73.3	6.7	45.5	54.5	0.0	2.412	2	0.2993	
21. Easily Angered										
n	5	8	2	6	4	1				
%	33.3	53.3	13.3	54.5	36.4	9.1	1.170	2	0.5571	
22. Depressed										
n	7	8	0	9	2	0				
%	46.7	53.3	0.0	81.8	18.2	0.0	3.313	1	0.0687	~
23. Social Contact										
n	1	3	11	0	6	5				
%	6.7	20.0	73.3	0.0	54.5	45.5	3.723	2	0.1555	
24. Restlessness										
n	7	5	3	9	2	0				
%	46.7	33.3	20.0	81.8	18.2	0.0	4.015	2	0.1343	~
25. Sleep Difficulties										
n	10	5	0	9	1	1				
%	66.7	33.3	0.0	81.8	9.1	9.1	3.179	2	0.2040	
26. Appetite Difficulties										
n	11	4	0	11	0	0				
%	73.3	26.7	0.0	100.0	0.0	0.0	3.467	1	0.0626	~
27. Anxiety										
n	3	11	1	8	3	0				
%	20.0	73.3	6.7	72.7	27.3	0.0	7.404	2	0.0247	*
28. Worry										
n	5	8	2	7	4	0				
%	33.3	53.3	13.3	63.6	36.4	0.0	3.125	2	0.2096	
29. Argumentative										
n	3	10	2	7	2	2				
%	20.0	66.7	13.3	63.6	18.2	18.2	6.471	2	0.0393	*
30. Short-tempered										
n	7	8	0	8	3	0				
%	46.7	53.3	0.0	72.7	27.3	0.0	1.766	1	0.1839	
31. Aggression										
n	13	2	0	11	0	0				
%	86.7	13.3	0.0	100.0	0.0	0.0	1.589	1	0.2075	

Postconcussive Symptomology: UNDER 21 RUGBY: FORWARDS versus BACKS

Table 4-48. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	FORWARDS			BACKS			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
1. Headaches										
n	3	8	0	6	2	0				
%	27.3	72.7	0.0	75.0	25.0	0.0	4.232	1	0.0397	*
2. Eyesight										
n	10	1	0	6	1	1				
%	90.9	9.1	0.0	75.0	12.5	12.5	1.565	2	0.4572	
3. Hearing										
n	10	1	0	6	1	1				
%	90.9	9.1	0.0	75.0	12.5	12.5	1.565	2	0.4572	
4. Weakness in Limbs										
n	7	4	0	6	2	0				
%	63.6	36.4	0.0	75.0	25.0	0.0	0.277	1	0.5988	
5. Clumsiness										
n	7	4	0	4	4	0				
%	63.6	36.4	0.0	50.0	50.0	0.0	0.353	1	0.5522	
6. Seizures										
n	11	0	0	8	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
7. Dizziness										
n	4	6	1	6	2	0				
%	36.4	54.5	9.1	75.0	25.0	0.0	3.001	2	0.2230	
8. Fatigue										
n	3	8	0	4	4	0				
%	27.3	72.7	0.0	50.0	50.0	0.0	1.028	1	0.3106	
9. Sensitivity to Noise										
n	6	4	1	5	3	0				
%	54.5	36.4	9.1	62.5	37.5	0.0	0.780	2	0.6772	
10. Hallucinations										
n	8	3	0	5	3	0				
%	72.7	27.3	0.0	62.5	37.5	0.0	0.224	1	0.6358	
11. Sexual Difficulties										
n	11	0	0	8	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
12. Speech Difficulties										
n	9	2	0	6	2	0				
%	81.8	18.2	0.0	75.0	25.0	0.0	0.130	1	0.7189	
13. Clumsy Speech										
n	4	7	0	4	4	0				
%	36.4	63.6	0.0	50.0	50.0	0.0	0.353	1	0.5522	
14. Stutter										
n	10	1	0	8	0	0				
%	90.9	9.1	0.0	100.0	0.0	0.0	0.768	1	0.3809	
15. Slurred Speech										
n	8	3	0	8	0	0				
%	72.7	27.3	0.0	100.0	0.0	0.0	2.591	1	0.1075	~
16. Memory										
n	5	6	0	6	2	0				
%	45.5	54.5	0.0	75.0	25.0	0.0	1.659	1	0.1978	

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

Continued overleaf

Table 4-48. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire (continued).

Question	FORWARDS			BACKS			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
17. Attention/Concentration										
n	4	6	1	4	4	0				
%	36.4	54.5	9.1	50.0	50.0	0.0	0.950	2	0.6219	
18. Sustained Attention										
n	2	8	1	4	2	2				
%	18.2	72.7	9.1	50.0	25.0	25.0	4.232	2	0.1205	~
19. Impatience										
n	3	6	2	3	4	1				
%	27.3	54.5	18.2	37.5	50.0	12.5	0.266	2	0.8753	
20. Irritability										
n	0	9	2	3	4	1				
%	0.0	81.8	18.2	37.5	50.0	12.5	4.905	2	0.0861	~
21. Easily Angered										
n	1	9	1	5	2	1				
%	9.1	81.8	9.1	62.5	25.0	12.5	6.817	2	0.0331	*
22. Depressed										
n	2	8	1	4	3	1				
%	18.2	72.7	9.1	50.0	37.5	12.5	2.529	2	0.2824	
23. Social Contact										
n	0	3	8	1	0	7				
%	0.0	27.3	72.7	12.5	0.0	87.5	3.685	2	0.1584	
24. Restlessness										
n	4	6	1	5	3	0				
%	36.4	54.5	9.1	62.5	37.5	0.0	1.679	2	0.4319	
25. Sleep Difficulties										
n	5	6	0	4	3	1				
%	45.5	54.5	0.0	50.0	37.5	12.5	1.679	2	0.4319	
26. Appetite Difficulties										
n	8	3	0	7	1	0				
%	72.7	27.3	0.0	87.5	12.5	0.0	0.608	1	0.4355	
27. Anxiety										
n	3	8	0	4	4	0				
%	27.3	72.7	0.0	50.0	50.0	0.0	1.028	1	0.3106	
28. Worry										
n	2	9	0	4	4	0				
%	18.2	81.8	0.0	50.0	50.0	0.0	2.170	1	0.1407	~
29. Argumentative										
n	3	6	2	4	3	1				
%	27.3	54.5	18.2	50.0	37.5	12.5	1.028	2	0.5981	
30. Short-tempered										
n	4	5	2	6	1	1				
%	36.4	45.5	18.2	75.0	12.5	12.5	3.001	2	0.2230	
31. Aggression										
n	5	6	0	6	2	0				
%	45.5	54.5	0.0	75.0	25.0	0.0	1.649	1	0.1978	

Postconcussive Symptomology: SPRINGBOK RUGBY versus UNDER 21 RUGBY

Table 4-49. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire.

Question	SPRINGBOK RUGBY			UNDER 21 RUGBY			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
1. Headaches										
n	17	9	0	9	10	0				
%	65.4	34.6	0.0	47.4	52.6	0.0	1.461	1	0.2268	
2. Eyesight										
n	25	0	1	16	2	1				
%	96.2	0.0	3.8	84.2	10.5	5.3	2.958	2	0.2278	
3. Hearing										
n	24	2	0	16	2	1				
%	92.3	7.7	0.0	84.2	10.5	5.3	1.549	2	0.4610	
4. Weakness in Limbs										
n	24	2	0	13	6	0				
%	92.3	7.7	0.0	68.4	31.6	0.0	4.285	1	0.0384	*
5. Clumsiness										
n	24	2	0	11	8	0				
%	92.3	7.7	0.0	57.9	42.1	0.0	7.522	1	0.0061	*
6. Seizures										
n	26	0	0	26	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
7. Dizziness										
n	18	7	1	10	8	1				
%	69.2	26.9	3.8	52.6	42.1	5.3	1.295	2	0.5234	
8. Fatigue										
n	22	4	0	7	12	0				
%	84.6	15.4	0.0	36.8	63.2	0.0	10.934	1	0.0009	*
9. Sensitivity to Noise										
n	19	7	0	11	7	1				
%	73.1	26.9	0.0	57.9	36.8	5.3	2.095	2	0.3508	
10. Hallucinations										
n	22	3	1	13	6	0				
%	84.6	11.5	3.8	68.4	31.6	0.0	3.305	2	0.1915	
11. Sexual Difficulties										
n	26	0	0	19	0	0				
%	100.0	0.0	0.0	100.0	0.0	0.0	*No Statistic*[1]			
12. Speech Difficulties										
n	24	2	0	15	4	0				
%	92.3	7.7	0.0	78.9	21.1	0.0	1.696	1	0.1928	
13. Clumsy Speech										
n	13	13	0	8	11	0				
%	50.0	50.0	0.0	42.1	57.9	0.0	0.275	1	0.6001	
14. Stutter										
n	23	3	0	18	1	0				
%	88.5	11.5	0.0	94.7	5.3	0.0	0.534	1	0.4650	
15. Slurred Speech										
n	24	2	0	16	3	0				
%	92.3	7.7	0.0	84.2	15.8	0.0	0.729	1	0.3933	
16. Memory										
n	19	7	0	11	8	0				
%	73.1	26.9	0.0	57.9	42.1	0.0	1.139	1	0.2859	

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

Continued overleaf

Table 4-49. Comparison of the Percentage of Subject Responses on the Postconcussive Symptomology Questionnaire. (continued).

Question	SPRINGBOK RUGBY			UNDER 21 RUGBY			x2	df	*p*	
	Never	Sometimes	Often	Never	Sometimes	Often				
17. Attention/Concentration										
n	18	6	2	8	10	1				
%	69.2	23.1	7.7	42.1	52.6	5.3	4.192	2	0.1229	~
18. Sustained Attention										
n	7	18	1	6	10	3				
%	26.9	69.2	3.8	31.6	52.6	15.8	2.330	2	0.3119	
19. Impatience										
n	4	15	7	6	10	3				
%	15.4	57.7	26.9	31.6	52.6	15.8	1.959	2	0.3756	
20. Irritability										
n	8	17	1	3	13	3				
ct Responses o	30.8	65.4	3.8	15.8	68.4	15.8	2.785	2	0.2485	
21. Easily Angered										
n	11	12	3	6	11	2				
%	42.3	46.2	11.5	31.6	57.9	10.5	0.641	2	0.7259	
22. Depressed										
n	16	10	0	6	11	2				
%	61.5	38.5	0.0	31.6	57.9	10.5	5.641	2	0.0596	~
23. Social Contact										
n	1	9	16	1	3	15				
%	3.8	34.6	61.5	5.3	15.8	78.9	1.992	2	0.3694	
24. Restlessness										
n	16	7	3	9	9	1				
%	61.5	26.9	11.5	47.4	47.4	5.3	2.174	2	0.3373	
25. Sleep Difficulties										
n	19	6	1	9	9	1				
%	73.1	23.1	3.8	47.4	47.4	5.3	3.159	2	0.2061	
26. Appetite Difficulties										
n	22	4	0	15	4	0				
%	84.6	15.4	0.0	78.9	21.1	0.0	0.241	1	0.6233	
27. Anxiety										
n	11	14	1	7	12	0				
%	42.3	53.8	3.8	36.8	63.2	0.0	0.977	2	0.6134	
28. Worry										
n	12	12	2	6	13	0				
%	46.2	46.2	7.7	31.6	68.4	0.0	3.024	2	0.2204	
29. Argumentative										
n	10	12	4	7	9	3				
%	38.5	46.2	15.4	36.8	47.4	15.8	0.012	2	0.9939	
30. Short-tempered										
n	15	11	0	10	6	3				
%	57.7	42.3	0.0	52.6	31.6	15.8	4.490	2	0.1059	~
31. Aggression										
n	24	2	0	11	8	0				
%	92.3	7.7	0.0	57.9	42.1	0.0	7.522	1	0.0061	*

CHAPTER 5 : DISCUSSION

The present research formed part of an ongoing research project into the effects of mild concussive and sub-concussive head injuries being conducted by Rhodes University, the South African Rugby Football Union (SARFU) and the South African Sports Science Institute. Phase one of this research involved a comparison of the Springbok rugby players with the Proteas cricket players (cricket's equivalent of the Springboks). Phase two, an extension of this research, expanded the sample of rugby players by including players from the national Under 21 rugby squad, and comparing the results of this larger rugby group with a new non-contact sport control group, national hockey players. Both phases of the research were concerned with investigating the cumulative effect of successive mild head injuries on cognitive performance and self-reported postconcussive symptomology. This was done by comparing the results obtained on a neuropsychological test battery and self-reported postconcussive symptom checklist by players of contact sport and non-contact sport. Further comparisons were made between the two groups of rugby players, the Springboks and the Under 21s, to determine whether there were any differences between these two groups. Finally, comparisons were made between the forwards and the backs amongst the rugby players to determine whether the positional variation found in phase one was replicated in this phase of the research.

The present study, one aspect of phase two of the larger research project, investigated the percentage of individuals with cognitive deficit and the presence of postconcussive symptoms. Specifically for this thesis the following hypotheses were posed, arising out of the literature review (see Chapter 2, p. 4). (i) Since rugby players, compared with hockey players, are exposed to more mild head injuries due to the nature of the game, either as a result of blunt trauma to the head (e.g. from knees or elbows, or from contact with the ground during play) or as a result of whiplash-like injuries (e.g. acceleration/deceleration resulting from tackling), it is expected that rugby players, relative to hockey players, will show higher proportions of deficit on tests sensitive to diffuse brain damage and will report higher proportions of postconcussive symptoms. (ii) Since rugby forwards, compared with rugby backs, are exposed to more collisions and impacts which can result in mild head injuries due to the nature of their role in the game, it is expected that the forwards at both Springbok and Under 21 level will, relative to the hockey players and the backs of both rugby groups, show higher proportions of deficit on tests sensitive to diffuse brain damage and will report higher proportions of postconcussive symptoms. (iii) Finally, since the Springbok rugby players, compared with the Under 21 rugby players, have had more exposure to collisions and impacts which can cause mild head injury, and are also generally heavier, faster and stronger such that their collisions involve greater forces, it is expected that the Springbok rugby players, relative to the Under 21 rugby players, will show higher proportions of deficit on tests sensitive to diffuse brain damage and will report higher proportions of postconcussive symptoms.

In order to test these hypotheses it was considered important to control for the variables of age, education and IQ because these can influence test results. For phase one of the research no significant differences were found between the contact sport group and the non-contact sport group with respect to these variables (age, education and IQ). For phase two of the research significant differences were found between some of the groups involved in the research when compared for age, education and IQ.

With respect to age, there were no significant differences between the forwards and the backs within the total rugby group, within the Springbok rugby group or within the Under 21 rugby group. Nor was there a significant difference between the rugby group as a whole and the hockey players. However, significant differences were found when the rugby players were split into their two groups and compared with the hockey players. When Springbok rugby was compared with hockey, the mean age of the Springbok rugby players (27,46) was older than that of the hockey players (23,24). When the Under 21 rugby players were compared with the hockey players, the mean age of Under 21 rugby (19,74) was younger than that of hockey (23,24). In addition, a significant difference was found when the Springbok rugby players were compared with the Under 21 rugby players, with the mean age of the Springbok rugby players (27,46) older than that of the Under 21 rugby players (19,74). It was, however, expected that the age of the Under 21 rugby players, when compared to the other groups, would result in significant differences because the Under 21 rugby players, by virtue of the age limit for the National Under 21 rugby squad, are younger than the Springbok rugby players and the hockey players, groups which have no age limit. Moreover, none of the age differences between groups were more than eight years whereas normative data is typically stratified in 10 year intervals working on the assumption that dramatic age changes do not occur in a decade.

With respect to education, there were no significant differences between the forwards and the backs within the total rugby group, within the Springbok rugby group or within the Under 21 rugby group. Nor was there a significant difference when the Springbok rugby players were compared with the hockey players. However, significant differences were found when the rugby players were compared with the hockey players with the mean level of education for the rugby players (13,4) being lower than that of the hockey players (14,3). When the Under 21 rugby players were compared with the hockey players, the mean level of education for the Under 21 rugby players (12,32) was lower than that of the hockey players (14,3). When the Springbok rugby players were compared with the Under 21 rugby players, the mean level of education for the Springbok rugby players (14,19) was higher than that of the Under 21 rugby players (12,32). It was, however, expected that the education level of the Under 21 rugby players, when compared to the other groups, would result in significant differences. Because of their age, those Under 21 rugby players who attend tertiary institutions have not yet completed this phase of their education. In addition, because rugby has become a professional sport some players do not continue studying after finishing school, opting instead

for a full time career as a rugby player. This option was not available to the Springbok rugby players until most had already finished their tertiary education. Because hockey is not a professional sport, hockey players cannot make a career solely out of playing the sport and generally continue with their education at tertiary level thus accounting for their higher level of education.

With respect to IQ (an estimated premorbid IQ based on two prorated subtest scores), there were no significant differences between the forwards and the backs within the total rugby group, within the Springbok rugby group or within the Under 21 rugby group. Nor was there a significant difference when the Springbok rugby players were compared with the hockey players. When the rugby players were compared with the hockey players a significant difference was found with the mean IQ of the rugby players (115,42) lower than that of the hockey players (122,0). When the Under 21 rugby players were compared with the hockey players a significant difference was found with the mean IQ for Under 21 rugby (110,26) lower than that of hockey (122,0). When the Springbok rugby players were compared with the Under 21 rugby players a significant difference was found with the mean IQ of the Springbok rugby players (119,19) higher than that of the Under 21 rugby players (110,26). With regard to the difference in mean IQ scores, in a clinical setting a 12 to 15 point difference in IQ scores is considered significant. Thus the 12 point difference in mean IQ scores between Under 21 rugby and hockey (the greatest difference in mean IQ scores) is approaching significance on clinical criteria with the Under 21 rugby mean IQ score within the 'above average' range and the hockey mean IQ scores within the 'superior' range. However, a measure of consistency has still been achieved in that all groups are at least in the above average range such that one would not expect scores to fall significantly below average as a consequence of average or below average IQ.

In the present study, *the differences between the forwards and the backs at both Springbok and Under 21 level are well controlled across age, education and IQ* such that differences in cognition and postconcussive symptoms within the forwards and backs cannot be attributed to these factors. On the other hand, due to the significant differences between some of the other groups on age, education and IQ (as stated above), it cannot be assumed that differences in cognition and postconcussive symptoms cannot be accounted for by these variables. This will be taken into account during the discussion of the results.

The neuropsychological results and the postconcussive symptom results will be discussed in more detail below, followed by the implications and the conclusions which can be drawn from these results.

5.1. NEUROPSYCHOLOGICAL ASSESSMENT

Comparisons which were either significant ($p < 0,05$) or approaching significance ($0,05 < p < 0,15$) will be discussed together for each test where this occurred. In that this analysis was concerned with a comparison

of the percentage of individuals with deficit (as distinct from a comparison of means), for explanatory purposes reference may be made to trends available purely from the descriptive statistics (i.e. the percentage of players with or without deficit). It was considered that to ignore these trends based on individual analysis of deficit as employed in a clinical setting would be to lose important information. When the actual percentage of impairment is utilised in the discussion, the figure will refer to a combination of all impairment present (including mild and moderate/severe impairment) unless otherwise stated.

5.1.1. DIGITS FORWARDS

The only finding on this test that was significant was the comparison between the Springbok rugby players and the Under 21 rugby players where the level of impairment of the Springbok rugby players (23,1%) was lower than that of the Under 21 rugby players (52,6%). It is of note that, in contrast, the comparison between the Springbok rugby players and the Under 21 rugby players on the Digits Backwards was neither significant nor approaching significance.

While Digits Forwards measures short-term memory for verbal material (McFie, 1975; Russell, 1986), Lezak (1995) states that this test is more closely related to the efficiency of attention than to memory. In practice, differentiating between attention, concentration and tracking is difficult as intact attention is an important element of both concentration and tracking. By observing the patients' general behaviour, as well as their performance on tests involving concentration and tracking, the beginnings of a distinction between the more global defects of attention and the more task-specific defects of concentration and tracking can be made (Lezak, 1995). Because of the Digits Forwards' relationship with attention, Lezak (1995) writes that it is not surprising that, in the first months following head trauma, the Digits Forwards span of some patients is likely to fall below normal limits, but that it is likely to return to normal levels during the subsequent years. It can also be reduced as a result of anxiety.

All rugby players were tested pre-season, at least three months after the end of the previous season. Any acute effects from mild head injuries suffered the previous season are likely to have resolved by this stage. In addition, although impaired attention and concentration is a symptom associated with mild head injury (for example, Rimel et al., 1981) the Digits Forwards is not as sensitive to the effects of diffuse brain damage as Digits Backwards and would, therefore, be expected to hold relative to Digits Backwards in the presence of diffuse brain damage (Lezak, 1995). However, there was no difference between the Springbok rugby players and the Under 21 rugby players on the Digits Backwards. Therefore, the relative deficit of the Under 21 rugby players on the Digits Forwards test is unlikely to be as a result of acute trauma.

The influence of age, education and IQ also needs to be considered for this test. If this test were affected by age, education and IQ, it would be expected that there would be equivalent lowering on both the Digits Forwards and the Digits Backwards which did not occur. However, it is possible that the combination of lower age, education and IQ (compared with the other groups) contributed to higher levels of anxiety in the Under 21 group. Thus because Digits Forwards is particularly affected by anxiety, it seems most likely that the difference on the Digits Forwards is due more to anxiety than to damage suffered as a result of mild head injuries.

5.1.2. WMS PAIRED ASSOCIATE LEARNING - HARD (IMMEDIATE)

When compared with the hockey players, who showed 0% impairment, the differences in levels of impairment of three of the rugby groups, the Springbok rugby players (19,2%), the Under 21 rugby players (10,5%), and the rugby forwards (19,2%), were approaching significance. The comparison between the rugby forwards and the rugby backs, while not significant nor approaching significance ($p = 0,5994$), did indicate, on a descriptive level, that the forwards (19,2%) showed greater impairment than the backs (10,5%). At the Under 21 level, all players showing impairment were forwards (18,2%) with the backs showing no impairment (0%). This test has consistently detected impairment in both the Springbok and Under 21 rugby groups.

Because the ability to remember the easy pairs relies primarily on old associate learning, while the hard pairs rely more on new learning ability (Lezak, 1995), the hard pairs are more susceptible to the effects of brain damage. Memory difficulties have been found, both in the acute stages (for example, Conkey, in Richardson, 1990; Gasquoine, 1997) and subacute stages (for example, Levin et al., 1987) following a mild head injury. Memory difficulties have also been found by other researchers studying mild head injuries in players of contact sports (for example, Ancer, 1999; Dickinson, 1998; Matser, 1998; Reid, 1998; Shuttleworth-Jordan et al., 1993; Tysvaer, 1992).

When taken together, the three findings which were approaching significance in the direction of the rugby players, and specifically the rugby forwards, performing poorly, provide strong support that players of contact sport exhibit verbal new learning problems relative to players of non-contact sport and that the position of the participant has some bearing on the deficit suffered. As these deficits were present during a pre-season assessment (approximately three months after the end of the previous season) it would appear likely that this could be evidence of chronic verbal memory deficit amongst a proportion of the players due to mild head injuries suffered in previous seasons. An important result to note on this test is that, in spite of their lower mean IQ score, there was less deficit present amongst the Under 21 rugby players (10,5%) than amongst the Springbok rugby players (19,2%).

5.1.3. SAWAIS DIGIT SYMBOL SUBSTITUTION INCIDENTAL RECALL

Four comparisons were approaching significance on this test. When the Springbok rugby players were compared with the hockey players, 30,7% of the Springbok rugby players were impaired compared with only 4,8% of the hockey players. When the rugby forwards were compared with the hockey players, 26,9% of the rugby forwards were impaired compared with only 4,8% of the hockey players. When the Springbok forwards were compared with the Springbok backs, 46,7% of the Springbok forwards were impaired compared with only 9,1% of the Springbok backs. When the Springbok rugby players were compared with the Under 21 rugby players, 30,7% of the Springbok rugby players were impaired compared with only 5,3% of the Under 21 rugby players. The major finding on this test was the poor performance of the Springbok rugby players, and more specifically, the Springbok forwards. Virtually all the deficit found amongst the rugby group was as a result of the poor performance of the Springbok forwards and resulted in all these results approaching significance. This test was not as consistent as the WMS Associate Learning - Hard (Immediate) test in detecting deficit across all the rugby groups.

This test is known to be susceptible to diffuse brain damage and has shown particularly good discriminating ability for the presence of cognitive deficit (Shuttleworth-Jordan & Bode, 1995). As this test taps recent memory it would suggest that the poor performance of the Springbok forwards could indicate the presence of deficits in visual memory amongst this group when compared with the other groups. This pattern is not replicated at the Under 21 rugby level. The result on this test supports the hypothesis posed earlier (see p. 110) and suggests that at the higher level of the game, it is the players in the more full contact positions who show higher proportions of deficit.

In addition, while the Under 21 rugby players performed significantly poorly compared to the Springbok rugby players on the Digits Forwards, which assesses attention and concentration, their performance on this test showed less deficit than the Springbok rugby players despite their lower mean IQ score. This would tend to lend support to the earlier argument that the poor performance of the Under 21 rugby players on the Digits Forwards was as a result of poor concentration or anxiety in association with lower age, education and IQ, rather than as a result of damage suffered following a mild head injury.

5.1.4. WMS VISUAL REPRODUCTION (DELAYED RECALL)

Two comparisons were approaching significance on this test. When the Springbok rugby players were compared with the hockey players, 23,1% of the Springbok rugby players were impaired compared with only 4,8% of the hockey players. When the Springbok rugby players were compared with the Under 21 rugby players, 23,1% of the Springbok rugby players were impaired compared with only 10,6% of the Under 21 rugby players.

Lezak (1995) reports that this test is sensitive to the effects of head trauma as delayed memory is typically more sensitive to the effects of diffuse brain damage than immediate memory. Stuss et al. (1985) have shown that this test can be used to significantly differentiate patients with mild head trauma from uninjured controls. The results on this test, caused by the poor performance of Springbok rugby, are consistent with the findings on the Digit Symbol Substitution Incidental Recall test. Replication of this deficit across two tests of visual memory supports the presence of diffuse brain damage and visual memory impairment amongst a proportion of the Springbok rugby players. The absence of impairment amongst the Under 21 rugby players on both these tests (Digit Symbol Substitution Incidental Recall test and the WMS Visual Reproduction Delayed Recall test) suggests that this is not a function where impairment is showing up yet at the lower level of the game. This would seem to provide evidence that extended exposure to opportunities for mild head injuries is required for this function to be compromised. Once again the Under 21 rugby players performed better than the Springbok rugby players thus suggesting that a global lowering of test scores is not evident despite their lower mean IQ score.

5.1.5. UNSTRUCTURED VERBAL FLUENCY

For this test, significant results were found on two comparisons. When the Under 21 rugby players were compared with the hockey players, 79% of the Under 21 rugby players were impaired compared with only 33,3% of the hockey players. When the Springbok rugby players were compared with the Under 21 rugby players, only 38,4% of the Springbok rugby players were impaired compared with 79% of the Under 21 rugby players. In addition, results approaching significance were found on two comparisons. When the rugby players were compared with the hockey players, 55,6% of the rugby players were impaired compared with only 33,3% of the hockey players. When the rugby forwards were compared with the hockey players, 65,3% of the rugby forwards were impaired compared with only 33,3% of the hockey players. The deficit present amongst the rugby group is heavily influenced by the poor performance of the Under 21 rugby players.

This test indirectly employs short-term memory in order to keep track of words already used and many patients who have suffered brain injury experience changes in the speed and ease of verbal production (Lezak, 1995). Although word finding difficulties can be a consequence of mild head injury (Lezak, 1995), in the absence of further evidence it is not possible on the basis of this test result alone, to state whether diffuse brain damage as a result of mild head injury has occurred.

Amongst the rugby group as a whole, the rugby forwards performed worse than the rugby backs which was in the direction expected, given their greater exposure to opportunities for mild head injuries. However, the Under 21 rugby players performed worse than the Springbok rugby players, a result which was in the

opposite direction than expected, given the Springbok rugby players' extended exposure to opportunities for mild head injuries. As verbal tests are more educationally loaded, it is possible that this result has been influenced by the lower levels of education amongst the Under 21 rugby players. In terms of Satz's brain reserve capacity theory (Satz, 1993) the Springbok rugby group, with their higher IQ and education levels, have a higher brain reserve capacity than the Under 21 rugby group. This acts as a protective factor, decreasing the risk of functional impairment. In terms of this theory, the Under 21 group, as a result of their lower age, education and IQ, have a lower brain reserve capacity and are, therefore, more vulnerable to showing symptoms of neurological impairment on educationally loaded tests as their threshold is lowered. Conversely, it can be argued that the Springbok rugby players, with their higher levels of education, are not as vulnerable as the Under 21 rugby players on a simple verbal fluency test. It can be hypothesised that a more complex or sensitive verbal test would begin to show the presence of impairment amongst the Springbok rugby group.

5.1.6. SAWAIS DIGIT SYMBOL SUBSTITUTION

For this test, significant results were found on five comparisons. When the Under 21 rugby players were compared with the hockey players, 42,2% of the Under 21 rugby players were impaired compared to only 4,8% of the hockey players. When the rugby forwards were compared with the hockey players, 46,1% of the rugby forwards were impaired compared to only 4,8% of the hockey players. When the rugby forwards were compared with the rugby backs, 46,1% of the rugby forwards were impaired compared to only 10,6% of the rugby backs. When the Springbok forwards were compared with the Springbok backs, 40% of the Springbok forwards were impaired compared with 0% of the Springbok backs. When the Springbok rugby players were compared with the Under 21 rugby players, 23,1% of the Springbok rugby players were impaired compared with 42,2% of the Under 21 rugby players. In addition, results approaching significance were found on two comparisons. When the rugby players were compared with the hockey players, 31,1% of the rugby players were impaired compared with only 4,8% of the hockey players. When the Springbok rugby players were compared to the hockey players, 23,1% of the Springbok rugby players were impaired compared with only 4,8% of the hockey players. Deficit amongst the Springbok rugby players was confined to the forwards (40%) with no backs showing any deficit (0%). The differences on this test appear mainly due to the influence of two main groups, the Springbok forwards and the Under 21 rugby players, with the Under 21 forwards (54,6%) playing a slightly more prominent role than the Under 21 backs (25%).

This is a test of complex visuoperceptual tracking (Lezak, 1995) and requires psychomotor problem solving and visual perceptual abilities (Barth et al., 1989). It is consistently sensitive to brain damage and its score is likely to be depressed even with minimal damage (Lezak, 1995; Russell, 1986). Maddocks et al. (1995) report that this is a most practical test of speed of information processing. Slowed information processing,

following a single mild head injury and multiple mild head injuries, has been found by many researchers (Gronwall & Wrightson, 1974; Gronwall & Wrightson, 1975; Leininger et al., 1990; Levin et al., 1987; Rimel et al., 1981). These results are consistent with studies of American football players (Barth et al., 1989) and Australian rules footballers (Maddocks & Saling, 1991) which have shown that information processing speed, as measured by tests such as the Digit Symbol Substitution test, is sensitive to the effects of mild head injury. However, unlike previous findings, Maddocks et al. (1995) argued that normal levels of performance on this test were found six months post-injury, and suggested that there were no residual effects from earlier head injuries, contrary to Gronwall and Wrightson's (1975) notion of cumulative effect from repeated concussive injury. However, Maddocks et al. (1995) did not make use of a control group in their study and merely compared concussed Australian rules footballers with non-concussed Australian rules footballers. This makes the assumption that the effect of a single head injury is being tested but ignores the possible influence of previous head injuries suffered by players in both the concussed and non-concussed groups, a highly likely scenario given the physical nature of the game.

The literature, the hypotheses posed and the results derived on this test are all in agreement and in the direction anticipated due to the presence of diffuse brain damage in players of contact sport. Thus it would appear that the Digit Symbol Substitution test, as indicated in the literature, is particularly useful in detecting the presence of diffuse brain damage expected in players of contact sports. The results derived on this test are extremely potent as all three rugby groups (total rugby, Springbok rugby and Under 21 rugby) indicate deficit amongst the contact sport players relative to the controls. The forwards at all levels (total rugby, Springbok rugby and Under 21 rugby) indicate deficit amongst the forwards relative to the backs. The only result not significant is the comparison between the Under 21 forwards and the Under 21 backs, although the trend here is in the direction of the forwards worse than the backs. *All the comparisons on this test either indicate deficit amongst contact sport players or positional variation indicating deficit amongst the forwards.* The consistent positional variation (of the forwards worse than the backs) is especially apparent at the Springbok rugby level. The difference between the forwards and backs at the Under 21 level is only a trend while there is a significant difference between the Springbok forwards and the Springbok backs, probably due to the extended exposure of the Springbok rugby players to mild head injuries. Although both the Under 21 rugby players and the Springbok rugby players performed worse than the hockey players, the Under 21 rugby players performed significantly poorly in comparison to the Springbok rugby players. This is surprising given the extended exposure to opportunities for mild head injuries amongst the Springbok rugby players. This can be explained in terms of brain reserve capacity theory (Satz, 1993) which would cause the Under 21 rugby players to be more vulnerable to a test which is known to present a high level of challenge to brain damaged subjects. Hence the Under 21 rugby players decompensated more than the Springbok rugby players because of their lower brain reserve capacity.

5.1.7. TRAIL MAKING TEST B

For this test, results approaching significance were found on four comparisons. When the rugby players were compared with the hockey players, 24,5% of the rugby players were impaired compared with only 4,8% of the hockey players. When the Under 21 rugby players were compared with the hockey players, 31,6% of the Under 21 rugby players were impaired compared with only 4,8% of the hockey players. When the rugby forwards were compared with the hockey players, 30,7% of the rugby forwards were impaired compared with only 4,8% of the hockey players. When the Springbok forwards were compared with the Springbok backs, 33,3% of the Springbok forwards were impaired compared with 0% of the Springbok backs. The statistically significant results on this test would appear to be due to the high levels of impairment found amongst the Under 21 rugby group and the Springbok forwards.

Part B of this test involves complex visuoperceptual tracking, the ability to shift a response set, and taps working memory function. This part of the test requires more information processing ability than Part A (Spreen & Strauss, 1991) and is extremely sensitive to diffuse brain damage (Lezak, 1995; Spreen & Strauss, 1991). As this is a test of visuoperceptual and visuomotor tracking, it involves both motor speed and attention. Part B involves more complex conceptual tracking and requires a greater degree of mental flexibility.

The results suggest, firstly, that contact sport players demonstrate greater proportions of deficit in mental flexibility, divided attention and visuoperceptual tracking than the non-contact sport controls. Secondly, at the highest level of the game, the forwards show greater proportion of deficit than the backs. Findings on this test provide support for the results found on the Digit Symbol Substitution test. The findings of deficit in the rugby players are also consistent with findings in other studies which reported that Trail Making Test times were slower in patients who had suffered a mild head injury than in controls (Leininger et al., 1990; Shuttleworth-Jordan et al., 1993). The findings of this study also agree with those reported by Kaste et al. (1982) on the Trail Making Test which supported the concept of cumulative effects from repeated head injuries. While the difference between the forwards and the backs is not apparent at the Under 21 rugby level, this could be as a result of their youth (mean age: 19,7) and over time, as the Under 21 forwards are exposed to more and more opportunities for mild head injuries, a similar pattern may emerge as found amongst the Springbok rugby players (mean age: 27,5).

5.1.8. FINGER TAPPING TEST

Two trials were performed of the Finger Tapping Test in order to obtain the participants' best score. In terms of the first trial of the preferred hand, the comparison between the Under 21 forwards and the Under 21 backs was the only result approaching significance. Amongst the Under 21 forwards, 45,5% were impaired while

there was no deficit amongst the Under 21 backs. On the first trial of the non-preferred hand significant results were found on two comparisons. When the Under 21 rugby players were compared with the hockey players, 36,8% of the Under 21 rugby players were impaired compared with 0% of the hockey players. When the Springbok Rugby players were compared with the Under 21 rugby players, 0% of the Springbok rugby players were impaired compared with 36,8% of the Under 21 rugby players. In addition, results approaching significance were found on three comparisons. When the rugby forwards were compared with the hockey players, 24% of the rugby forwards were impaired compared with 0% of the hockey players. When the rugby forwards were compared with the rugby backs, 24% of the rugby forwards were impaired compared with 5,3% of the rugby backs. When the Under 21 forwards were compared with the Under 21 backs, 54,6% of the Under 21 forwards were impaired compared with 12,5% of the Under 21 backs. These results were due to the high levels of impairment found amongst the Under 21 rugby players, and more specifically the Under 21 forwards, as neither Springbok rugby nor the hockey players showed any deficit on this trial.

On the second trial of the preferred hand, results approaching significance were found on two comparisons. When the Under 21 rugby players were compared with the hockey players, 10,5% of the Under 21 rugby players were impaired compared with 0% of the hockey players. When the Springbok rugby players were compared with the Under 21 rugby players, 0% of the Springbok rugby players were impaired compared with 10,5% of the Under 21 rugby players. All the deficit amongst the Under 21 rugby players was due to the poor performance of the Under 21 forwards (18,2%), with the Under 21 backs showing no deficit (0%). No comparisons were significant or approaching significance on the second trial of the non-preferred hand.

Lezak (1995) reports that as this is a timed test, bilateral slowing would be an indication of diffuse brain damage. While there is evidence of bilateral slowing on the first trial of this test amongst the Under 21 forwards when compared to the Under 21 backs, by the second trial there was no evidence of this slowing. There is a strong trend for the forwards to have higher levels of impairment than the backs on this test. No deficit is present amongst the rugby backs, the Springbok rugby players and the hockey players on the second trial of this test. In addition, on the first trial of this test the Springbok rugby players and the hockey players show no deficit for the non-preferred hand.

A consistent picture which thus emerges from these trials is the presence of significant deficit on hand-motor function amongst the Under 21 forwards relative to the Under 21 backs. The poor performance of the Under 21 forwards on this test cannot be explained by age, level of education or IQ because there was no significant difference on any of these variables between the forwards and backs. Consequently, these results suggest that while this test can be sensitive to the presence of diffuse brain damage, the Springbok rugby

players, due to the nature of the game, have developed their hand-motor skills enough to compensate for some deficit. Any deficit present amongst the Springbok rugby players is not severe enough to be measured by this test. The Under 21 rugby group, on the other hand, have not played rugby for long enough to have developed sufficient hand-motor skills to compensate for the diffuse brain damage present. Alternatively, another explanation could be that the Under 21 rugby players are more vulnerable to diffuse brain damage because of their lower brain reserve capacity due to their age, education and IQ levels. It is possible that the results on this test are as a result of a combination of these factors.

5.2. POSTCONCUSSIVE SYMPTOM RESULTS

Where symptoms were reported which resulted in comparisons which were either significant ($p < 0,05$) or approaching significance ($0,05 < p < 0,15$) these will be discussed together for each symptom where this occurred. In that this analysis was concerned with a comparison of the percentage of individuals reporting symptoms (as distinct from a comparison of means), on occasion reference will be made to trends available purely from the descriptive statistics (i.e. the percentage of players with or without the symptom). It was considered that to ignore these trends would be to lose important information. If the discussion is elaborated on in terms of the actual percentage of individuals who report a symptom, this figure will include the combined figure for both the categories, 'sometimes' and 'often', unless otherwise stated.

5.2.1. HEADACHES

For this symptom, only one comparison was significant. When the Under 21 forwards were compared with the Under 21 backs, 72,7% of the Under 21 forwards reported experiencing this symptom compared with only 25% of the Under 21 backs. Results approaching significance were found on three comparisons. When the rugby players were compared with the hockey players, 42,2% of the rugby players reported experiencing this symptom compared with 61,9% of the hockey players. When the Springbok rugby players were compared with the hockey players, 34,6% reported experiencing this symptom compared with 61,9% of the hockey players. When the rugby forwards were compared with the rugby backs, 53,8% of the rugby forwards reported experiencing this symptom compared with only 26,3% of the rugby backs. The comparisons between the rugby players and the hockey players and between the Springbok rugby players and the hockey players, which were both approaching significance due to the lower incidence of symptoms reported by the Springbok rugby players, are anomalous results. While it was expected that the comparison between the forwards and the backs would be in the direction of the forwards reporting more headaches, the comparison between the Springbok rugby players and the hockey players, with the hockey players reporting more headaches, was clearly an anomalous result which influenced the comparison between the rugby players and the hockey players in the same direction. The difference between the rugby forwards and the rugby backs,

with the rugby forwards reporting more headaches, is mainly due to the reported difference between the Under 21 forwards and the Under 21 backs.

Headaches have consistently been reported as the most commonly occurring symptom in the acute post-injury phase following mild head injuries in general (Alves et al., 1993; Barth et al., 1989; Levin et al., 1987; Lishman, 1987; McLean et al., 1983; Rimel et al., 1981; Rutherford et al., 1977) and following sports-related mild head injuries (Barnes et al., 1998; Shuttleworth-Jordan et al., 1993). In the present research, the findings amongst the Under 21 rugby players are supported by the overall findings between the rugby forwards and the rugby backs with a consistent trend present amongst all forwards and backs. These results suggest that the Under 21 forwards are exposed to more head injuries than the Under 21 backs, thus accounting for their higher incidence of headaches. While not statistically significant, a similar trend is evident within the Springbok rugby group between the forwards (40%) and the backs (27,3%). The higher proportion of headaches reported by the forwards is in keeping with findings in the literature on mild head injuries in sportsmen which show that forwards suffer more mild head injuries than backs (Alexander et al., 1979; Dickinson, 1998; Gissane et al., 1997; McQuillan, 1992; Stephenson et al., 1996).

However, the anomalous result in this study is that the hockey players (non-contact sport control group) report more headaches than the Springbok rugby players and, as a result of this, the rugby group as a whole. This anomaly is only present at the Springbok rugby level and not at the Under 21 rugby level. Dickinson (1998) reports a similar finding where the cricket players (the non-contact sport controls) reported a higher incidence of headaches than the Springbok rugby players (the contact sport group). The author suggests the possibility that the anomaly was caused by post-season fatigue and stress rather than postconcussive symptomatology because the cricket players, unlike the Springbok rugby players, were assessed post-season. In the current research, however, the non-contact sport controls (hockey players) were assessed pre-season as were all the rugby players. The results of this study suggest, therefore, that Dickinson's explanation may not be completely correct and an alternative explanation needs to be sought for the replication of the anomalous finding that the cricket players and the hockey players report more head injuries than the Springbok rugby players. There are two further explanations which can assist in explaining this anomaly (to be discussed in more detail below) as follows: (1) the fear of possible negative consequences arising as a result of reported symptoms; and (2) elite sportsmen continue to train and play despite the presence of symptomatology.

Firstly, phase one of this research (under which Dickinson's study was conducted) was instituted at the request of SARFU who required brief reports containing individual results for the sports physician of the Sports Science Institute and which formed part of a comprehensive report for SARFU. It is possible,

therefore, that the Springbok rugby players were motivated to under-report any symptomatology in order to avoid possible consequences arising as a result of their self-reported symptomatology which might affect their careers. These reports, however, did not form part of phase two of this research (under which this study was conducted) and it is possible that the Under 21 rugby players, without fear of the consequences, might have reported their symptomatology more accurately.

Secondly, Watson (1993) states that elite athletes have a tendency to tolerate discomfort and continue to exercise under circumstances that would discourage the recreational participant. It is possible that the Springbok rugby players are accustomed to suffering postconcussive symptoms, that the presence of some of these symptoms might almost be 'normal' and, therefore, not considered significant or worth reporting. The Springbok rugby players, the elite level players, who have more at stake in terms of prestige and financial incentives than the Under 21 rugby players, may tend to ignore the presence of any postconcussive symptoms and continue playing the game, whereas the Under 21 rugby players would report the symptom(s) and stop playing the game until it resolves.

These two different, but closely related, reasons must be considered seriously as numerous authors have commented on the under-reporting of symptoms by sportsmen, for a variety of reasons, following a sports-related mild head injury (Anderson, 1996; Barth et al., 1989; MacLeod, 1993; Roy, 1974; Ruchinskas et al., 1997; Sturmi et al., 1990; Van Heerden, 1976; Watson, 1993; Wrightson & Gronwall, 1980). Hence both these reasons provide a very plausible explanation of why the hockey players (the non-contact sport control group) report more headaches than the elite level Springbok rugby players (the contact sport group). These alternative explanations, not explored by Dickinson, all suggest that the cause of the anomaly is that the Springbok rugby players under-reported their true symptomatology.

The under-reporting of symptoms appears to cast doubt on comparisons where the Springbok rugby players reported lower proportions of symptoms than the Under 21 rugby group and/or the hockey group. However, comparisons between the Springbok forwards and the Springbok backs still appear to be valid as the under-reporting of postconcussive symptomatology by the Springbok rugby players appears to be common to both the forwards and the backs within the team. This is not incompatible with the argument that has been provided above, that the Springbok rugby players under-report their symptoms, in that the issue is one of under-reporting, not a total absence of symptoms. It would appear that the Springbok forwards and backs consistently under-reported their symptomatology and the difference between these two subgroups is still in the direction expected, with the Springbok forwards reporting more headaches than the Springbok backs. Dickinson (1998) found that the Springbok forwards, as expected due to their greater exposure to opportunities for mild head injuries, generally reported a higher proportion of headaches than the Springbok

backs. In turn, this is also consistent with findings within the Under 21 rugby group where the forwards report more headaches than the backs.

5.2.2. WEAKNESS IN LIMBS

For this symptom, a significant result was found on the comparison between the Springbok rugby players and the Under 21 rugby players, where only 7,7% of the Springbok rugby players report experiencing this symptom compared with 31,6% of the Under 21 rugby players. A result approaching significance was found on the comparison between the Springbok rugby players and the hockey players, where only 7,7% of the Springbok rugby players report experiencing this symptom compared with 23,8% of the hockey players. As in the case of headaches discussed above, both the Under 21 rugby players and the hockey players report a higher incidence of this symptom than the Springbok rugby players. It is suggested, as discussed in detail above, that this is due to the under-reporting of their symptomatology by the Springbok rugby players. However, of note is the fact that there was no difference between the Under 21 rugby players and the hockey players. This suggests that weakness in limbs is not a persisting symptom amongst the Under 21 rugby players as they were assessed about three months post-season, by which time any acute symptoms should have resolved. This finding supports that of Barth et al. (1989) who report an increase in weakness 24 hours post-injury which resolved over a 10 day period. Further investigation would be required to determine whether the even more pronounced absence of this symptom amongst the Springbok rugby players is due to the acute time-span of the symptom or as a result of under-reporting by the Springbok rugby players.

5.2.3. CLUMSINESS

For this symptom, two significant comparisons were found. When the Under 21 rugby players were compared with the hockey players, 42,1% of the Under 21 rugby players reported experiencing this symptom compared with only 9,5% of the hockey players. When the Springbok rugby players were compared with the Under 21 rugby players, only 7,7% of the Springbok rugby players reported experiencing this symptom compared with 42,1% of the Under 21 rugby players. It is suggested that the higher incidence of symptoms amongst the Under 21 rugby players, when compared with the Springbok rugby players, is due to the under-reporting amongst the latter group as seen above for 'weakness in limbs'. The higher incidence of clumsiness amongst the Under 21 rugby players, when compared to the hockey players, suggests that this is a chronic symptom as any acute symptoms should have resolved by the time of the assessment, about three months post-season. This appears to be one of the first studies to find this although authors have alluded to it. The subjects in the study by Critchley (in Jordan, 1987) did not report 'clumsiness', but did report suffering from transient 'imbalance' (separate from 'dizziness') following a boxing bout. In a study by Kaste et al. (1982), the authors suggested that while none of the subjects reported suffering from clumsiness of movement, as the subjects were young, they were possibly still at risk for this to occur. The Under 21 rugby

players are already reporting a significant incidence of clumsiness in comparison to the non-contact sport controls which suggests, as expected, that they have been exposed to more mild head injuries than the hockey players.

5.2.4. FATIGUE

For this symptom, a significant result was found on the comparison between the Springbok rugby players and the Under 21 rugby players, where only 15,4% of the Springbok rugby players report experiencing this symptom compared with 63,2% of the Under 21 rugby players. A result approaching significance was found on the comparison between the Under 21 rugby players and the hockey players, where 63,2% of the Under 21 rugby players reported experiencing this symptom compared with only 33,4% of the hockey players. Here too the hockey players reported a higher incidence of this symptom than the Springbok rugby players, raising the possibility of under-reporting by the Springbok rugby players as seen above for two other physical symptoms (weakness in limbs and clumsiness). Cumulatively, this fits the picture of reticence on the part of the Springbok rugby players to admit to symptoms which could have an adverse effect on the selectors. On the other hand, the comparison between the Under 21 rugby players and the hockey players indicates that contact sport players (who arguably are reporting more accurately) suffer more fatigue than players of non-contact sports and suggests that the Under 21 rugby players have been exposed to more cumulative mild head injuries than the hockey players. As the assessment was conducted about three months post-season, by which time acute symptoms would likely have resolved, the suggestion is that this is a chronic symptom. The presence of this symptom has been reported following mild head injuries in general (Levin et al., 1987; McLean et al., 1983; Rutherford, 1989; Rutherford et al., 1977). Shuttleworth-Jordan et al. (1993) report that fatigue, present three days post-injury in university level rugby players, had resolved three months post-injury. While the Under 21 rugby players are of a similar age to the players used in the Shuttleworth-Jordan study, symptoms appear to be persisting amongst the Under 21 rugby group but not in the university players. A possible explanation for this difference is that the Under 21 rugby players have been exposed to cumulative mild head injuries at a higher level of the game where the intensity of matches is greater. This appears to support studies which state that the incidence of injury is higher amongst top level players (Cantu, 1995; Nathan et al., 1983; Stephenson et al., 1996).

5.2.5. SENSITIVITY TO NOISE

For this symptom, results approaching significance were found on four comparisons. When the rugby players were compared with the hockey players, 33,3% of the rugby players reported experiencing this symptom compared with only 9,5% of the hockey players. When the Springbok rugby players were compared with the hockey players, 26,9% of the Springbok rugby players reported experiencing this symptom compared with only 9,5% of the hockey players. When the Under 21 rugby players were compared

with the hockey players, 42,1% of the Under 21 rugby players reported experiencing this symptom compared with only 9,5% of the hockey players. When the rugby forwards were compared with the hockey players, 34,6% of the rugby forwards reported experiencing this symptom compared with only 9,5% of the hockey players. The presence of this symptom seems to apply to all rugby players, both forwards and backs, while very few of the non-contact sport control group, the hockey players, report suffering from sensitivity to noise. It would appear that for this symptom, in contrast to the three aforementioned physical symptoms, the Springbok rugby players have not under-reported the incidence of the symptom. The reason for the more accurate reporting of this symptom by Springbok rugby players is unknown. However, two possible explanations are suggested. Firstly, unlike physical symptoms which could be seen to affect a player's performance on the field (such as weakness in limbs, clumsiness and fatigue), this symptom may be viewed as not having much effect on playing performance and the Springbok rugby players therefore reported its presence accurately. Furthermore, it is even possible that the presence of this symptom could be viewed in a positive manner by team management especially in view of the personal experiences of some of the Springbok rugby players. In the past much media attention has focussed on Springbok rugby players who have gone to bars or nightclubs the night before a match, with negative consequences for the players' careers (common public knowledge from media reports over the last five years). Therefore, the reported presence of this symptom could conceivably be seen by team management as a positive sign.

This highly consistent finding, that the contact sport players, particularly the forwards, may report being more sensitive to noise, is supported by the hypothesis and by research into symptoms following mild head injuries in general (Bohnen & Jolles, 1992; Dacey & Dikmen, 1987; McLean et al., 1983; Richardson, 1990) and following sports-related mild head injuries (Shuttleworth-Jordan et al., 1993). While Dacey and Dikmen (1987) and McLean et al. (1983) report that the incidence of this symptom increased between the initial assessment (three days post-injury) and the follow-up assessment (one month post-injury), the possible long-term persistence of this symptom was not assessed. Shuttleworth-Jordan et al. (1993) on the other hand, report that this symptom had resolved one month post-injury. However, subjects for the latter study were only university level rugby players and a possible explanation for the positive findings in this research, i.e. Springbok rugby players and Under 21 rugby players showing chronic sensitivity to noise, is the influence on the symptom presentation amongst the *national* level players of the greater intensity of the game at this level, both in the way the game is played and the number of matches played.

5.2.6. HALLUCINATIONS

For this symptom, a significant result was found on the comparison between the Under 21 rugby players and the hockey players, where 31,6% of the Under 21 rugby players reported experiencing this symptom compared with only 4,8% of the hockey players. A result approaching significance was found on the

comparison between the rugby forwards and the hockey players, where 23,1% of the rugby forwards reported experiencing this symptom compared with only 4,8% of the hockey players.

Although the presence of psychoses following a closed head injury is not common it may occur in patients with no history of psychiatric illness (Richardson, 1990). According to Lishman (1987), the presence of psychiatric symptoms, which can follow even trivial head injuries, appears to be a chronic problem. Schizophrenia-like hallucinoses can occur in which affect is preserved and thought disorder is not intrusive. Levin et al. (1982) report the presence of schizophrenic-like psychosis following a mild head injury during which hallucinations may occur. To the author's knowledge, with respect specifically to mild head injuries, published research indicates that hallucinations are not a commonly reported symptom following a mild head injury but this may be because it has not specifically been researched. Thus the positive finding in the present study is very interesting given the general absence of reference to this symptom in the mild head injury literature. It would seem likely that some people with a mild head injury might suffer prodromal psychotic episodes, as occurs following head injuries in general. Further investigation is needed, therefore, to determine the incidence of this symptom following mild head injury and to substantiate what appears to be a novel finding.

5.2.7. CLUMSY SPEECH AND SLURRED SPEECH

These two symptoms will be discussed together as most authors merely refer to speech difficulties and do not differentiate between clumsy speech and slurred speech. For clumsy speech, results approaching significance were found on three comparisons. When the rugby players were compared with the hockey players, 53,3% of the rugby players reported experiencing this symptom compared with only 33,3% of the hockey players. When the Under 21 rugby players were compared with the hockey players, 57,9% of the Under 21 rugby players reported experiencing this symptom compared with only 33,3% of the hockey players. When the rugby forwards were compared with the hockey players, 61,5% of the rugby forwards reported experiencing this symptom compared with only 33,3% of the hockey players. It would appear that the forwards, at both the Springbok rugby and Under 21 rugby level, are the main contributors to these statistics. For slurred speech, a result approaching significance was found on the comparison between the Under 21 forwards and the Under 21 backs, where 27,3% of the Under 21 forwards reported experiencing this symptom compared with 0% of the Under 21 backs. Although all these results in the area of speech are only approaching significance, taken together across a series of comparisons, all in the expected direction, they gain in the robustness of the indications.

Critchley (in Jordan, 1987) reports that following a bout, boxers may report transient speech difficulties that soon resolve. This finding is supported by the study by Shuttleworth-Jordan et al. (1993) who stated that

speech problems were reported three days post-injury but had resolved one month post-injury. The reported incidence of speech difficulties among the Under 21 rugby players and the rugby forwards corroborate the findings on the Unstructured Verbal Fluency test. Both these groups, in comparison with the hockey players, performed poorly on this test, a test regarded as more sensitive to diffuse brain damage than the Structured Verbal Fluency test. The presence of speech difficulties pre-season, after at least a three month layoff, shows the possibility of chronic diffuse brain damage amongst the Under 21 rugby players, especially amongst the Under 21 forwards, and amongst the rugby forwards as a whole. The corroboration between the reported speech difficulties by the significant objective test findings adds further weight to the series of findings for reported speech difficulties although they were only approaching significance.

5.2.8. MEMORY

For this symptom, significant results were found on two comparisons. When the rugby forwards were compared with the rugby backs, 50% of the rugby forwards reported experiencing this symptom compared with only 10,5% of the rugby backs. When the Springbok forwards were compared with the Springbok backs, 46,7% of the Springbok forwards reported experiencing this symptom compared with 0% of the Springbok backs.

Memory problems have been reported following mild head injuries in general (Dacey et al., 1989; Levin et al., 1987; McLean et al., 1983; Rutherford et al., 1977; Rutherford, 1989) and following mild head injuries to sportsmen (Barth et al., 1989; Critchley, in Jordan, 1987; Macciocchi et al., 1996). Critchley (in Jordan, 1987) reports that following a bout, boxers may report transient memory difficulties which soon resolve. This finding is supported by the study by Barth et al. (1989) which found a considerable increase in reported memory problems 24 hours post-injury but this symptom had returned to pre-season rates by 10 days post-injury. The follow-up study by Macciocchi et al. (1996) also found a significant increase in reported memory problems 24 hours post-injury. However, while the authors report that most symptoms had resolved by 10 days post-injury, there was actually a slight increase in self-reported memory problems at this time. Unfortunately, no further follow-up was conducted to determine the persistence of this trend.

There is very clear differentiation between the Springbok forwards and the Springbok backs on this test which suggests the presence of diffuse brain damage in almost half the Springbok forwards. The self-reported incidence of memory difficulties amongst the Springbok forwards provides extremely strong reciprocal corroboration for the objective findings on three of the objective memory tests (WMS Associate Learning Hard - Immediate, Digit Symbol Substitution Incidental Recall test and WMS Visual Reproduction test). In contrast, the Under 21 rugby players do not report significant memory problems and only show deficit on one objective memory test (WMS Associate Learning Hard - Immediate). It is suggested that this is because

the Under 21 rugby players have not been as exposed to opportunities for mild head injuries as the Springbok rugby players. The presence of memory difficulties pre-season, after at least a three month layoff, suggests the possibility of chronic diffuse brain damage amongst the Springbok forwards.

5.2.9. ATTENTION/CONCENTRATION AND SUSTAINED ATTENTION

These symptoms will be discussed together as most authors do not differentiate between attention and concentration, and sustained attention. For attention and concentration, a significant result was found on the comparison between the Springbok rugby players and the hockey players, where only 30,8% of Springbok rugby players reported experiencing this symptom compared with 57,1% of the hockey players. A result approaching significance was found on the comparison between the Springbok rugby players and the Under 21 rugby players, where only 30,8% of the Springbok rugby players reported experiencing this symptom compared with 57,9% of the Under 21 rugby players. Again it is possible that the Springbok rugby players under-reported their postconcussive symptomatology, although it is less clear why this should be so given that memory problems are not under-reported. However, players may have conceptualised these as two separate entities, possibly because the presence of attention/concentration problems, unlike memory problems, could be seen to affect match performance.

For sustained attention, a result approaching significance was found on the comparison between the Under 21 forwards and the Under 21 backs, where 79,8% of the Under 21 forwards reported experiencing this symptom compared with only 50% of the Under 21 backs. This result supports the results of those objective tests requiring a degree of sustained attention and freedom from distractibility such as the Digits Forwards, the Digit Symbol Substitution test and the Trail Making Test Part B.

Attention and concentration problems have been reported following mild head injuries in general (Dacey et al., 1989; Levin et al., 1987; McLean et al., 1983; Rutherford et al., 1977; Rutherford, 1989) and following mild head injuries to sportsmen (Shuttleworth-Jordan et al., 1993). In the study by Shuttleworth-Jordan et al. (1993), problems with attention and concentration were present three days post-injury but had resolved by the three month follow-up. The presence of attention difficulties amongst the Under 21 rugby forwards, more than three months post-season, by which time any acute effects of mild head injuries should have resolved, suggests that this is a chronic symptom. The trend here amongst the Under 21 forwards supports the hypothesis that forwards, because of greater exposure to opportunities for mild head injuries, should report a higher incidence of problems with sustained attention.

5.2.10. IRRITABILITY, EASILY ANGERED, ARGUMENTATIVE, SHORT-TEMPERED, AGGRESSION

These symptoms are grouped together as they are all aspects involved in 'lowered frustration tolerance'. For **irritability**, results approaching significance were found on two comparisons. When the rugby forwards were compared with the rugby backs, 88,4% of the rugby forwards reported experiencing this symptom compared with only 57,9% of the rugby backs. When the Under 21 forwards were compared with the Under 21 backs, 100% of the Under 21 forwards reported experiencing this symptom compared with only 62,5% of the Under 21 backs. For **easily angered**, the same trend was evident. A significant result was found on the comparison between the Under 21 forwards and the Under 21 backs, where 90,9% of the Under 21 forwards reported experiencing this symptom compared with only 37,5% of the Under 21 backs. A result approaching significance was found on the comparison between the rugby forwards and the rugby backs, where 76,9% of the rugby forwards reported experiencing this symptom compared with only 42,1% of the rugby backs. For **argumentative**, significant results were found on two comparisons. When the rugby forwards were compared with the rugby backs, 76,9% of the rugby forwards reported experiencing this symptom compared with 42,1% of the rugby backs. When the Springbok forwards were compared with the Springbok backs, 80% of the Springbok forwards reported experiencing this symptom compared with only 36,4% of the Springbok backs.

For the symptom of being **short-tempered**, results approaching significance were found on three comparisons. When the Under 21 rugby players were compared with the hockey players, 15,8% of the Under 21 rugby players reported *often* experiencing this symptom compared with 0% of the hockey players. When the rugby forwards were compared with the rugby backs, 57,7% of the rugby forwards reported experiencing this symptom compared with only 26,4% of the rugby backs. When the Springbok rugby players were compared with the Under 21 rugby players, 42,3% of the Springbok rugby players reported *sometimes* experiencing this symptom compared with the Under 21 rugby players, where 31,6% reported *sometimes* and 15,8% reported *often* experiencing this symptom. For **aggression**, significant results were found on two comparisons. When the Under 21 rugby players were compared with the hockey players, 42,1% of the Under 21 rugby players reported experiencing this symptom compared with only 14,3% of the hockey players. When the Springbok rugby players were compared with the Under 21 rugby players, only 7,7% of the Springbok rugby players reported experiencing this symptom compared with 42,1% of the Under 21 rugby players. A result approaching significance was found on the comparison between the rugby forwards and the rugby backs, where 30,8% of the rugby forwards reported experiencing this symptom compared with only 10,5% of the rugby backs.

This cluster of symptoms (irritability, easily angered, argumentative, short-tempered, aggression) has been reported in the literature following mild head injuries in general (McLean et al., 1983; Rutherford et al., 1977; Rutherford, 1989) and following mild head injuries in sport (Critchley, in Jordan, 1987; Dickinson, 1998). The strongest trend to emerge from these results is the tendency of the forwards, at both Springbok and Under 21 level, to report a higher proportion of these symptoms than the backs. It is a strong indication that the forwards have lowered frustration tolerance in comparison with the backs, which is such a classic symptom of frontal lobe damage following head injury (Lezak, 1995). This supports the hypothesis that players involved with more physical contact are sustaining more mild head injuries and hence are reporting a higher proportion of postconcussive symptoms. It could be argued that the nature of the forward position, involving as it does more physical contact, would attract the naturally more aggressive personality type. In addition, it can be argued that players may be socialised into more aggressive behaviour by the very game itself. However, players are usually allocated to positions on the basis of physical characteristics and not personality traits. Generally, the heavier, taller, slower players are more suited to forward positions where physical size is a requirement, while the lighter, faster, more agile players are more suited to back positions where speed and agility are required most. However, the influence of personality traits, whether pre-existing or as a result of socialisation, cannot be ignored. Future research would be needed to investigate whether the trend seen here is due to postconcussion symptoms, personality traits, or a combination of both of these.

5.2.11. DEPRESSED

For this symptom, results approaching significance were found on three comparisons. When the rugby forwards were compared with the rugby backs, 65,3% of the rugby forwards reported the presence of this symptom compared with only 31,6% of the rugby backs. When the Springbok forwards were compared with the Springbok backs, 53,3% of the Springbok forwards reported the presence of this symptom compared with only 18,2% of the Springbok backs. When the Springbok rugby players were compared with the Under 21 rugby players, only 38,5% of the Springbok rugby players reported the presence of this symptom compared with 68,4% of the Under 21 rugby players. Once again the Springbok rugby players appear to have under-reported their true symptomatology due to possible negative consequences. Despite this, there is a consistent pattern present amongst the rugby forwards and backs, and the Springbok forwards and backs, with the forwards reporting a higher incidence of depression than the backs. Both these results are approaching significance in the expected direction which adds to the robustness of this trend.

This symptom is often associated with the sequelae of mild head injuries in general (Dacey et al., 1989; Levin et al., 1982; Rutherford et al., 1977; Rutherford, 1989) and mild head injuries in sport (Dickinson, 1998). There is a strong trend for the forwards to report a higher incidence of depression than the backs, especially at the Springbok level, and it is clear that this symptom is more prevalent amongst those players

in the more full contact positions. However, with a symptom such as depression, it is not possible to say whether it is as a direct result of a mild head injury or as a secondary result of the presence of neurocognitive deficit and/or other postconcussive symptoms. A number of authors have suggested that postconcussive symptoms begin on an organic basis but persist on a psychological basis (Binder, 1986; Levin et al., 1982; Lishman, 1987). This is particularly relevant to a symptom such as depression. It is possible that the Springbok rugby players are becoming aware of their memory problems and this may be interacting with their psychological make-up, resulting in the presence of symptoms related to depression such as lowered frustration tolerance.

5.2.12. SOCIAL CONTACT

Three comparisons were significant for this symptom, which asked subjects to rate the frequency, never, sometimes, or often, with which they *enjoy* having social contact. While 100% of the hockey players reported often *enjoying* social contact, 31,1% of the rugby players reported never or only sometimes *enjoying* social contact, 38,4% of the Springbok rugby players reported never or only sometimes *enjoying* social contact, and 26,9% of the rugby forwards reported never or only sometimes *enjoying* social contact. A result approaching significance was found when the Under 21 rugby players were compared with the hockey players, where 21,1% of the Under 21 rugby players reported never or only sometimes *enjoying* social contact compared with 100% of the hockey players who reported often *enjoying* social contact. There is a strong trend here for contact sport players, at both the Springbok and Under 21 level, towards never or only sometimes *enjoying* social contact while all the non-contact sport players report often *enjoying* social contact.

Lezak (1995) states that diffuse brain damage tends to compromise mental speed, attentional functions and cognitive efficiency. Following a mild head injury many patients are acutely aware of their mental inefficiency and this realisation may cause these patients to avoid stressful (i.e. highly stimulating) situations - such as cocktail parties, the local pub and shopping malls. The result is that these patients become more socially withdrawn (Lezak,1995). The results on the self-report questionnaire strongly suggest that the rugby forwards and the Under 21 rugby players are aware of their mental inefficiency in memory and attention and concentration respectively, which supports Lezak's point of view. In addition, it is possible that the lack of enjoyment of social contact is influenced by the presence of some of the other postconcussive symptoms, especially those such as depression, sensitivity to noise, clumsy speech and lowered frustration tolerance, which might interfere with the contact sport players' ability to enjoy social contact.

5.2.13. RESTLESSNESS

For restlessness, results approaching significance were found on two comparisons. When the rugby forwards were compared with the rugby backs, 57,7% of the rugby forwards reported the presence of this symptom compared with only 26,3% of the rugby backs. When the Springbok forwards were compared with the Springbok backs, 53,3% of the Springbok forwards reported the presence of this symptom compared with only 18,2% of the Springbok backs. A strong trend is present between the rugby forwards and the rugby backs with the rugby forwards reporting a higher incidence of restlessness. This same trend is evident, and in the same direction, amongst the Springbok forwards and the Springbok backs.

The presence of this symptom is not, to the author's knowledge, reported following mild head injuries in general but has been reported following mild head injuries in sport. Shuttleworth-Jordan et al. (1993) report that this symptom was present three days post-trauma but had resolved by the three month follow-up. However, the significant reporting of this symptom in the present study (approximately three months following the end of the season) suggests that it is a chronic symptom, unlike that found by Shuttleworth-Jordan et al. (1993). This is probably as a result of extended exposure to mild head injuries at a higher level of the game than the subjects used by Shuttleworth-Jordan (university level rugby players). The strong trend for the rugby forwards to report a higher incidence of this symptom than the rugby backs, especially at the Springbok rugby level, suggests that in this study, positional variation has a strong influence on the presence of this symptom. Those players exposed to greater opportunities for mild head injuries, the forwards, report a higher incidence of this symptom. Feelings of restlessness amongst these players may be linked to the subjective reports of depression discussed above. There is no positional variation in the incidence of this symptom at the Under 21 rugby level which suggests that the extended exposure to opportunities for mild head injuries has an effect on this symptom.

5.2.14. SLEEP DIFFICULTIES

For sleep difficulties, a result approaching significance was found when the rugby forwards were compared with the rugby backs. Of the rugby forwards, 42,3% reported the presence of this symptom compared with only 31,6% of the rugby backs.

Insomnia has been reported following mild head injuries in general (McLean et al., 1983; Rutherford, 1989; Rutherford et al., 1977) and following mild head injuries in sport (Barnes et al., 1998; Shuttleworth-Jordan et al., 1983). Shuttleworth-Jordan et al. (1993) report that this symptom was present three days post-injury but had resolved by the one month follow-up. There is a general trend amongst the forwards and the backs for the forwards to report higher proportions of this symptom than the backs. This suggests some positional variation due to the more full contact role played by the forwards. Difficulty sleeping reported by these

players may be linked to the subjective reports of depression amongst the rugby forwards. In addition, the presence of this symptom more than three months post-season suggests that it is a chronic symptom, unlike that found by Shuttleworth-Jordan et al. (1993). It is likely that this is a result of extended exposure to mild head injuries at a higher level of the game than the subjects used by Shuttleworth-Jordan (university level rugby players).

5.2.15. APPETITE DIFFICULTIES

For appetite difficulties, results approaching significance were found on two comparisons. When the rugby forwards were compared with the rugby backs, 26,9% of the rugby forwards reported the presence of this symptom compared with only 5,3% of the rugby backs. When the Springbok forwards were compared with the Springbok backs, 26,7% of the Springbok forwards reported the presence of this symptom compared with 0% of the Springbok backs.

The presence of this symptom is not, to the author's knowledge, reported following mild head injuries in general. However, Shuttleworth-Jordan et al. (1993) report the presence of this symptom following mild head injury in sport. The authors report that this symptom was present three days post-injury but had resolved by the one month follow-up. The presence of this symptom more than three months post-season in this study suggests that it is a chronic symptom, unlike that found by Shuttleworth-Jordan et al. (1993). This is probably due to these players' extended, and very intensive, exposure to opportunities for mild head injuries. In addition, this provides additional support for the studies which have shown that more head injuries occur at the higher level of the game (Cantu, 1995; Nathan et al., 1983; Stephenson et al., 1996). Strong positional variation is evident amongst the forwards and the backs, with the forwards reporting higher proportions of this symptom than the backs, as expected given their more full contact role in the game. The presence of self-reported appetite difficulty may be related to the self-reported experience of depression, and all the symptoms associated with depression, amongst the rugby forwards and the Springbok forwards.

5.2.16. ANXIETY AND WORRY

For anxiety, significant results were found on two comparisons. When the rugby forwards were compared with the rugby backs, 76,9% of the rugby forwards reported the presence of this symptom compared with only 36,8% of the rugby backs. When the Springbok forwards were compared with the Springbok Backs, 80% of the Springbok forwards reported the presence of this symptom compared with only 27,3% of the Springbok backs. A result approaching significance was found on the comparison between the rugby forwards and the hockey players, where 76,9% of the rugby forwards reported the presence of this symptom compared with only 47,6% of the hockey players.

For worry, results approaching significance were found on two comparisons. When the rugby forwards were compared with the rugby backs, 73,1% of the rugby forwards reported the presence of this symptom compared with only 42,1% of the rugby backs. When the Under 21 forwards were compared with the Under 21 backs, 81,8% of the Under 21 forwards reported the presence of this symptom compared with only 50% of the Under 21 backs.

Anxiety or worry have been reported following mild head injuries in general (McLean et al., 1983; Rutherford, 1989; Rutherford et al., 1977) and mild head injuries in sport (Dickinson, 1998; Shuttleworth-Jordan et al., 1993). Shuttleworth-Jordan et al. (1993) report that anxiety was present three days post-injury but had resolved by the one month follow-up. In contrast, Dickinson's study (1998) amongst higher level rugby players found anxiety present at least three months post-season, by which stage the acute effects of any head injuries are likely to have resolved. In this study there is once again strong positional variation, with the forwards reporting a higher incidence of symptoms than the backs. This provides further support for the hypothesis that those players exposed to more full physical contact, the forwards, suffer more mild head injuries than those involved in less physical contact, the backs. In addition, anxiety and worry form part of a cluster of symptoms which are related to the depression symptom cluster which has emerged quite clearly (irritability, lowered frustration tolerance, social contact, restlessness, sleep difficulty and appetite difficulty). These symptoms, all in the direction expected (higher incidence amongst contact sport players than non-contact sport players, and amongst the forwards than the backs) and either significant or approaching significance, add to the robustness of the results found on the individual symptoms.

5.3. OVERALL INDICATIONS FROM THE RESEARCH

The overall indications from the research are best addressed in terms of the hypotheses which were posed: (i) contact sport players will show more deficit and report more symptomatology relative to non-contact sport players; (ii) the rugby forwards will show more deficit and report more symptomatology relative to both the rugby backs and non-contact sport players; and (iii) the Springbok rugby players will show more deficit and report more symptomatology relative to the Under 21 rugby players.

An important finding in relation to the neuropsychological test results were the significant differences found between some of the groups used in the research on the variables of age, education and IQ. In particular, the mean age, mean level of education and mean IQ for the Under 21 rugby players was significantly lower than that of both the Springbok rugby players and the hockey players. While this appears to have influenced some of the test results, there was no evidence of a global lowering of scores for the Under 21 rugby players across all the tests. It would appear that the lower age, level of education and IQ of the Under 21 rugby group contributed to an inability to compensate for deficit on some of the tests due to their lower brain reserve

capacity. Because of the absence of global lowering on all the test scores, attributions concerning the presence of deficit due to brain injury could be made using the Under 21 rugby group in spite of these differences. Importantly, the forwards and backs within all three of the contact sport groups, namely rugby, Springbok rugby and Under 21 rugby, were well controlled for age, education and IQ, with no significant differences present between these positional groups. Hence attributions concerning the presence of deficits due to brain injury could be made in a relatively unconfounded manner, as regards these potentially confounding variables, between the forwards and backs in all the rugby groups.

An important finding in relation to postconcussive symptomatology was the strong indication that the Springbok rugby players under-reported their true symptom presentation, especially on physical symptoms, which could be interpreted as having a direct bearing on match performance, including fatigue, weakness in limbs and clumsiness. The Under 21 rugby players, on the other hand, appear to have reported their postconcussive symptoms accurately. The common anomaly of the headaches reported by Dickinson (1998) and the present study, serves to highlight the under-reporting of their symptomatology by the Springbok rugby players. This could be attributed to the Springbok rugby players worrying about possible consequences of accurate reporting because their assessments were conducted with the purpose of writing clinical reports (unlike for the Under 21 rugby players), and because elite athletes tolerate symptoms which would prevent recreational participants from playing the game.

The neuropsychological test results clearly distinguish between contact sport players and non-contact sport players. The postconcussive symptoms did not distinguish between the Springbok rugby players and the hockey players, probably due to the under-reporting of symptoms by the Springbok rugby players, but did distinguish between the Under 21 rugby players and the hockey players. However, positional variation was clearly evident from both the neuropsychological test results and the postconcussive symptoms. No clear differentiation between the Springbok rugby players and the Under 21 rugby players was present on the neuropsychological tests or on the postconcussive symptoms. While the Springbok rugby players showed more visual memory deficit than the Under 21 rugby players (Digit Symbol Substitution Incidental Recall and WMS Visual Reproduction) , the Under 21 rugby players showed more deficit than the Springbok rugby players on tests that were either educationally loaded (Unstructured Verbal Fluency), known to be particularly challenging and sensitive to diffuse brain damage (Digit Symbol Substitution), or which tapped highly developed skills amongst the Springbok rugby players (Finger Tapping Test). Thus the lower level of education and IQ seem to predispose the Under 21 rugby players in some instances to more deficit. Taking this point further, the implication of this study raises the concern that in patients with even lower IQ or learning disability, the negative effects would be even more extreme than those seen with the Under 21

rugby players in the areas of verbal memory, verbal fluency, visuoperceptual ability and hand-motor dexterity.

While there were some anomalous results, there were a series of results which were consistent across contact sport versus non-contact sport and across forwards versus backs. These were strongly reinforced by the direction of the self-reported postconcussive symptoms. In terms of the actual tests, the most significant neurocognitive deficits were found in the areas of information processing speed, attention and concentration, mental flexibility, visual memory and verbal new learning. The most significant neuropsychiatric complaints were reported in the areas of memory, social contact, sensitivity to noise, lowered frustration tolerance, anxiety and worry, and depression. Thus the effect of cumulative mild head injuries sustained by the contact sport players, particularly those in the forward positions, is clearly evident from the combination of objectively measured deficits and self-reported postconcussive symptoms. The comparisons of percentages of individuals with deficit (or symptomatology) proved to be highly effective in detecting the presence or absence of deficit (or symptomatology). Moreover, the ability to report the actual percentage allowed for a pattern of individual incidence to emerge which would otherwise have been lost in the group picture.

In terms of the actual tests, the most sensitive neuropsychological test used in the present study was the Digit Symbol Substitution test, which clearly distinguished contact sport players from non-contact sport players, and forwards from backs across six out of seven of the comparisons (the only exception being the Under 21 forwards and Under 21 backs which showed a trend in the right direction). Amongst the contact sport group nearly *one third* (31,1%) showed impairment compared with less than 5% of the non-contact sport group. Amongst the total rugby forwards nearly *half* (46,1%) showed impairment compared with just over 10% of the backs. The Trail Making Test Part B and the Digit Symbol Substitution Incidental Recall test were also sensitive to the effects of diffuse brain damage and distinguished contact sport players from non-contact sport players, and forwards from backs on four out of the eight comparisons. The most sensitive postconcussive symptom appears to be memory, where *half* (50%) the total rugby forwards report experiencing memory problems compared with just over 10% of the rugby backs. At the Springbok rugby level, almost *half* (46,7%) the Springbok forwards report experiencing memory problems compared with 0% of the Springbok backs.

All the postconcussive symptoms reported in the study by Shuttleworth-Jordan et al. (1993) were present in phase one (Dickinson, 1998) and were replicated in phase two by the present study. These highly consistent findings across three separate studies suggest that these can be attributed to cumulative mild head injuries sustained while playing the game of rugby. With regard to the literature, the only symptom which is commonly reported to follow a mild head injury (for example, Levin et al., 1987; McLean et al., 1983;

Rutherford, 1989; Rutherford et al., 1977) that was not reported in either phase one (Dickinson, 1998) or phase two (this study) was 'dizziness'. The assessments for both phase one and phase two of the present research were conducted at least three months post-season, by which time it is likely that any acute effects following a mild head injury would have resolved. It is suggested, therefore, that 'dizziness' is an acute symptom only. The results of the study by Dickinson (1998), together with the results of the present study, imply that the symptomatology present is evidence of medium- to long-term effects and could indeed be permanent. The only finding by Dickinson (1998) which was not replicated in this study was that of 'eyesight', an anomalous result where the non-contact sport control group reported a higher incidence of this symptom than the contact sport group. However, this anomaly appeared to be due to Dickinson's very feasible explanation that the cricket players spend many hours playing cricket in harsh sunlight and, in addition, the cricket players were assessed post-season after a long and tiring (and ultimately unsuccessful) tour while the rugby players were assessed pre-season.

As stated above, all the players who took part in this study were assessed pre-season, which is at least three months post-season. This suggests that any deficit found or postconcussive symptom reported in this study is of a chronic nature as it is likely that, as reported in the literature (for example, Alves et al., 1986, Binder, 1986; Evans, 1992; Levin et al., 1987), the acute effects of a mild head injury would have resolved by this time. As this is a cross-sectional study, pre-selection effects cannot be ruled out. However, comparisons between the forwards and backs are controlled for age, education and IQ and, in addition, the test results of the Under 21 rugby players were not globally lower on all tests, only selected tests which are highly consistent with the expected results following diffuse brain damage. The results of this study, therefore, can be seen to provide compelling evidence of the medium- to long-term deleterious effects of mild head injuries in players of contact sport. The manner in which these deficits might affect the everyday occupational and home lives of the players can be speculated on but are not specifically answered by this study, as this would involve formally investigating everyday functioning which was beyond the scope of this research. However, research amongst HIV+ patients found that even mild cognitive impairment can cause problems in everyday functioning, leading to diminished quality of life and increased unemployment (Grant & Marcotte, 1999). It is very likely that the contact sport players, and particularly the forwards, have the potential to show similar problems and this is borne out by the presence of chronic self-reported postconcussive symptoms which strongly imply that this may be the start of such a process. Implications concerning the latent effects of cumulative mild head injuries, such as the possible early onset of Alzheimer's Disease (Spear, 1995), would need further longitudinal research.

5.4. CONCLUSIONS

This research strongly suggests the presence of neuropsychological deficit and chronic postconcussive symptomatology amongst a proportion of rugby players, notably amongst rugby forwards. The idea behind studying these players is not to prevent people from playing the game, merely for them to be aware of the risks involved in playing the game and the possible consequences of their actions. The challenge which must now be taken up by the administrators and team management is to openly build these risk factors into contracts and to work towards minimising these risks while still maintaining the popularity and spectacle of the game.

5.5. EVALUATION OF THIS RESEARCH

5.5.1. METHODOLOGICAL STRENGTHS

The methodological strengths of this study include:

1. The large sample size (n = 45) of the contact sport group is an improvement over phase one of the research which had a smaller contact sport sample size (n = 26).

2. All participants were from well defined homogenous groups of generally high functioning, physically fit subjects, thus controlling for extraneous variables that can have bearing on cognitive performance. Strict exclusion criteria were also applied to the participants in order to prevent further confounding variables: history of substance abuse; neurological or psychiatric disorder; previous moderate to severe non-sport related head injury.

3. This study made use of the South African National Hockey squad as the non-contact sport control group (n = 21). This is an improvement over other studies which did not use a control group and over the phase one study by Dickinson (1998) which used the cricket players as the control group. Most of the players had played very little rugby, if any, and those that had, played at a low level while at primary school.

4. All participants were assessed pre-season thus eliminating the confounding variables of fatigue and depression found amongst the cricket players, the non-contact sport control group for phase one of this research.

5. Levels of deficit or impairment were calculated according to appropriate norms for the participants given their age and generally high level of functioning. The normative data were based on high functioning university students between the ages of 18 - 25 years.

6. Using neuropsychological assessment together with postconcussive symptoms is a strong method of providing cross-validation between the objectively measured cognitive deficit (if any) and the self-reported symptoms, whether the nature of the self-reported symptoms is supported by the objective levels of deficit found and vice versa.

7. The wide range of tests used allows for the replication of possible deficit within modalities (e.g. two tests of visual memory used) and for the dissociation of effects between modalities (e.g. Digits Forwards and Digits Backwards).

8. Comparing individual players to the normative data and calculating individual levels of impairment allows one to see information that gets lost when merely comparing group means to the normative data. It allows a picture of individual variation to emerge which would otherwise have been lost in the group picture.

5.5.2. METHODOLOGICAL WEAKNESSES

The methodological weaknesses of this research include the following:

1. The relatively small size of the non-contact sport sample should be expanded for future research. According to Satz et al. (1997) the minimum sample size for research into head injuries is 20 participants, therefore the non-contact sport sample size is barely adequate. Although the contact sport group was bigger, it was split into two smaller groups (Springbok rugby and Under 21 rugby), both of which were split into two even smaller groups (forwards and backs) for purposes of analysis. The size of these smaller groups was less than ideal according to Satz et al. (1997).

2. Whereas it was attempted to extend the rugby group by including the Under 21 rugby players, the two groups of rugby players (Springboks and Under 21s) were not totally equivalent for age, education and IQ. The Under 21 rugby players were not, therefore, an ideal group to use to increase the size of the top level rugby group. Despite these differences, analyses could be made with the Under 21 group and both the similarities and differences between the Under 21 group and the Springbok rugby and hockey groups were used in the comparisons.

3. The estimated premorbid IQ scores were based on only two Subtests of the SAWAIS. A wider range of tests may have been preferable in estimating the premorbid IQ, as this method tends to predict a higher level of premorbid functioning than methods which take factors such as level of education into account (Vanderploeg, 1994). However, given the time constraints,

employing Lezak's (1983) 'best performance' method, using two tests which are relatively unaffected by prior head injury, is considered a good estimate of general level of intellectual functioning. Moreover, using this method gave the present study an advantage over other studies which have used baseline premorbid data based on pre-season assessments but using tests sensitive to the effects of mild head injury.

4. This research is a cross-sectional research study of possible brain damage in rugby players. Whilst it would seem there is a strong argument that deficit is as a result of multiple mild head injuries, it cannot be ruled out that the differences are not the result of preselected differences between these groups. This could only be done on the basis of longitudinal research.

5. Whilst the cognitive tests and postconcussive symptomatology provided ratification for the presence of deficit, it cannot be said what effect this deficit would have in the work or home situation. This would be far too extensive to investigate given the parameters of the present thesis and would form a large research project on its own.

5.6. RECOMMENDATIONS FOR FURTHER RESEARCH

1. It would be beneficial to assess the present Springbok squad as only two or three players from the original squad assessed remain in the current squad. The additional players could be added to the present sample and used for further comparisons. In addition, comparisons could be made with the original Springbok group assessed to determine what effect turning professional at a younger age is having on the players. It would also be necessary to increase the sample size of the hockey playing control group at the same time.

2. Long-term research into the effects of mild head injuries is of vital importance. It would be useful to follow up the Under 21 rugby group now, two years after the initial assessment, to determine what changes, if any, have occurred during this period. In addition, if all the players who have already been assessed could be permanently followed up until death, a picture would begin to emerge as to the long-term effects of mild head injuries suffered at a younger age.

3. It would benefit the research to assess Under 21 hockey players in order to have a control group which is matched for age with the Under 21 rugby players. It is possible that a similar trend of more previously disadvantaged players and lower levels of education will occur amongst the hockey players which would provide a more appropriate matched control group for the rugby group.

4. Research into the effect of mild head injury at schoolboy level is an important area that needs to be investigated using matric rugby players and controls. Anderson (1996) states that there is increasing concern that youngsters under 16 years represent a high risk group for sustaining head injuries in a variety of sporting and recreational activities. This could be having a serious impact on their academic performance at a time in their lives when performance during matric exams etc is of crucial importance to their future. Contact sport participants in this age group need to be made aware of possible negative consequences of mild head injuries (e.g. slowed information processing might only become apparent during times of stress such as during their matric exams). Failure to perform to expectations might lead to subsequent psychosocial problems which could be avoided.

5. It would be useful to assess players of similar levels from other rugby-playing countries to see whether the results and trends found in this research are replicated elsewhere or whether this is strictly a South African phenomenon.

6. It is important to find ways of making the game safer for the players while at the same time sustaining the popularity of the game amongst the general public. While the rules of the game have changed over the years in order to improve the safety of the players, players should be assessed after any rule changes in order to ensure that the changes are indeed having the desired effect. The effectiveness of protective clothing in preventing injuries, such as some form of helmet, needs to be investigated.

CHAPTER 6 : REFERENCES

About Rugby League: How does it differ with Rugby Union. (1999, June 15). Available: http://www.personal.u-net.com/~interzone/faq.htm

Answers to Frequently Asked Questions and other Trivia. (1999, November, 15). Available: http://www.uidaho.edu/clubs/womens_rugby/RugbyRoot/rugby/FAQ/faq.html

Abreau, F., Templer, D. I., Schuyler, B. A. and Hutchison, H. T. (1990). Neuropsychological assessment of soccer players. *Neuropsychology. 4,* 175-181.

Adams, J. H., Mitchell, D. E., Graham, D. I. and Doyle, D. (1977). Diffuse brain damage of immediate impact type. Its relationship to 'primary brain-stem damage' in head injury. *Brain. 100,* 489 -502.

Alexander, D., Kennedy, M. and Kennedy, J. (1979). Injuries in rugby league football. *The Medical Journal of Australia. 2,* 341-342.

Alves, W. M., Rimel, R. W. and Nelson, W. E. (1987). University of Virginia prospective study of football-induced minor head injury: status report. *Clinics in Sports Medicine. 6*(1), 211-218.

Ancer, R. (1999). *A study on mild head injury in rugby: Cognitive test profiles of professional rugby and cricket players*. Unpublished Master's thesis, Rhodes University, Grahamstown.

Anderson, S. D. (1996). Postconcussional disorder and loss of consciousness. *Bull Am Acad Psychiatry Law. 24*(4), 493-504.

Anderson, S. J. (1996). Sports-related head injuries: A neuropsychological perspective. *Sports Medicine, September,* 23-27.

Barnes, B. C., Cooper, L., Kirkendall, D. T., McDermott, T. P., Jordan, B. D. and Garrett, W. E. (1998). Concussion history in elite male and female soccer players. *The American Journal of Sports Medicine. 26*(3), 433-438.

Baroff, G. S. (1998). Is heading a soccer ball injurious to brain function? *Journal of Head Trauma Rehabilitation. 13*(2), 45-52.

Barth, J. T., Macciocchi, S. N., Giordani, B., Rimel, R., Jane, J. A., and Boll, T. J. (1983). Neuropsychological sequelae of minor head injury. *Neurosurgery. 13*(5), 529-532.

Barth, J. T., Alves, W. M., Ryan, T. V., Macciocchi, S. N., Rimel, R., Jane, J. A. and Nelson, W. E. (1989). Mild head injury in sports: neuropsychological sequelae and recovery of function. In H. S. Levin, H. M. Eisenberg and A. L. Benton (Eds.), *Mild Head Injury* (pp.257-275). Oxford: Oxford University Press.

Binder, L. M. (1986). Persisting symptoms after mild head injury: a review of postconcussive syndrome. *Journal of Clinical and Experimental Neuropsychology. 8*(4). 323-346.

Binder, L. M. (1997). A review of mild head trauma. Part II: clinical implications. *Journal of Clinical and Experimental Neuropsychology, 19*(3), 432-457.

Binder, L. M., Rohling, M. L. and Larrabee, G. J. (1997). A review of mild head trauma. Part I: meta-analytic review of neuropsychological studies. *Journal of Clinical and Experimental Neuropsychology, 19*(3), 421-431.

Bless, C., and Kathuria, R. (1993). *Fundamentals of Social Statistics: An African Perspective.* Cape Town: Juta & Co, Ltd.

Boden, B. P., Kirkendall, D. T. and Garrett, W. E. (1998). Concussion incidence in elite college soccer players. *The American Journal of Sports Medicine. 26*(2), 238-241.

Bohnen, N. and Jolles, J. (1992). Neurobehavioral aspects of postconcussive symptoms after mild head injury. *The Journal of Nervous and Mental Disease. 180(11),* 683-692.

Bohnen, N., Jolles, J. and Twijnstra, A. (1992). Neuropsychological deficits in patients with persistent symptoms six months after head injury. *Neurosurgery. 30*(5), 692-696.

Boll, T. J. (1983). Minor head injury in children - out of sight but not out of mind. *Journal of Clinical Child Psychology, 12*(1), 74-80.

Boll, T. J. (1985). Developing issues in clinical neuropsychology. *Journal of Clinical and Experimental Neuropsychology. 7*(5), 473-485.

Brooks, N., Kupshik, G., Wilson, L., Galbraith, S., and Ward, R. (1987). A neuropsychological study of active amateur boxers. *Journal of Neurology, Neurosurgery, and Psychiatry. 50*, 997-1000.

Burbach, F. R. (1987). *Neuropsychological sequelae of aneurysmal sub-arachnoid haemorrhage in patients without neurological deficits 6 to 8 months post operatively - an exploratory study.* Unpublished masters thesis, University of Cape Town.

Butler, R. J., Forsythe, W. I., Beverly, D. W., Adams, L. M. (1993). A prospective controlled investigation of the cognitive effects of amateur boxing. *Journal of Neurology, Neurosurgery, and Psychiatry. 56,* 1055-1000.

Cantu, R. C. (1995). Head and spine injuries in youth sport. *Clinics in Sports Medicine. 14*(3), 517-532.

Cantu, R. C. (1996). Head injuries in sport. *British Journal of Sports Medicine. 30*, 289-296.

Cantu, R. C. (1998). Second impact syndrome. *Clinics in Sports Medicine. 17*(1), 37-44.

Casson, I. R., Sham, R., Campbell, E. A., Tarlau, M. and DiDomenico, A. (1982). Neurological and CT evaluation of knocked-out boxers. *Journal of Neurology, Neurosurgery, and Psychiatry. 45*, 170-174.

Casson, I. R., Siegel, O., Sham, R., Campbell, E. A., Tarlau, M. and DiDomenico, A. (1984). Brain damage in modern boxers. *Journal of the American Medical Association. 251*(20), 2663-2667.

Council on Scientific Affairs. (1983). Brain injury in boxing. *Journal of the American Medical Association. 249*(2), 254-257.

Cremona-Meteyard, S. L. and Geffen, G. M. (1994). Persistent visuospatial attention deficits following mild head injury in Australian Rules Football players. *Neuropsychologia, 32,* 649-662.

Dacey, R. G. and Dikmen, S. S. (1987). Mild head injury. In P. R. Cooper (Ed) *Head Injury. 2nd Edition.* (pp.72-88). London: Williams and Wilkins.

Denckla, M. B. (1973). Development of speed in repetitive and successive finger movements in normal children. *Developmental Medical Child Neurology, 15,* 635-645.

Dickinson, A. (1998). *Postconcussive sequelae in Contact Sport: Rugby versus Non-contact Sport Controls.* Unpublished Master's thesis, Rhodes University, Grahamstown.

Dikmen, S. S.; Temkin, N. and Armsden, G. (1989). Neuropsychological recovery: relationship to psychosocial functioning and postconcussional complaints. In H. S. Levin, H. M. Eisenberg and A. L. Benton (Eds.), *Mild Head Injury* (pp.229-241). Oxford: Oxford University Press.

Editorial. (1998). A salute to Morné. *Eastern Province Herald*, 23 May 1998, p6.

Erlanger, D. M., Kutner, K. C., Barth, J. T. and Barnes, R. (1999). Neuropsychology of sports-related injury: dementia pugilistica to post concussion syndrome. *The Clinical Neuropsychologist. 13*(2), 193-209.

Evans, R. W. (1992). The post concussion syndrome and the sequelae of mild head injury. *Neurologic Clinics, 10*(4), 815-847.

Ferguson, G. A. (1988). *Statistical Analysis in Psychology and Education.* Auckland: M°Graw-Hill.

Galbraith, S. (1985). *Head injuries in sport - their nature and management.* Proceedings of the VIII Commonwealth and International Conference on Sport, Physical Education, Dance, Recreation and Health. In J. A. MacGregor and J. A. Moncur (Eds), *Sport and Medicine.* London: E. & F. N. Spon

Garraway, M. and MacLeod, D. (1995). Epidemiology of rugby football injuries. *Lancet, 345,* 1485-1487.

Gasqoine, P. G. (1997). Postconcussion symptoms. *Neuropsychology Review. 7*(2), 77-85.

Gennarelli, T. A. (1987). Cerebral concussion and diffuse brain damage. In P. R. Cooper (Ed.), *Head Injury* (2nd Edition). (pp.108-124). Baltimore: Williams and Wilkins.

Gentilini, M., Nichelli, P., and Schoenhuber, R. (1989). Assessment of attention in mild head injury. In H. S. Levin, H. M. Eisenberg and A. L. Benton (Eds.), *Mild Head Injury* (pp.163-175). Oxford: Oxford University Press.

Gentilini, M., Nichelli, P., Schoenhuber, R., Bortolotti, P., Tonelli, L., Falasca, A. and Merli, G. A. (1985). Neuropsychological evaluation of mild head injury. *Journal of Neurology, Neurosurgery and Psychiatry, 48,* 137-140.

Gerberich, S. G., Priest, J. D., Boen, J. R., Straub, C. P. and Maxwell, R. E. (1983). Concussion incidences and severity in secondary school varsity football players. *American Journal of Public Health. 73*(12), 1370-1375.

Gibbs, N. (1993). Injuries in professional rugby league. A three-year prospective study of the South Sydney Professional Rugby League Football Club. *The American Journal of Sports Medicine. 21*(5), 696-700.

Gissane, C., Jennings, D. C., Cumine, A. J., Stephenson, S. E. and White, J. A. (1997). Differences in the incidence of injury between rugby league forwards and backs. *The Australian Journal of Science and Medicine in Sport. 29*(4), 91-94.

Gleave, J. (1986). Head injuries. In B. H. Helal, J. King and W. Grange (Eds.), *Sports injuries and Their Treatment.* (pp.47-59). London: Chapman and Hall.

Graham, D. I., Adams, J. H., Path F. R. C. and Gennarelli, T. A. (1987). Pathology of brain damage in head injury. In P. R. Cooper (Ed) *Head Injury. 2nd Edition.* (pp.72-88). London: Williams and Wilkins.

Grant, I. G. and Marcotte, T. D. (1999). HIV infection: medical, neuropsychological and neuropsychiatric aspects. Workshop presented at the twenty-second mid-year meeting of the International Neuropsychological Society, Durban, South Africa.

Green, G. A. and Jordan, S. E. (1998). Are brain injuries a significant problem in soccer? *Clinics in Sports Medicine. 17*(4), 795-809.

Gronwall, D. (1989). Effects of concussion on attention and cognition. In H. S. Levin, H. M. Eisenberg and A. L. Benton (Eds.), *Mild Head Injury* (pp.153-162). Oxford: Oxford University Press.

Gronwall, D. and Wrightson, P. (1974). Delayed recovery of intellectual function after minor head injury. *The Lancet. September 14, 1974,* 605-609.

Gronwall, D. and Wrightson, P. (1975). Cumulative effects of concussion. *The Lancet, 1975,* 995-997.

Haglund, Y. and Eriksson, E. (1993). Does amateur boxing lead to chronic brain damage? *The American Journal of Sports Medicine. 21*(1), 97-109.

Heilbronner, R. L., Henry, G. K. and Carson-Brewer, M. (1991). Neuropsychologic test performance in amateur boxers. *The American Journal of Sports Medicine. 19*(4), 376-380.

Hinton-Bayre, A. D., Geffen, G. and McFarland, K. (1997). Mild head injury and speed of information processing: a prospective study of professional rugby league players. *Journal of Clinical and Experimental Neuropsychology, 19*(2), 275-289.

Jacobson, G. and Speechley, E. (1988). Concussion in rugby. *Sports Medicine, 3,* 18-19.

Jacobson, R. R. (1995). The post-concussional syndrome: physiogenesis, psychogenesis and malingering. An interactive model. *Journal of Psychosomatic Research. 39*(6), 675-693.

Jennett, B. (1989). Some international comparisons. In H. S. Levin, H. M. Eisenberg and A. L. Benton (Eds.), *Mild Head Injury* (pp.257-275). Oxford: Oxford University Press.

Jordan, A. B. (1998). Rugby death a wake-up call for parents. *Eastern Province Herald*, 23 May 1998, p4.

Jordan, B. D. (1987). Neurologic aspects of boxing. *Archives of Neurology, 44,* 453-459.

Jordan, S. E., Green, G. A., Galanty, H. L., Mandelbaum, B. R. and Jabour, B. A. (1996). Acute and chronic brain injury in United States national team soccer players. *The American Journal of Sports Medicine. 24*(2), 205-210.

Kaste, M., Vilkki, J., Sainio, K., Kuurne, T., Katevuo, K. and Meurala, H. (1984). Is chronic damage in boxing a hazard of the past? *Lancet, 2,* 1186-1188.

Kibby, M. Y. and Long, C. J. (1996), Minor head injury: attempts at clarifying the confusion. *Brain Injury. 10*(3), 159-86.

King, N. (1997). Mild head injury: Neuropathology, sequelae, measurement and recovery. *British Journal of Clinical Psychology. 36,* 161-184.

Kraus, J. F., and Nourjah, P. (1989). The epidemiology of mild head injury. In H. S. Levin, H. M. Eisenberg and A. L. Benton (Eds.), *Mild Head Injury* (pp.257-275). Oxford: Oxford University Press.

Lehman, L. B. and Ravich, S. J. (1990). Closed head injuries in athletes. *Clinics in Sports Medicine. 9*(2), 247-261.

Leininger, B. E., Grammling, S. E., Farrell, A. D., Kreutzer, J. S. and Peck, E. A. (1990). Neuropsychological deficits in symptomatic minor injury after concussion and mild concussion. *Journal of Neurology, Neurosurgery, and Psychiatry, 53*(4), 293-296.

Levin, H. S., Benton, A. L., Grossman, R. G. (1982). *Neurobehavioral Consequences of Closed Head Injury.* Oxford: Oxford University Press.

Levin, H. S., Mattis, S., Ruff, R. M., Eisenberg, H. M., Marshall, L. F., Tabaddor, K., High, W. M. and Frankowski, R. F. (1987). Neurobehavioral outcome following minor head injury: a three-center study. *Journal of Neurosurgery. 66,* 234-243.

Lezak, M. D. (1995). *Neuropsychological Assessment.* (3rd ed.). Oxford: Oxford University Press.

Lishman, W. A. (1978). Physiogenesis and psychogenesis in the "postconcussional syndrome". *British Journal of Psychiatry, 153,* 460-469.

Lishman, W. A. (1987). *Organic Psychiatry: the Psychological Consequences of Cerebral Disorder.* London: Blackwell Scientific Publications.

Lovell, M. R. and Collins, M. W. (1998). Neuropsychological assessment of the college football player. *The Journal of Head Trauma Rehabilitation. 13*(2), 9-26.

Macciocchi, S. N., Barth, J. T., Alves, W., Rimel, R. W. and Jane, J. A. (1996). Neuropsychological functioning and recovery after mild head injury in collegiate athletes. *Neurosurgery, 39*(3), 510-514.

Macciocchi, S. N., Barth, J. T. and Littlefield, L. M. (1998). Outcome after Mild head injury. *Clinics in Sports Medicine. 17*(1), 27-36.

MacFlynn, G., Montgomery, E. A., Fenton, G. W. and Rutherford, W. (1984). Measurement of reaction time following minor head injury. *Journal of Neurology, Neurosurgery and Psychiatry. 47,* 1326-1331.

MacLeod, D. A. D. (1993). Risks and injuries in rugby football. In G. R. McLatchie and C. M. E. Lennox (Eds.). *The Soft Tissues: Trauma and Sports Injuries.* (pp.372-381). Oxford: Butterworth-Heinemann Ltd.

Maddocks, D. L. and Saling, M. M. (1991). Neuropsychological sequelae following concussion in Australian Rules Footballers. *Journal of Clinical and Experimental Psychology. 13,* 439-42.

Maddocks, D. L., Saling, M. M. and Dicker, G. D. (1995). A note on normative data for a test sensitive to concussion in Australian Rules Footballers. *Australian Psychologist, 30*(2), 125-127.

Martland, H. S. (1928). Punch-drunk. *Journal of the American Medical Association. 19,* 1103-7.

Matser, J. T., Kessels, A. G. H., Jordan, B. D., Lezak, M. D. and Troost, J. (1998). Chronic traumatic brain injury in professional soccer players. *Neurology. 51,* 791-796.

Meuller, F. O. and Blyth, C. S. (1987). Fatalities from head and cervical spine injuries occurring in tackle football: 40 years experience. *Clinics in Sports Medicine, 6,* 185-196.

McCunney, R. J., and Russo, P. K. (1984). Brain injuries in boxers. *The Physician and Sportsmedicine. 12*(5), 53-67.

McFie, J. (1975). *Assessment of Organic Intellectual Impairment.* London: Academic Press.

McLatchie, G., Brooks, N., Galbraith, S., Hutchison, J. S. F., Wilson, L., Melville, I. and Teasdale, E. (1987). Clinical neurological examination, neuropsychology, electroencephalography and computed tomographic head scanning in active amateur boxers. *Journal of Neurology, Neurosurgery, and Psychiatry. 50,* 96-99.

McLean Jnr, A., Temkin, N. R., Dikmen, S. and Wyler, A. R. (1983). The behavioral sequelae of head injury. *Journal of Clinical Neuropsychology. 5*(4), 361-376.

McQuillan, R. J. M. (1992). A survey of rugby injuries attending an accident and emergency department. *Irish Medical Journal. 85*(2), 72-73.

Miller, R. (1981) *Simultaneous Inference.* 2nd Edition. New York: Springer-Verlag.

Murelius, O. and Haglund, Y. (1991). Does Swedish amateur boxing lead to chronic brain damage? *Acta Neurol Scand. 83,* 9-13.

Nathan, M., Goedeke, R., and Noakes, T. D. (1983). The incidence and nature of rugby injuries experienced at one school during the 1982 rugby season. *South African Medical Journal, 64,* 132-137.

Oppenheimer, D. R. (1968). Microscopic lesions in the brain following head injury. *Journal of Neurology, Neurosurgery and Psychiatry. 31,* 299-306.

Parasuraman, R., Mutter, S. A. and Molloy, R. (1991). Sustained attention following mild closed head injury. *Journal of Clinical and Experimental Neuropsychology. 13*(5), 789-811.

Reid, I. (1998). *Tackling Mild Head Injury in Rugby: A Comparison of the Cognitive Profiles of Professional Rugby and Cricket Players*. Unpublished Master's thesis, Rhodes University, Grahamstown.

Reid, S. E., Tarkington, J. A., Epstein, H. M. and O'Dea, T. J. (1971). Brain tolerance to impact in football. *Surgery, Gynecology and Obstetrics. 133*(6), 929-936.

Reitan, R. M. (1956). *The Trail Making Test: Manual for Administration, Scoring and Interpretation.* Indiana: Indiana Medical Centre, Indiana University.

Richardson, J. T. E. (1990). *Clinical and Neuropsychological Aspects of Closed Head Injury.* London: Taylor & Francis.

Rimel, R. W., Giordani, B., Barth, J. T., Boll, T. J., and Jane, J. A. (1981). Disability caused by minor head injury. *Neurosurgery. 9,* 221-228.

Ross, R. J., Casson, I. R., Siegel, O. and Cole, M. (1987). Boxing injuries: neurologic, radiologic, and neuropsychologic evaluation. *Clinics in Sports Medicine. 6*(1), 41-51.

Roux, C. E., Goedeke, R., Visser, G. R., van Zyl, W. A. and Noakes, T. D. (1987). The epidemiology of schoolboy rugby injuries. *South African Medical Journal. 71,* 307-313.

Roy, S. P. (1974). The nature and frequency of rugby injuries: a pilot study of 300 injuries at Stellenbosch. *South African Medical Journal. 48,* 2321-2327.

Ruchinskas, R. A., Francis, J. P., and Barth, J. T. (1997). Mild Head Injury in Sports. *Applied Neuropsychology. 4,* 43-49.

Ruff, R. M., Levin, H. S., Mattis, S., High Jr., W. M., Marshall, L. F., Eisenberg, H. M. and Tabaddor, K. (1989). Recovery of memory after mild head injury: a three center study. In H. S. Levin, H. M. Eisenberg and A. L. Benton (Eds.), *Mild Head Injury* (pp.176-188). Oxford: Oxford University Press.

Russell, E. W. (1986). The psychometric foundation of clinical neuropsychology. In S. B. Filskov and T. J. Boll (Eds.), *Handbook of Clinical Neuropsychology, Volume 2.* (pp.45-80). New York: John Wiley and Sons.

Rutherford, W. H. (1989). Postconcussion symptoms: relationship to acute neurological indices, individual differences, and circumstances of injury. In H. S. Levin, H. M. Eisenberg and A. L. Benton (Eds.), *Mild Head Injury* (pp.217-228). Oxford: Oxford University Press.

Rutherford, W. H., Merrett, J. D. and McDonald, J. R. (1977). Sequelae of concussion caused by minor head injuries. *The Lancet. January 1977,* 1-4.

Ryan, A. J. (1987). Intracranial injuries resulting from boxing: a review (1918 - 1985). *Clinics in Sports Medicine. 6*(1), 31-40.

Satz, P. (1993). Brain reserve capacity on symptom onset after brain injury: a formulation and review of evidence for threshold theory. *Neuropsychology, 7*(3), 273-295.

Satz, P., Zaucha, K., McCleary, C., Light, R., Asarnow, R. and Becker, D. (1997). Mild head injury in children and adolescents: a review of studies (1970 - 1995). *Psychological Bulletin. 122*(2), 107-131

Schoenhuber, R. and Gentilini, M. (1989). Neurophysiological assessment of mild head injury. In H. S. Levin, H. M. Eisenberg and A. L. Benton (Eds.), *Mild Head Injury* (pp.257-275). Oxford: Oxford University Press.

Segalowitz, S. J. and Lawson, S. (1995). Subtle symptoms associated with self-reported mild head injury. *Journal of Learning Disabilities. 28*(5), 309-319.

Seward, H., Orchard, J., Hazard, H. and Collinson, D. (1993). Football injuries in Australia at the élite level. *The Medical Journal of Australia. 159,* 298-301.

Shuttleworth-Jordan, A. B. (1992). *Age and second-language effects on Digit Span Backwards relative to Digit Span Forwards and Digit Supraspan.* Paper presented at the European meeting of the International Neuropsychological Society, Durham, England.

Shuttleworth-Jordan, A. B. (1996). On not reinventing the wheel: a clinical perspective on culturally relevant test usage in South Africa. *South African Journal of Psychology. 26*(2), 96-102.

Shuttleworth-Jordan, A. B. (1999). *When a little becomes too much: a prospective theoretical context for cumulative mild head injury effects. A response to apparently null outcomes.* Paper presented at the twenty-second mid-year meeting of the International Neuropsychological Society, Durban, South Africa.

Shuttleworth-Jordan, A. B. and Bode, S. G. (1995). Taking account of age related differences on digit symbol and incidental recall for diagnostic purposes. *Journal of Clinical and Experimental Psychology. 17*(3), 439-448.

Shuttleworth-Jordan, A. B., Balarin, E. and Puchert, J. (1993). Mild Head Injury Effects in Rugby: Is Playing the Game Really Worth the Cost? Paper presented at the International Neuropsychological Society 16th European Conference, Island of Madeira (Portugal).

South African Wechsler Adult Intelligence Scale (SAWAIS) Manual. (1969). Johannesburg: National Institute for Personnel Research, Human Sciences Research Council.

Spear, J. (1995). Are professional footballers at risk of developing dementia? *International Journal of Geriatric Psychiatry. 10,* 1011-1014.

Spreen, O. and Strauss, E. (1991). *A Compendium of Neuropsychological Tests: Administration, Norms, and Commentary.* Oxford: Oxford University Press.

Stephenson, S. E., Gissane, C. and Jennings, D. (1996). Injury in rugby league: a four-year prospective survey. *The British Journal of Sports Medicine. 30,* 331-334.

Strich, S. J. (1961). Shearing of nerve Fibres as a cause of brain damage due to head injury. *The Lancet. Saturday 26 August 1961,* 443-447.

Sturmi, J. E., Smith, C. and Lombardo, J. A. (1998). Mild brain trauma in sports: diagnosis and treatment guidelines. *Sports Medicine. 25*(6), 351-358.

Stuss, D. T., Ely, P., Hugenholtz, H., Richard, M. T., LaRochelle, S., Poirer, C. A. and Bell, I. (1985). Subtle neuropsychological deficits in patients with good recovery after closed head injury. *Neurosurgery. 17,* 41-47.

Szymanski, H. V. and Linn, R. (1992). A review of postconcussive syndrome. *International Journal of Psychiatry in Medicine, 22,* 357-375.

Terman, L. M. and Merrill, M. A. (1973). *Standford-Binet Intelligence Scale. Manual for the Third Revision, Form L-M.* Boston: Houghton-Mifflin Co.

Torg, J. S., Truex, R., Quedenfield, T. C., Burstein, A., Spealman, A. and Nichols, C. (1978). The National football head and neck injury registry: reports and conclusions 1978. *The Journal of the American Medical Association. 241*(14), 1477-1479.

Torg, J. S., Vegso, J. J. and Sennett, B. (1987). The National football head and neck injury registry: 14-year report on cervical quadriplegia (1971-1984). *Clinics in Sports Medicine. 6*(1), 61-72.

Tysvaer, A. T. (1992). Head and neck injuries in soccer: impact of minor trauma. *Sports Medicine. 14*(3), 200-213.

Tysvaer, A. T. and Løchen, E. A. (1991). Soccer injuries to the brain. A neuropsychologic study of former players. *The American Journal of Sports Medicine. 19*(1), 56-60.

Tysvaer, A. T. and Storli, O. (1992). Association football injuries to the brain: a preliminary report. *The British Journal of Sports Medicine. 15*(3), 163-166.

Tysvaer, A. T., Storli, O. and Cachen, N. I. (1989). Soccer injuries to the brain: a neurologic and electroencephalographic study of former players. *Acta Neurologica Scandinavia, 80*(2), 151-156.

Vanderploeg, R. D. (1994). Estimating premorbid level of functioning. In R. D. Vanderploeg (Ed.). *Clinicians guide to Neuropsychological Assessment.* Hillsdale: Lawrence Erlbaum Associates inc., Publishers.

Van Heerden, J. J. (1976). ń Ontleding van rugbybeserings. *South African Medical Journal. 50,* 1374-1379

Vegso, J. J. and Lehman, R. C. (1987). Field evaluation and management of head and neck injuries. *Clinics in Sports Medicine. 6*(1), 1-15.

Walker, R. D. (1985). Sports injuries: rugby league may be less dangerous than union. *The Practitioner. 229,* 205-206.

Walsh, K. (1987). *Neuropsychology: A Clinical Approach.* 2nd Edition. London: Churchill Livingstone.

Warren, W. L. and Bailes, J. E. (1998). On the field evaluation of athletic head injuries. *Clinics in Sports Medicine. 17*(1), 13-26.

Watson, A. W. S. (1993). Incidence and nature of sports injuries in Ireland: analysis of four types of sport. *The American Journal of Sports Medicine. 21*(1), 137-143.

Wechsler, D. (1945). A standardized memory scale for clinical use. *The Journal of Psychology, 19,* 87-95.

Weight, D. G. (1998). Minor head trauma. *The Psychiatric Clinics of North America. 21*(3), 609-624.

Wilberger, J. E. (1988). Minor head injury in athletes. *Neurotrauma Medical Report. 2*(1), 3-4.

Wilberger, J. E. (1993). Minor head injuries in American Football: prevention of long term sequelae. *Sports Medicine. 15*(5), 338-343.

Wrightson, P. and Gronwall, D. (1980). Attitudes to concussion in young New Zealand men. *New Zealand Medical Journal. November, 1980,* 359-361.

Yarnell, P. R. and Lynch, S. (1973). The 'ding': amnestic states in football trauma. *Neurology. 23,* 196-197.

Yeudal, L. T. (1986). Normative data stratified by age and sex for 12 neuropsychological tests. *Journal of Clinical Psychology, 42*(6), 918-946.

Appendix A

Prorated Estimated IQ Scores:

Springbok Rugby, Under 21 Rugby, and Hockey

PRO-RATED IQ SCORES AND LEVEL OF EDUCATION - SPRINGBOK RUGBY

NO	C	PC	PREMORBID IQ	EDUCATION (years)
1 F	12.5	11.0	114	13
2 F	15.0	11.0	125	12
3 F	11.0	14.5	122	12
4 F	11.5	11.0	110	13
5 F	8.5	12.5	104	13
6 F	10.5	13.0	114	13
7 B	12.5	14.5	129	15
8 F	13.0	15.0	133	15
9 B	12.5	15.0	132	15
10 F	11.5	11.0	110	14
11 B	11.0	12.5	114	16
12 B	10.5	15.0	122	15
13 B	12.5	15.0	132	15
14 B	15.5	12.5	133	16
15 B	11.0	12.5	114	15
16 B	10.5	12.5	113	12
17 B	12.5	12.5	121	15
18 F	11.5	14.5	125	16
19 F	12.0	12.5	118	14
20 F	12.5	14.5	129	15
21 F	9.0	9.5	94	12
22 F	9.5	6.5	96*	15
23 B	12.5	15.0	132	15
24 F	14.0	14.0	133	15
25 F	13.5	12.5	125	16
26 B	10.5	8.5	96	12

Key:

C - Comprehension
PC - Picture Completion
F - Forward
B - Backline

* - Estimated IQ calculated using highest single subtest score.

PRO-RATED IQ SCORES AND LEVEL OF EDUCATION - UNDER 21 RUGBY

NO	C	PC	PREMORBID IQ	EDUCATION (years)
1 B	12.0	8.5	102	12
2 B	10.0	10.0	100	12
3 F	10.0	12.5	110	14
4 F	14.0	14.5	136	14
5 F	10.5	9.5	100	13
6 B	9.5	10.0	98	10
7 F	9.5	14.5	117*	15
8 F	11.0	12.5	114	12
9 F	11.5	11.0	110	12
10 B	13.0	12.5	122	13
11 F	8.5	10.5	96	11
12 F	12.0	9.5	106	13
13 F	13.0	10.5	114	13
14 B	10.0	12.5	110	14
15 B	8.0	12.5	121*	12
16 F	10.5	8.5	96	8
17 F	11.0	14.5	122	12
18 B	11.0	11.0	108	12
19 B	8.5	12.5	104	12

Key:

C - Comprehension
PC - Picture Completion
F - Forward
B - Backline

* - Estimated IQ calculated using highest single subtest score.

PRO-RATED IQ SCORES AND LEVEL OF EDUCATION - HOCKEY

NO	C	PC	PREMORBID IQ	EDUCATION (years)
1	14.0	12.5	128	13
2	7.5	12.5	121*	12
3	12.0	12.5	118	16
4	14.0	14.5	136	15
5	10.0	15.0	121	15
6	13.5	14.5	133	14
7	14.0	14.5	136	12
8	15.5	11.0	128	16
9	11.5	14.5	125	14
10	13.5	10.0	114	12
11	13.0	12.5	122	15
12	10.0	12.5	110	16
13	11.5	14.5	125	15
14	13.5	12.5	125	14
15	11.5	11.0	110	15
16	13.0	15.0	133	15
17	11.5	14.5	125	15
18	11.5	10.0	106	14
19	9.5	12.5	108	14
20	12.5	12.5	121	15
21	10.0	12.5	110	14

Key:

C - Comprehension
PC - Picture Completion
F - Forward
B - Backline

* - Estimated IQ calculated using highest single subtest score.

Appendix B

Assessment schedule:

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)

Appendix C

Protocols:

Neuropsychological Test Battery

DIGIT SYMBOL SUBSTITUTION

Testee's Name:_____

Requirements: Test sheet
Pencil
Stop watch

TIMED

Time Limit: 90 seconds (1 minute 30 seconds)

Instructions: *Place the Digit Symbol sheet in front of the subject and indicate the key at the top.*
"Look at these little boxes or squares. You will notice that each has a number in the upper part and a sign or mark in the lower part. Every number has a different sign *(indicate)*. Now, down here *(point to the sample)* there are some more of the boxes, but this time they only have the numbers at the top and the spaces below are empty. You have to put into each of the spaces the mark that belongs (corresponds) to the number at the top. The first number is 2, so we have to put in this mark *(pointing to the key - examiner fill in the 2-sign)*. The next is a 1, so we put in this mark *(indicating the sign and filling it in)*.

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

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

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

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

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

AAM / NAME ..... Datum / Date .....

SLEUTEL
KEY

1	2	3	4	5	6	7	8	9
–	⋀	⊐	L	∪	O	∧	X	=

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

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

DIGIT SYMBOL SUBSTITUTION - INCIDENTAL RECALL

Testee's Name: _____

Requirements: Test sheet
Pencil

NOT TIMED

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

SCORE:

Number remembered correctly: _____

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

NAAM
NAME .....

Datum
Date .....

SLEUTEL
KEY

1	2	3	4	5	6	7	8	9

TRAIL MAKING

Requirements: test sheets (4 pages)
pencil
Stop watch

TIMED

Instructions:

TRAIL A:

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

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

Now draw a line to connect the circles consecutively from 1 to 25, without lifting your pencil, and do it as fast as you can.

Record time

TRAIL B:

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

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

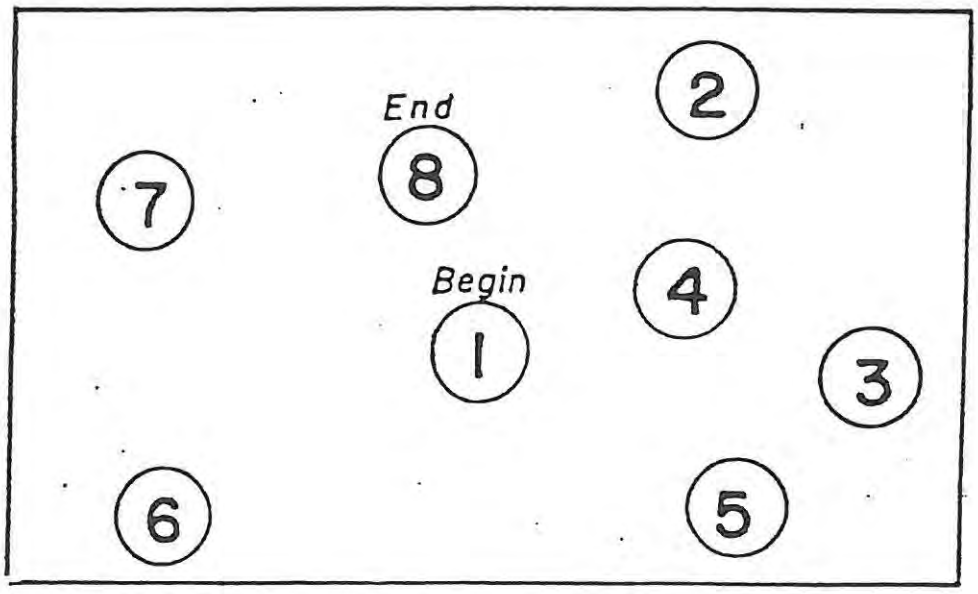
Draw a line to join the circles consecutively by alternating between 1 and A, as fast as you can.

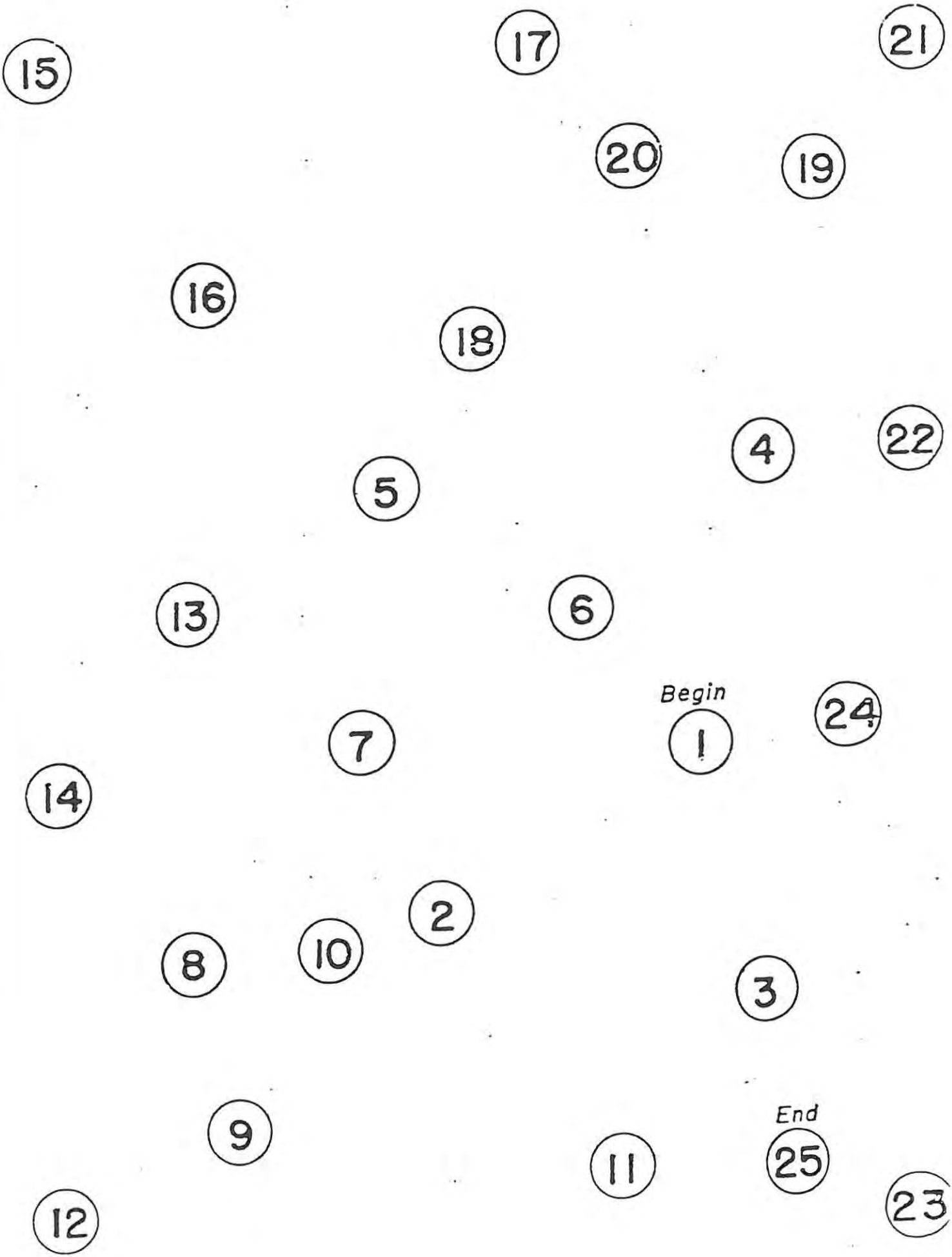
(Note: If subject makes mistake, **don't stop timing**; point out mistake and subject carries on).

TRAIL MAKING

Part A

SAMPLE

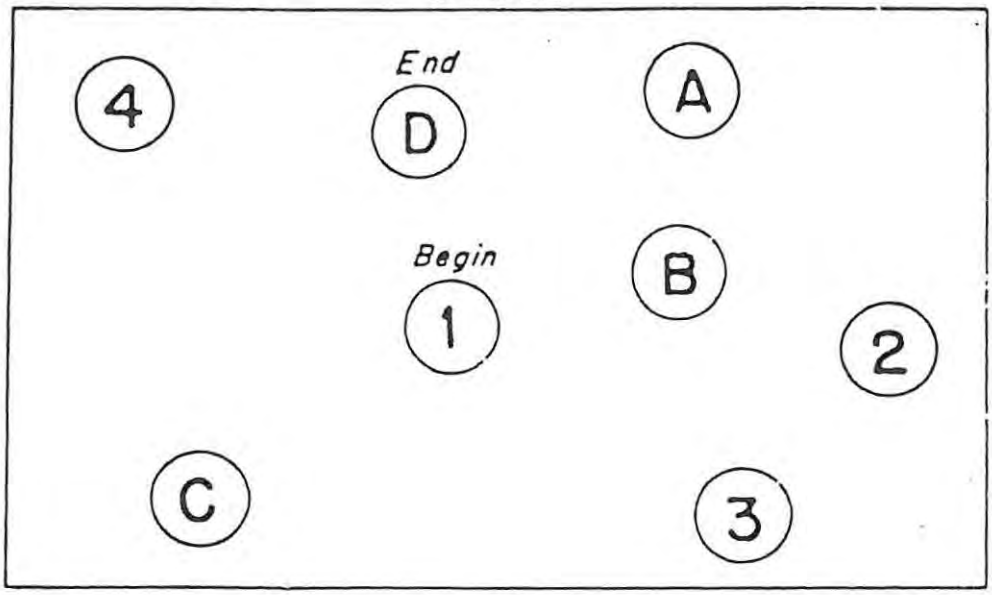


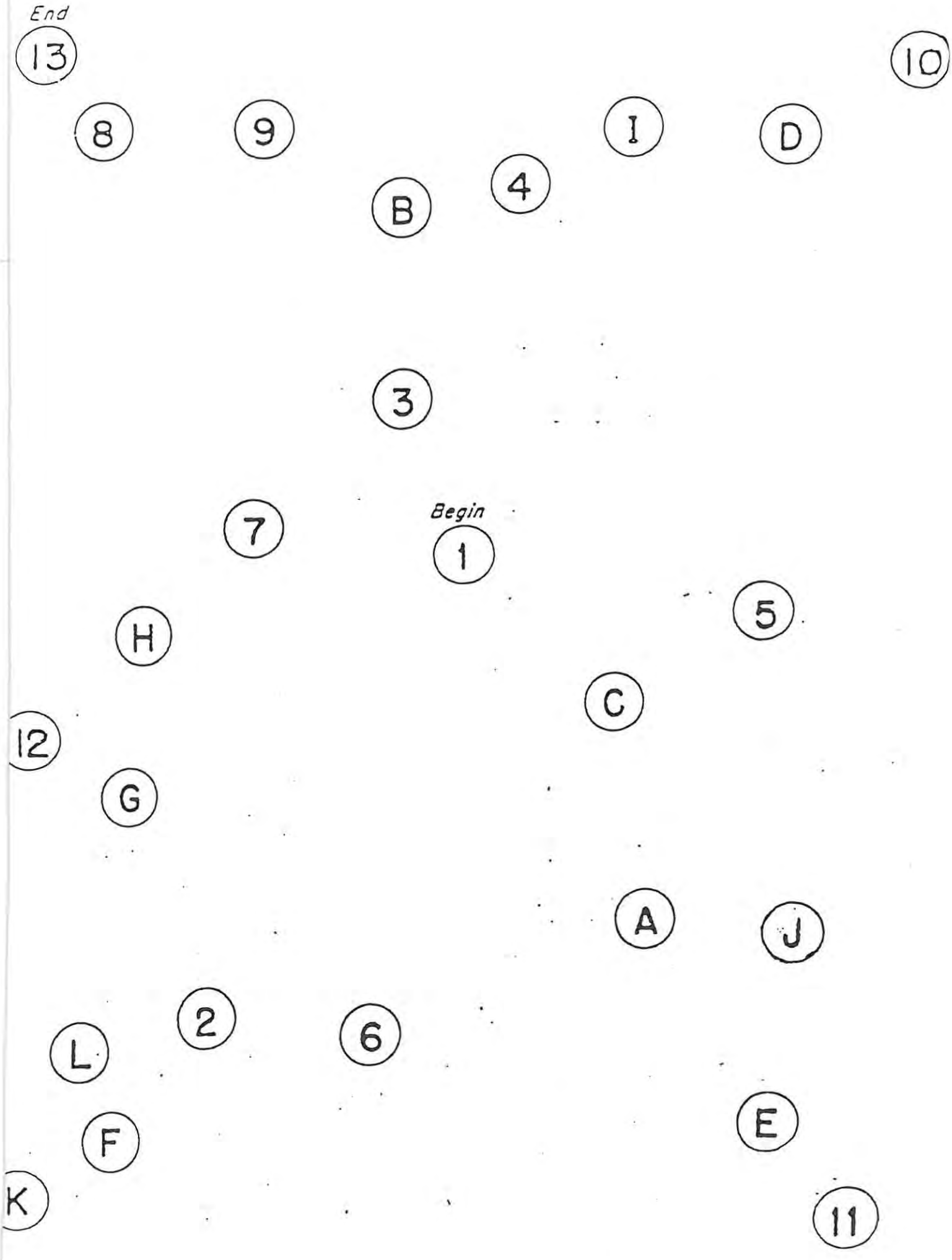

15
17
21
20
19
16
18
4
22
5
13
6
Begin
24
7
1
14
2
8
10
3
End
9
11
25
23
12

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

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

Hard: 1. ____
2. ____
3. ____
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 D

Consent Form:

Phase I and Phase II

NEUROPSYCHOLOGICAL ASSESSMENT

CONSENT FORM

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

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

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

Signed: Date:

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:_____

Appendix E

Questionnaire:

Demographic Data and Injury History

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 F

Questionnaire:

Postconcussive Symptom Checklist

RHODES UNIVERSITY PSYCHOLOGY DEPARTMENT

Symptom Check List

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

NAME:_____

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

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

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

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

18. Does your attention wander while following a conversation

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

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

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

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

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

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

0 Never 0 Sometimes 0 Often

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

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

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

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

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

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

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

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