DO CUMULATIVE MILD HEAD INJURIES IN RUGBY AFFECT NEUROPSYCHOLOGICAL PERFORMANCE? A COMPARATIVE STUDY BETWEEN CLUB RUGBY PLAYERS AND NON-CONTACT SPORT ATHLETES.

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Abstract

Context: Concussion is a major sports medicine concern that is currently under scrutinisation worldwide. Well-publicised cases of careers ending due to multiple concussions, and the potential for permanent, disabling neurocognitive deficits have raised concerns and encouraged further research to take place.

Objectives: This study aimed to investigate subjects exposed to mild head injuries with the aim of determining if neurological sequelae are detectable. The objectives of this study were to evaluate changes in neuropsychological performance over a period of playing rugby for one full season, which extended over nine months. This study investigated the relationship between concussion history and neuropsychological performance relating to the possible cumulative effects of concussion. Neuropsychological functioning in recently concussed athletes was compared with that of non-injured (control) athletes to detect whether neurological sequelae were present. Investigation into the relationship between post-concussion symptoms and neuropsychological performance was evaluated. The position of play was analysed to see if there were any measurable differences in neuropsychological performance present between forward and backline players.

Design, Setting, and Participants: 35 club rugby players and 35 non-contact sports athletes were assessed over a period of 9 months. Both groups underwent pre-season baseline testing and post-season testing. A comprehensive battery of reliable and valid neuropsychological tests was used to assess these subjects, with particular focus on the following 5 areas of cognition: planning, visuo spatial and constructional ability; attention and concentration; memory; verbal fluency and speed of information processing.

Results: The data showed that significant differences occurred in rugby players participating regularly in the sport over one full season in terms of changes in neuropsychological test performance in a range of cognitive domains, including planning, visual spatial and constructional ability, attention and concentration, memory and verbal fluency. Numerous significant relationships were found between certain Post Concussion Symptom Scale (PCSS) scores and poor neuropsychological performance, which were considered indicative of subtle effects of sub-concussive injuries and mild head injury (MHI). Surprisingly, following the assessment of concussed players during the season, the data did not show any reliable significant declines in cognitive performance compared to their baseline testing. However, mean scores of the concussed group did show a trend of

decreased neuropsychological performance in almost every cognitive domain following the concussive injury. The data did not show any significant relationship between a history of three or more previous concussions and neuropsychological performance. Furthermore, no significant differences in neuropsychological performance between backline and forward players were evident.

Conclusion: This research demonstrates that concussion can present serious consequences for athletes and warrants the attention it has received. This present study gives a clear description of the potential negative consequences of playing rugby, which are evident by looking at the change in scores between pre- and post-season testing and poorer performance in most neuropsychological measures following a concussive injury. Although this study dealt mainly with 'normal' players, the results shown here are a cause for concern. What has become evident is that the player need not be exposed to severe concussion in order to experience some form of cognitive impairments. Even if these impairments are minimal, they are however still present and have the potential of accumulating, which could lead to disastrous permanent deficits.

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INTRODUCTION

Over the past few years, extensive research has been done on rugby league players regarding cognitive impairments following concussive injuries (Barth, Alves, Ryan, Macciochi, Rimel, Jane, & Nelson, 1989; Maddocks & Sailing, 1991, 1995; Hinton-Bayre, Geffen & McFarland, 1997; Edwards, 1993; Collins & Hawn, 2002; Iverson, Gaetz, Lovell & Collins, 2004), and the findings have often been complimentary as well as contradictory. The need for further development and more evidence-based research was emphasised at the Symposium on Concussion in Prague in 2004. There, they concluded that such research is vital in contributing to the current field of knowledge regarding the phenomenon of concussion, and will potentially provide valuable information which will inform important issues such as: educating athletes and their health care providers regarding the detection of concussion, its clinical features and assessment techniques, clinical management, rehabilitation, return-to-play guidelines, and long-term outcomes of concussive injuries. Other reasons to conduct this type of research include that it aids proper management of concussed players, which aims to minimise the potential damaging and long-term negative consequences of mild head injury which rugby players risk sustaining during their sporting career (Symposium on Sports Concussion, Prague, 2004).

The clinical nature of sport-related concussion will be discussed in this paper. I will highlight the difficulties with definitions, classification of injury, injury severity grading, and the understanding of clinical symptoms. In addition, I will discuss in detail the well-recognized sequelae of concussion including the neuropsychological effects of various cognitive domains. Where possible, an evidence-based approach is adopted to assist the

understanding of the literature in this complex area. Due to the multiplicity of published and non-published articles in this area, specific articles were selected in order to give an overview of the complexity and contradictory nature of this developing area of 'sports concussion'.

Seventy research participants took part in this study. The rugby sample consisted of 35 rugby league players and the control group consisted of 35 non-contact sport athletes. The two groups were randomly selected and the following demographic details were taken into consideration when comparing baseline neuropsychological test results: age, level of education, any professionally diagnosed learning difficulty, use of medication, years playing rugby and previous number of concussions. Neuropsychological data were collected pre-season (to obtain baseline scores) and post-season, for both the control and rugby groups. Their baselines scores were compared to their end-of-season scores to observe if there were any measurable differences in neuropsychological functioning as a result of possible mild head injury sustained during play. Players who had sustained a concussion were re-assessed within 36 hours of their injury and differences in neuropsychological performance were noted. The reason the subjects were re-assessed following a concussive injury was to observe if there were any measurable declines in neuropsychological functioning, to assess what area of cognitive functioning is most affected by concussion, and to note which neuropsychological tests are most sensitive in detecting the subtle signs of mild head injury sustained in rugby play. During post-season testing, all research subjects were re-assessed using alternate forms of these tests as a means to minimise the benefits of practice effects. This was done because there is thought to be a relationship between mild head injury and accumulating subtle neuropsychological deficits.

The purpose of this study is multi-fold in that it aims to expand our understanding of the signs, symptoms and cognitive effects of concussive injuries. This research was conducted in order to try and improve the current understanding and identification of mild head injuries, which aims to help with clinical management of injured players and return-to-play decisions. Within this study, cognitive change focuses specifically on: planning, verbal fluency, visual and auditory memory, speed of information processing, and attention and concentration. The reason for this choice of cognitive functions as well as the rationale for the below-mentioned objectives will be explained in detail within the literature review.

Research Hypotheses

1. To assess what happens to the rugby players' neuropsychological performance over a 9-month period of playing rugby. It is hypothesised that the control group scores will remain constant or may benefit from practice effects between pre- and post-testing sessions over the testing interval; however it is presumed that the rugby players' performance would remain the same over that testing interval, or show possible deterioration in post-season test scores.

2. When players report concussion during the rugby season and are assessed within 36 hours of their injury, is there a significant change from their baseline levels of performance? If so, on what measures do these individuals differ? It is hypothesised that concussion scores should be significantly poorer than their baseline scores, particularly in relation to areas of planning, visuo spatial and constructional ability; attention and concentration; memory; verbal fluency; and speed of information processing. Are there

specific areas of neuropsychological functioning that are more severely affected than others as a result of this concussion?

3. To assess the relationship between the individually rated Post-concussion Symptom Scale (PCSS) scores and post-season neuropsychological scores. It is hypothesised that the more severe the post-concussion symptoms, the more the post-season test results will be negatively affected.

4. To assess whether players reporting a history of 3 or more previous concussions have lower scores at baseline compared to the control group. It is hypothesised that players with a history of previous concussion will have lower baseline scores as a result of the possible cumulative effects of concussion.

5. To assess whether there are any differences between the forward and backline players' neuropsychological performance at both or either of the measurement intervals. It is predicted that as the 'forwards' participate in more scrums, mauls and tackles, and as such are exposed to more impact and thus may be more prone to mild head injuries, their neuropsychological performance in sensitive domains of performance will be more affected than the backline players' performance in the same domains.

Rationale for the Research Objectives

The mechanisms involved in mild head injuries sustained by rugby players are a result of diffuse brain damage of nerve fibres and blood vessels, without local signs ((National Health and Medical Research Council (NHMRC), 1994)). This diffuse brain damage may

lead to generalised reductions in memory, decision-making, speed of information processing, and memory storage and retrieval deficits (Lezak, Howieson, Loring, Hannay, & Fischer, 2004). Thus, a comprehensive battery of neuropsychological tests was selected that has been proven to be sufficiently sensitive to detect the neuropsychological effects of concussion and mild head injury (Lovell, Iverson, Collins, McKeag & Maroon, 1999; Maddocks & Sailing, 1995; Maddocks & Sailing, 1996; Collie, Makdissi, Maruff, Bennell & McCrory, 2006; Lovell et al., 2003; Barth et al., 1989 & Gronwall & Wrightson, 1981). Research around the subtleties of the various neuropsychological tests is raised in the literature review.

As a result of playing rugby over one full season, and due to the underreporting of possible concussive injuries (Lovell et al., 2003), it is likely that some players were indeed exposed to some form of mild head injury although they did not feel the need to report the possible injury, or underestimated the neurological symptoms they were experiencing. This could become evident when looking at the ***difference between pre- and post-season test scores.*** Previous research conducted in the field of sports-related concussion is discussed in the literature review. Conclusions from a variety of sources, namely previous research, propose that it is indeed likely for the control group scores to remain constant over a season, or even improve over this period due to "practice" effects, whereas the players' performance would remain the same, or even deteriorate, thus suggesting that they did not benefit from practice effects. These findings are explained by the possible subtle effects of mild head injury some of the players endured during the season of playing rugby.

The traditional approach to concussive brain injury utilising loss of consciousness as the primary measure of injury severity, has acknowledged limitations in assessing the severity of concussive injury (McCrory, Makdissi, Davis & Collie, 2005). The fact that

the effects of concussion can be subtle and are often not recognised by the athlete does not necessarily mean that the consequences for an individual will be trivial (Collins et al., 1999). Boll (1983) refers to this as the 'quiet' or 'silent' minor head injury. Edwards (1993, pp.41) considers the lack of obvious visibility of effects in itself to be 'particularly pernicious'. In order to assess if players were experiencing any subtle effects of concussion, they were required to fill out a ***Post-concussion Symptom Scale*** **(PCSS)** following every game played during the season. The purpose of this was to assess if the players did indeed experience any subtle effects of concussion, which would possibly affect their post-season test results, even when they were unaware of the presence and seriousness of their post-concussion symptoms.

It is a common assumption in sports medicine that a ***prior history of concussion*** is predictive of a lowered threshold and worse outcome following subsequent concussive injury (Lovell, et al., 2003). The possible cumulative effects of mild head injury is further explained in the literature review. Since it was stated by Quigley's Rule (Schneider, 1973, as cited in Lovell et al., 2004) that athletes should discontinue participation in sports following three consecutive concussions due to the possible negative and dangerous cumulative effects of concussion, I decided to use three and more concussions as a cut-off level for assessing this hypothesis. As a result, this study aimed to assess if any differences in neuropsychological performance were present between those players who had a history of 3 or more previous concussions and the control group who had no history of previous concussions. The time differences between the previous concussion/s and baseline assessment varied from 1 year to 15 years, depending on the player's cumulative years of playing ruby. This hypothesis focused on the rugby group at large, and thus calculated the group mean scores; hence individual differences were not taken into account with regards to length of times between each individual concussion. Adequate

time for post concussion recover was provided between the players last concussion and their pre-season baseline test.

In terms of the ***positional influences*** on exposure to concussion, it has been confirmed that for rugby, and football at adult level, forward players are involved in more rucks, mauls, scrums and tackles compared to the backline players (Shuttleworth-Edwards, Border, Reid & Radloff 2004). In Shuttleworth-Edwards et al., 2004, they highlight previous studies which have found forwards to sustain significantly higher frequency of injuries, and more injuries to their heads and necks compared to the backline players (Davies & Gibson, 1978; Gissane, Jennings, Cumine, Stephenson & White, 1997; Lingard, Sarrock & Salmond, 1976; Jakoet & Noakes, 1998; Seward et al., 1993). Impacts at these sites often lead to the shearing of neurons that reportedly affect neuropsychological performance (Shuttleworth-Edwards et al., 2004), and thus this study aimed to see if any differences were evident between the neuropsychological functioning of forward and backline players.

LITERATURE REVIEW

Brain injury suffered in organised sports has been the focus of increasing attention from medical personnel of the administrative bodies of various sporting codes. It appears that a significant proportion of rugby players will receive mild concussive head injuries during a season due to stresses and impacts on the head and neck during scrumming, tackling and collision between players (Edwards, 1993). The two most serious sequelae to mild traumatic brain injury (concussion) are possible irreversible and crippling cognitive deficits, or death due to Second Impact Syndrome (SIS) (Grindel, Lovell & Collins, 2001). Since the majority of the players will receive 'knocks' to their head over a period of years of participation in this sport, it seems imperative that aspirant rugby players should be made fully aware of any potential negative consequences of such injury (Edwards, 1993), because successive head injuries may place the athletes at risk of permanent neurological damage, and they need to make an informed decision about their participation in the sport.

Efforts to protect athletes from prematurely ending their career due to injury, or experiencing possible permanent disabling neurological injuries have led to increased efforts in professional sport to evaluate the injured athlete more effectively and thoroughly (Lovell & Collins, 2001). This means that players, coaches, and teams will need to be more aware about monitoring, assessing and managing concussive injuries, and are strongly advised to follow validated return-to-play protocols, with the aim being to minimise the potential serious short-term and long-term consequences of concussive injuries.

Incident Studies

Head trauma and fatal injuries have been noted in numerous contact sports such as boxing, soccer, rugby, wrestling, grand prix motor racing, baseball, to name but a few. The morbidity and mortality linked with traumatic brain injury have been labelled a 'silent epidemic' because to date, they have received surprisingly little attention compared with other neurological illnesses (Mueller, 2001). It is conservatively estimated that 300,000 sports-related brain injuries occur per year in the United States, 250,000 of which are seen in high school football alone (Mueller, 2001). The results of a study conducted by Mueller (2001) showed that a football-related fatality has occurred every year from 1945 to 1998. The study also showed that from 1984 to 1999, 69 football head-related injuries resulted in permanent disability.

While it appears that football has received the most attention, it is worth noting that from 1982 to 1999, 20 deaths and 19 permanent disability injures occurred in a variety of other sports (Mueller, 2001). Incidental studies reveal that concussion in Rugby League[1] accounts for at least 8.5% of all injuries and approximately 8 injuries per 1000 hours played (Hinton-Bayre, Geffen & McFarland, 1991). In Shuttleworth-Edwards et al. (2004), Jakoet and Noakes (1998) focused their study on assessing the frequency of injury sustained in the 1995 Rugby World Cup by 416 rugby players from 16 different countries. Their results suggested a very high injury risk in Rugby Union,[2] especially amongst the best players, which challenges the previously held view that a player's experience, fitness and skill lower the risk of rugby injury (Shuttleworth-Edwards et al., 2004).

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[1] **Rugby league football** is a full-contact team sport played with a prolate spheroid-shaped ball by two teams of thirteen on a rectangular grass field. Rugby league is one of the two major codes of rugby football, the other being rugby union.

[2] **Rugby union** (short for **rugby union football**) is an outdoor sport played with a prolate spheroid-shaped ball by two teams of fifteen players.

According to Jakoet and Noakes (1998), cited in Shuttleworth-Edwards et al. (2004), most Rugby Union injuries occur during the 'tackling' phase of play, followed by rucks and mauls, and there appears to be dangerously high positive relationships between concussive injury and speed of the game, size of players (height and weight), and level of competitiveness. Shuttleworth-Edwards et al. (2004) report that the majority of studies conducted in the area of injury incidence in Rugby Union showed that a high proportion of injuries (25-50%) occurred to the head, face and neck. According to Shuttleworth-Edwards et al. (2004), from comparative studies it has become evident that compared with soccer, American football, Rugby League and Australian Rules football, Rugby Union games appear to be the most dangerous sport, and also the most susceptible to incidences of concussion.

While most of these reported statistics involve mild traumatic brain injury (MTBI), there were an estimated 900 deaths per year in sports and recreational activities due to an injury to the brain (Grindel, Lovell & Collins, 2001). Although the majority of athletes who experience a concussion are likely to recover, the incidence of chronic cognitive and neurobehavioral difficulties related to the current injury is not yet clearly known. Thus, the long-term negative consequences of such injuries are currently under investigation.

The statistics presented are a major cause for concern and warrant more effective management strategies and return-to-play protocols. This is especially critical since research has shown that, in reality, the prevalent rates of concussion within rugby have been severely under-reported (Marshall & Spencer, 2001), which highlights the added importance for health professional and athletes to take heed of new data published, and reiterates the need for researchers to continue their work in this area.

Definitions of Concussion

No universal agreement on the standard definition or nature of concussion exists (McCrory & Johnston, 2002). Over the past few decades the definition of concussion has changed and further developed as the understanding and epidemiology of concussion is under constant review. Various definitions of concussion have been reported in the literature over the past 100 years. Contemporary definitions of this injury assume a neurophysiological rather that neuroanatomical basis for concussion (Lovell et al., 2006). The term 'concussion' has been used interchangeably with the term 'mild head injury' (MHI) and 'traumatic brain injury' (TBI). The Committee on Head Injury Nomenclature of the Congress of Neurological Surgeons (1966) proposed the following definition of concussion: 'a clinical syndrome characterised by immediate and transient impairment of neural functions, such as alteration of consciousness, disturbance of vision, equilibrium, etc., due to mechanical forces' (p. 387). However, this definition was criticised as being too narrow: it did not address the common symptoms of concussion, such as headache, nausea, and so on, and it did not include minor impact injuries that result in long-term physical or cognitive symptoms (Aubry et al., 2002). The American Academy of Neurology (AAN) Guidelines defines concussion as 'a trauma-induced alteration in mental status that may or may not involve loss of consciousness' (Maroon et al., 2000). The American Orthopaedic Society for Sports Medicine (AOSSM) Concussion Workshop Group defined concussion as "any alteration in cerebral function caused by a direct force or indirect (rotation) force transmitted to the head resulting in one or more of the following acute signs or symptoms: a brief loss of consciousness (LOC), light-headedness, vertigo, cognitive and memory dysfunction, tinnitus, blurred vision, difficulties in concentrating, amnesia, headache, nausea, vomiting, photophobia or a

balance disturbance. Delayed signs and symptoms may also include sleep irregularities, fatigue, personality changes, an inability to perform usual daily activities, depression or lethargy" (Wojtys et al., 1999 p.676).

The area of sports concussion has been under investigation due to the possible health concerns for those players who experience one or more concussions. These concerns led to the development of a multi-disciplinary working party consisting of neurologists, neurosurgeons, sports psychologists and other professionals, called the 'Concussion in Sport Group' (CIS) (Aubry et al., 2002). The first meeting was held in Vienna in 2001, and focused on providing recommendations for the safety and health of athletes who suffer concussion. The multi-disciplinary team addressed issues of "epidemiology, basic and clinical science, grading systems, cognitive assessment, new research methods, protective equipment, management, prevention, and long-term outcome, and to discuss a unitary model for understanding concussive injury" (Aubry et al., 2002, p. 6). It also provided incentives for researchers by concluding that there was insufficient research to establish evidence-based guidelines for return-to-play (Aubrey et al., 2002). The second meeting was held in Prague in 2004, and aimed to expand on principles highlighted in the Vienna symposium and to further develop conceptual understanding of concussive injuries occurring within sport (McCrory, Johnston, Meeuwisse, Aubry, Cantu, Dvorak, Graf-Baumann, Kelly, Lovell & Schamasch, 2005). This meeting produced new definitions of concussion and a more precise description of return-to-play protocols. A general revision of the Vienna recommendations was also discussed (McCrory et al., 2005). The Prague group described two types of concussion: ***simple concussions***, which resolve within 7 to 10 days of injury, and ***complex concussions***, which cause persistent symptoms and are consistent with what is described as post-concussion syndrome.

As a result of shortfalls in the various 'concussion definitions', the CIS tried to standardise the definition and defined concussion as: "a complex pathophysiological process affecting the brain, induced by traumatic biochemical forces. Several common features that incorporate clinical, pathological and biochemical injury constructs that may be used in defining the nature of a concussive head injury include:

1) Concussion may be caused by a direct blow to the head, face, neck or elsewhere on the body with an 'impulsive' force transmitted to the head.
2) Concussion typically results in the rapid onset of short-lived impairment of neurological functioning that resolves spontaneously.
3) Concussion may result in neuropathological changes, but the acute clinical symptoms reflect a functional disturbance rather than a structural injury.
4) Concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course.
5) Concussion is typically associated with grossly normal structural neuroimaging studies" (McCrory et al., 2005, p.7).

This definition has now become the most widely recognised and accepted definition of concussion within the field of sports-related concussive injuries, and is well used throughout the new literature.

Mechanisms involved in Mild Head Injury

Despite more than 1000 years of medical research, the nature and pathophysiological basis of the clinical symptoms of mild head injury still remain unclear and are continuously under debate (McCrory et al., 2005). Mild traumatic brain injury (MTBI) is

characterised by immediate physiological changes conceptualised as a multilayered neurometabolic cascade in which affected cells usually recover, although under certain circumstances a small percentage of these cells may degenerate and die. The primary pathophysiologies include ionic shifts, abnormal energy metabolism, diminished cerebral blood flow, and impaired neurotransmissions (Iverson, 2005).

According to Lovell & Collins (2001), in terms of neuropathology, four basic mechanisms may account for concussion or MHI: 1) rotational/acceleration forces, 2) linear or translational acceleration, 3) carotid injury, and 4) impact deceleration. Maximum brain injury occurs beneath the point of cranial impact (coup injury) when a forceful blow hits the resting, movable head. This is the situation when the head is in a resting state and is forcibly struck by another object such as an opponent's football helmet of "left hook" (Poirer, 2003). This can be referred to as *contact force* resulting in static injuries. Damage occurs as a result of the inward moulding of the skull at the point of impact as well as the compensatory adjacent out bending followed by rebound effects (Lezak et al., 2004). When a moving head hits a moving object, maximum brain injury is produced on the side opposite the site of cranial impact (contrecoup injury) as the brain shifts within the bony skull (Lezak et al., 2004). When the head is accelerated before impact, the brain lags towards the trailing surface, thus squeezing away the cerebral spinal fluid (CSF) and creating maximal shearing forces at this site (Guskiewicz et al., 2004). This can also be referred to as *inertial forces,* which involve translational acceleration whereby the head moves in a straight line with the brain's centre of gravity, or rotational acceleration whereby the brain rotates around its centre of gravity often causing neuronal damage via the shearing of neurons (Lezak et al., 2004).

The majority of sports-related concussions are the result of a combined coup and contrecoup mechanism, involving damage to the brain on both the sides of initial impact

and the opposite side of the brain due to brain lag (Guskiewicz et al., 2004). An applied force to the brain such as a boot to the head, or the head striking the ground during a tackle, results in the injured brain being subjected to three types of stresses: compressive, tensile, and shearing stress. Compression stress involves a crushing force where the tissue cannot absorb any additional force or load; tensile stress or tension involves the pulling or stretching of neural tissue; and shearing stress involves a force that moves across the parallel organization of the neural tissue (Guskiewicz et al., 2004). Brief, uniform compressive stresses are thought to be fairly well tolerated by neural tissue, but tension and shearing stresses are very poorly tolerated, and thus the type of stress on the brain has a direct effect on the severity of the injury (Guskiewicz et al., 2004).

The National Health and Medical Research Council (NHMRC) suggest that the mechanisms involved in mild head injuries sustained by rugby players are a result of diffuse brain damage of nerve fibres and blood vessels, without local signs (Lovell, Collins, & Bradley, 2004). Diffuse brain injury can result in widespread or global disruption of neurologic function and are not usually associated with macroscopically visible brain lesions except in the most severe cases (Guskiewicz et al., 2004). An important type of brain damage that occurs in closed head injury results from the combination of translatory force and rotational acceleration of the brain within the bony structure of the skull, which Edwards (1993) refers to as the Acceleration-Deceleration model. The effects of these immediate disturbances in neurological functions created by the mechanical forces of rapid acceleration/deceleration are called "concussion".

The movement of the brain within the skull places tension on delicate nerve fibres, and blood vessels stretch to the point of shearing. Shearing effects, in the form of microscopic lesions, occur throughout the brain, and tend to be concentrated in the frontal and temporal lobes (Lezak *et al.,* 2004). Acceleration and deceleration, with additional

rotational forces, cause damage to delicate axons in the form of tearing and shearing, and are specifically focused in the cerebral and brain stem white matter, such as the reticular formation structures, which are areas responsible for breathing, heart rate, and wakefulness (Lezak et al., 2004). Thus, rotational velocity appears to play a significant role in producing loss of consciousness in concussion (Lezak et al., 2004). The damage of these axons is referred to as *diffuse axonal injury* (DAI), which is the most severe type of diffuse injury because of its negative consequences on cognitive performance (Guskiewicz et al., 2004). This diffuse brain damage becomes the site of degenerative changes and scar tissue, and thus is likely to lead to generalised reductions in a range of cognitive domains, namely attention, decision making, speed of information processing, and memory storage and retrieval deficits (Lezak et al., 2004). Cognitive difficulties associated with diffuse damage resulting from concussive injuries have become apparent in relevant neuropsychological tests, which will be highlighted later in the literature review.

Clinical Symptoms of Concussion and Post Concussive Syndrome

Concussion is characterised by a number of post-concussive symptoms that have a range of features which are *cognitive* (memory, concentration and processing speed problems), *typical* (vomiting, dizziness, nausea), *physical* (balance problems, numbness or tingling sensations) and *emotional* (anxiety, sleep disturbance, depression) in nature (Aubry et al., 2002). Within the American Academy of Neurology (AAN) practice parameter guidelines (1997), the symptoms that an athlete may experience can be divided into "early" and "late" categories, although the time periods of the presence of these symptoms vary according to each individual and the severity of the injury sustained.

Early symptoms last from minutes to days, and include headaches, dizziness, lack of awareness of surroundings, nausea, or vomiting. Late symptoms last from days to weeks and include persistent low-grade headache, light-headiness, poor attention and concentration, memory dysfunction, easy fatigability, irritability and low frustration tolerance, intolerance of bright light or loud noises, emotional disturbances such as anxiety or depressed mood, and sleep disturbances. According to McCrory & Johnston (2002), the only validated symptoms are headache, dizziness, nausea, blurred vision, attention deficit, amnesia, and loss of consciousness. Despite these symptoms being linked to concussive injury, surprisingly few athletes recognise the link, which results in the frequent underreporting of concussive injures (McCrory & Johnston, 2002). In 1997, Delaney and his colleagues conducted a study on Canadian footballers, and found that more that 4 out of 5 players did not realise they had experienced a concussion (Delaney, Lacroix, Leclerc & Johnston, 2000). This has consequences for the management of concussive injuries and future research in this field. Due to players' reluctance to report symptoms, the use of neuropsychological testing as objective indices of concussion may be useful in helping to diagnose and grade a concussion (McCrory, Makdissi, Davis & Collie, 2005).

Post Concussive Syndrome (PCS), a sequelae of mild head injury, was first proposed in the 19th century and refers to prolonged, disabling and sometimes permanent symptoms such as headaches, dizziness, tinnitus, depression, irritability, slowed mental processing, impaired attention, and deficits in memory (Thurman, Branche & Sniezek, 1998). Since the 19th century, PCS has been a controversial diagnosis due to conflicting findings regarding symptom duration, an absence of objective neurological findings, inconsistencies in presentation, poorly understood etiology and a significant amount of methodological problems with literature (Legome & Wu, 2006). No one definition is

accepted by all health professionals, and it is still an area of continual debate: are the symptoms cause by microscopic lesions, or are the symptoms driven by psychological reasons? (Legome & Wu, 2006). It is commonly understood that physiological and psychological factors before, during, and after the injury all take part in the development of PCS (Ryan & Warden, 2003). McCrory & Johnston (2002) report that there are two distinct schools of thought regarding the pathophysiologies of PCS, however the relative contribution of these two mechanisms remain unclear. The first group proposes that the symptoms associated with PCS are an immediate result of concussion. The second group refers to the symptoms as functional and as such are indicative of psychological or emotional sequelae of the concussive injury.

In a study conducted by Gouvier, Cubic, Jones, Brantley & Cutlip (1992), researchers found no difference between MHI and normal groups on the frequency, intensity, or duration of PCS symptoms, although interestingly each of these co-varied with fluctuating levels of daily stress. Gouvier et al. (1992) concluded that the presentation of post-concussion symptoms are directly affected by a variety of external and intrapersonal factors, such as an individual's level of psychosocial stress, particular coping style or cognitive appraisal strategies, and thus should be considered on an individual level. Although the initial cause of PCS may be physiological, psychological factors appear to play a key role in the presentation and maintenance of these PCS.

In a study conducted by Ferguson, Mittenberg & Barone (1999), 209 males from college and high school amateur collision sports programmes completed a 30-item symptom checklist related to post-concussion complaints. They were also required to complete a demographics questionnaire to collect information about their head injury history, which was used to assign groups: those athletes who suffered MHI during the course of the research, and those athletes who did not suffer any form of MHI over the

research period (control group). Athletes in the mild head injury group were asked to indicate symptoms that they experienced at the time of completing the questionnaire, and then to estimate which symptoms they experienced before the injury. The control group was asked to list the symptoms they experienced at the time of interview. They were then asked to 'imagine' experiencing a concussion and make a list of the symptoms they thought they would experience after such an injury. When analysing data to examine the hypothesised relationship between imagined or expected symptoms and reported concussive symptoms, it was found that the mean number of symptoms expected by controls significantly exceeded the reported symptom base rate by the injured group ($p<.0001$), and they imagined symptoms following MHI to increase by 102%. The study also found that athletes who expected an increase in post-concussion symptoms, but did not experience any actual increase, ended up underestimating their pre-morbid symptom incidence by 97% ($p<.001$) compared to the control group. The authors explained these results as a means of reconciling their perceptions and expectations, and used a cognitive-behavioural model to explain the persistence of post-concussion syndrome.

Other researchers, however, disagree, and have found that there is a direct comparison between post-concussion symptoms and impaired neuropsychological test performance, indicating the presence of some form of mild head injury. These authors highlight the benefit of regularly administering post-concussion symptom scales as a means of aiding the clinical assessment and management of possibly injured athletes. Collins et al. (2003) conducted a study in which 110 high school athletes who had suffered concussion took part, in order to investigate whether post-concussive headaches are associated with neurocognitive deficits and/or the presence of other post-concussion symptoms at approximately one week post-injury. The group were separated into those with symptoms seven days post-injury, and those without symptoms. The groups were administered the

computerized program, Immediate Post-concussion Assessment and Cognitive testing (ImPACT), within 5-10 days post-injury. The Post-concussion Symptom Scale, consisting of 19 different symptoms, was included in the ImPACT testing intervals. The results concluded that the athletes who had been concussed and who experienced post-traumatic headache performed significantly worse on reaction time ($p<.001$) and memory tasks ($p<.02$), compared to athletes reporting no symptoms. The separate groups did not differ on processing speed. The athletes with moderate to severe headaches reported a significantly greater number of other post-concussion symptoms relative to those with mild head injury ($p=.001$). The results of this study suggested that post-concussion headache is likely to be indicative of incomplete recovery from concussion. Despite the high prevalence of post-concussive headache, no current concussion grading scale includes headaches as a criterion defining the severity of injury (Collins et al., 2003).

Collie et al. (2006) conducted research on 615 male Australian footballers to assess the effects of post-concussion symptoms on neurocognitive performance, compared to those players who were asymptomatic at the time of assessment. At pre-season, a baseline battery was administered, which included both CogSport (computer-generated test), and two pencil and paper cognitive tasks (Digit Symbol Substitution Test and the Trail Making Test, part B). Sixty-one athletes (25 symptomatic and 36 asymptomatic) who had been concussed were reassessed within 11 days post-injury, and 84 controls were reassessed. The results showed that the symptomatic group performed less well on the computerized tests of simple, choice and complex reaction times, compared with the asymptomatic and control groups. On pencil and paper cognitive tasks, the symptomatic group showed no improvement in cognitive tasks; however the control and asymptomatic groups did show improvement, which was explained by their benefiting from learning and practice effects (Collie et al., 2006). Based on their research, the authors

recommended that all athletes should be withheld from further contact sport until their post-concussive and cognitive symptoms had been fully resolved and had returned to baseline levels.

Post-concussive syndrome can have disastrous consequences, as in the case of Second Impact Syndrome, which has led to approximately 30-40 deaths over the past decade in the United States alone (ImPACT, 2004, www.impacttest.com). SIS refers to the symptoms arising from suffering a second concussion to the head while still recovering from an initial injury. SIS, or rapid brain swelling and herniation after a second head injury, and is more common than previous reports in the medical literature have suggested (Lovell et al., 2003). The majority of victims have been between the ages of 13-18, suggesting greater vulnerability to severe injury in children and adolescents (Lovell et al., 2003). Returning to play while the athlete is not clear of post-concussion symptoms, which possibly indicates that the brain has not yet recovered from the initial injury, may lead to irreversible cognitive deficits or even death, although prolonged cognitive deficits can be seen after a single insult (Grindel et al., 2001).

One of the most disturbing features of mild concussive head injury is that the effects are cumulative (Edwards, 1993); that is, if a player suffers repeated injuries of this type within the space of days or weeks, the effects may be 'disastrous' (De Villiers, 1987, p164). The most probable explanation of the cumulative effects of concussion is that each event destroys neurons, diminishing the reserve available and making loss evident under the stress of further brain injury (Gronwall & Sampson, 1975; Ferguson, Mittenberg & Barone, 1999). Players who have sustained a concussion are at greater risk of impaired neuropsychological performance (Garnham, 1992; Iverson, Gaetz, Lovell & Collins, 2004a), further injury (Cremona-Meteyard & Geffen, 1994) and a possible catastrophic outcome due to SIS (Erlanger, 1999). Schulz et al. (2004) were the first researchers to

identify that a history of concussions is a potential risk factor for the prospective elevation of the incidence of future concussions. They provide two explanations for this relationship. First, if the brain has been exposed to previous insults, the ability of the brain to respond to traumatic insults may be compromised, thus making those individuals more susceptible to further concussions. The second reason relates to environmental and behavioural factors: some athletes play more minutes in a game, or are more competitive and aggressive than fellow team-mates, which place them at higher risk of stronger falls and heavier exposure to impact.

The overall issues of the cumulative effects of concussion can be understood by examining '*animal models of neuro-trauma*' (Hovda et al., as cited in Lovell et al., 2004). These models suggest that limited but significant damage to neurons can result from a single concussion. Hovda et al. (as cited in Lovell et al., 2004) explains that a metabolic dysfunction occurs within the brain when cells are immediately injured upon concussion. This results in dramatic evolving changes in both the intracellular and extracellular environments within the brain structure. The notion of a 'continuum of injury' implies that as the acceleration/deceleration forces increase, the number of cells damaged will increase, and damage will progressively occur in deeper structures causing more serious and permanent damage (Hovda, et al., as cited in Lovell et al., 2004). Numerous animal research models have shown that cholinergic fibres are more susceptible to biomechanical trauma than other neurotransmitter fibre systems, frequently leading to acetylcholine depletion and hypo function of the hippocampus. This suggests that hippocampal function and the cholinergic system may be affected in MTBI, giving us a reason for memory deficits found during neuropsychological testing (Hanlon, Demery, Martinovich & Kelly, 1999). Clearly, it is very difficult to extrapolate from animal research to concussion sustained by humans in sport, however any type of research in the field of mild or

traumatic brain injury aids the further development of theory and understanding of these complex injuries. It also raises many imperative questions regarding the threat of vulnerability, how long the symptoms last and if these injuries are accompanied by any specific, identifiable markers of both injury and recovery (Hanlon, Demery, Martinovich & Kelly, 1999). Nonetheless, a basic scientific foundation for the possible cumulative effects of concussion is apparent, and thus further investigation in this field is warranted.

The cumulative effects of mild head injury have been highlighted in various research studies, and it has been argued that players who have sustained a previous concussion are generally at greater risk of impaired performance and are likely to suffer further injury, including more concussions (Schulz et al., 2004). 'Risk factors' for concussion have been under investigation, and the question of whether a previous concussive injury has an effect on further concussive injuries has been in the spotlight in recent years.

Collins, Lovell, Douglas, & McKeag (1999) did find evidence of long-term cognitive deficits among football players with a history of two or more concussions, compared to those with none; Macciocchi, Barth, Littlefield & Cantu (2001), however, found no similar neuropsychological deficits among football players who had a history of two or more concussions compared to those athletes with only one previous concussion. Guskiewicz et al. (2002) support this claim, and in their study they found no association between chronic cognitive impairment and a history of mild concussions among collegiate players. In a study conducted by Schulz et al. (2004), on a group of high school athletes, concussion rates were found to be elevated for athletes with a history of concussion, and they increased with the increasing level of body contact permitted in the sport. After adjustment for sport, body mass index, and year in school, history of concussion(s) remained a moderately strong risk factor for concussion (rate ratio = 2.28, 95% confidence interval: 1.24, 4.19). They reported that the risk of concussion could also

be greater among those with a history of concussion for environmental and behavioural reasons, i.e. some athletes may play more games, be exposed to more intense athletic activity, and so forth. Studies of football players have shown support for this hypothesis; however, the effect strata for history of concussion within football is much stronger than the effect estimate for history of concussion for other sports, since football players are exposed to more forceful collisions than athletes in other studies (Schulz et al., 2004). The fact that concussion history is an important predictor of concussion incidence and future injury, it emphasises the importance of primary prevention measures, accurate identification, and careful clinical management of these injuries (Schulz et al., 2004).

Is Neuropsychological Testing Useful in the Management of Concussion?

Traditional neuroimaging techniques vs. neuropsychological testing

Research has shown that concussion is related to neurophysiologic factors rather than a neuroanatomical basis, and that traditional neuroimaging procedures are ineffective in detecting subtle features of concussion and monitoring injury (Lovell et al., 2006). Concussion is generally considered a functional disorder of the brain and is therefore mostly associated with normal X-rays, CAT scans, and MRIs. Seventy-five percent of a group of MTBI patients with persistent post-concussion symptoms had a normal MRI or CAT scan at the time of injury, yet later displayed temporal (75%), frontal (30%), or fronto-temporal (40%) abnormalities on PET and SPEC (Umile, Sandel, Alavi, Terry & Plotkin, 2002). Presently, no neuroanatomic or physiologic measurements can be used to determine the severity of a concussion, or are able to detect when complete recovery has occurred in an athlete after a concussion (Lovell et al., 2006). The concussed player is advised to follow through with neuroimaging techniques only if: 1) structural brain

damage is indicated, 2) the patient shows a rapid deterioration in his clinical condition, 3) the patient is displaying seizure activity, or 4) the patient has experienced a prolonged period of loss of consciousness (LOC) of more than 5 minutes (Guskiewicz et al., 2004). Although newer functional brain imaging (fMRI) protocols show promise as a diagnostic technology, fMRI is currently not available for widespread clinical use due to the lack of availability and extensive costs involved (Lovell et al., 2004). Therefore, the latest CIS group symposium concluded that neuropsychological testing and assessment has become the "cornerstone" and a "golden standard" in the role of concussion management in sports-related injuries (McCrory et al., 2004). Neuropsychological tests have been found to further enrich the management of concussion by providing an objective measure of cognitive recovery, as well as aiding understanding of the brain structures and processes underlying concussion, including post-concussion syndrome.

Neuropsychological testing has become a valuable method for evaluating symptoms of subtle concussion, and is sensitive to the subtleties of cognitive decrements associated with concussion in sports (Collins et al., 1999; Collie et al., 2006; Collins et al., 2003; Cremona-Meteyard et al., 1994; Echemendia et al., 2001; Gronwall et al., 1981). In recent literature there appears to be an emerging pattern of cognitive deficits after sports-related concussion. These include alterations in attention and concentration, speed of information processing, learning and memory, working memory, executive functioning and verbal fluency, and visuo-motor reaction times. Tests of attention and concentration (Collie et al., 2006; Collins et al., 1999; Maddocks et al., 1995; Maddocks, 1996) and memory (Barth et al., 1989; Gronwall et al., 1981; Lovell et al., 2003) have been found to be highly sensitive to change following injury. In an article written by McCrory et al. (2005), the authors report that research conducted by Barth et al. (2001) and Lenginger et al. (1990) found that tasks involving visuospatial constructional ability, language, and

sensory motor function were partially sensitive to the effects of concussion and significant differences indicating poor performance after concussion were found on certain measures. In a neurological evaluation of soccer players on the United States football team, conducted by Iverson and his colleagues (2004), the Complex Figure test proved to be sensitive in detecting symptoms of neurocognitive impairment. According to the standard Osterrieth, as used in clinical practice, 7% of the control subjects and 45% of the professional soccer players showed moderate to impaired scores (Iverson et al., 2004). Measures such as the Paced Auditory Serial Addition Test (PASAT), Choice Reaction Time, Digit Symbol subtest of the WAIS, and Smith Symbol Digit Modalities test (SDMT), which all measure reduced attention and speed of processing, were also found to be highly sensitive to the effects of concussion (Barth et al., 1989; Gronwall et al., 1981; Maddocks et al., 1989).

Based on the above literature, the tests selected for this particular study were thus purposefully selected to measure areas of cognition that have been shown to be sensitive to the effects of sports-related concussion. The SDMT has been routinely used in sport concussion research, and has been found on numerous occasions to detect the mild subtleties of concussion, and, although the results are not always significant, particular trends in performance were evident across studies (e.g., Collins et al., 1999; Hinton-Bayre et al., 1997; Macciocchi et al., 2001; McCrea et al., 2003).

Paper based vs. computer: advantages & disadvantages

The wide scale use of paper and pencil tests in sports is limited by the requirement that test administration and interpretation be undertaken by trained professionals, and that administration is usually done on a one to one basis. This makes neuropsychological testing of entire sporting teams extremely time consuming, expensive and beyond the means of most junior and amateur contact sporting organisations (Collie & Maruff,

2003). Collie & Maruff (2003) point out that the pencil and paper test batteries are also not ideal for sporting settings as they suffer from psychometric confounds that make them less ideal for serial use in sport, including a lack of equivalent alternative forms, poor test-retest reliability, and susceptibility to inter-rater biases and practice effects. However, the advantage of pencil and paper tests is that they can be performed at a stadium, do not require sophisticated equipment and can be scored immediately (Collie et al., 2003). These practical limitations have led to the development of a number of computerised neuropsychological test batteries, namely: ImPACT (Lovell & Collins, 1998), CogSport (Cogstate, 1999), and Concussion Resolution Index (Erlanger, Feldman & Kutner, 1999). These computerised batteries are designed specifically for widespread use in sports medicine, and provide a relatively inexpensive alternative as they can assess a larger quantity of athletes simultaneously. In some cases tests can be self-administered (Collie & Maruff, 2003). According to McCrory et al. (2005) there are numerous advantages of computerised testing compared to conventional pencil and paper neuropsychological tests. These include: 1) standardisation of stimulus presentation (computer software designed to control for stimulus presentation and contingency onset by minimising any inter-assessor or intra-assessor variability/unreliability); 2) quick administration; 3) heightened sensitivity of computerised programmes due to detecting deficits below measurement capabilities of traditional pencil and paper clinical neuropsychology tests; 4) minimizes the players benefiting from 'practice effects' due to presentation of multiple forms and equivalent alternative forms of a test; 5) accurate analysis of performance stability/variability; 6) accurate and efficient computerised analysis; 7) centralised data storage, analysis and reporting; and 8) quick and efficient delivery of tests due to potential internet based delivery (McCrory et al., 2005, p.5).

Implications of neuropsychological testing

Neuropsychological tests have not been found to be beneficial in assessing concussion when they are individually used, and thus 'test batteries' are designed to look for comparisons and consistencies in symptoms among different test scores (Barr, 2001). The use of multiple instruments, which measure a range of cognitive functions, offer the clinician greater potential for recognising any cognitive deficits resulting from the injury (Barr, 2001). No clear indications exist as to which individual test is most sensitive to detecting MHI, due to the multiple different presentations of concussive injuries in individual players (Guskiewicz et al., 2004). Test batteries measuring sport-related concussion should include tests that are most sensitive and susceptible to change following concussive injury (Maddocks et al., 1996; Maddocks et al., 1995; Macciocchi et al., 1996; Collins et al., 1999).

Many researchers reiterate the value of obtaining a detailed clinical history interview as part of the formal assessment process, taking demographic information into account prior to analysing and interpreting test scores (Lovell et al., 1989; Hinton-Bayre et al., 1999; Hinton-Bayre et al., 1997). Details gathered during at baseline should include information about previously diagnosed learning difficulties, neurological disorders such as ADHD, history of concussive injuries (LOC, amnesia, symptoms, recovery time, time lost from participation, etc.), as well as an understanding of multiple concussions (Lovell et al., 1989; Hinton-Bayre et al., 1999; Hinton-Bayre et al., 1997). These factors are important to consider when interpreting baseline and post-injury scores (Guskiewicz et al., 2004), as well as for determining increased risk of further injury. In a study on college footballers by Collins et al. (1999), the researchers demonstrated that learning difficulties are prevalent and effective in influencing neuropsychological test performance. During their research, a significant interaction was found between students who had a history of

learning difficulties and a history of multiple concussions on two neuropsychological measures (Trail-Making Test, Form B ($p=0.007$) and SDMT ($p=0.009$). The results also indicated poorer performance for the group who had learning difficulties and multiple concussions, compared to groups without any learning difficulties. They concluded their study suggesting that neuropsychological assessment is a useful indicator of cognitive functioning in athletes, and that both athletes with a history of repeated concussions or learning difficulties are likely to display poorer cognitive performance on a range of neuropsychological measures.

According to Grindel et al. (2001), learning effects (practice effects) must be considered when selecting and administering neuropsychological tests, especially concerning tests of memory. Other researchers have also highlighted the possibility of 'practice effects', especially when using pencil and paper tests that do not have alternative forms. Practice effects refer to the athlete improving their performance following additional testing sessions as a result of previous exposure. Grindel et al. (2001) advise that re-testing of athletes should be minimised, and equivalent forms used whenever possible.

Despite the theoretical rationale for the use of neuropsychological testing in the management of sports-related concussion, Randolph et al. (2005) disagrees with neuropsychological testing being used as the *'cornerstone'* for concussion management, as they report that no neuropsychological tests have met the necessary criteria to support a clinical application of assessing concussion at this time. He and his colleagues conducted research whereby they collated all literature on sports-concussion and neuropsychological testing between 1990 and 2004. Their data synthesis concluded that the effects of concussion on neuropsychological test performance were so subtle even during that acute phase of injury (1-3 days post-injury), that the majority of studies failed

to reach statistical evidence within group studies. They also reported that it is unclear whether neuropsychological testing can in fact detect impairment in players once concussion-related symptoms (e.g. headaches) have resolved. They believe additional research is needed to establish the utility of these tests before they can be considered part of routine standard care, and concussion recovery should be monitored via the standard clinical examination and subjective symptom checklists until neuropsychological testing or other methods are proven effective for this use.

Guskiewicz et al. (2004) believe the clinician should also be aware that any concussion assessment tool, either brief screening instruments or more extensive neuropsychological testing, comes with some degree of risk for false negatives (e.g. a player performs within what would be considered the normal range on the measure before actually reaching a complete clinical recovery after concussion). Therefore, test results should always be interpreted in the context of all clinical information, including the player's full medical history (McCrory et al., 2005). This detailed clinical history should include information about previous possible head injuries, as well as details of injuries to the neck, face or head area as these impacts could have an effect on the current clinical presentation (McCrory et al., 2004).

Assessment and Management of Concussion

Over the past few years there has been considerable amount of research conducted regarding the implementation of standardised concussion assessment strategies, various grading scales and return-to-play protocols. However, it is concerning that there are still no clear guidelines as to how to assess and manage concussion. Within the past few decades, 19 different concussion symptom scales and over 15 grading system scales and

return-to-play parameters have been published since 1973. This has led to the misuse and misdiagnosis of concussive injuries in sports due to many of the scales having differing criteria and recommendations, thus causing confusion among athletes and untrained coaches (Guskiewicz et al., 2004).

A key issue in the management of head injury is determining when to resume contact sport. It has been advised that after three repeated concussions, participation in any particular sport should be discontinued due to the dangers of SIS or the negative and possible permanent cognitive deficits experienced as a results of the cumulative effects of concussion (Barth et al., 1989). Many other studies speculate that it is safe to return to contact and collision sports 5-7 days post-injury, provided the athlete has been symptom-free and their neurological exam is normal (Barth et al., 1989; Collins et al., 1999; Macciocchi et al., 1996). It has been argued by Grindel et al. (2001) that multiple traumatic brain injuries change these recommendations due to multiple injuries prolonging the signs and symptoms of concussion and worsening their severity. The Cantu Grading Scale for Concussion (Cantu, 1986) and the AAN Guidelines (AAN, 1997) suggests that because of these complications, precautions must be taken in assessing the injured player and making return-to-play decisions. In cases of severe concussion, extreme or prolonged symptoms, or multiple concussions, neuropsychological testing may be of advantage, in conjunction with a detailed clinical evaluation (Grindel et al., 2001).

At the forefront of proper concussion management is the implementation of baseline and/or post-injury neurocognitive assessment (Barr, 2001). This enables coaches and medical personnel to track the player's rate of recovery for safe return-to-play, thus preventing the cumulative effects of concussion. Baseline testing on concussion assessment measures is recommended to establish the individual athlete's ''normal'' pre-

injury performance and to provide the most reliable benchmark against which to measure post-injury recovery (Guskiewicz et al., 2004). Barr (2001) reports that obtaining baseline data on a player enables the researcher to shorten the test battery by avoiding tedious time-consuming tests for evaluating overall intelligence and estimating pre-morbid level of functioning. With baseline tests, the researcher is able to make informed decisions about the presence or absence of cognitive change over time by using the athletes previous functioning as a starting point (Barr, 2001). Normative data for competitive athletes on conventional (i.e. paper-and-pencil) and computerised neuropsychological tests and other concussion assessment measures are these days readily available from large-scale research studies, but baseline data on an individual athlete still provides the greatest clinical accuracy in interpreting post-injury test results (Guskiewicz et al., 2004).

Grading scales

The purpose of designing and using a set of 'grading scales' with potentially concussed athletes is to assess the severity of their injury, and to devise follow-up and appropriate management strategies for these injured players (Guskiewicz et al., 2004). There is currently a wide variety of grading scales (over 20) that have been used; however, only a few of the scales seem to have been validated as 'formal' return-to-play guidelines (Lovell et al. 2004). Grading systems represent expertise of clinicians and researchers, yet a consensus of scientific evidence is lacking (LeClerc et al., 2001). The only exception to this is the Glasgow Coma Scale, which was validated as a 6-hour assessment for moderate to severe brain injury (LeClerc et al., 2001).

Most of these grading systems include an assessment of a range of concussion parameters, including loss of consciousness (LOC), orientation, and posttraumatic amnesia (PTA) (McCrory & Johnston, 2002).

LOC: the majority of grading systems used in the sporting arena, such as the American Academy of Neurology guidelines (AAN), rely heavily on LOC as a predictor for injury severity (Kelly & Rosenberg, 1997). **Orientation:** recent literature has concluded that the standard orientation questions such as, time, place and person, are less sensitive in discriminating concussed from non-concussed football players when compared with questions of recently acquired memory (McCrory & Johnston, 2002). Tests relating to sports specific concepts, such as Maddocks' Questions, have proven to be sensitive to concussive injuries (McCrea et al., 1998). **PTA:** there has been mixed information regarding whether PTA can be used as an effective and reliable symptom of concussion. At this point, PTA as a symptom is under current review and thus it is ill advised to use this symptom as a primary indicator of any concussive injury (Maddocks et al., 1995).

The majority of these grading classification systems indicate that most severe head injuries are associated with LOC or amnesia; however, few studies have been done for sport-related concussion (LeClerc et al., 2001). Research suggests that these two factors, either alone or in combination, are not good predictors of injury severity in sport-related injury (Maddocks et al., 1995). The AAN and Cantu Evidence-Based grading scales are the most used classification systems currently in sports medicine. Examples of each are provided in the following table:

Table 1: AAN and Cantu Grading Scales of Concussion (As cited in LeClerc et al., 2001)

Grade	*American Academy of Neurology (1997)*	*Cantu evidence-based grading system (1986)*
Grade 1 (mild)	No LOC*; transient confusion; concussion symptoms or mental status abnormality resolved in < 15 min	No LOC*, PTA† ,30 min, PCSS‡ ,24 h
Grade 2 (moderate)	No LOC; transient confusion; concussion symptoms Or mental status abnormality last > 15 min	LOC <1 min **or** PTA <30 min ,24 h **or** PCSS 24 h ,7 days
Grade 3 (severe)	Any LOC, either brief or prolonged	LOC> 1 min **or** PTA <24 h **or** PCSS 7 days

*LOC indicates loss of consciousness.
†PTA indicates posttraumatic amnesia (anterograde/retrograde).
‡PCSS indicates post-concussion signs and symptoms other than amnesia.

The difference between the Cantu and AAN classifications relates to their emphasis on PTA or LOC. The duration of these two symptoms are prime determinants of injury severity. However from the above two examples, it is not clear how to grade a player who has had a concussion without loss of consciousness (LOC) and PTA, but with prolonged PCS such as headaches, dizziness and problems with memory, concentration and balance (LeClerc et al., 2001). This results in difficulties of diagnosing mild concussion, in which there is transient confusion but no LOC. This problem is verified by incidence studies revealing that more than 75% of all sport-related brain injuries are a result of mild head injury, with no signs of LOC or PTA (Cantu et al., 1986, as cited in LeClerc et al., 2001).

Post-concussion Symptom Scale (PCSS)

Traditionally, the diagnosis and management of concussion has relied heavily on the athlete's presentation of post-concussive symptoms such as headaches, nausea, etc. This led to the development of numerous Post-concussion Symptom Scales, which usually list

around 20 symptoms, whereby the athlete is required to mark the intensity of the symptoms he is experiencing, usually in the form of a likert scale. The problem is that these symptoms often go unrecognised by team medical personnel, or the player underreports these symptoms for fear of being excluded from the team until his symptoms cease (McCrory et al., 2005). It has also been found that the reliance on team-mates or coaches to report these concussive injuries has also been unreliable (Lovell & Collins, 1998). This underreporting can occur for a variety of reasons including: 1) athletes underestimate their symptoms; 2) athletes do not have regular access to medical staff and symptoms often settle within 24 hours, or they may not have ongoing symptoms that prompt a medical consultation; 3) athletes are used to seeing professional players return-to-play after being knocked out and do not understand the risks associated with returning to play too soon (Lovell & Collins, 1998). As a result of underreporting these symptoms or downplaying the severity of these symptoms, the athlete may be returned to the field prematurely, which can have disastrous neurological consequences (Kelly & Rosenberg, 1997), as has already been highlighted in this review.

In a study conducted by Field et al., (2003), the researchers found that self-reports of post-concussion symptoms by student athletes were not predictive of poor performance in neuropsychological testing. However, in contrasting findings, Lovell et al. (2003) found that post-concussive symptoms were positively correlated with memory decline, when the athletes exhibited longer than 5-minute on-field mental status changes. As a result, it is imperative for players, coaches and clinical physicians to maintain a high index of suspicion for concussion, while educating the athlete, athletic trainer, parents, and coach about the signs and symptoms (Terrell, 2004).

Concussion assessment tools

Sports physicians and clinically trained athletic coaches are increasingly using standardised methods to obtain a more objective measurement of post-concussion signs and symptoms, cognitive dysfunction, and postural instability (Guskiewicz et al., 2004). Different strategies of cognitive assessment are required when diagnosing concussion compared to determining recovery to baseline performance (McCrory et al., 2005). The need for different tests highlights the nature of the deficits and the practicalities of assessing athletes.

Sports clinicians require a simple and valid tool that can be administered in an 'on-field' situation to help with the diagnosis of concussion, which would indicate whether the athlete should be removed from the field to be assessed further. McCrory et al. (2005), list a variety of sideline assessment tools that have been developed in the past, most of which have not been validated nor widely published. These include: 1.) Sideline evaluation for concussion (Colorado Head Injury Foundation, Inc.); 2) Management of concussion sports palm card (American Academy of Neurology and Brain Injury Association); 3) Sideline Concussion Check (Sports Medicine New Zealand, Inc.) (unpublished); 4) McGill Abbreviated Concussion Evaluation (Unpublished); 5) National Hockey League Physician evaluation form (unpublished); and 6) The UK Jockey Club Assessment of Concussion.

The two most popular and well-researched sideline assessment tools include the Standard Assessment of Concussion (SAC) (McCrea et al., 1998) and Maddocks' Questions (Maddocks et al., 1995), both of which have been published and validated in recent studies. The assessment of memory (as in Maddocks' Questions) and attention (as in SAC) has been proven to be critical in neuropsychological testing of concussion; however, the assessment of recovery warrants a different test strategy (McCrory et al.,

2005). For the purpose of this study it would not be beneficial to go into detail of all these measures. The SAC will suffice as an example of the 'typical' sideline assessment tool.

The *Standardized Assessment of Concussion* (SAC) protocol was developed by McCrea et al. (1998), and is considered a convenient and effective tool to use on the sideline field. It has consequently been recommended by the CIS group due to its reliability and validity. The SAC is used primarily as a neuropsychological method for evaluating symptoms immediately following concussion. This test is a standardised measure of *orientation* (day, month, year, time); *concentration* (repeating in reverse order strings of digits that increase from 3 to 6 numbers, and reciting the months of the year in reverse order); *immediate memory* (5-word list); and *delayed recall* (of the original 5-word list). This test in total takes five minutes to administer. It is administered by trained coaches or team officials on the sideline following suspicion of a concussive injury, and these results are compared to their baseline test scores (Barr, 2001). McCrea et al. (1998) demonstrated that the SAC was sensitive to detecting mental status abnormalities and differentiating injured from non-injured players in mild concussion. Alternative measures of this instrument are presented as a means of preventing the occurrence of 'practice effects' which could affect the reliability of the data (Barr, 2001). These results are used with other clinical criteria to make decisions regarding the clinical management of the athlete and return-to-play protocols.

According to the National Athletic Training Association (NATA) (2004), the optimum Concussion Assessment Battery should include a combination of tests for cognition that have been proven sensitive to concussion, postural stability, and self-reported symptoms in the form of Post-concussion Symptom scales, with symptoms that are known to be a predictor of concussion. During the NATA meeting in 2004, it was agreed that a

combination of brief sideline screening tools including the symptom checklist, Balance Error Scoring Symptom, Standardised Assessment of Concussion (SAC), as well as more extensive measures, such as detailed neuropsychological test batteries, would be the most effective in assessing possible injury, as well as determining individual recovery rates.

As a result of the limited empirical evidence surrounding the above-mentioned grading scales, the CIS group in Prague in 2004 deliberately did not endorse any of these measures. Rather, the CIS highlighted the importance of using 'combined measures' as a means of assessing injury severity and prognosis, and then to 'individually' asses, review and guide return-to-play decisions depending on each player's clinical presentation at the time (McCrory et al., 2005). These combined measures involve sideline evaluations which include mental status testing and neurological assessment, as well as brief neuropsychological testing alongside the field, which focus on measures of memory and attention which have been shown to be effective in predicting concussive injury (McCrory et al., 2005). It must be emphasised that these brief assessment measures do not replace comprehensive neuropsychological testing that measures subtle forms of deficits that are likely to persist beyond the acute episode (McCrory et al., 2005).

Return-to-play guidelines

Although numerous concussion-rating scales compete with separate return-to-play guidelines, they are all in agreement that athletes should be symptom-free before returning to play – including both neuropsychological symptoms as well as post concussive symptoms. The various guidelines mainly differ only in factors involving rating the severity of a concussion and in how long the player should be free of any symptoms prior to returning to play (Randolph et al., 2005).

Collins et al. (1999) report that at least 14 return-to-play scales have been published

since 1973, including the Cantu Guidelines, the Colorado Guidelines, and the Practice Parameter American Academy of Neurology. NATA recommended that returning the athlete to play should follow a progression once the athlete is completely symptom-free, and that if an injury is repeated, especially within the season, the athlete should be withheld for at least seven days (Guskiewicz et al., 2004). All signs and symptoms should be evaluated using a graded symptom scale or checklist such as the Post-concussion Symptom Scale, as already described. Baseline measurements of neuropsychological tests should be compared to post-injury results, and any differences noted and further explored (Guskiewicz et al., 2004). The CIS group advised that following a mild head injury, a step-wise process should be followed as a means of helping the player back to play where the player is only advised to progress to the next level, provided he is asymptomatic at the current level: 1) no activity, complete rest, once asymptomatic proceed to level 2; 2) light aerobic exercise such as walking or stationary cycling, no resistance to training; 3) sport-specific exercise; 4) non-contact training drills; 5) full contact training after medical clearance; 5) game play (Aubry et al., 2004).

A table of the clinical/management recommendations for the various grading scales has been provided below which highlight the differences between the Cantu, Colorado and AAN guidelines.

Table 2: Clinical/Management recommendations for grading scales (As cited in Lovell et al., 1999)

Guideline	Grade 1 severity	Grade 2 severity	Grade 3 severity
Cantu	Athlete may return-to-play that day in selected situations if normal clinical examination at rest and exertion. If symptomatic, athlete may return-to-play in 7 days	Athlete may return-to-play in 2 weeks if asymptomatic at rest and exertion for 7 days	Athlete may return-to-play in 1 month if asymptomatic at rest and exertion for 7 days
Colorado	Remove athlete from contest and evaluate immediately and every 5 min. Allow athlete to return if amnesia or symptoms do not appear for 20 min	Remove athlete, not allow athlete to return. Examine athlete next day. Permit athlete to return to practice after 1 week if asymptomatic	Transport athlete to hospital. Perform neurological examination. Permit athlete to play after 2 weeks if asymptomatic
Practice Parameter AAN	Examine athlete immediately for mental status changes. Return-to-play if no symptoms or mental status change at 15 minutes	Remove athlete, not allow athlete to return. Examine athlete on site for symptoms/mental status changes. Athlete can return in 1 week if asymptomatic	Remove athlete and transport to hospital. Perform neurologic examination. Permit athlete to play if asymptomatic after 1 week (if LOC brief), or 2 weeks if LOC prolonged

The National Athletic Trainers Association (NATA) Position Statement on concussion management in sport (2004) notes three current approaches to managing concussion: 1) Grading at the time of injury following one of the current guidelines (such as the AAN) on the basis of signs and symptoms present in the first 15 minutes of injury. The injury is graded based on LOC and provides an estimation of injury severity; 2) Grading of the injury after all the concussion signs and symptoms have resolved, as in the Cantu system. This scale places less emphasis on LOC as a predictor of impairment, and more emphasis on overall symptom duration; 3) A third approach does not use a grading scale but highlights whether the athlete is symptomatic or asymptomatic. When the athlete's symptoms appear to be resolved, a step-wise programme should be followed before the athlete resumes full contact sport. This multi-tiered approach was also suggested by the CIS Prague Statement in 2005, where the implementation of a combination of assessment

measures such as symptoms scales, neuropsychological tests, and postural-stability tests, with a focus on individual recovery, was recommended. All the above-mentioned assessment tools and grading systems were collaborated into a single sideline assessment tool developed by the Prague consensus group (McCrory et al., 2005) to form the "Sport Concussion Assessment Tool (SCAT)". The purpose of this assessment measure was to have a standardised tool that would focus on athlete education and physician assessment of sports concussion.

Collins & Hawn (2002) conclude that concussion management guidelines do not appear to have scientifically evolved to the extent that they can be relied upon to make accurate and safe return-to-play decisions. They feel that there is no uniformity between current grading systems, which result in communication difficulties with clinicians. Sports physicians, coaches and athletes need to bear in mind that no two concussions will present with identical features, and that the resulting symptoms may be very different, depending on the force of impact to the brain, the degree of metabolic dysfunction, the tissue damage, duration of time needed to recover, the number of previous concussions, and the time between injuries (Guskiewicz et al., 2004). All these factors must be considered when managing an athlete suffering from cerebral concussion (Guskiewicz et al., 2004).

Critique and Limitations of Previous Research

The MTBI literature is enormous, complex, methodologically flawed, and controversial. Studies on neuropsychological assessment of rugby players have been subject to a number of limitations inherent in their research design: in some, appropriate control groups were not employed, or baseline data was not obtained and utilised for

ipsative comparison (Edwards, 1993). In other studies, participants were volunteers, thereby raising the possibility of selection bias (Erlanger, 1999). Often studies have shown significant results, however it has been argued that such studies cannot be interpreted as examining the effects of a single concussion, because the majority of contact sport players have a history of head injury in their sporting careers, and significant values could be attributed to the possible cumulative effects of concussion rather than findings being explained by a single concussion sustained during the testing interval (Hinton-Bayre, Geffen & McFarland, 1997).

In a study conducted by Rutherford et al. (2003), they mentioned that the majority of neuropsychological studies conducted thus far suffer from methodological problems, and numerous studies should only be considered as 'exploratory'. They mention that a major factor contributing to methodological limitations is the inappropriate use and selection of subject groups whereby the subject groups are too small, resulting in the statistical power of the results being less than optimal. Other methodological limitations include low or unknown response rates, and inappropriate statistical methods, such as type 1 errors, or adjusting for multiple comparison or potential confounders (Rutherford et al., 2003). Rutherford et al. (2003), gives an example of this by criticising a study carried out by Master et al. (1999), for conducting up to 283 statistical tests without proper adjustment for the level of significance.

In a critical review of sports-related concussion literature by Kirkendall and Garrett (2001), they reported that a number of studies failed to include a variety of confounding variables that could potentially affect neuropsychological test performance scores, which has led to the inaccurate reporting and explanation of data. They claim that negative neuropsychological values are often incorrectly deemed to be a result of concussion or heading exposure. They argue that these negative values are more likely attributable to a

range of different factors. The first factor they consider relates to alcohol abuse and malnutrition, which are known to lead to cognitive impairments. The second factor revolves around the history of previous concussions and possible long-term negative consequences of previous concussive head injuries. Another problem the authors refer to relates to what constitutes the definition of 'concussion', which impacts on the diagnosis and further management of the injury. As already mentioned in the literature review, many concussions go unnoticed by coaches, trainers and players themselves due to the lack of consensus regarding concussion diagnosis and its clinical management, which leads to the misdiagnosis and underreporting of injuries. Kirkendall and Garrett (2001), mention that the third factor relates to learning difficulties, including dyslexia and attention-deficit-hyperactivity disorder. They referred to a study conducted by Frith (1998) of children with dyslexia, and another study by Nigg et al. (1998) of subjects with attention-deficit/hyperactivity disorders, and reported that these subjects tested poorly when compared with healthy controls. Kirkendall and Garrett also referred to a study conducted by Beer et al. (1998) who found that college students with learning disorders or mild brain injury performed below healthy students on a range of neuropsychological test measures. They concluded their critical review of sports concussion literature by highlighting the importance of being aware of the possible confounding factors that could have an effect on neuropsychological test performance scores when planning the research design and interpreting the results, and thus emphasised the importance of taking a detailed clinical history prior to any assessment.

Design measures to improve previous criticisms of previous research

When planning the design of this study I sought to rectify some of these limitations. A

detailed demographics questionnaire was administered to all subjects prior to any neuropsychological assessment. This questionnaire aimed to gather information about the individual's age, level of education, diagnosed learning difficulties, medical history, use of medication, total years playing rugby, position of play and a detailed history of the number and characteristics of previous concussions. These variables were considered as possible 'confounding factors', and were objectively taken into account when selecting the subjects and analysing the data. The control group consisted of individuals who had no prior history of head injury and who did not participate in any form of contact sport - thus differences in neuropsychological performance could be attributed to the effects of MHI within the rugby-playing group. The control and rugby groups were matched according to age and education in order to control for educational effects on cognitive performance. The control group's raw scores were presumed to provide a 'reliable normative sample' against which the rugby groups could be compared. It was decided against using foreign or international norms, standards and z scores, as they are not considered to be representative of the normative South African distribution and could have skewed the results. The time of testing and the conditions of testing were the same for both the rugby and control samples, and factors such as time of day and possible alcohol consumption were taken onto account during the testing sessions.

Concerning the critique by Rutherford et al. (2003), where researchers conducted excessive statistical tests without proper adjustment of the level of significance, this present study aimed to compensate for this limitation by using the Bonferroni statistical test. This is a test that controls for multiple comparisons, and thus provides corrections for countless of tests of significance.

Additionally, prior to analysing and interpreting the data in terms of the research objectives, all data was analysed to assess if any significantly measurable differences

were present between the control and rugby group concerning neuropsychological performance. This had the potential of placing the groups as 'different' from the outset, and thus further comparability across the season would have elicited unfair and unrealistic data. Thus, this 'pre-analysis' aimed to ensure the two groups were matched from the start, and further analysis of data could take place.

METHODOLOGY

Subjects

The study group consisted of 35 rugby players and 35 control subjects from the same geographical area.

The researcher approached a local rugby club and gave an informational talk to both the players and coaches. The discussion highlighted the nature of concussive injuries, various rating scales of concussive injuries, the seriousness of Second Impact Syndrome, sideline examinations, management styles of concussion and various return-to-pay protocols. Following the informational talk, the researcher asked which players would be willing to participate in this study. As a result, the rugby players volunteered to participate within this research, and were randomly selected from two of the top teams in KwaZulu-Natal. The names of the clubs nor individuals will not be revealed in order to protect the anonymity of the players as this relates to the ethics of confidentiality. Neither formal advertising nor incentivising strategies were used to obtain subjects in this study. This sample is considered to be representative of rugby players in general as there was no direct benefit to those who participated and those who did not. The players group consisted of 21 forward and 14 backline positions. An equal cross section of these positions was not possible to obtain due to the availability of willing subjects to participate within this study.

The researcher approached the local athletic club and gym to obtain a sample of control athletes and gave an informational talk to members regarding the purpose and objectives of this study. Neither formal advertising nor incentivising strategies to obtain control subjects were used in this study. This sample is considered representative of non

contact sport athletes in general as these athletes were keen sports men who did not participate in any form of contact sport. There was also no direct benefit to those who participated in the study and those who did not. The control subjects volunteered to be randomly selected. Control group selection criteria included subjects clear of any neurological disorders, professionally diagnosed learning difficulties and with no history of prior head injury.

Both control and experimental groups were selected from similar socio-economic backgrounds, of similar ages (18-28 years) and the average level of education of the subjects was taken into account. This was done by selecting participants who live in the same geographical area, with similar educational histories and who shared a common interest in sporting activities.

All participants were required to fill in a demographics questionnaire as a means of collating this information. Subjects who had a history of a professionally diagnosed learning difficulties and those who used prescriptive medication (with possible side effects that could suppress attention, concentration, and information processing ability) were excluded from the study prior to commencing any neuropsychological testing. These requirements were part of a strategy to avoid any biases in possible differences of neuropsychological functioning. The purpose of matching the controls and players on the same dimensions ensured that when comparing the players against the control group, any resulting differences in their cognitive functioning could be more easily attributed to the effect of neurological impact the players endured during the season. The results of the demographic details are presented in table 1 below.

Table 1: Demographic details for Rugby and Control groups

Personal Details	Control Mean	Control SD	Rugby Mean	Rugby SD
Age	23.90	2.50	21.43	2.20
Level of Education (Total Years from Grade 1 to university level)	14.30	1.82	13.29	1.50
Total years playing rugby	0	0	13.09	3.50
Average Number of Concussions in sporting history	0	0	2.09	3.14
Professionally diagnosed Learning Difficulty	0	0	0	0
Use of any prescriptive/sedative Medication	0	0	0	0
Backline players	0	0	14	
Forward players	0	0	21	

The rugby and control group were required to fill in a consent form stating that they clearly understood the purpose of the study, were willing to participate in the research, understood and accepted the terms of confidentiality, and were free to withdraw from the study at any point in time if they decided to do so. (Appendix A)

Assessment Instruments

1. **Post Concussion Symptom Scale (PCSS)**
2. **Assessment of concussion (Practice Parameter of the AAN)**
3. **Neuropsychological tests**

Post Concussion Symptom Scale (PCS): The scale was originally developed in the 1980s within the context of the Pittsburg Steelers (a professional American football team) concussion management program, and a variety of different versions have been adapted

by the various hockey and automobile racing leagues, as well as numerous schools and universities (Lovell et al., 2006). It is now used throughout professional and amateur sports as a reliable assessment tool in conjunction with other assessment tools such as neuropsychological testing (Lovell, M. R., & Collins, M.W, 1998). This scale has been a dependent measure in several published studies (Collins *et al.,* 2003; Iverson, Gaetz, Lovell, & Collins, 2004a, 2004b; Lovell *et al.,* 2003; Lovell, Collins *et al.,* 2004). According to Lovell *et al.,* 2006, the PCSS was developed to provide a formal method of documenting post-concussion symptoms, as subjectively perceived and experienced by the player. They also mention it is important to carefully evaluate self-reported symptoms in athletes with known or suspected concussion due to the subjective nature of this scale.

The PCSS consists of 22-item scales, specifically designed to measure the severity of symptoms in the acute phase of recovery from concussion (Lovell, 1999; Lovell & Collins, 1998). It is designed as a Likert Scale, graded 0-6, where zero indicates no symptoms, 3 indicates moderate and 6 indicates severe symptoms. The PCSS is separated into *cognitive features* (confusion, amnesia, LOC, memory disturbances etc), *typical symptoms* (headache, dizziness, nausea, light and noise sensitivity etc), *physical signs* (poor balance, poor attention and concentration, nausea, vomiting etc.) and *emotional symptoms* (increased irritability, sadness, feeling more emotional) (Aubry et al., 2002). This scale was thoughtfully designed for the athletes themselves, and used non- medical jargon, i.e. 'fogginess', which could be easily understood by both university and primary school students. This assessment scale has also been suggested as a valuable management tool by the CIS group (Aubry et al., 2002). In a study conducted by Lovell et al., 2006, normative data was gathered from 1746 high school and university athletes. The students completed the computerised version of the PCSS as presented in the ImPACT Version 1 computer programme, which follows an identical format to the paper-based version used

in the current study. A further clinical sample consisted of 260 concussed athletes, who completed the PCSS within 5 days post injury. The results suggested that there was no difference between self-reporting of university students and school goers, however a significant difference was evident across gender, suggestive that females report higher symptoms compared to males, in both the university and school going groups. The internal consistency reliability of the PCSS varied from .88 to .94 across the sample of healthy school goers and university students (Lovell et al., 2006, p 6). At a 80% confidence interval, the total score was +/- 3.4 points for young men, and at a 80% confidence interval, the total score was 4.4 points for young women (Lovell et al., 2006, p 6). For the concussed athletes, the internal consistency of the PCSS was reportedly very high (r=.93). The standard error of measurement was 5.3, and the 80% confidence level was 6.8 points (Lovell et al., 2006, p 6). Refer to Appendix C for an example of the PCSS.

Assessment of Concussion: Concussion in this particular study has been defined as a "traumatically induced alteration in mental status that may or may not be accompanied by a loss of consciousness," based on the standard American Academy of Neurology nomenclature, AAN Guidelines (1997) (Maroon *et al.*, 2000). A concussion was diagnosed if the player experienced either LOC or reported other symptoms such as headache, dizziness, nausea, visual disturbances etc. There was no trained physician or coach present whom was able to professionally diagnose the concussion at the time of injury, nor was there anyone available who was trained to administer a brief sideline mental status examination on the injured player. Thus, the player self reported their own injuries directly to the researcher.

Neuropsychological tests: Several factors were taken into account in test selection. Since there was a limited amount of time available for testing athletes, the neurocognitive domains that were assessed targeted those cognitive systems that are known to be at risk following MTBI. The test instruments chosen needed to be short in duration, easily administered without extensive neuropsychological training, have sound psychometric properties (see references for each test), have a history of use with athletes, and produce minimal levels of frustration. Each of the measures described below has been used extensively with athletes and has demonstrated adequate levels of reliability and validity (Lovell, M.R., & Collins, M.W, 1998). These measures were also found to be useful and recommended for the assessment of sports-related MTBI by the Sports Neuropsychology Panel (Lovell, M.R., & Collins, M.W, 1998).

The following tests were administered in succession: Rey Complex Figure (copy), Stroop Colour Word Test, Rey Complex Figure (2-minute recall trial), Rey Auditory Verbal Learning Test (RAVLT), Symbol Digit Modalities Test Written and Oral (SDMT), Digits – forwards and backwards, Trail Making part A & B, Controlled Oral Words Association Tests (COWAT), Rey Complex Figure (30 minute delayed recall trial).

Measures

In this section, the neuropsychological test instruments that were used in this study will be described in terms of the publisher, purpose, test constructs, development, validity and reliability. The administration will be described, and issues of administration which arose in this study will be discussed.

Symbol Digit Modality Test (SDMT)

This test was developed by Aaron Smith (1973) and was originally published by Western Psychological Services, USA, and revised in 1982. The purpose of the SDMT is used to assess complex visual scanning and tracking; and motoric speed and agility. This test gives an overall indication of processing speed and efficiency. It consists of a series of nine meaningless geometric designs where the examinee needs to search for a key which corresponds with the digit, both orally then verbally. The SDMT manual presents the means and standard deviations by age and educational level. Impaired performance has been associated with a variety of conditions including depression, learning difficulties, dementia, as well as closed head injury (Hinton-Bayre *et al.,* 1997; Ponsford & Kinsella, 1992, cited in Spreen and Strauss 1998, p. 254.).

As cited in Spreen and Straus, 1998, pg 255, Smith (1991) provides data based on a sample of 1,307 normal adults, aged 18-78 years. Smith (1991) suggests that scores of 1-1.5 SD below the mean age norms should be considered suggestive of cerebral dysfunction. In normal adults, the correlation between the written and oral forms is above 0.78, suggesting that the two forms are fairly equivalent (Spreen and Strauss, 1998).

Digit Span

Digit Span is a subtest of the Wechsler Memory Scale. David Wechsler and Calvin P. Stone are the authors of this test. It was originally published in 1974 by the Psychological Corporation, and revised in 1987. The purpose of this subtest is to provide a measure of immediate memory and verbal recall, and is useful in an early investigation of attention difficulties. The digits backwards trial may be useful in uncovering tracking difficulties. This test consists of 9 digits that are called out in a specific order with equal spacing between the digits. The examinee is required to remember as many digits as possible and

repeat them back to the examiner in the same order as cited. Digits backwards require the examinee to recall the digits back to the examiner in the reverse order. This process is repeated, while increasing the number of digits on each trail, until the examinee fails on two consecutive trials. According to Wechsler, 1987 the average reliability coefficient across age groups for individual subtests of Digit Span was 0.88. Patients with left hemisphere damage and patients with visual field defects have shorter reversed spans than those without such defects (F.W Black, 1986; Newcombe, 1969; Weinberg, Diller, *et al.*, 1972, as cited in Lezak *et al.*, 2004).

Trail Making Test

This test was developed by U.S Army Psychologists and was initially a subtest of the Army Individual Battery Test, 1944. It was originally constructed in 1938 as a 'Divided Attention Test', which formed part of the Army individual Test Battery, 1994. The purpose of this test is to assess visual conceptual tracking, and visuo-motor tracking. It consists of two parts, A and B. In part A the subject is instructed to draw connections between 25 encircled numbers randomly arranged on a page. In part B, the subject must draw the lines alternating between matched numbers and letters of the alphabet. The examinee needs to work as quickly as possible without lifting the pencil from the paper.

Reported reliability coefficients vary greatly, with the majority above 0.60 but several in the 0.90's and more in the 0.80's (Spreen and Strauss, 1998). By contrast, inter-rater reliability has been reported as 0.94 for Part A and 0.90 for Part B (Fals-Stewart, 1991, as cited in Spreen and Strauss 2004). Normative data varies substantially and thus Mitrushina *et al,* 1999 (as cited in Lezak et al., 2004) recommend care in selecting the most appropriate data set for clinical comparisons. Parts A and B correlate only 0.49 with

each other, indicating they measure different cognitive processes (Heilbronner *et al.*, 1991 in Spreen and Strauss 2004, pg 536).

The test has been reported to be sensitive to closed head injury (des Rosiers & Kavangh, 1987 as cited in Spreen and Strauss, 2004). Part B is reported to be more sensitive to brain damage compared to the simpler task posed by part A. This is due to part B being more complex in design, in that it assesses the examinees ability to shift course during an ongoing activity and their ability to deal with more than one stimulus at a given time.

STROOP Colour Word Test (1935)

There is a variety of versions of the Stroop test which differ in the number of cards used as well as use of colours. Within this study, the Victoria version was administered. This revised version was written by M. Regard, 1981, Canada, Department of Psychology, University of Victoria.

The purpose of the STROOP Colour Word Test is designed to assess cognitive flexibility, attention and information processing, and the ease with which a person can shift their perceptual set to conform to changing demands and suppress a habitual response in favour of an unusual one. It consists of three cards: dots, words, and colours that are always presented in the same order. The examinee is required to say the colour name (not the word) as quickly as possible. The time taken for each section and the total number of errors are taken into consideration when scoring.

Uttl and Graf (1997) researched healthy individuals with regards to trial-to-trial reliabilities. They found the estimated reliabilities for the average of the three trials were above 0.75. Test-retest reliability coefficients were found to be 0.90, 0.83 and 0.91 for the three parts of the test (Spreen and Strauss, 2004, p214).

Rey-Osterrieth Complex Figure (RCF)

The Rey–Osterrieth Complex Figure Test (RCF) was developed by Rey in 1941 and standardised by Osterrieth in 1944. The main purpose of this test is to assess visuo-spatial constructional ability and visual memory. The RCF also assesses a variety of cognitive processes, including planning, organisational skills, problem solving strategies, and perceptual, motor and memory functions (Waber & Holmes, 1986; Meyers & Meyers, 1995a as cited in Spreen and Strauss, 2004). Recently, the RCF has been a useful tool for measuring executive function that is mediated by the prefrontal lobe (Shin *et al.,* 2006). The pre-season test included the 'Rey-Osterrieth Complex Figure Test, Form A' (Rey Figure), and the post-season and post-concussion test included the 'Rey-Osterrieth Complex Figure Test, Form B' (Taylor Alternate Version).

The RCF consists of three test conditions: Copy, Immediate Recall and Delayed Recall. At the first step, subjects are given the RCF stimulus card, and then asked to draw the same figure. Subsequently, they are instructed to draw what they remembered. Then, after a delay of 20-30 minutes, they are required to draw the same figure once again. Both immediate and delayed recall trials have a strong visual memory component. According to Lezak et al. (2004) the RCF recall is sensitive to mild neuropsychological impairment, and this could be useful in detecting cognitive deficits resulting from concussive and sub concussive injuries sustained in rugby.

According to D.T.R. Berry, Allen, & Schmitt, 1991; Loring, Martin et al., 1990 and Shorr et al., 1992, as cited in Lezak, et al. 2004, inter-scorer reliability is good ($r = .91$ to .98) and test-retest reliabilities using alternate forms (CF-RO, CF-T) were .60 to .76.

Rey Auditory Verbal Learning Test (RAVLT)

The Auditory Verbal Learning Test was developed by André Rey in 1941 and first published in France in the 1964. According to Boake, 2000, as cited in Lezak et al. (2004) Andre Rey adapted and further developed the test composed by Edouard Claparede, whereby the 15 original French words were translated to English. The purpose of this test is to assess verbal learning and memory: retrieval, storage, and acquisition (Lezak et al., 2004). This test is easily administered, and assesses learning and retention over a 5 trial presentation of 15 words, followed by an interference list, a 20-minute delayed recall trial and a recognition memory list where the examinee is required to filter out distractor words.

This test has high test-retest reliability, as shown by studies conducted by Delaney Prevey, Cramer *et al.,* 1992, as cited in Lezak et al., 2004. They noted that using alternate forms with a retest interval of one month, correlation coefficients ranged from .1 to .86 for trials I-V and from .51 to .72 for delayed recall and recognition. Learning measures of the RAVLT (V, VI, recognition) are shown to correlate significantly with values of .50 to .65 with other learning measures (Macartney-Filgate & Vriezen, 1998; J.J. Ryan, Rosenberg, and Mittenberg, 1984, as cited in Lezak et al., 2004).

Controlled Oral Word Association Test (COWAT)

This test was originally developed by Benton and Hamsher (1976). It was updated in 1983 and again by Benton, Hamsher and Sivan in 1994. The purpose of this test is to assess an individual's spontaneous production of words and verbal fluency under restricted conditions such as the given letter of the alphabet.

This test consists of three word-naming trials. The original set of letters used were F, A, S, however Benton, Hamsher & Sivan (1994) as cited in Spreen an Strauss, 2004, p.

447, further developed the FAS test, to formulate a similar version called the Multilingual Aphasia Examination which provides norms for two sets of letters, namely C, F, L, and P, R, W. These letters were selected based on the frequency of English words beginning with letters. In each set, words beginning with the first letter have a relatively high frequency of usage, the second letter a lower frequency, and the third has the lowest. The examinee asks the research subject to think of as many words as they can beginning with that particular letter of the alphabet, excluding proper nouns, numbers, and the same word with a different suffix, within the time period of 60 seconds. The score is the sum of all acceptable words produced in the 3 one-minute trials. Word fluency is a sensitive measure of brain dysfunction, and low scores could indicate frontal lobe lesions, especially within the left hemisphere (Mansfield, 2002). Category (animal) naming is part of the Boston Diagnostic Aphasia Examination and the Standford-Binet test.

According to Snow *et al.,* 1988, as cited in Spreen and Strauss 2004, p. 449, inter scorer reliability on this test is near perfect and 1-year retest reliability in older adults has been reported as .70, and after 19-42 days as .88 (des Rosiers & Kavaagh, 1987, as cited in Spreen and Strauss, 2004, p. 490).

Procedure

The neuropsychological test battery was administered twice to the control and rugby subjects over a 9-month period, at pre- and post-season testing intervals. Each neuropsychological test battery was administered to individual players and controls, by a trained university psychology graduate. Each psychology graduate had been given extensive training by a qualified psychologist specialising in neuropsychological assessment prior to the commencement of the project. This training focused on the

purpose of each assessment tool as well as the correct administration rules for each assessment tool. All tests were administered according to standardised procedures. Those assessments that had alternative forms available were used in the post-season and post-concussion testing sessions, with the purpose being to minimise the consequences of 'practice effects'. At both pre- and post-season testing intervals, the tests were administered at equivalent times. The testing took place between 4 pm and 6 pm, before practice on a Tuesday and Thursday night. The tests were administered in various rooms provided by the rugby club. The testing environment was free from noise and distractions which could have affected the examinees' performance. The control group was tested under analogous testing conditions, including similar time intervals, times of the day, and in rooms free from distracting variables.

Throughout the season each player filled in a self-reported Post Concussion Symptom Scale (PCSS), within a 24-hour period after each game played. The player was asked to choose the rating scale that most accurately reflected his status with regard to each symptom. The controls' scores with regards to this scale were presumed zero, due to the fact that none of them were exposed to any neurological impact that could have led to concussion or any mild head injury during the testing period. These forms were collected weekly and tallied. Periodically meetings with the coaches and players were held every two to three weeks. The ideal would have been to hold weekly meetings, but this was difficult due to periods of 'away games', university holidays and exam periods. Frequent contact between the present researcher and players allowed for regular discussions regarding possible concussions of various players, signs and symptoms of the concussions and return-to-play guidelines. Whenever there was cause for concern, players were referred to a medical doctor with the necessary degree of expertise, for decisions about returning to play. No results were disclosed to the coach, or any other 3rd party.

The individual players informed the researcher of a possible concussive injury using the ANN guidelines provided by the researcher, within 12 hours of the injury via phone call. The player was retested within 48 hours of the injury, using an alternate test battery, measuring the same areas of cognition as in the pre- and post-season test battery. This testing took place at the same time, in the same venue as the baseline testing session and was free from external distractions. The researcher compiled a qualitative report from the rugby player about the nature of the injury and their post-concussion symptoms as a means of assessing the severity of the injury.

RESULTS

Due to there being a relatively large number of neuropsychological dependent variables in this research, it was decided to group the various dependent variables. This grouping was for reasons of logic, and also to achieve a degree of theoretical coherence. The grouping of the variables involved combining the scores arithmetically, and they were organised in various cognitive domains for reasons of theoretical structure. The framework follows a rationale established by Matser et al., (1999). These authors grouped the neuropsychological measures into 5 areas of cognitive functioning which are believed to be relevant to the effects of mild concussion as discussed in the literature review above. The following 5 areas of cognitive functioning used in this present study are:

1) ***Planning, visuo spatial, constructional ability*:** Rey Complex Figure Test (RCF)
2) ***Attention and Concentration*:** Stroop Colour, Words, Dots, Error; Trail Making part A and B; Symbol Digit Modality Test (SDMT) written and oral, digits backwards and forwards
3) ***Memory (STM and LTM)*:** Rey Complex Figure Test (RCF); Rey Auditory Verbal Learning Test (RAVLT), digits backwards and forwards
4) ***Verbal Fluency:*** Controlled Oral Word Association Test (COWAT) letters and animals
5) ***Speed of Processing:*** Stroop Colour, Words, Dots, Errors; SDMT oral and written, Trail Making part A and B

This present study employed mean raw scores for all 7 neuropsychological tests as the dependent variables, as these measures have shown great reproducibility and sensitivity to mild head injury as already highlighted within the literature review. The independent

variables were Rugby vs. Control groups, and Pre/Post (testing before the rugby season vs. testing after the rugby season).

The data analysis commenced with descriptive statistics and data exploration, using means plots and observing confidence intervals. Inferential statistical analysis presented some challenges because of the relatively high number of dependent variables and the large total number of comparisons that needed to be done. Multiple tests of significance bring about a danger of Type 1 errors (falsely rejecting the null hypothesis) in cases where significant findings occur by chance. In order to compensate for this source of statistical errors, the Bonferroni adjustment was used in the initial stages of hypothesis testing where an *SPSS* Custom Table Model was used to do multiple t-tests. The Bonferroni correction controlled for multiple comparisons and provided corrections for the overall number of tests of significance.

The Z scores of each subtest were initially taken into consideration when analysing the initial set of results to plot the distribution of scores, but since these norms were not developed locally within the South African context, they were seen as a potential source of artefacts and thus these scores were not used for the analysis.

The research design tried to achieve matching, as far as possible, in terms of age and level of education between the rugby and control group. The 'matched' control group was used in order to avoid any problems with normative comparisons and also as a more powerful research strategy. While there are significant differences between the rugby and control group on both these variables, the normative comparison group generally would be the same for both groups, and the differences are relatively small (age: control group mean age was 2.43 greater than the rugby group; education: controls' average education was 0.97 years more than the rugby group). The ANOVA results are as follows: Age $F(1) = 18.3$, $p < 0.0001$; Education $F(1) = 16.51$, $p < 0.05$. Nevertheless, the data collected

during this study may have value for standardising the tests for this population. Refer to tables 1A & 1B and 2A & 2B.

Table 1 A: South African norms for Rugby Group Pre-Season

Neuropsychological Test	Mean	Median	Mode	Std. Deviation	Variance	Range
RCF Copy	32.4000	32.0000	34.00	2.17540	4.732	10.00
STROOP Dots	12.7874	12.2300	12.00	1.82906	3.345	8.21
STROOP Dots Error	.1143	.0000	.00	.32280	.104	1.00
STROOP Words	14.3963	14.5000	10.00(a)	2.76123	7.624	12.10
STROOP Words Error	.1714	.0000	.00	.38239	.146	1.00
STROOP Colour	21.5406	20.3800	22.00	4.29369	18.436	21.43
STROOP Colour Error	.7429	.0000	.00	1.03875	1.079	4.00
RCF 2 min recall	23.2029	24.0000	25.00	4.11371	16.923	19.00
RAVLT Total	52.0286	52.0000	50.00	7.19267	51.734	27.00
RAVLT Learn	5.8857	6.0000	7.00	1.87509	3.516	9.00
RAVLT Recognition	13.7143	14.0000	14.00(a)	1.20224	1.445	4.00
RAVLT 20 min delay recall	10.8571	11.0000	11.00	2.43918	5.950	10.00
RAVLT Immediate	6.9143	7.0000	7.00	1.35845	1.845	5.00
RAVLT V1/A6 2 min delay recall	10.9714	11.0000	11.00	2.46726	6.087	9.00
RAVLT B1 distractor list	7.0000	7.0000	6.00	2.41320	5.824	12.00
SDMT Written	54.3714	55.0000	57.00	7.31672	53.534	34.00
SDMT Oral	60.1429	61.0000	62.00	8.14986	66.420	41.00
Digits forwards	6.3143	6.0000	7.00	.99325	.987	3.00
Digits backwards	5.0000	5.0000	5.00	1.05719	1.118	4.00
COWAT F	12.1714	13.0000	13.00	3.58498	12.852	14.00
COWAT A	9.9714	10.0000	11.00	2.87469	8.264	14.00
COWAT S	13.9714	14.0000	10.00(a)	3.11057	9.676	13.00
COWAT FAS	35.8286	35.0000	38.00(a)	7.81584	61.087	34.00
COWAT Animals	17.9143	18.0000	19.00	2.83229	8.022	12.00
Trail Making A	27.5171	27.2000	20.00(a)	6.60208	43.587	26.61
Trail Making B	62.7123	57.0000	49.00(a)	20.84742	434.615	80.00
RCF 20 min delay recall	22.3286	23.0000	23.00	4.98519	24.852	19.50

Table 1 B: South African norms for Control Group Pre-Season

Neuropsychological Test	Mean	Median	Mode	Std. Deviation	Variance	Range
RCF Copy	32.3000	33.0000	34.00	2.78705	7.768	10.00
STROOP Dots	13.7166	13.4800	10.02(a)	2.83283	8.025	15.33
STROOP Dots Error	.2571	.0000	.00	.65722	.432	3.00
STROOP Words	15.3243	14.4800	1.20(a)	5.42618	29.443	30.32
STROOP Words Error	.2000	.0000	.00	.47279	.224	2.00
STROOP Colour	24.0194	23.0700	13.68(a)	6.78454	46.030	31.32
STROOP Colour Error	1.0286	.0000	.00	1.42428	2.029	5.00
RCF 2 min recall	20.1857	20.5000	13.00(a)	6.82350	46.560	27.50
RAVLT Total	51.6857	50.0000	48.00	7.61489	57.987	34.00
RAVLT Learn	6.2286	6.0000	7.00	2.19740	4.829	11.00
RAVLT Recognition	13.1714	14.0000	14.00	1.87060	3.499	7.00
RAVLT 20 min delay recall	10.8571	11.0000	11.00	2.71318	7.361	10.00
RAVLT Immediate	6.7143	7.0000	5.00	1.84026	3.387	10.00
RAVLT B1 distractor list	5.8571	6.0000	5.00	1.62956	2.655	8.00
RAVLT V1/A6 2 min delay recall	10.8000	11.0000	10.00	2.49470	6.224	10.00
SDMT Written	51.9429	53.0000	56.00	6.99976	48.997	29.00
SDMT Oral	57.7429	60.0000	60.00	6.32615	40.020	23.00
Digits forwards	6.7143	7.0000	6.00(a)	.98731	.975	3.00
Digits backwards	4.9429	5.0000	5.00	1.21129	1.467	5.00
COWAT F	12.4680	12.0000	11.00	4.55334	20.733	27.38
COWAT A	11.6286	10.0000	9.00(a)	9.00681	81.123	56.00
COWAT S	13.7143	14.0000	11.00	3.90754	15.269	16.00
COWAT FAS	37.8109	34.0000	29.00(a)	13.83441	191.391	81.38
COWAT Animals	17.4857	18.0000	16.00(a)	3.92107	15.375	20.00
Trail Making A	28.3603	27.0000	28.53	8.87429	78.753	45.26
Trail Making B	64.3131	60.3200	70.00(a)	21.78192	474.452	102.00
RCF 20 min delay recall	20.3857	21.5000	15.00	5.39756	29.134	20.00

Table 2 A: South African norms for Rugby Groups Post-Season

Neuropsychological Test	Mean	Median	Mode	Std. Deviation	Variance	Range
RCF Copy	31.6000	33.0000	33.00(a)	3.21943	10.365	12.50
STROOP Dots	12.6160	12.3300	13.00	2.16438	4.685	10.50
STROOP Dots Error	.0571	.0000	.00	.23550	.055	1.00
STROOP Words	15.1009	14.3100	11.00	3.51783	12.375	14.00
STROOP Words Error	.2000	.0000	.00	.47279	.224	2.00
STROOP Colour	19.7534	19.6600	20.00	4.95390	24.541	23.90
STROOP Colour Error	.4857	.0000	.00	.78108	.610	3.00
RCF 2 min recall	25.2857	26.0000	28.00(a)	5.08788	25.887	22.00
RAVLT Total	49.7429	49.0000	49.00	5.88789	34.667	24.00
RAVLT Learn	5.6286	6.0000	6.00	2.40203	5.770	12.00
RAVLT Recognition	13.2571	14.0000	14.00	1.57821	2.491	7.00
RAVLT 20 min delay recall	10.9143	11.0000	13.00	2.29285	5.257	9.00
RAVLT Immediate	6.4286	6.0000	6.00	1.52017	2.311	6.00
RAVLT B1 distractor list	5.3429	5.0000	5.00	1.62595	2.644	7.00
RAVLT A6P 2 min delay recall	10.8857	11.0000	12.00	2.15258	4.634	8.00
SDMT Written	56.3143	55.0000	51.00	8.14129	66.281	29.00
SDMT Oral	63.0857	62.0000	57.00	8.10364	65.669	35.00
Digits forwards	6.1143	6.0000	6.00	1.07844	1.163	5.00
Digits backwards	4.6857	5.0000	4.00(a)	1.10537	1.222	4.00
COWAT F	12.0571	12.0000	8.00(a)	3.26247	10.644	13.00
COWAT A	11.6857	11.0000	11.00	3.21551	10.339	13.00
COWAT S	11.1429	12.0000	14.00	3.39674	11.538	14.00
COWAT FAS	34.8857	35.0000	28.00	8.56385	73.339	36.00
COWAT Animals	16.7714	17.0000	16.00	3.12566	9.770	13.00
Trail Making A	26.5389	25.7500	22.00	6.00233	36.028	28.93
Trail Making B	61.4911	56.5100	120.00	19.38580	375.809	87.60
RCF 20 min delay recall	24.3571	24.0000	24.00	4.41850	19.523	18.00

Table 2 B: South African norms for Control Groups Post-Season

Neuropsychological Test	Mean	Median	Mode	Std. Deviation	Variance	Range
RCF Copy	33.1714	33.0000	33.00	1.69750	2.882	7.50
STROOP Dots	12.8551	12.5100	11.03	2.56924	6.601	10.14
STROOP Dots Error	.3143	.0000	.00	.58266	.339	2.00
STROOP Words	14.0554	13.5000	9.02(a)	3.00107	9.006	14.30
STROOP Words Error	.0857	.0000	.00	.28403	.081	1.00
STROOP Colour	19.1634	18.8900	7.65(a)	4.41189	19.465	20.58
STROOP Colour Error	.2857	.0000	.00	.57248	.328	2.00
RCF 2 min recall	25.4286	25.0000	28.00(a)	5.30558	28.149	18.50
RAVLT Total	50.8000	50.0000	50.00	7.95872	63.341	38.00
RAVLT Learn	5.9714	6.0000	7.00	1.97761	3.911	9.00
RAVLT Recognition	13.6286	14.0000	14.00	1.11370	1.240	4.00
RAVLT 20 min delay recall	10.2571	10.0000	8.00	2.53613	6.432	9.00
RAVLT Immediate	6.6857	6.0000	6.00	1.65869	2.751	9.00
RAVLT B1 distractor list	5.9143	5.0000	5.00	2.22778	4.963	11.00
RAVLT A6P 2 min delay recall	10.4286	11.0000	9.00	2.45292	6.017	11.00
SDMT Written	57.2286	55.0000	54.00	8.17128	66.770	32.00
SDMT Oral	60.7714	60.0000	71.00	9.29923	86.476	32.00
Digits forwards	7.0000	7.0000	7.00	.93934	.882	3.00
Digits backwards	4.9143	5.0000	5.00	.98134	.963	4.00
COWAT F	13.7143	14.0000	12.00	3.69874	13.681	18.00
COWAT A	13.8857	14.0000	14.00	3.99832	15.987	15.00
COWAT S	12.9714	13.0000	10.00(a)	3.91442	15.323	18.00
COWAT FAS	40.5714	40.0000	33.00(a)	10.39877	108.134	46.00
COWAT Animals	18.2571	18.0000	18.00	2.82159	7.961	12.00
Trail Making A	26.8743	24.3600	40.17	7.70665	59.392	30.39
Trail Making B	59.4080	57.2800	60.03	18.86683	355.957	74.94
RCF 20 min delay recall	25.2286	25.5000	28.00	5.39927	29.152	22.00

Information gathered from the demographics questionnaire was considered as potential 'confounding variables' and was taken into consideration when analysing and interpreting the data. These 'confounding variables' include age, level of education, learning difficulties, neurological diseases, possible use of medication, the total number

of previous concussions, and the individual's history of previous concussions. The rationale for collecting this information is highlighted in the literature reviewed above, and it has been found that these confounding variables do indeed appear to be associated with alterations in individuals' neuropsychological test performance. Refer to table 3.

Table 3: Demographic Details of the Control and Rugby Group

Personal Details	**Control Mean**	**Control SD**	**Rugby Mean**	**Rugby SD**	**Sig. P <= 0.05**
Age	23.90	2.50	21.43	2.20	p <0.0001
Level of Education (Total Years from Grade 1 to matric, university level)	14.30	1.82	13.29	1.50	p <0.05
Total years playing rugby	0	0	13.09	3.50	
Average Number of Concussions in sporting history	0	0	2.09	3.14	
Professionally diagnosed Learning Difficulty	0	0	0	0	
Use of any prescriptive/sedative Medication	0	0	0	0	

DATA ANALYSIS

Data was captured using Microsoft Excel worksheets, and these were imported into *SPSS* Version 15 and analysed. *SPSS* allows long variable labels, but only 8-letter variable names, and in this study with so many scores, it was more convenient to use shortened names like B1, V1, A6 etc. for the various measures. A list of these shortened *SPSS* variables and the equivalent full names has been provided in Appendix H.

Prior to analysing and interpreting the data in terms of answering the research objectives, it was considered imperative by the researcher to overview the data of both the rugby and control group to assess if these two groups were indeed similar and 'comparable'. There was a potential concern that the research could be confounded by possible baseline differences between the two groups. This analysis aimed at ruling out the possibility that any noticeable differences in neuropsychological performance found during further data analysis could be attributed to the fact these two groups were already different and 'incomparable' from the start. One reason for possible pre-season differences is that the rugby group could have suffered multiple concussions in the past which could have affected their baseline sores. This analysis helped to ensure that the rugby and control group were indeed comparable, and thus any further significant differences found between these two groups could be attributed to conditions, or events, such as mild head injury sustained in play over the 9-month assessment period of playing rugby.

Hypothesis 1: *To assess what happens to the rugby players' neuropsychological performance over a 9-month period of playing rugby. It is hypothesised that the control group scores will remain constant or may benefit from practice effects between pre- and post-testing sessions over the testing interval; however it is presumed that the players' performance would remain the same over the testing interval, or show possible deterioration in post-season test scores as a result of being exposed to continual neurological impact over the season of playing rugby.*

The data analysis proceeded in 3 phases:

1) Descriptive statistics in the form of error bars were computed and graphs of the

distribution produced. These were used as a means of 'eye-balling' the data to look for possible differences on the dependent variables at the two test intervals (Pre and Post), between the two groups (Rugby and Control). The error bars were used graphically to illustrate the upper and lower bounds within which the means fell, and a 95% confidence level of the mean was used. The advantage of using the error bars is that they consider the extreme cases when analysing the data, and thus via 'eyeballing' the graphs it becomes evident which factors are likely to show statistically significant group differences ($p < .05$) on further investigation. Only significant values were reported and interpreted. Refer to figure 1 and 2 below (Bar graphs of the Pre- and Post-season group differences for both the rugby and control group).

2) The first hypothesis was tested in order to examine whether there were significant differences between the rugby and control groups at each measurement interval: a) before the season, b) after the season. After examining the plots mentioned above, a Custom Table design was generated in *SPSS* to systematically test all variables at both pre- and post-test intervals. Due to the vast number of independent t-tests being conducted, the Bonferroni correction was used to control for the number of comparisons. This helps to control for type 1 errors which has been one of the main criticisms of previous research in this particular field of sporting concussions, as mentioned previously in the 'critique of previous research'. Alpha was set to 0.05 for all comparisons.

3) The first hypothesis also aimed to systematically test whether there were group differences and within-subjects differences attributable to the rugby season over the 9-month testing interval. This was done in *SPSS* using a repeated measure ANOVA (player/control x pre/post) which is a more powerful model at detecting both group differences, within subject's differences (pre-post-season) and where relevant, between subjects interactions between these variables. It was also used as a means for further

investigating the patterns seen in the findings of the descriptive statistics conducted previously. For the purpose of this study, only significant values, using the alpha level of $p < 0.05$ are noted when writing the results.

Figure 1 below shows that within the ***pre-season*** group difference error bars there were 2 possible variables, namely the 1) RCF 2 minute recall and 2) RAVLT B1 distractor list, that showed some possibility of being significant *(p=<0.05)*. The variable A6/V1 (RAVLT 2 min recall) has been presented in the error bars to show that this variable, among the rest of the variables, would not be likely to show any significant difference in neuropsychological test scores between the rugby and control group due to the higher level of overlap between the upper and lower bounds of the two groups. Refer to figure 1 below.

Figure 2 below shows that within the ***post-season*** group difference error bars it became evident that 7 variables showed some possibility of being significant *(p=<0.05)* on further investigation, namely 1) ReyCopyP (Rey Complex Figure Copy), 2) StroopErrorsP (Stroop Dot Errors), 3) DIGITFP (Digit Forward), 4) FPOST, 5) LPOST, 6) CFLPOST, 7) ANIMALSP (COWAT for the letters F, L, CFL and Animals). This increase in significant values form the pre- to post-season testing suggests that the rugby players cognitive performance was negatively affected over the season of playing rugby. Refer to figure 2 below.

Figure 1: Pre-season group mean differences

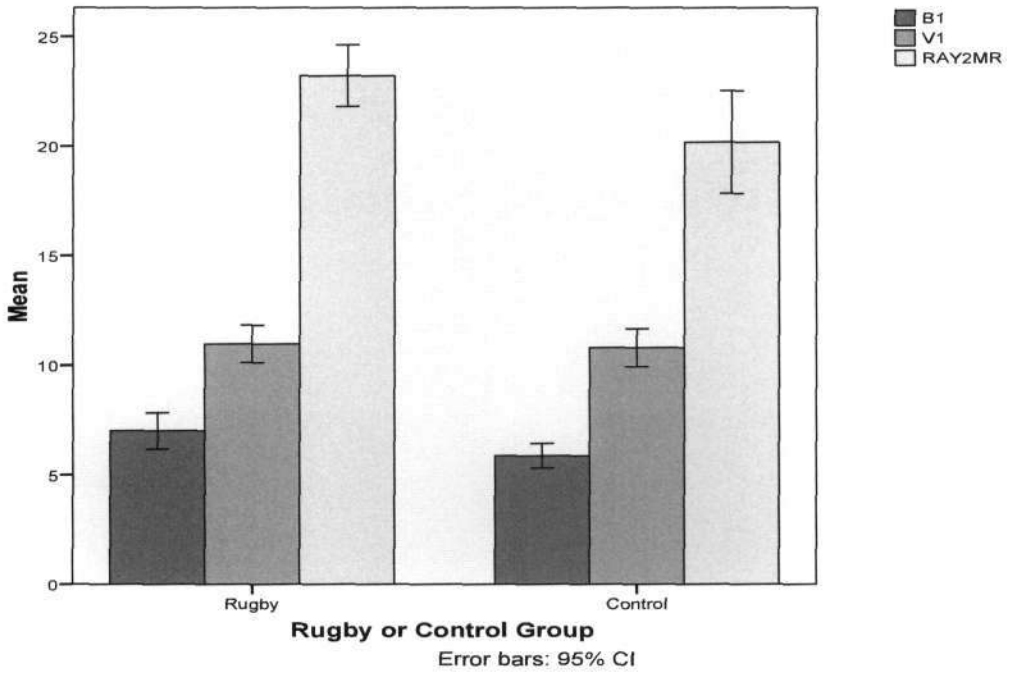
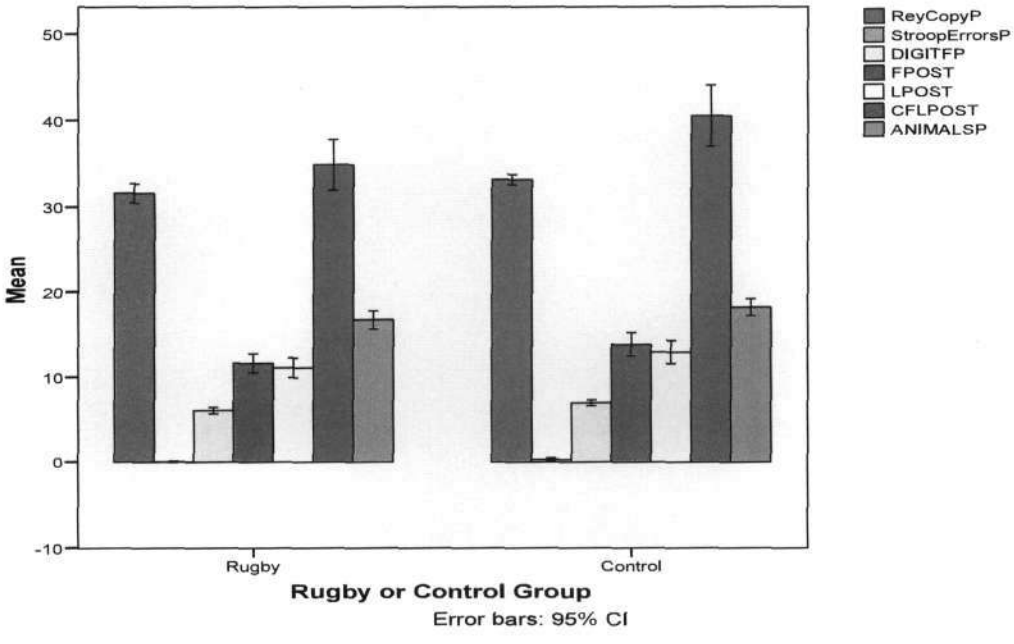


Figure 2: Post-season group mean differences



Since the magnitude of the different scales varied considerably thus making it difficult to obtain a clear picture of overlap between the individual error bars, plots of the variables that were promising were done individually to see if possible significant differences exist. The mean plots of the *pre-season* and *post-season* differences between the groups are presented below. Refer to figures 3 – 12.

Figure 3: Pre-Season Differences between means plots – Rey Complex Figure 2 minute recall: Group

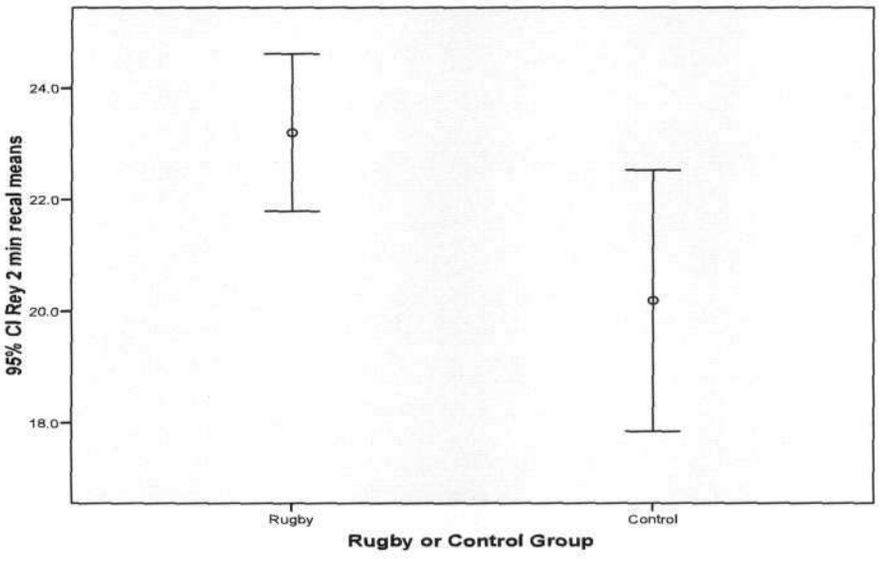


Figure 4: Pre-Season Differences between means plots – B1 (RAVLT Distractor list): Group

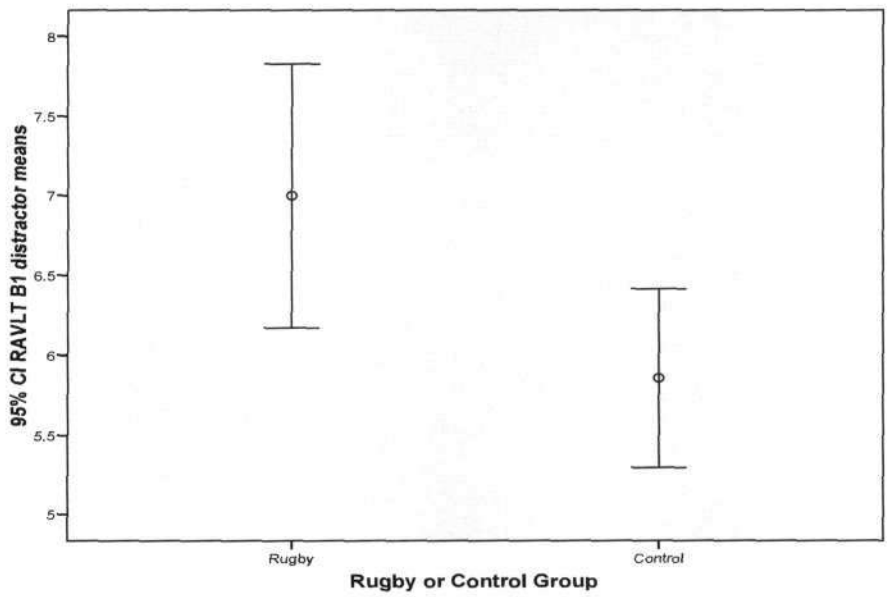
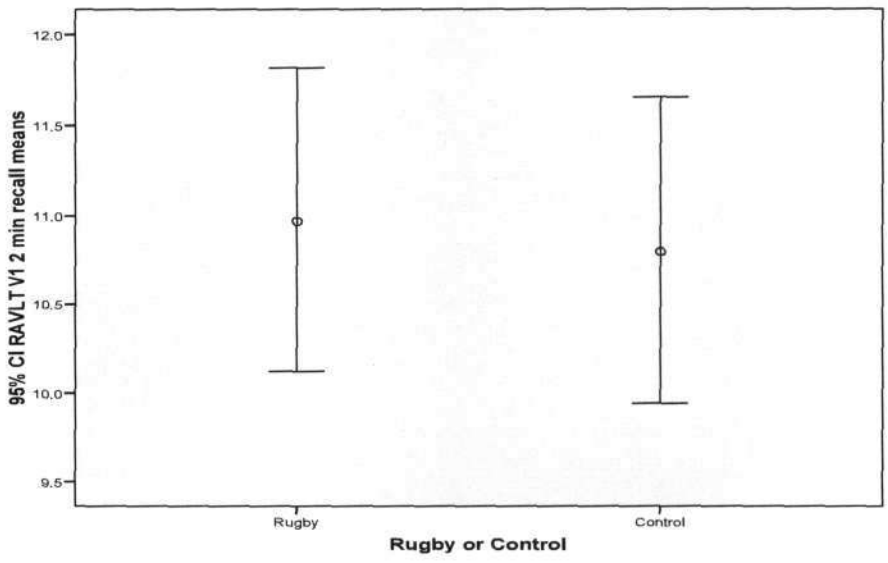


Figure 5: Pre-Season Differences between means plots – V1 (RAVLT 2 min recall): Group



Results from the pre-season testing session are presented above in figures 3 - 5. Overall, these initial exploratory findings of the analysis of differences between groups in the pre-season testing session showed some differences between the two groups on the

following two variables: 1) Rey Complex Figure 2 minute recall and 2) RAVLT B 1 distractor list. There were no significant variations evident between the rugby and control group on all other variables. The graphical representation of the errors bars for both groups of subjects illustrate that these two variables showed some prospect of being found significant on further investigation. This is evident by looking at the level of overlap between the lower and upper bounds of each group i.e., minimal to no overlap between groups is suggestive of probable significant values. An example of a high level of overlap has been submitted to show that this variable (RAVLT V1 2 minute recall), among the rest of the variables, was unlikely to show any evidence of a significant difference between the rugby and control group at the pre-season testing interval. This confirmed that the two groups were indeed similar and thus comparable on almost every measure.

Figure 6: Post-Season Differences between means plots – Rey Complex Figure Copy: Group

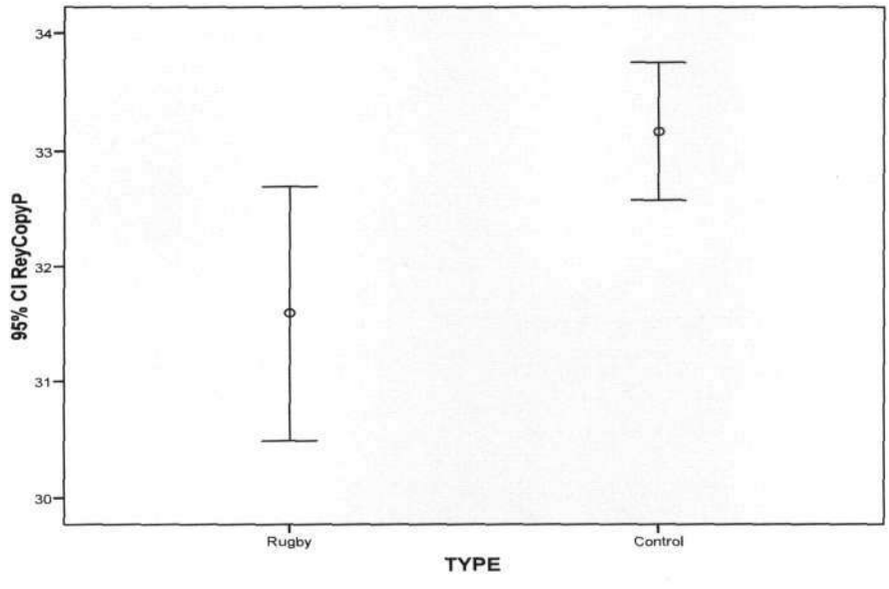


Figure 7: Post-Season Differences between means plots – Digit Forwards: Group

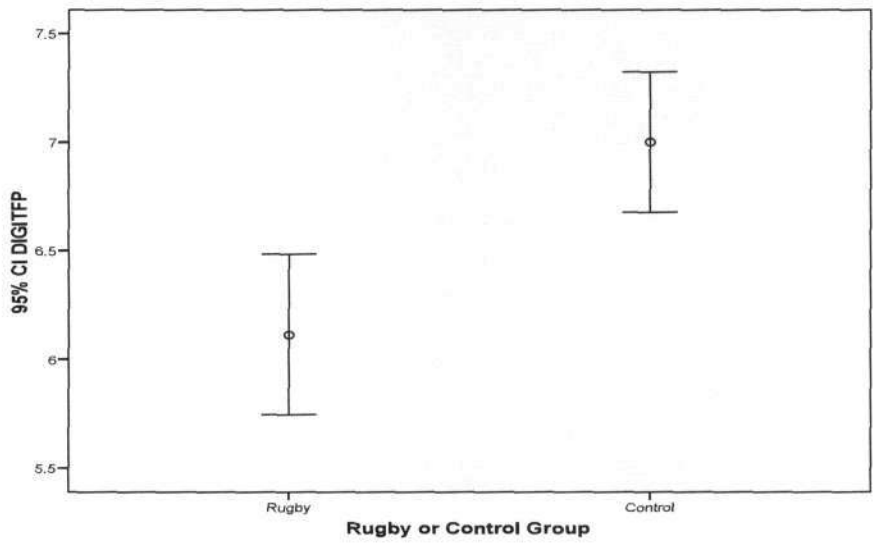


Figure 8: Post-Season Differences between means plots – Stroop Dots Error: Group

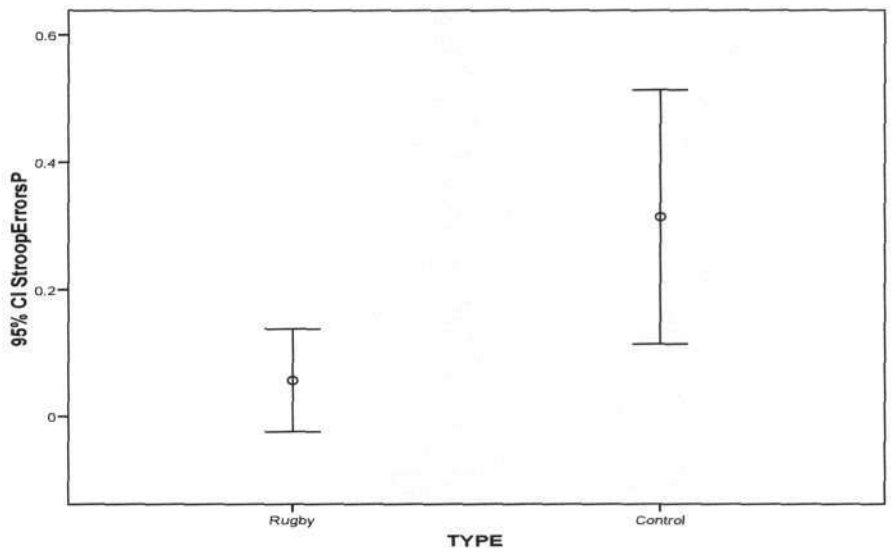


Figure 9: Post-Season Differences between means plots – COWAT letter F: Group

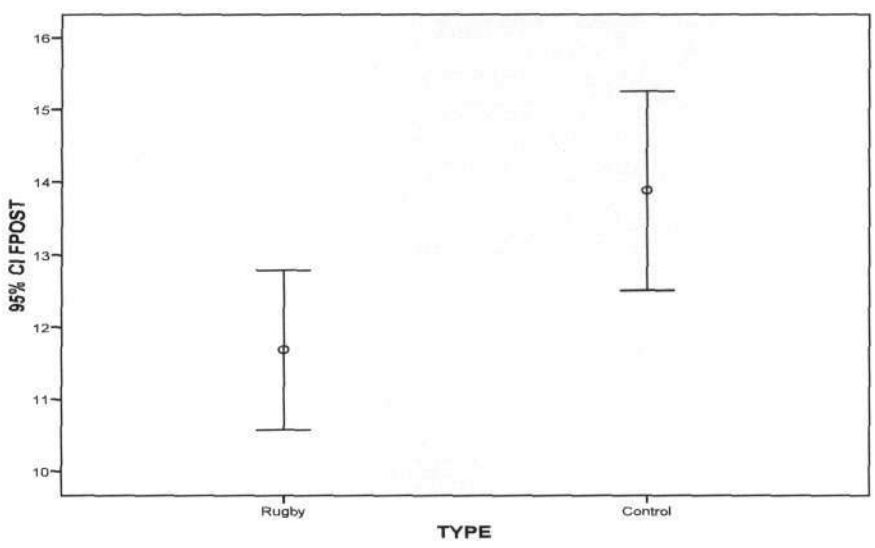


Figure 10: Post-Season Differences between means plots – COWAT letter L: Group

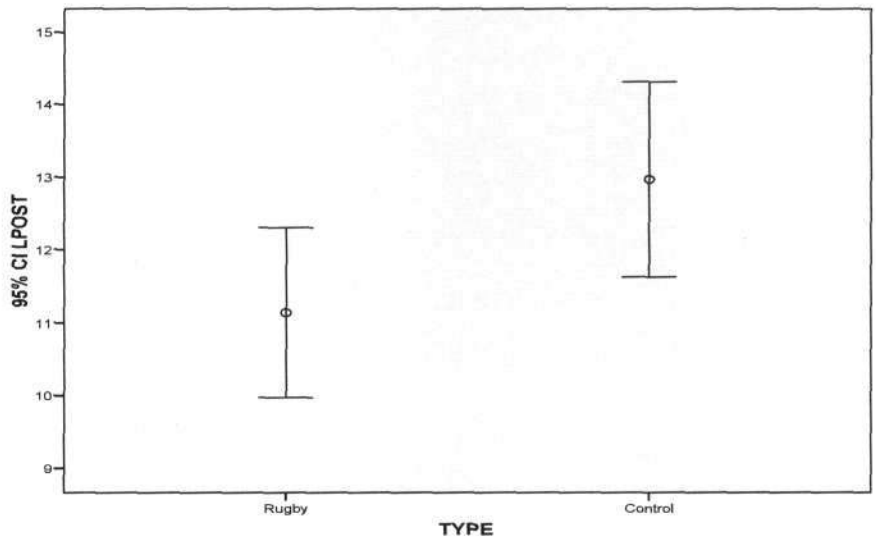


Figure 11: Post-Season Differences between means plots – COWAT letters CFL: Group

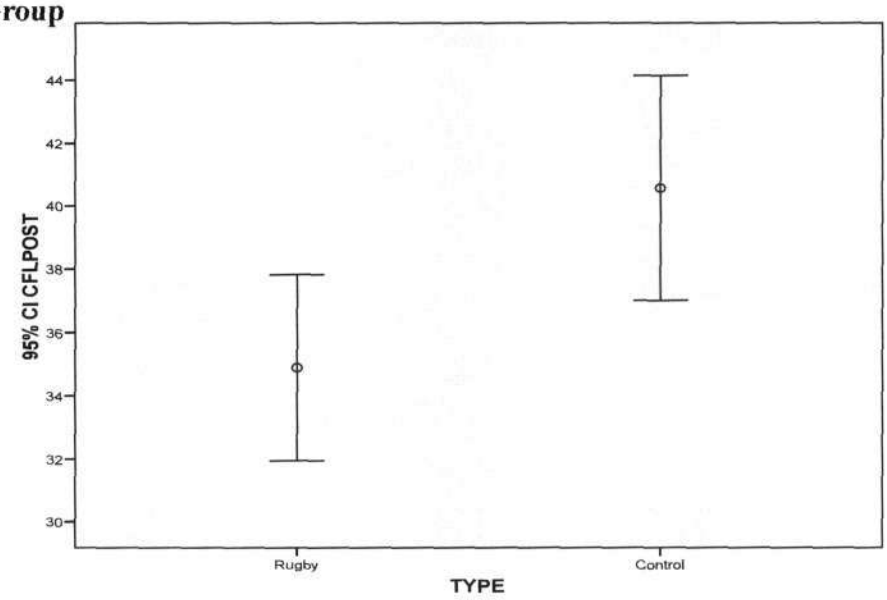
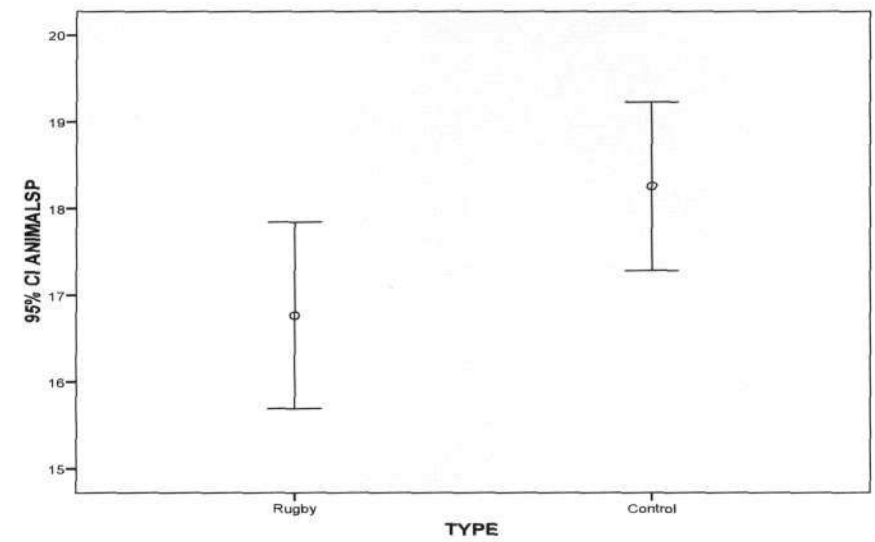


Figure 12: Post-Season Differences between means plots – COWAT Animals: Group



Contrasting to the pre-season test results, the post-season results presented above in figure 6 - 12 suggest that there are numerous differences present between the rugby and control group with regards to neuropsychological functioning on the following 7 variables: 1) Rey Complex Figure (RCF) Copy, 2) Stroop Dot Errors, 3) Digit Forwards, 4) COWAT –F, 5) COWAT-L, 6) COWAT-CFL, 7) COWAT Animals. These results suggest that the rugby group were exposed to some form of MHI/sub-concussive injury over the season that had affected the neuropsychological performance of the players. These results thus encourage the researcher to further analyse and interpret the data, with the aim being to prove or disprove the hypotheses set out in this research.

The independent variable 'Group' (Rugby vs. Control) was further analysed using the SPSS Custom Tables model and the Bonferroni adjustment, which calculated a set of t-tests to assess where any significant differences were present between the two groups i) during the pre-season testing interval and ii) during the post-season testing interval. Table 4 below illustrates that at Pre-season; only 2 of the total 26 variables showed a significant difference between the players and control groups, suggestive that except for these variables, these two groups are likely to be similar in terms of overall neuropsychological functioning. The areas of neuropsychological functioning that appear different between the groups relate to 1) visual memory, planning, visuo spatial and constructional ability (RCF 2 minute recall) and 2) immediate memory (RAVLT B1 distractor list).

The general trend of mean scores suggests that the rugby group as a whole performed better than the control group on most of the measures of the *pre-season* testing interval. Refer to table 4 below.

Table 4 Custom Table: Pre-season Analysis of differences between groups

Dependent Variables	Rugby Mean	SD	Control Mean	SD	Significant Values *p*	Higher rugby scores
RCF Copy	32.4000	2.17540	32.3000	2.78705		#
STROOP Dots	12.7874	1.82906	13.7166	2.83283		#
STROOP Dots Error	.1143	.32280	.2571	.65722		#
STROOP Words	14.3963	2.76123	15.3243	5.42618		#
STROOP Words Error	.1714	.38239	.2000	.47279		#
STROOP Colour	21.5406	4.29369	24.0194	6.78454		#
STROOP Colour Error	.7429	1.03875	1.0286	1.42428		#
RCF 2 min recall	23.2029	4.11371	20.1857	6.82350	*	#
RAVLT Total	52.0286	7.19267	51.6857	7.61489		#
RAVLT Learn	5.8857	1.87509	6.2286	2.19740		
RAVLT Recognition	13.7143	1.20224	13.1714	1.87060		#
RAVLT 20 min delay recall	10.8571	2.43918	10.8571	2.71318		Tie
RAVLT Immediate	6.9143	1.35845	6.7143	1.84026		#
RAVLT B1 distractor list	7.000	2.41320	5.8571	1.62956	*	#
RAVLT V1 2 min delay recall	10.9714	2.46726	10.8000	2.49470		#
SDMT Written	54.3714	7.31672	51.9429	6.99976		
SDMT Oral	60.1429	8.14986	57.7429	6.32615		
Digits forwards	6.3143	.99325	6.7143	.98731		
Digits backwards	5.0000	1.05719	4.9429	1.21129		#
COWAT F	12.1714	3.58498	12.4680	4.55334		
COWAT A	9.9714	2.87469	11.6286	9.00681		
COWAT S	13.9714	3.11057	13.7143	3.90754		#
COWAT FAS	35.8286	7.81584	37.8109	13.83441		
COWAT Animals	17.9143	2.83229	17.4857	3.92107		#
Trail Making A	27.5171	6.60208	28.3603	8.87429		#
Trail Making B	62.7123	20.84742	64.3131	21.78192		#
RCF 20 min delay recall	22.3286	4.98519	20.3857	5.39756		#

***Significance level $p < 0.05$**

 # Mean scores on which the rugby group performed better than the control group

Table 5 below illustrates that during the *post-season* testing interval; 7 of the total 26 dependent variables present with a significant difference between the players and control groups, suggestive that it is possible that some change in cognitive performance took place between these two groups over the period of the rugby season.

During the post-season testing interval the control group appeared to have significantly higher scores compared to that of the rugby group in a range of cognitive domains, 1) including planning (Rey Complex Figure – Copy), 2) visuo spatial constructional ability (Rey Complex Figure – Copy), 3) attention and memory (Digits Forwards), 4) immediate memory (Digit Forwards), and 5) verbal fluency (COWAT – F, L, CFL, Animals). However, on the STROOP Dots Errors task the rugby group appear to have made significantly less mistakes compared to the control group, showing ability for speed of processing, and good levels of attention and concentration on that particular task.

The cognitive domain that appeared to be most sensitive to the 9-month testing interval relates to verbal fluency. On this neuropsychological measure, 4 of the 5 verbal fluency subtests showed significant differences present ($p<.05$) between the rugby and control group. The control group appears to have performed significantly better than the rugby group in this particular cognitive domain.

The rugby group did not appear to have statistically significant lower scores on the other measures compared to the control group, nor did they show a dramatic decrease in their performance between the pre- and post-season testing interval. However, analysis of mean scores suggests a general trend of the control group performing better than the rugby group in a range of cognitive areas. These post-season test results suggest that there is a possibility that the rugby group had been exposed to some type of mild head injury over the nine month period of playing rugby, which has negatively affected their neuropsychological performance. It also shows that the control group appeared to have

benefitted more from learning and 'practice effects' which resulted in improved overall cognitive performance compared to the rugby group, again reiterating the possibility that the rugby group suffered from possible sub-concussive injuries which affected their ability to benefit for 'practice effects'. Refer to table 5 below.

Table 5 Custom Table: Post-season Analysis of differences between groups

Dependent Variables	Rugby Mean	SD	Control Mean	SD	Significant Values *p*	Higher rugby scores
RCF Copy	31.6000	3.21943	33.1714	1.69750	*	
STROOP Dots	12.6160	2.16438	12.8551	2.56924		#
STROOP Dots Error	.0571	.23550	.3143	.58266	*	#
STROOP Words	15.1009	3.51783	14.0554	3.00107		
STROOP Words Error	.2000	.47279	.0857	.28403		
STROOP Colour	19.7534	4.95390	19.1634	4.41189		
STROOP Colour Error	.4857	.78108	.2857	.57248		
RCF 2 min recall	25.2857	5.08788	25.4286	5.30558		
RAVLT Total	49.7429	5.88789	50.8000	7.95872		
RAVLT Learn	5.6286	2.40203	5.9714	1.97761		
RAVLT Recognition	13.2571	1.57821	13.6286	1.11370		
RAVLT 20 min delay recall	10.9143	2.29285	10.2571	2.53613		#
RAVLT Immediate	6.4286	1.52017	6.6857	1.65869		
RAVLT B1 distractor list	5.3429	1.62595	5.9143	2.22778		
RAVLT A6 2 min delay recall	10.8857	2.15258	10.4286	2.45292		#
SDMT Written	56.3143	8.14129	57.2286	8.17128		#
SDMT Oral	63.0857	8.10364	60.7714	9.29923		
Digits forwards	6.1143	1.07844	7.0000	.93934	*	
Digits backwards	4.6857	1.10537	4.9143	.98134		
COWAT C	12.0571	3.26247	13.7143	3.69874		
COWAT F	11.6857	3.21551	13.8857	3.99832	*	
COWAT L	11.1429	3.39674	12.9714	3.91442	*	
COWAT CFL	34.8857	8.56385	40.5714	10.39877	*	
COWAT Animals	16.7714	3.12566	18.2571	2.82159	*	
Trail Making A	26.5389	6.00233	26.8743	7.70665		#
Trail Making B	61.4911	19.38580	59.4080	18.86683		
RCF 20 min delay recall	24.3571	4.41850	25.2286	5.39927		

***Significance level $p < 0.05$**

 # Scores on which the rugby group performed better than the control group

A multivariate statistical model was used to systematically control for a within (pre/post) and between (control/rugby) factor design, and to analyse how the groups' cognitive functioning changed over the 9-month period. Within this Repeated Measures model, multivariate tests of within subjects effects ('Pre/Post' – i.e. both pre- and post-season measurements) and between subjects ('Group' – Rugby vs. Control groups) effects were examined. For the purpose and depth of this study, only the significant values ($p<.05$) will be explained below.

Rey Complex Figure (RCF) – Copy

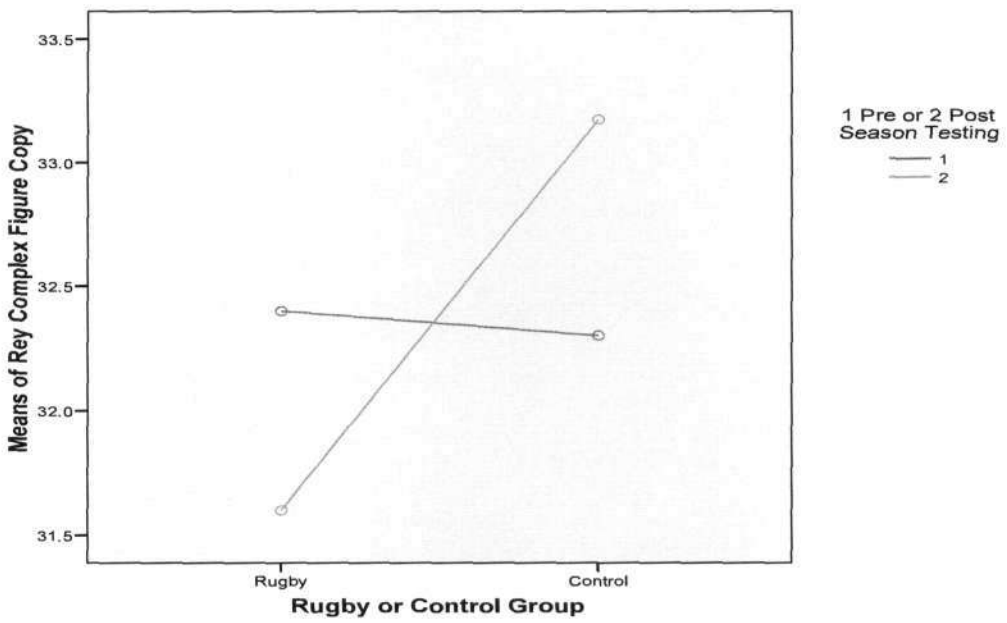
The Rey Complex Figure Copy sub-test Pre-Post main effect was not significant (Pre-Post: $F(1) = 0.009$, $p = 0.925$), however the interaction between Pre-Post and Group factors was significant (Pre-Post by Group $F(1) = 4.912$; $p = 0.030$). Refer to table 6 below. It appears that the rugby scores showed a relative decrease and the control scores showed a relative increase between the testing intervals indicating that the control group benefited from learning and practice effects whereas the rugby group did not (see Figure 13).

Table 6: Multivariate Tests for Rey Complex Figure - Copy

Effect		Value	F	Hypothesis df	Error df	Sig.
PrePost	Pillai's Trace	.000	.009(a)	1.000	68.000	.925
	Wilks' Lambda	1.000	.009(a)	1.000	68.000	.925
	Hotelling's Trace	.000	.009(a)	1.000	68.000	.925
	Roy's Largest Root	.000	.009(a)	1.000	68.000	.925
PrePost * Group	Pillai's Trace	.067	4.912(a)	1.000	68.000	.030
	Wilks' Lambda	.933	4.912(a)	1.000	68.000	.030
	Hotelling's Trace	.072	4.912(a)	1.000	68.000	.030
	Roy's Largest Root	.072	4.912(a)	1.000	68.000	.030

Significance level p <0.05
a Exact statistic
b Design: Intercept+TYPE
Within Subjects Design: factor1

Figure 13 estimated Marginal Mean of Rey Complex Figure (RCF) - Copy



Stroop Dot Error

The main effect between the Between Subjects Test was significant (F=5.357; p=0.024), thus the two groups were different from each other regardless of the measurement interval. Refer to table 7 below. Players group pre-season mean score was 0.1143 and the players post-season mean score was .0571. Control groups pre-season mean score was 0.2571 and the control post-season mean score was 0.3143. Refer to table 7 below.

Table 7: Tests of Between-Subjects Effects for Stroop Dot Error

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power (a)
Intercept	2.414	1	2.414	18.476	.000	.214	18.476	.989
Group	.700	1	.700	5.357	.024	.073	5.357	.626
Error	8.886	68	.131					

Significance level $p < 0.05$
Transformed Variable: Average
a Computed using alpha = .05

Stroop Colour

The Stroop Colour test Pre-Post main effect was significant (Pre-Post: F(1) = 19.83, p = <0.001), and the interaction between Pre-Post and Group was also significant (Pre-Post by Group F(1) = 4.23; p = 0.043). Refer to table 8 below. It appears that the control group's timed scores were initially poorer than the rugby group's performance at pre-season, however the control group improved significantly over those of the rugby group at the post-season measurement interval. This indicates that the control group benefited more from learning and practice effects than did the rugby group. (see Figure 14 below).

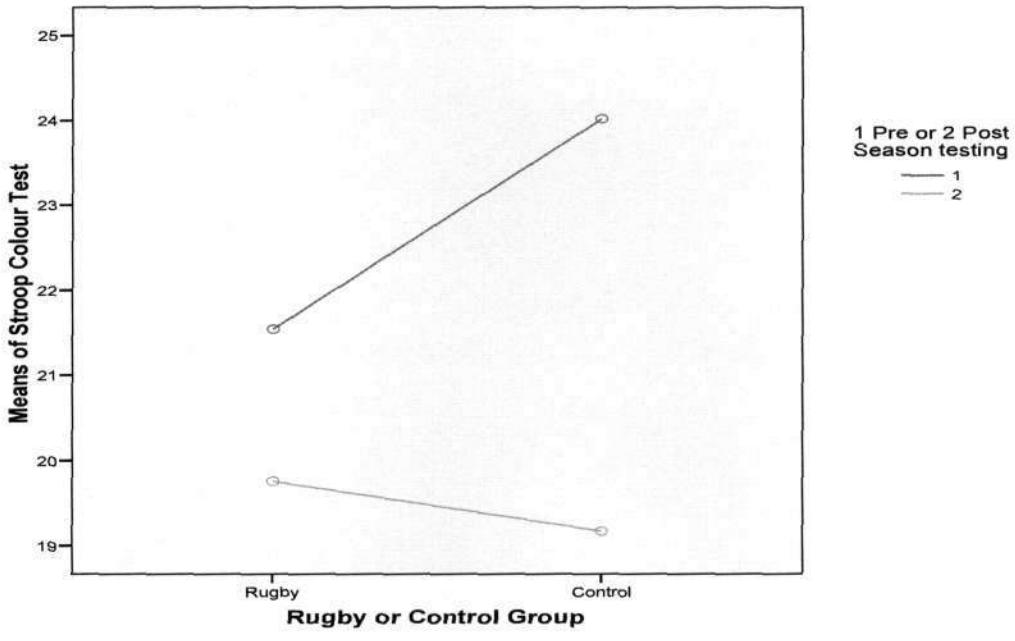
Table 8: Multivariate Tests for Stroop Colour

Significance level p <0.05

Effect		Value	F	Hypothesis df	Error df	Sig.
PrePost	Pillai's Trace	.226	19.832(b)	1.000	68.000	.000
	Wilks' Lambda	.774	19.832(b)	1.000	68.000	.000
	Hotelling's Trace	.292	19.832(b)	1.000	68.000	.000
	Roy's Largest Root	.292	19.832(b)	1.000	68.000	.000
PrePost * Group	Pillai's Trace	.059	4.232(b)	1.000	68.000	.043
	Wilks' Lambda	.941	4.232(b)	1.000	68.000	.043
	Hotelling's Trace	.062	4.232(b)	1.000	68.000	.043
	Roy's Largest Root	.062	4.232(b)	1.000	68.000	.043

a Computed using alpha = .05
b Exact statistic
c Design: Intercept+Group
Within Subjects Design: PrePost

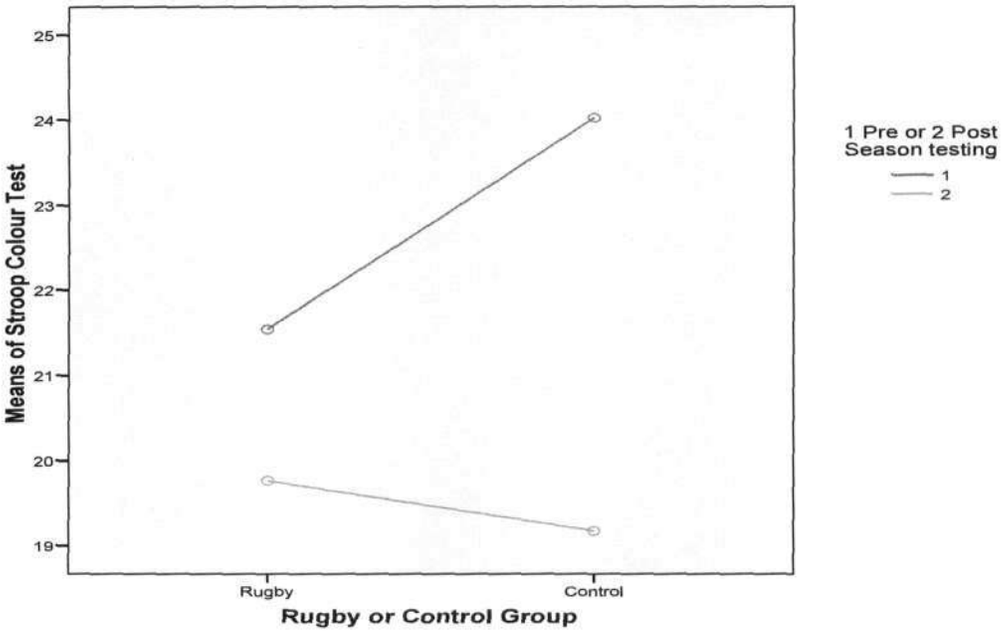
Figure 14 Estimated Marginal means of STROOP COLOUR TEST



Stroop Colour Error

The Stroop Colour Error test PrePost main effect was significant (Pre-Post: F(1) = 9.969, p= 0.002), however the interaction between PrePost and Group was not significant (Pre-Post by group F(1) = 2.352, p= .130). Refer to table 9 below. This shows that both the control and rugby group scores improved, in that they made fewer errors on this test at the post-season testing interval. The control group initially performed poorer than the rugby group at the pre-season testing session, however it appears that the control group made fewer errors over the post-season measurement interval than did the rugby group but this was not statistically significant (see Figure 15 below). The results show that the control group benefited more from practice effects than did the rugby group. (see Figure 15 below).

Figure 14 Estimated Marginal means of STROOP COLOUR TEST



Stroop Colour Error

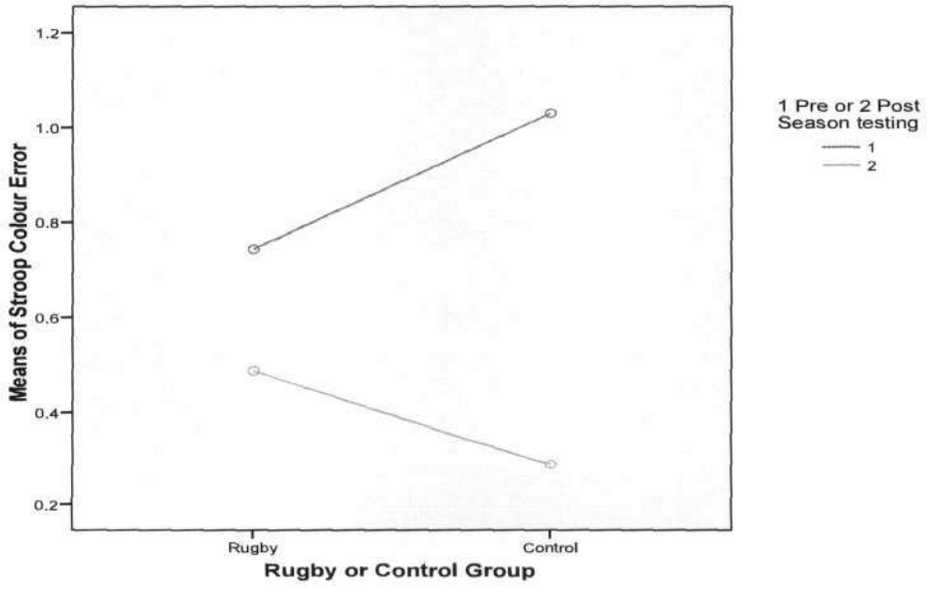
The Stroop Colour Error test PrePost main effect was significant (Pre-Post: F(1) = 9.969, p= 0.002), however the interaction between PrePost and Group was not significant (Pre-Post by group F(1) = 2.352, p= .130). Refer to table 9 below. This shows that both the control and rugby group scores improved, in that they made fewer errors on this test at the post-season testing interval. The control group initially performed poorer than the rugby group at the pre-season testing session, however it appears that the control group made fewer errors over the post-season measurement interval than did the rugby group but this was not statistically significant (see Figure 15 below). The results show that the control group benefited more from practice effects than did the rugby group. (see Figure 15 below).

Table 9: Multivariate Tests For Stroop Colour Error

Effect		Value	F	Hypothesis df	Error df	Sig.
PrePost	Pillai's Trace	.128	9.969(b)	1.000	68.000	.002
	Wilks' Lambda	.872	9.969(b)	1.000	68.000	.002
	Hotelling's Trace	.147	9.969(b)	1.000	68.000	.002
	Roy's Largest Root	.147	9.969(b)	1.000	68.000	.002
PrePost * Group	Pillai's Trace	.033	2.352(b)	1.000	68.000	.130
	Wilks' Lambda	.967	2.352(b)	1.000	68.000	.130
	Hotelling's Trace	.035	2.352(b)	1.000	68.000	.130
	Roy's Largest Root	.035	2.352(b)	1.000	68.000	.130

Significance level $p < 0.05$
a Computed using alpha = .05
b Exact statistic
c Design: Intercept+Group
Within Subjects Design: PrePost

Figure 15 Estimated Marginal means of STROOP COLOUR WORD ERROR TEST



Rey Complex Figure 2 Minute Delayed Recall

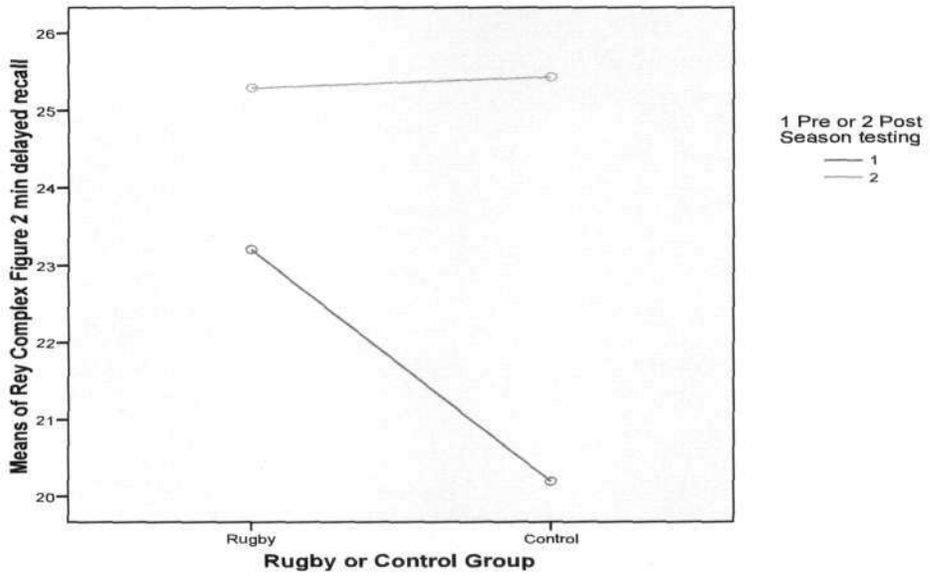
The REY COMPLEX FIGURE 2 Minute Recall test Pre-Post main effect was significant (Pre-Post: F(1) = 35.228, p = <0.001), and the interaction between Pre-Post and Group was also significant (Pre-Post by Group F(1) = 6.566; p = 0.013). Refer to table 10 below. Although the control group preformed less well than the rugby group at the pre-season testing session, it appears that the control group's recall scores improved significantly over those of the rugby group at the post-season measurement interval (see Figure 16 below). This indicates that the control group benefited more from learning and practice effects compared to the rugby group.

Table 10: Multivariate Tests for REY COMPLEX FIGURE 2 Minute Delayed Recall

Effect		Value	F	Hypothesis df	Error df	Sig.
PrePost	Pillai's Trace	.342	35.288(b)	1.000	68.000	.000
	Wilks' Lambda	.658	35.288(b)	1.000	68.000	.000
	Hotelling's Trace	.519	35.288(b)	1.000	68.000	.000
	Roy's Largest Root	.519	35.288(b)	1.000	68.000	.000
PrePost * Group	Pillai's Trace	.088	6.566(b)	1.000	68.000	.013
	Wilks' Lambda	.912	6.566(b)	1.000	68.000	.013
	Hotelling's Trace	.097	6.566(b)	1.000	68.000	.013
	Roy's Largest Root	.097	6.566(b)	1.000	68.000	.013

Significance level p <0.05
a Computed using alpha = .05
b Exact statistic
c Design: Intercept+Group
Within Subjects Design: PrePost

Figure 16: Estimated Marginal means of REY COMPLEX FIGURE 2 MINUTE DELAYED RECALL



RAVLT B – Distractor List

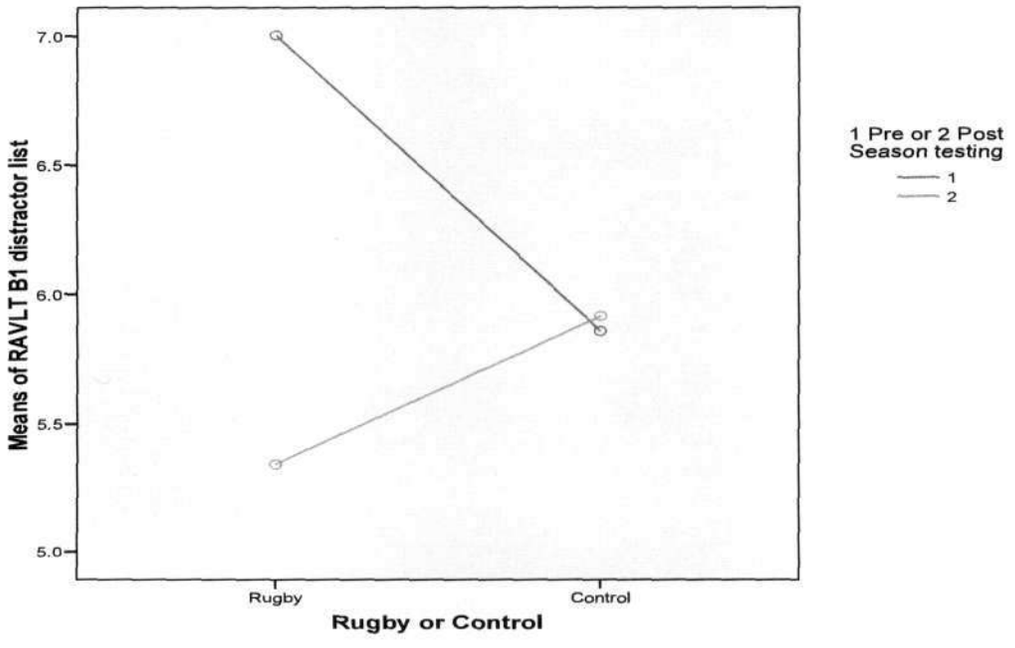
The RAVLT B distractor list test Pre-Post main effect was significant (Pre-Post: F(1) = 6.120, $p = 0.016$), and the interaction between Pre-Post and Group was also significant (Pre-Post by Group F(1) = 7.026; $p = 0.010$). Refer to table 11 below. It appears (see Figure 17 below) that the rugby group show a significant decline in scores over the measurement interval, whereas the control groups show no significant decline in memory performance. The results show that neither group benefited from learning and practice effects on this particular measure.

Table 11: Multivariate Tests for RAVLT B Distractor list

Effect		Value	F	Hypothesis df	Error df	Sig.
Prepost	Pillai's Trace	.083	6.120(a)	1.000	68.000	.016
	Wilks' Lambda	.917	6.120(a)	1.000	68.000	.016
	Hotelling's Trace	.090	6.120(a)	1.000	68.000	.016
	Roy's Largest Root	.090	6.120(a)	1.000	68.000	.016
PrePost*Group	Pillai's Trace	.094	7.026(a)	1.000	68.000	.010
	Wilks' Lambda	.906	7.026(a)	1.000	68.000	.010
	Hotelling's Trace	.103	7.026(a)	1.000	68.000	.010
	Roy's Largest Root	.103	7.026(a)	1.000	68.000	.010

Significance level p <0.05
a Exact statistic
b Design: Intercept+TYPE
Within Subjects Design: factor1

Figure 17 Estimated Marginal means of RAVLT B Distractor List



SDMT Written

The SDMT Written sub-test Pre-Post main effect was significant (Pre-Post: F(1) = 25.473, p = <0.001), and the interaction between Pre-Post and Group was also significant (Pre-Post by Group F(1) = 5.448; p = 0.023). Refer to table 12 below. Both the control

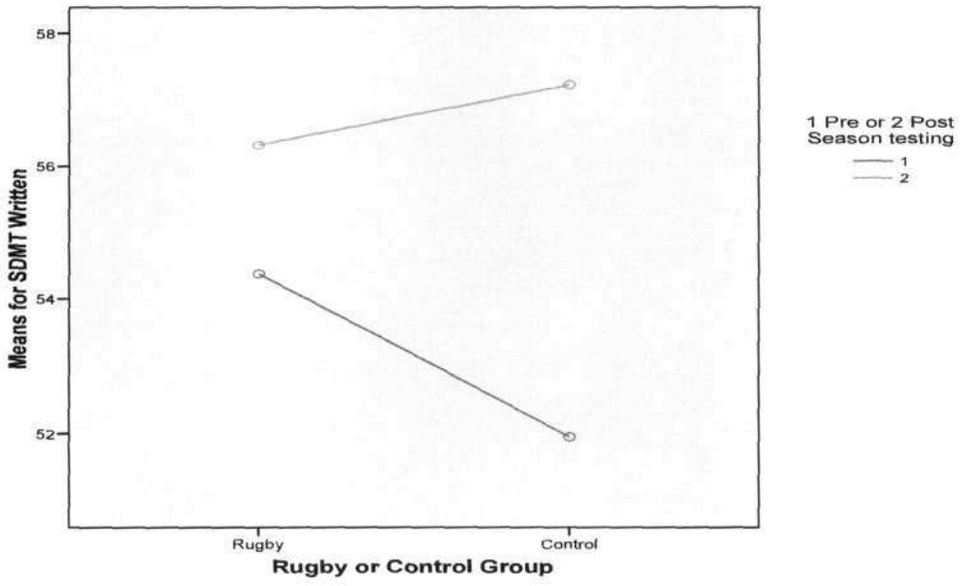
and rugby group show an improvement in scores over the measurement interval. It appears (see Figure 18 below) that the control group's speed of processing scores improved significantly over those of the rugby group at the post-season measurement interval. This indicates that both groups benefited from learning and practice effects, however the control appear to have benefited more from these effects than did the rugby group.

Table 12: Multivariate Tests for SDMT Written

Effect		Value	F	Hypothesis df	Error df	Sig.
PrePost	Pillai's Trace	.273	25.473(b)	1.000	68.000	.000
	Wilks' Lambda	.727	25.473(b)	1.000	68.000	.000
	Hotelling's Trace	.375	25.473(b)	1.000	68.000	.000
	Roy's Largest Root	.375	25.473(b)	1.000	68.000	.000
PrePost * Group	Pillai's Trace	.074	5.448(b)	1.000	68.000	.023
	Wilks' Lambda	.926	5.448(b)	1.000	68.000	.023
	Hotelling's Trace	.080	5.448(b)	1.000	68.000	.023
	Roy's Largest Root	.080	5.448(b)	1.000	68.000	.023

Significance level $p < 0.05$
a Computed using alpha = .05
b Exact statistic
c Design: Intercept+Group
Within Subjects Design: PrePost

Figure 18 Estimated Marginal means for SDMT Written



SDMT Oral

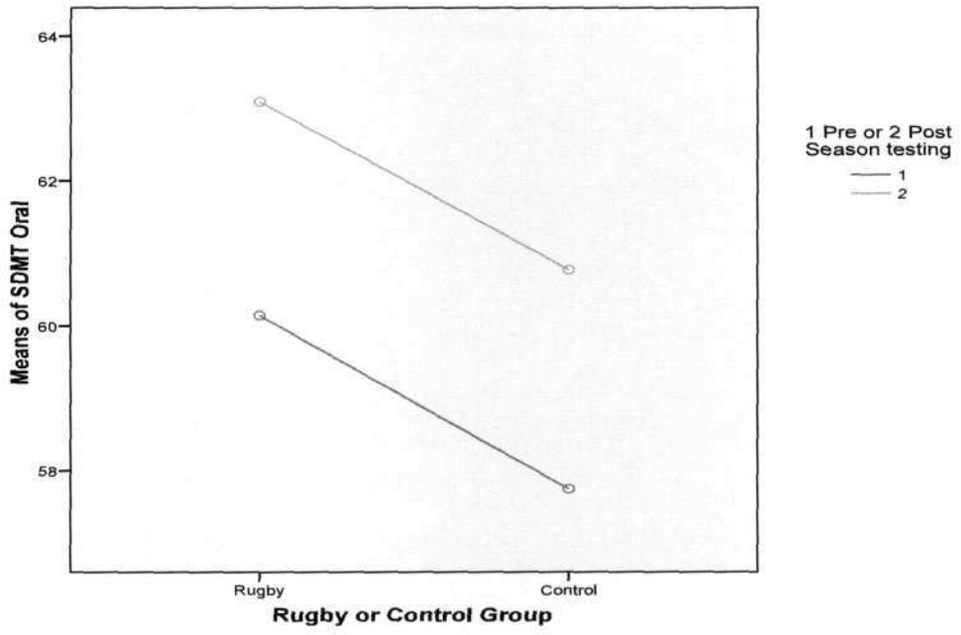
The SDMT Oral sub-test Pre-Post main effect was significant (Pre-Post: $F(1) = 8.740$, $p = 0.004$), and the interaction between Pre-Post and Group was not significant (Pre-Post by Group $F(1) = 0.002$; $p = 0.966$). Refer to table 13 below. The control group performed better than the rugby group on both the testing intervals. Both the control and rugby group show an improvement in speed of processing scores over the measurement interval. It appears (see Figure 19 below) that both the control and rugby group's scores improved significantly over the post-season measurement interval, indicating that both groups benefited from learning and practice effects on this particular measure.

Table 13: Multivariate Tests for SDMT ORAL

Effect		Value	F	Hypothesis df	Error df	Sig.
PrePost	Pillai's Trace	.114	8.740(b)	1.000	68.000	.004
	Wilks' Lambda	.886	8.740(b)	1.000	68.000	.004
	Hotelling's Trace	.129	8.740(b)	1.000	68.000	.004
	Roy's Largest Root	.129	8.740(b)	1.000	68.000	.004
PrePost * Group	Pillai's Trace	.000	.002(b)	1.000	68.000	.966
	Wilks' Lambda	1.000	.002(b)	1.000	68.000	.966
	Hotelling's Trace	.000	.002(b)	1.000	68.000	.966
	Roy's Largest Root	.000	.002(b)	1.000	68.000	.966

Significance level $p < 0.05$
a Computed using alpha = .05
b Exact statistic
c Design: Intercept+Group
Within Subjects Design: PrePost

Figure 19 Estimated Marginal means for SDMT ORAL



Digits Forwards

The main effect between the Between Subjects Test was significant (F=9.850; p=0.003), thus the two groups are different from each other regardless of the measurement interval. Refer to table 14 below. The players group pre-season mean score was 6.3143 and the players group post-season mean score was 6.1143. The control groups pre-season mean score was 6.7143 and the control groups post-season mean score was 7.0000.

Table 14: Tests of Between-Subjects Effects for Digits Forwards

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power(a)
Intercept	2990.089	1	2990.089	4072.339	.000	.984	4072.339	1.000
Group	7.232	1	7.232	9.850	.003	.127	9.850	.872
Error	49.929	68	.734					

Significance level $p < 0.05$
Transformed Variable: Average
a Computed using alpha = .05

COWAT – Animal

The COWAT animal sub-test Pre-Post main effect was not significant (Pre-Post: F(1) = 0.169, $p = 0.682$), however the interaction between Pre-Post and Group was significant (Pre-Post by Group F(1) = 4.484 ; p 0.038). Refer to table 15 below.

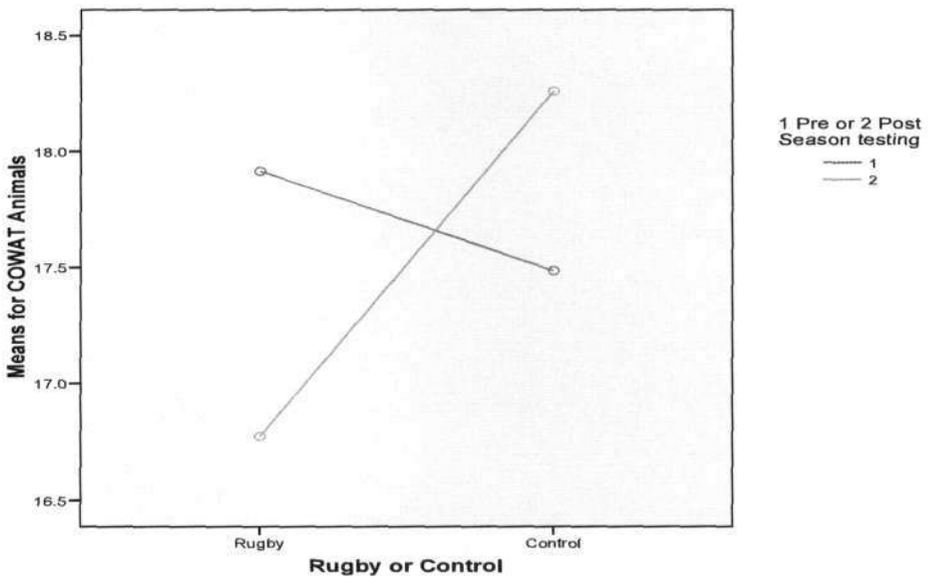
It appears (see Figure 20 below) the rugby scores showed a relative decrease in scores and the control scores showed a relative increase in scores between the testing intervals. This indicates that the control group benefited from learning and practice effects whereas the rugby group did not.

Table 15: Multivariate Tests for COWAT Animals

Effect		Value	F	Hypothesis df	Error df	Sig.
Pre-post	Pillai's Trace	.002	.169(a)	1.000	68.000	.682
	Wilks' Lambda	.998	.169(a)	1.000	68.000	.682
	Hotelling's Trace	.002	.169(a)	1.000	68.000	.682
	Roy's Largest Root	.002	.169(a)	1.000	68.000	.682
Pre-post * Group	Pillai's Trace	.062	4.484(a)	1.000	68.000	.038
	Wilks' Lambda	.938	4.484(a)	1.000	68.000	.038
	Hotelling's Trace	.066	4.484(a)	1.000	68.000	.038
	Roy's Largest Root	.066	4.484(a)	1.000	68.000	.038

Significance level p <0.05
a Exact statistic
b Design: Intercept+TYPE
Within Subjects Design: factor1

Figure 20 : Estimated Marginal means for COWAT Animals



REY Complex Figure 20 minute delayed recall

The Rey Complex Figure test Pre-Post main effect was significant (Pre-Post: $F(1) = 33.851$, $p = <0.001$), and the interaction between Pre-Post and Group was also significant (Pre-Post by Group $F(1) = 5.678$; $p = 0.020$). Refer to table 16 below.

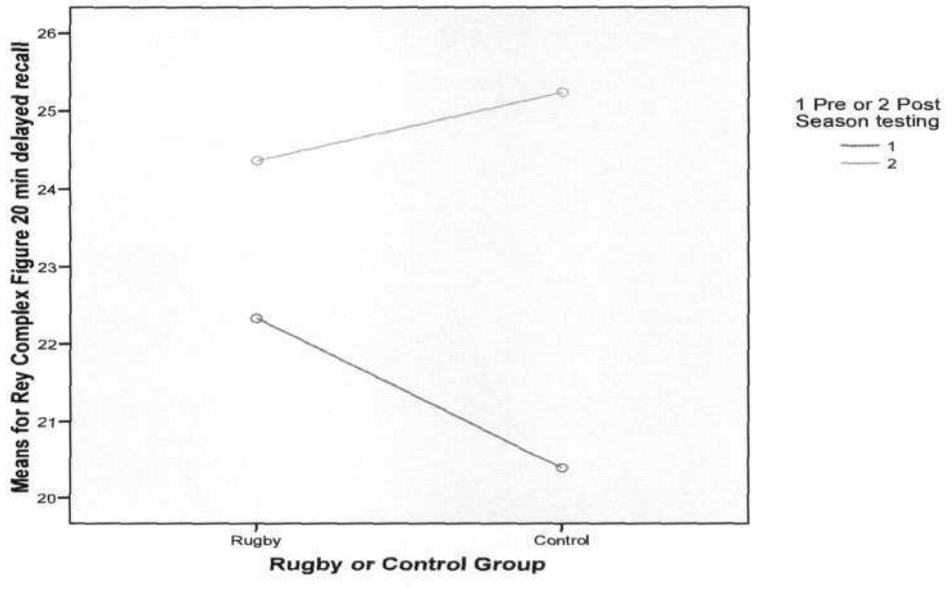
It appears (see Figure 21 below) that both the control and rugby group increased their scores over the testing interval, and that the control group's long term memory scores improved significantly over those of the rugby group at the post-season measurement interval. This indicates that both the control and rugby group benefited from learning and practice effects, however the control group appear to have benefited more from these effects than did the rugby group.

Table 16: Multivariate Tests for RCF 20 minute delayed recall

Effect		Value	F	Hypothesis df	Error df	Sig.
PrePost	Pillai's Trace	.332	33.851(b)	1.000	68.000	.000
	Wilks' Lambda	.668	33.851(b)	1.000	68.000	.000
	Hotelling's Trace	.498	33.851(b)	1.000	68.000	.000
	Roy's Largest Root	.498	33.851(b)	1.000	68.000	.000
PrePost * Group	Pillai's Trace	.077	5.678(b)	1.000	68.000	.020
	Wilks' Lambda	.923	5.678(b)	1.000	68.000	.020
	Hotelling's Trace	.084	5.678(b)	1.000	68.000	.020
	Roy's Largest Root	.084	5.678(b)	1.000	68.000	.020

Significance level p <0.05
a Computed using alpha = .05
b Exact statistic
c Design: Intercept+Group
Within Subjects Design: PrePost

Figure 21: Estimated Marginal means for RCF 20 minute delayed recall



To summarize, mean score analysis of the post-season testing scores via the use of custom tables showed that there were numerous significant differences present between the two groups, on a range of cognitive domains, namely planning, visuo spatial and constructional ability (RCF copy), attention and concentration (Stroop Dot errors, digits

forwards), memory (digits forwards) and verbal fluency (COWAT CFL, Animals), whereby the rugby players performed more poorly compared to the control group.

The ANOVA Analysis highlighted an interesting aspect of the 'learning and practice effect' over the 9-month period. The rugby playing group appears to have benefited from 'practice and learning effects' in only 7 out of the 27 sections, compared with the controls group who benefited from learning effects in 19 of the 27 sections. These results is suggestive that numerous rugby players were exposed to some from of sub-concussive or MHI over the season which affected their neuropsychological performance.

HYPOHESIS 2:

When players report concussion during the rugby season and are assessed within 48 hours of their injury, is there a significant change from their baseline levels of performance? If so, on what measures do these individuals differ? It is hypothesised that concussion scores should be significantly less than their baseline scores, particularly in relation to areas of planning, visuo spatial and constructional ability; attention and concentration; memory; verbal fluency and speed of information processing. Are there specific areas of cognitive functioning that are more severely affected as a result of this concussion?

The data analysis proceeded in 3 phases:

1) Descriptive statistics to 'eyeball' the data via the use of error bars
2) Repeated Measures ANOVA
3) Table of means – descriptive analysis

Due to the number of concussed players being low n=5; it did not prove to be statistically sound to initially run t-tests on these measures. The researcher thus primarily

forwards), memory (digits forwards) and verbal fluency (COWAT CFL, Animals), whereby the rugby players performed more poorly compared to the control group.

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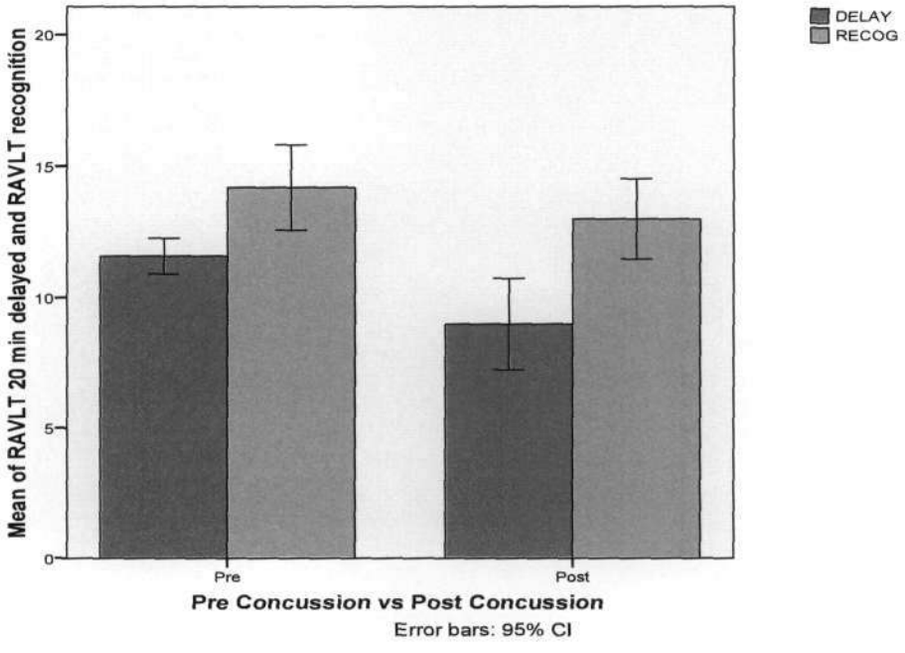
2) Repeated Measures ANOVA
3) Table of means – descriptive analysis

Due to the number of concussed players being low n=5; it did not prove to be statistically sound to initially run t-tests on these measures. The researcher thus primarily

explored the data via the use of error bars to see if any noticeable differences among the dependent variables were present. However, no variable, except RAVLT 20 minute delayed recall seemed likely to show a significant difference. An example of one of the variables (RAVLT Recognition) that did not look like it would approach significance level has been added to Figure 22 below. This variable was represented as a means of explaining how all variables were visually assessed using this error bar method. Due to the high level of overlap between the upper and lower bounds of the RAVLT Recognition scores for the rugby and control group at pre and post-season testing, we can presume that this measure, among all remaining variables, were unlikely to reach significance levels even upon further statistical analysis. Refer to Figure 22 below.

Figure 22: Player's RAVLT (20 min delayed recall & recognition) mean scores pre and post concussive injury



Following the error bars analysis, a Repeated Measures ANOVA was run with all the dependent variables, and a single IV (Pre-post) to identify any significant values. This test

is generally deemed a more powerful measure of detecting significant differences between the player's baseline and concussed scores. However, it is important to note that this tests results should be interpreted with great caution, as it has no corrections for multiple comparisons, such as using the Bonferroni test, and there was an extremely small sample of concussed players, n=5. Thus the initial error bars are strongly relied upon to suggest possible group differences. It is further evident from the ANOVA analysis that RAVLT 20 minute delayed is the only variable that appeared to show a significant difference between baseline testing and post concussion testing ($F=14.7$; $p=0.005$). Refer to table 17 below.

Table 17: ANOVA for Pre and Post concussion testing

ANOVA Table [a,b,c,d,e,f,g,h,i,j,k,l,m,n,o,p,q,r,s,t,u,v,w,x,y,z,aa,bb,cc]

			Sum of Squares	df	Mean Square	F	Sig.
RAY * PrePost	Between Groups	(Combined)	8.100	1	8.100	1.728	.225
	Within Groups		37.500	8	4.688		
	Total		45.600	9			
Dots * PrePost	Between Groups	(Combined)	.812	1	.812	.056	.819
	Within Groups		116.214	8	14.527		
	Total		117.026	9			
Error * PrePost	Between Groups	(Combined)	.400	1	.400	1.000	.347
	Within Groups		3.200	8	.400		
	Total		3.600	9			
Words * PrePost	Between Groups	(Combined)	4.436	1	4.436	.252	.629
	Within Groups		140.628	8	17.578		
	Total		145.063	9			
Error * PrePost	Between Groups	(Combined)	.100	1	.100	1.000	.347
	Within Groups		.800	8	.100		
	Total		.900	9			
Colour * PrePost	Between Groups	(Combined)	87.143	1	87.143	3.033	.120
	Within Groups		229.884	8	28.736		
	Total		317.027	9			
Error * PrePost	Between Groups	(Combined)	4.900	1	4.900	1.256	.295
	Within Groups		31.200	8	3.900		
	Total		36.100	9			
RAY * PrePost	Between Groups	(Combined)	60.025	1	60.025	1.450	.263
	Within Groups		331.200	8	41.400		
	Total		391.225	9			
TOTAL * PrePost	Between Groups	(Combined)	2.500	1	2.500	.179	.683
	Within Groups		111.600	8	13.950		
	Total		114.100	9			
LEARN * PrePost	Between Groups	(Combined)	.400	1	.400	.133	.724
	Within Groups		24.000	8	3.000		
	Total		24.400	9			
RECOG * PrePost	Between Groups	(Combined)	3.600	1	3.600	2.250	.172
	Within Groups		12.800	8	1.600		
	Total		16.400	9			
DELAY * PrePost	Between Groups	(Combined)	16.900	1	16.900	14.696	.005
	Within Groups		9.200	8	1.150		
	Total		26.100	9			
IMM * PrePost	Between Groups	(Combined)	2.500	1	2.500	.833	.388
	Within Groups		24.000	8	3.000		
	Total		26.500	9			
VI * PrePost	Between Groups	(Combined)	.400	1	.400	.127	.731
	Within Groups		25.200	8	3.150		
	Total		25.600	9			
B1 * PrePost	Between Groups	(Combined)	1.600	1	1.600	.314	.591
	Within Groups		40.800	8	5.100		
	Total		42.400	9			
SDMT WRITTN * PrePost	Between Groups	(Combined)	12.100	1	12.100	.248	.632
	Within Groups		390.400	8	48.800		
	Total		402.500	9			
SDMT ORAL * PrePost	Between Groups	(Combined)	3.600	1	3.600	.220	.651
	Within Groups		130.800	8	16.350		
	Total		134.400	9			
DIGIT FORWARD * PrePost	Between Groups	(Combined)	.400	1	.400	.615	.455
	Within Groups		5.200	8	.650		
	Total		5.600	9			
DIGIT BACKWARDS * PrePost	Between Groups	(Combined)	2.500	1	2.500	2.381	.161
	Within Groups		8.400	8	1.050		
	Total		10.900	9			
MAZE1 * PrePost	Between Groups	(Combined)	52.946	1	52.946	3.473	.099
	Within Groups		121.950	8	15.244		
	Total		174.896	9			
MAZE2 * PrePost	Between Groups	(Combined)	25.059	1	25.059	2.013	.194
	Within Groups		99.609	8	12.451		
	Total		124.668	9			
MAZE3 * PrePost	Between Groups	(Combined)	13.110	1	13.110	.420	.535
	Within Groups		249.695	8	31.212		
	Total		262.805	9			
MAZE4 * PrePost	Between Groups	(Combined)	47.524	1	47.524	.552	.479
	Within Groups		688.997	8	86.125		
	Total		736.521	9			
MAZE5 * PrePost	Between Groups	(Combined)	78.288	1	78.288	.099	.761
	Within Groups		6340.991	8	792.624		
	Total		6419.279	9			
C * PrePost	Between Groups	(Combined)	3.600	1	3.600	.206	.662
	Within Groups		140.000	8	17.500		
	Total		143.600	9			
F * PrePost	Between Groups	(Combined)	.100	1	.100	.006	.938
	Within Groups		124.000	8	15.500		
	Total		124.100	9			
L * PrePost	Between Groups	(Combined)	44.100	1	44.100	1.747	.223
	Within Groups		202.000	8	25.250		
	Total		246.100	9			
CFL * PrePost	Between Groups	(Combined)	67.600	1	67.600	.600	.461
	Within Groups		902.000	8	112.750		
	Total		969.600	9			
Animal * PrePost	Between Groups	(Combined)	10.000	1	10.000	1.117	.321
	Within Groups		71.600	8	8.950		
	Total		81.600	9			
TRAIL A * PrePost	Between Groups	(Combined)	.357	1	.357	.093	.768
	Within Groups		30.775	8	3.847		
	Total		31.132	9			
TRAIL B * PrePost	Between Groups	(Combined)	36.902	1	36.902	.392	.549
	Within Groups		753.981	8	94.248		
	Total		790.884	9			
20 min DELAYED RAY * PrePost	Between Groups	(Combined)	27.225	1	27.225	1.056	.334
	Within Groups		206.300	8	25.788		
	Total		233.525	9			

Finally, it was presumed more effective to look at the five concussed player's mean scores to get a clearer understanding of how their neuropsychological functioning was effected within 36 hours post concussion (Refer to table 18 below). Although poorer performance did not appear to reach significance level ($p=<0.05$) via the use of error bars or ANOVA analysis, during analysis of the concussed players' mean scores there did indeed appear to be a general decline in their neuropsychological functioning in all cognitive domains assessed in this research. These cognitive domains included: 1) planning, visuo spatial and constructional ability; 2) attention and concentration; 3) memory; 4) verbal fluency and 5) speed of processing skills.

The following variables indicated a poorer cognitive performance in post-concussive injury assessment scores compared to baseline testing, as highlighted by the yellow shaded areas in table 18 below: Rey Complex Figure copy, Stroop Dots, Stroop Dots Errors, Stroop Words Error, Stroop Colour, Stroop Colour Error, Rey Complex Figure 2 min recall, RAVLT total, RAVLT recognition, RAVLT 20 minute delay recall, RAVLT immediate, RAVLT V1 2 minute delay recall, SDMT written, SDMT oral, Digits forwards, Digits backwards, COWAT FAS / CFL, COWAT Animal, Rey Complex Figure 20 min recall.

From analysing the data below, and using the grouping system employed by Master et al. (1999) we can conclude that all areas of neuropsychological functioning appeared to have been negatively affected by concussion. However, three areas of neuropsychological functioning did not appear to be completely affected by concussion, as no indication of decreased performance was present between baseline and post-concussion testing in a few of the sub-tests measuring these domains. The three cognitive domains relate to 1) memory (RAVLT B1 distractor list and RAVLT learn), 2) speed of processing, and 3) attention and concentration (Trail Making A & B, Stroop Words). There are 2 domains

that did indeed prove to be most sensitive to the effects of concussion. These include 1) planning, visuo spatial and constructional ability, and 2) verbal fluency.

Table 18: Means for Baseline and Post Concussion Scores of the 5 Concussed Players

	Pre Concussion Mean scores			Post Concussion Mean scores			
Neuropsychological Test	Mean	N	Std. Deviation	Mean	N	Std. Deviation	Mean
RCF Copy	32.60	5	1.673	30.80	5	2.564	31.70
STROOP Dots	13.9720	5	3.76459	14.5420	5	3.85762	14.2570
STROOP Dots Error	.00	5	.000	.40	5	.894	.20
STROOP Words	16.4680	5	4.99588	15.1360	5	3.19345	15.8020
STROOP Words Error	.00	5	.000	.20	5	.447	.10
STROOP Colour	21.1580	5	4.89737	27.0620	5	5.78678	24.1100
STROOP Colour Error	.60	5	1.342	2.00	5	2.449	1.30
RCF 2 min recall	26.000	5	4.6368	21.100	5	7.8294	23.550
RAVLT Total	50.80	5	3.271	49.80	5	4.147	50.30
RAVLT Learn	6.200	5	2.1679	6.600	5	1.1402	6.400
RAVLT Recognition	14.20	5	1.304	13.00	5	1.225	13.60
RAVLT 20 min delay recall	11.600	5	.5477	9.000	5	1.4142	10.300
RAVLT Immediate	7.00	5	2.121	6.00	5	1.225	6.50
RAVLT V1 2 min delay recall	11.40	5	1.817	11.00	5	1.732	11.20
RAVLT B1 distractor list	6.200	5	2.1679	7.000	5	2.3452	6.600
SDMT Written	58.600	5	5.6391	56.400	5	8.1117	57.500
SDMT Oral	61.20	5	4.494	60.00	5	3.536	60.60
Digits forwards	6.400	5	.8944	6.000	5	.7071	6.200
Digits backwards	5.60	5	1.140	4.60	5	.894	5.10
COWAT FAS/CFL	39.80	5	11.798	34.60	5	9.290	37.20
COWAT Animals	19.80	5	1.643	17.80	5	3.899	18.80
Trail Making A	22.6840	5	2.15571	22.3060	5	1.74549	22.4950
Trail Making B	51.9540	5	7.74037	48.1120	5	11.33940	50.0330
RCF 20 min delay recall	24.500	5	3.1225	21.200	5	6.4672	22.850

Yellow shaded areas: Variables indicating decreased neuropsychological performance following post concussive injury

HYPOTHESIS 3

To assess the relationship between the individually-rated Post Concussion Symptom Scale scores (PCSS) and post-season neuropsychological scores. It is hypothesised that the more severe the post-concussion symptoms, the more the post-season test results will be negatively affected.

In order to find out if high scores on the post concussion symptom scale correlate with low post-season neuropsychological scores, we make use of the Spearman's rank correlation coefficient computed by SPSS version 15 (Refer to table 19 below). Due to the vast scale of the correlation table, only the significant values have been selected here for discussion. Refer to appendix G for the full correlation table.

All 21 post-concussion symptom scale scores for the control group were presumed zero due to these subjects non-involvement in contact sport over the testing interval. Thus, no correlation table needed to be calculated for the control group.

There appears to be numerous significantly correlated relationships between the post-concussion symptom scale scores and the post-season neuropsychological scores of the rugby-playing group. This indicates that there does indeed appear to be a relationship between poor post-season neuropsychological performance and high post-concussion symptom scale scores on certain variables. We can therefore assume that the severity of neurological impact a player is exposed to during a game or over a season can be related to and predictive of further cognitive performance.

Although significant ($p<.05$), the strength of the correlation coefficient is weak to moderate. A possible reason for the low correlation coefficient could be due to a low

number of players who experienced a concussion during the season (n=5), as well as the player's underestimation or downplaying of their post concussive symptoms in fear of being re-tested or excluded from further games. Refer to Table 19 of correlations below. All significant (p) values are highlighted in green (p=<0.05), negative correlations are highlighted in pink, and positive correlations are highlighted in blue. All anomalies have been highlighted in red.

Most of the correlations for the rugby group's post-season test scores are in the predicted direction of having a negative correlation, but some may appear anomalous via the presents of a positive correlation. However, this is not the case, and can be explained by the fact that some of the neuropsychological tests look at 'performance speed' and 'error rate' as an indicator of success or higher functioning. The following variables are timed tests and thus a positive correlation will support the hypothesis above that high post-concussion test scores are related to higher/longer timed scores i.e. poor neuropsychological test performance: STROOP colour, dots and words; and TRAILS A and B. The Stroop Errors for dots, words and colour should also show a positive correlation in order to support the hypothesis above, as high scores on this test is indicative of poor performance due to the player making more errors on that particular task.

Summary of Table 19's Correlation Table:

Below is a list of variables that that showed significant p values (p=<0.05) and have been *negatively correlated* with high post concussive symptom scale scores thus supporting the above hypothesis. These variables show that the following areas of cognitive performance appear sensitive to the effects of post-concussion symptoms, and thus sensitive to detecting the possible presents of MHI in rugby players:

Neuropsychological Test	**Cognitive Domain Affected**	**Post Concussion Symptom Spearman's Rank correlation co-efficient and significant value ($p <= 0.05$)**
RCF Copy	Planning, visuo spatial and constructional ability	Increased nervousness (r= -0.46; p= 0.00) Tingling (r= -0.33; p= 0.05) Visual disturbances (r= -0.37; p= 0.03)
RAVLT 20 min delay	Recall memory	Increased nervousness (r= -0.37; p= 0.03)
RAVLT Immediate	Immediate memory	Tingling (r= - 0.34; p= 0.04)
RAVLT B1 distractor list	Immediate memory	Increased sadness (r= -0.33; p= 0.05)
SDMT written	Speed of processing, attention and concentration	Light sensitivity (r= -0.34; p= 0.05) Increased irritability (r= -0.38; p= 0.03) Increased nervousness (r= -0.34; p= 0.05) Feeling emotional (r= -0.36; p= 0.03)
SDMT oral	Speed of processing, attention and concentration	Noise sensitivity (r= -0.37; p= 0.03) Feeling emotional (r=-0.43; p= 0.01) Difficulty concentrating (r= -0.38; p= 0.03)
Digits forwards	Immediate memory	Visual disturbances (r= -0.38; p= 0.03)
Digits backwards	Immediate memory	Increased drowsiness (r= -0.36; p= 0.03) Noise sensitivity (r= -0.39; p= 0.02) Increased nervousness (r= -0.49; p= 0.00) Fogginess (r= -0.37; p= 0.03) Difficulty concentrating (r= -0.40; p= 0.02) Difficulty remembering (r= -0.47; p= 0.00) Light sensitivity (r= -0.45; p= 0.01)

Below is a list of variables that showed significant p values (p=<0.05) and have been ***positively correlated*** with high post concussive symptom scale scores. Due to these three variables falling under the 'timed performance' and 'error rate' category, the presence of a positive correlation supports the above hypothesis. These variables show that the following areas of cognitive performance appear sensitive to the effects of post-concussion symptoms and thus are presumed sensitive to detecting the presents of MHI in rugby players:

Neuropsychological Test	**Cognitive Domain Affected**	**Post Concussion Symptom**
Stroop Words Error	Attention and concentration	Increased nervousness (r= 0.30; p= 0.02)
Stroop Colour Error	Attention and concentration	Balance problems (r= 0.54; p= 0.00) More sleep (r= 0.48; p= 0.00) Drowsiness (r= 0.51; p= 0.00) Irritability (r= 0.36; p= 0.04) More emotional (r= 0.44; p= 0.01) Slowed thought (r= 0.37; p= 0.03) Difficulty concentrating (r= 0.36; p= 0.03) Difficulty remembering (r= 0.42; p= 0.01)
Trail Making B	Attention and concentration and speed of information processing	Noise sensitivity (r= 0.35; p= 0.04) Increased irritability (r= 0.44; p= 0.01)

The following two tests appear to be ***anomalies***, as they presented with significant positive correlations, when were expected to present with negative correlations to support the above hypothesis. We are unable to say whether these two variables are the result of a neuropsychological deficit, or occurred due to chance. If you compare the results of this hypothesis with the ANOVA computed for hypothesis 1, it is evident that RAVLT B1

distractor list also appeared as an anomaly and thus this variable should be interpreted with caution. These anomalies could be further interpreted by looking at the type of post-concussion symptom experienced: feelings of drowsiness and feeling mentally 'slowed'. Due to the rugby participants experiencing these particular symptoms it is possible that they felt the additional need to increase their level of motivation and concentration to complete these two tasks, and thus ended performing better than expected.

Neuropsychological Test	**Cognitive Domain affected**	**Post Concussion Symptom**
RAVLT B1 distractor list	Immediate memory	More sleep (r= 0.37; p= 0.03) Slowed thinking (r= 0.39; p= 0.02) Mental fogginess (r= 0.34; p= 0.04)
RAVLT 2 minute delay recall	Recall memory	Feeling fatigued (r= 0.37; p= 0.03)

The results show that no particular post-concussion symptom was most frequently experienced by the rugby group. High scores on the PCSS appeared to have affected all cognitive domains assessed, except for verbal fluency. The cognitive domain 'attention and concentration' appeared most sensitive to high scores on PCSS.

Table 19: Correlation table - Neuropsychological tests and post concussion symptoms

		Head-ache	Naus-ea	Vomit-ing	Bal-ance	Dizzy-ness	Fati-gue	diff fall asleep	more sleep	Drow si-ness	light sensi tivity	noise
Stroop Color Error	Correlation Coefficient	0.32	0.26	0.42	0.54	0.23	0.31	0.23	0.48	0.51	0.32	0.32
	Sig. (2-tailed)	0.06	0.13	0.01	0.00	0.18	0.07	0.18	0.00	0.00	0.06	0.06
RAVLT B1 Distractor	Correlation Coefficient	0.31	0.09	0.22	0.30	0.23	0.24	0.05	0.37	0.12	0.25	0.17
	Sig. (2-tailed)	0.07	0.61	0.20	0.09	0.18	0.17	0.76	0.03	0.50	0.14	0.32
RAVLT 2 min delay recall	Correlation Coefficient	0.10	-0.01	-0.03	-0.05	-0.15	0.37	-0.12	-0.08	0.06	-0.20	-0.11

	Sig. (2-tailed)	0.59	0.97	0.88	0.78	0.39	0.03	0.49	0.65	0.74	0.25	0.52
SDMT Written	Correlation Coefficient	0.05	-0.08	-0.05	-0.08	-0.08	0.16	-0.13	-0.25	-0.21	-0.34	-0.31
	Sig. (2-tailed)	0.76	0.65	0.76	0.64	0.65	0.36	0.44	0.15	0.23	0.05	0.07
SDMT Oral	Correlation Coefficient	-0.06	-0.21	-0.03	-0.19	-0.29	0.06	-0.16	-0.15	-0.31	-0.30	-0.37
	Sig. (2-tailed)	0.74	0.22	0.88	0.28	0.09	0.73	0.37	0.39	0.07	0.08	0.03
Digits Backwards	Correlation Coefficient	-0.17	-0.15	-0.14	-0.25	-0.13	0.06	-0.25	-0.11	-0.36	-0.45	-0.39
	Sig. (2-tailed)	0.34	0.40	0.43	0.15	0.45	0.75	0.15	0.53	0.03	0.01	0.02
Trail B	Correlation Coefficient	-0.12	-0.01	0.19	0.25	-0.08	-0.04	0.06	0.26	0.29	0.26	0.35
	Sig. (2-tailed)	0.48	0.96	0.29	0.16	0.65	0.83	0.74	0.14	0.09	0.13	0.04

Significance level p <0.05 – shaded areas

Shaded areas: Significant p value Positive correlation Negative Correlation Anomolie

		Irritab-ility	Sad-ness	Increa-sed nerves	more emo-tional	tingle	slow	Fog-gy	Diffic ulty conc entra te	Diffic ulty reme mber	Visual disturb ance
RCF Copy	Correlation Coefficient	-0.32	-0.22	-0.46	-0.20	-0.33	-0.21	-0.04	-0.29	-0.27	-0.37
	Sig. (2-tailed)	0.06	0.20	0.00	0.25	0.05	0.23	0.82	0.09	0.12	0.03
Stroop Words Error	Correlation Coefficient	0.15	-0.05	0.39	0.15	0.04	0.13	0.21	0.32	0.20	-0.03
	Sig. (2-tailed)	0.40	0.77	0.02	0.40	0.84	0.45	0.22	0.07	0.26	0.85
Stroop Colour Error	Correlation Coefficient	0.36	0.09	0.28	0.44	0.19	0.37	0.16	0.36	0.42	0.17
	Sig. (2-tailed)	0.04	0.59	0.10	0.01	0.27	0.03	0.34	0.03	0.01	0.34
RAVLT 20 min delay recall	Correlation Coefficient	-0.09	-0.18	-0.37	-0.25	-0.02	-0.08	-0.03	-0.15	-0.29	-0.10
	Sig. (2-tailed)	0.62	0.30	0.03	0.14	0.93	0.66	0.86	0.38	0.09	0.57
RAVLT Immediate	Correlation Coefficient	0.05	-0.14	-0.10	-0.05	-0.34	0.19	-0.02	0.03	-0.22	-0.30
	Sig. (2-tailed)	0.79	0.41	0.57	0.76	0.04	0.29	0.89	0.88	0.20	0.08

RAVLT B1 Distractor	Correlation Coefficient	0.23	-0.39	-0.06	0.17	-0.12	0.39	0.39	0.22	-0.10	-0.31
	Sig. (2-tailed)	0.19	0.05	0.72	0.32	0.50	0.02	0.04	0.21	0.57	0.07
SDMT Written	Correlation Coefficient	-0.38	-0.11	-0.34	-0.36	-0.02	-0.16	-0.24	-0.30	-0.12	-0.16
	Sig. (2-tailed)	0.03	0.53	0.05	0.03	0.92	0.35	0.16	0.09	0.49	0.37
SDMT Oral	Correlation Coefficient	-0.32	-0.03	-0.31	-0.43	-0.10	-0.15	-0.30	-0.38	-0.28	-0.21
	Sig. (2-tailed)	0.06	0.87	0.07	0.01	0.57	0.39	0.08	0.03	0.10	0.22
Digits Forwards	Correlation Coefficient	-0.07	-0.27	-0.24	-0.20	-0.05	0.18	0.00	-0.13	-0.27	-0.38
	Sig. (2-tailed)	0.67	0.12	0.17	0.26	0.79	0.31	1.00	0.46	0.11	0.03
Digits Backwards	Correlation Coefficient	-0.26	-0.20	-0.49	-0.33	-0.09	-0.22	-0.37	-0.40	-0.47	-0.29
	Sig. (2-tailed)	0.13	0.25	0.00	0.06	0.60	0.20	0.03	0.02	0.00	0.09
Trail B	Correlation Coefficient	0.44	0.13	0.12	0.25	-0.15	0.15	0.21	0.30	0.05	0.22
	Sig. (2-tailed)	0.01	0.45	0.51	0.15	0.38	0.40	0.22	0.09	0.77	0.20

Significance level p <0.05
Shaded areas: Significant p value Positive correlation Negative Correlation Anomolie

HYPOTHESIS 4

To assess whether players reporting a history of three or more previous concussions have lower scores at baseline compared to the control group. It is hypothesised that players with a history of previous concussion will have lower baseline scores as a result of the possible cumulative effects of concussion.

In order to answer this hypothesis a multivariate analysis of variance was computed by SPSS Version 15, whereby a process of grouping variables took place. There were multiple dependent variables (each individual neuropsychological test) and one independent grouping variable (players whom have had a history of three or more

concussions). 11 of the total 35 rugby players were selected and compared against all 35 control subjects who had no prior history of head injury (refer to table 20).

Table 20: Total concussed players (3 or more concussions) and Total control group

		Value Label	N
CONG	.00		24
	1.00	Player Concussed >=3	11
	2.00	Control No Concussion	35

Looking at the multivariate analysis of variance in table 21 below it is evident that there were surprisingly no areas of significance identified between the players' who did report a history of 3 or more previous concussions and the control groups' neuropsychological test scores. On closer analysis it appeared that there were a few variables that did come close to reaching significance level *(p=<0.05)*. They were the Rey Complex Figure 2 minute recall, F==2.28; p=0.64; RAVLT B1 distractor list, F=3.077; p=0.053 and FAS Animal F=2.788; p=0.69 (refer to the shaded values in table 21). These findings appear to follow a similar pattern to the findings in the other hypotheses suggesting that areas of 1) planning, visuo spatial and constructional ability; 2) immediate and recall memory; and 3) verbal fluency are shown to be sensitive to the effects of concussion.

Table 21: Multivariate Analysis of Variance of players with 3 or more concussions compared to the control group with 0 concussions: Tests of Between-Subjects Effects

Source	Dependent Variable	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	RCF Copy	4.790(a)	2	2.395	.382	.684
	STROOP Dots	15.137(b)	2	7.568	1.312	.276
	STROOP Dots Error	.430(c)	2	.215	.794	.456
	STROOP Words	15.373(d)	2	7.687	.409	.666
	STROOP Words Error	.179(e)	2	.089	.483	.619
	STROOP Colour	114.513(f)	2	57.257	1.756	.181
	STROOP Colour Error	1.520(e)	2	.760	.482	.620
	RCF 2 min recall	182.515(g)	2	91.258	2.864	.064
	RAVLT Total	2.070(h)	2	1.035	.019	.982
	RAVLT Learn	3.464(d)	2	1.732	.411	.665
	RAVLT Recognition	5.160(i)	2	2.580	1.028	.363
	RAVLT 20 min delay recall	5.725(j)	2	2.863	.429	.653
	RAVLT Immediate	.818(k)	2	.409	.154	.857
	RAVLT V1 2 min delay recall	26.172(l)	2	13.086	3.077	.053
	RAVLT 2 B1 distractor list	4.800(a)	2	2.400	.388	.680
	SDMT Written	113.749(m)	2	56.875	1.096	.340
	SDMT Oral	101.677(n)	2	50.838	.941	.395
	Digits forwards	3.600(o)	2	1.800	1.831	.168
	Digits backwards	.057(h)	2	.029	.022	.978
	COWAT FAS	94.328(p)	2	47.164	.369	.693
	COWAT Animals	61.362(q)	2	30.681	2.788	.069
	Trail Making A	114.890(r)	2	57.445	.949	.392
	Trail Making B	71.075(s)	2	35.538	.077	.926
	RCF 20 min delay recall	111.994(t)	2	55.997	2.096	.131

Significance level $p < 0.05$
Shaded areas represent values that are approaching significance level

HYPOTHESIS 5

To assess whether there are any differences between the forward and backline players' neuropsychological performance at both or either of the measurement intervals. It is predicted that as the 'forwards' participate in more scrums, mauls, and tackles, and are exposed to higher impact, they are likely to be more prone to sustaining mild head injuries, which will affect neuropsychological performance in sensitive domains of performance.

In order to answer this question we make use of the independent sample t-test to test if there is a difference between the frontline and back players with respect to pre- and post-season neuropsychological test scores. The reason for analysing the pre-season scores was to assess if there were any noticeable differences present between the positions of play before testing began, which would be indicative of the possible cumulative effects of concussion within the forwards group. However, no noticeable differences were found on the pre-season testing, except on RAVLT Recognition. In isolation, this result may be attributed to chance as much as due to neurological dysfunction. There is no particular reason why this memory sub-test would show up significant when none of the others did, and is therefore regarded as a possible anomaly (refer to table 22 below).

Neither was any significant differences found in the cognitive performance between the backline and the forward players on any of the neuropsychological measures during the post-season testing interval. Thus, the forward and backline players were considered 'similar' in terms of neuropsychological functioning during both testing intervals.

We are thus unable to hypothesize that the position of play in this particular rugby sample had an effect on neuropsychological functioning. It is probable that this particular group of forwards were not exposed to excessive amounts of neurological impact, or regular blows to their heads through the season as was initially predicted. We are unable

to conclude that the forward players had been exposed to more scrums, mauls or tackles compared to the backs over the season which would have had the potential of lowering neuropsychological performance as a result of bruising, shearing or tearing of delicate neurons.

Pre-season Analysis for 'Position of Play': refer to table 22 below

H_0: there is no difference in the cognitive performance between the backs and the forwards (pre-season scores)

H_1: there is a difference in the cognitive performance between the backs and the forwards (pre-season scores)

If p is less than 0.05 reject Ho in favour of H_1,

If the p values are greater than 0.05 accept Ho, and reject H_1

Table 22: Independent Samples Test for the 'Position of Play' during the pre-season testing interval

		Levene's Test for Equality of Variances				
		F	Sig.	t	df	Sig. (2-tailed)
REY COPY	Equal variances assumed	.937	.340	1.098	33	.280
	Equal variances not assumed			1.176	32.834	.248
STROOP DOTS	Equal variances assumed	3.893	.057	-.844	33	.405
	Equal variances not assumed			-.929	32.791	.360
DOTS ERROR	Equal variances assumed	.708	.406	.422	33	.676
	Equal variances not assumed			.406	24.307	.688
STROOP WORDS	Equal variances assumed	1.089	.304	-.860	33	.396
	Equal variances not assumed			-.947	32.747	.350
WORDS ERRORS	Equal variances assumed	9.012	.005	1.468	33	.152
	Equal variances not assumed			1.347	20.129	.193
STROOP COLOUR	Equal variances assumed	1.302	.262	.136	33	.892
	Equal variances not assumed			.124	19.573	.902
COLOUR ERROR	Equal variances assumed	.163	.689	.196	33	.845
	Equal variances not			.188	23.767	.852

	assumed					
REY 2 MIN RECALL	Equal variances assumed	.003	.957	-.020	33	.984
	Equal variances not assumed			-.020	28.631	.984
RAVLT TOTAL	Equal variances assumed	.435	.514	-.831	33	.412
	Equal variances not assumed			-.801	24.523	.431
RAVLT LEARN	Equal variances assumed	.584	.450	-.993	33	.328
	Equal variances not assumed			-.994	28.097	.329
RAVLT RECOG	Equal variances assumed	.184	.670	-2.461	33	.019
	Equal variances not assumed			-2.477	28.655	.019
RAVLT 20 MIN DELAY	Equal variances assumed	.021	.884	-1.285	33	.208
	Equal variances not assumed			-1.290	28.345	.208
RAVLT IMMEDIATE	Equal variances assumed	.418	.522	.050	33	.960
	Equal variances not assumed			.049	25.286	.962
RAVLT 2 MIN DELAY	Equal variances assumed	1.693	.202	-1.510	33	.141
	Equal variances not assumed			-1.429	22.774	.167
RAVLT DISTRACTOR	Equal variances assumed	.056	.815	.000	33	1.000
	Equal variances not assumed			.000	29.660	1.000
SDMT WRITTEN	Equal variances assumed	.075	.786	-.475	33	.638
	Equal variances not assumed			-.456	24.021	.652
SDMT ORAL	Equal variances assumed	.031	.862	-.545	33	.590
	Equal variances not assumed			-.528	25.070	.602
DIGIT FORWARD	Equal variances assumed	.026	.872	-1.188	33	.243
	Equal variances not assumed			-1.184	27.688	.246
DIGIT BACKWARD	Equal variances assumed	.272	.605	.647	33	.522
	Equal variances not assumed			.655	29.182	.517
COWAT F	Equal variances assumed	.000	.995	1.121	33	.271
	Equal variances not assumed			1.102	26.438	.280
COWAT A	Equal variances assumed	1.618	.212	-.189	33	.851
	Equal variances not assumed			-.202	32.684	.841
COWAT S	Equal variances assumed	.114	.738	-.175	33	.862
	Equal variances not assumed			-.176	28.738	.861
COWAT FAS	Equal variances assumed	2.352	.135	.104	33	.917
	Equal variances not assumed			.113	32.988	.911
COWAT ANIMAL	Equal variances assumed	.032	.859	-.701	33	.488
	Equal variances not assumed			-.691	26.542	.496
TRAIL A	Equal variances assumed	1.085	.305	-.038	33	.970
	Equal variances not assumed			-.037	25.757	.971

TRAIL B	Equal variances assumed	1.672	.205	.486	33	.630
	Equal variances not assumed			.463	23.367	.647
REY 20 MIN RECALL	Equal variances assumed	.799	.378	-.869	33	.391
	Equal variances not assumed			-.845	25.309	.406

Significance level p <0.05 – shaded areas

At the 5% significance level, we will accept H_0 for the un-shaded areas above and reject H_1 for only one of the variables (RAVLT Recognition). It seems that there is no difference in the cognitive performance between the backline and the forward players (pre-scores), except for RAVLT Recognition (F=0.184; p=0.019). We are therefore able to conclude that the forward group is not likely to be suffering from any cumulative effects of concussion.

Post-season Analysis for 'Position of Play': refer to table 23 below

H_0: there is no difference in the cognitive performance between the backs and the forwards (post-season scores)

H_1: there is difference in the cognitive performance between the backs and the forwards (post-season scores)

If p is less than 0.05 reject Ho in favour of H_1,

If the p values are greater than 0.05 accept Ho, and reject H_1

Table 23: Independent Samples Test for the 'Position of Play' during the post-season testing interval

		Levene's Test for Equality of Variances				
		F	Sig.	t	df	Sig. (2-tailed)
REY COPY	Equal variances assumed	3.198	.083	1.780	33	.084
	Equal variances not assumed			1.956	32.813	.059
STROOP DOTS	Equal variances assumed	2.140	.153	.940	33	.354

	Equal variances not assumed			1.035	32.770	.308
DOTS ERRORS	Equal variances assumed	6.943	.013	-1.179	33	.247
	Equal variances not assumed			-1.451	20.000	.162
STROOP WORDS	Equal variances assumed	.029	.866	1.197	33	.240
	Equal variances not assumed			1.211	29.115	.236
WORDS ERRORS	Equal variances assumed	.260	.614	.144	33	.887
	Equal variances not assumed			.134	21.286	.895
STROOP COLOUR	Equal variances assumed	1.203	.281	-.319	33	.751
	Equal variances not assumed			-.348	32.970	.730
COLOUR ERROR	Equal variances assumed	.685	.414	.087	33	.931
	Equal variances not assumed			.082	21.901	.936
REY 2 MIN RECALL	Equal variances assumed	.419	.522	1.053	33	.300
	Equal variances not assumed			1.113	32.354	.274
RAVLT TOTAL	Equal variances assumed	2.029	.164	-.370	33	.714
	Equal variances not assumed			-.401	32.998	.691
RAVLT LEARN	Equal variances assumed	2.554	.120	1.185	33	.245
	Equal variances not assumed			1.289	32.992	.206
RAVLT RECOG	Equal variances assumed	.001	.971	-.783	33	.439
	Equal variances not assumed			-.755	24.511	.458
RAVLT DELAY	Equal variances assumed	.089	.767	.626	33	.535
	Equal variances not assumed			.622	27.370	.539
RAVLT IMMEDIATE	Equal variances assumed	4.130	.050	-.675	33	.504
	Equal variances not assumed			-.746	32.661	.461
RAVLT DISTRACTOR	Equal variances assumed	.296	.590	.042	33	.967
	Equal variances not assumed			.040	23.691	.968
RAVLT 2 MIN RECALL	Equal variances assumed	.783	.383	.253	33	.802
	Equal variances not assumed			.246	25.412	.808
SDMT WRITTEN	Equal variances assumed	6.007	.020	-.775	33	.444
	Equal variances not assumed			-.835	32.940	.410
SDMT ORAL	Equal variances assumed	2.362	.134	-1.075	33	.290
	Equal variances not assumed			-1.147	32.717	.260
DIGIT FORWARD	Equal variances assumed	.582	.451	-.189	33	.851
	Equal variances not assumed			-.200	32.242	.843
DIGIT BACKWARD	Equal variances assumed	.064	.802	1.735	33	.092
	Equal variances not assumed			1.698	25.940	.101
COWAT C	Equal variances assumed	.105	.748	-.188	33	.852
	Equal variances not			-.192	30.184	.849

	assumed					
COWAT F	Equal variances assumed	.190	.666	1.462	33	.153
	Equal variances not assumed			1.416	24.934	.169
COWAT L	Equal variances assumed	3.134	.086	1.781	33	.084
	Equal variances not assumed			1.904	32.781	.066
COWAT CFL	Equal variances assumed	.141	.710	1.158	33	.255
	Equal variances not assumed			1.194	30.741	.242
COWAT ANIMAL	Equal variances assumed	.002	.966	.022	33	.983
	Equal variances not assumed			.022	29.309	.983
TRAIL A	Equal variances assumed	.263	.612	-.289	33	.774
	Equal variances not assumed			-.300	31.162	.766
TRAIL B	Equal variances assumed	.187	.669	.530	33	.600
	Equal variances not assumed			.533	28.565	.598
REY 20 MIN RECALL	Equal variances assumed	.001	.973	.541	33	.592
	Equal variances not assumed			.555	30.272	.583

Significance level p <0.05

At the 5% significance level, we will accept H_0 for the un-shaded areas above and reject H_1. We can conclude that there appears to be no differences in neuropsychological functioning between the forward and backline players during the post-season testing interval.

To summarize, the above statistics show that there are no profound neuropsychological differences between the forward and backline players at either of the testing intervals. This is suggestive that the forward players have not been exposed to more serious levels of neurological impact that could have negatively affected their cognitive performance.

DISCUSSION

The present study was carried out on 35 club rugby players and 35 control group subjects. Data was collected on these players, and adjustments were made for potential confounding factors such as education, alcohol consumption, age, diagnosed learning difficulties, history of previous concussion, total years of playing rugby etc. Proven reliable and valid neuropsychological tests were selected that were known to be sensitive to detecting subtle forms of mild head injury in both sporting and non-sporting subjects. The data was analysed, interpreted and regular adjustments for multiple statistical tests of comparison were used, such as the Bonferroni correction. Since the focus of this study was not on the clinical evaluation of the subjects, the discussion will focus on the raw score trends which have at times been represented graphically in this paper.

The data showed that a significant relationship was present between rugby players participating regularly in the sport over one full season and decreased neuropsychological test performance in a range of cognitive domains, including planning, visual spatial and constructional ability, attention and concentration, memory and verbal fluency. Verbal fluency appeared to be most sensitive to the post-season testing session, whereby 4 out of the 5 sub-tests showed a significant relationship. Numerous significant relationships were also found between certain PCSS scores and poor neuropsychological performance, which were considered indicative of the subtle effects of sub-concussive injuries or even possible MHI.

Surprisingly, following the assessment of players who were concussed during the season, the data did not show any reliable significant declines in cognitive performance compared to their baseline testing. When analysing the means scores of the concussed group, a trend did become evident suggesting there was indeed a decrease in these players

neuropsychological test scores amongst almost every measure and cognitive domain. No particular neuropsychological test appeared to be more sensitive to the effects of concussion.

The data did not show any trend towards a history of previous concussions and poorer neuropsychological performance suggesting that these players had recovered from the potential cumulative effects of concussion they could have experienced in the past, as a result of suffering three or more concussions. Nor were there any significant differences found in neuropsychological performance between the backline and forward players.

Many of the results in this present study are consistent with the findings in recent literature, although hypothesis 5 is not in line with the 'commonly accepted' literature. A possible reason for these inconsistencies could be supported by the comprehensive review of studies conducted by Rutherford et al., (2003). These researchers found that numerous psychological studies undertaken so far suffer from extensive methodological problems, and they suggest that at best, some of the research conducted and published should only be regarded as exploratory studies (Rutherford et al., 2003). For this reason the findings of hypothesis 5 needs to be considered as 'additions' to the present knowledge base, and not as 'different or contradictory' to previous research. In the context of the present study, additional reasons for these findings will be presented shortly in the discussion.

Discussion of the Separate Hypotheses

Hypothesis 1

The aim of the initial hypothesis was to assess whether there were any changes in neuropsychological functioning between the rugby and control group at both the pre and post-season testing interval, and to see how each of these groups' cognitive functioning improved, deteriorated or remained constant over the season i.e. between the pre and

post-season testing interval. It was hypothesised that the control group scores would remain constant, or improve due to benefiting from 'practice and learning effects', and it was presumed that the rugby group's scores would remain the same or even deteriorate over the season. If the rugby group did not appear to benefit from the practice and learning effects on the majority of the measures, it would suggest that some of the players were suffering from the effects of a sub-concussive or MHI, which was affecting their neuropsychological performance.

The general trend of the results in this section did support the above hypothesis, and were in an agreement with results found by numerous other researchers in this field.

The groups were assessed using error bars and the Custom Table model to see if any significant differences were present between the groups at pre and post-season. During pre-season testing, only two values were found to be statistically significant, namely the RCF 2 minute recall and the RAVLT B1 distractor list, suggesting that the two groups initially performed similarly with regards to all areas of cognitive functioning. It is important to note that this stage of testing was prior to the beginning of the season and the players had not yet been exposed to a vast number of "knocks". It is presumed that adequate time had lapsed since the previous season which would have allowed for recoveries to take place. Diffuse brain damage at this stage of testing would have been minimal for the players, excluding those players who were suffering from the longer lasting cumulative effects of concussion and hence began the season with already existing cognitive impairments. The researcher was thus able to ascribe any further differences in neuropsychological performance between the groups to the possible effects of MHI some of the player's would endure during the season ahead. The effects of mild head injury was expected to be shown in the difference between 'Pre' and 'Post' test scores over the 9-month period of playing rugby.

Due to the RCF 2 minute recall and the RAVLT B1 distractor list scores appearing in isolation, with none of the other variables assessing the same domain of cognitive functioning appearing significant, it is likely that these two variables appeared with significant values due to chance. As with the above two measures, numerous other scores indicated that the rugby group had performed marginally better than the control group on the majority of the measures. These results can be explained by the possibility that the rugby group were more determined and motivated than the control group at the onset of testing, which would have resulted in elevated scores. When comparing all the mean scores of pre-season testing session, it became evident that the rugby group performed better than the control group in all areas of cognitive functioning, again supporting the fact that the rugby players approached this initial testing session with a more serious and determined attitude. Due the rugby group being aware that this study aimed to identity possible neuropsychological deficits, specifically within their group, and considering the fact that many of them had suffered from concussive injuries in the past (60% of team), they were given reason to be more motivated than the control group and perform to the best of their ability.

During analysis of the post-season testing scores via the use of custom tables, it became evident that there were numerous significant differences present between the two groups, on a range of cognitive domains, namely planning, visuo spatial and constructional ability (RCF copy), attention and concentration (Stroop Dot errors, digits forwards), memory (digits forwards) and verbal fluency (COWAT CFL, Animals), whereby the rugby players performed more poorly compared to the control group. These results are suggestive that some type of neuropsychological change did take place over the season, and are likely to be the direct result of sub-concussive or MHI sustained by the rugby-playing group during the season.

The ANOVA Analysis highlighted another interesting aspect of the 'learning and practice effect'. On further review of the groups' mean scores it became evident that in only 7 out of the 27 sections did the players' seem to have benefited from practice effects, compared with the controls' who benefited from the effects of practice in 19 of the 27 sections. This is suggestive that the numerous rugby players were exposed to some from of sub-concussive or MHI over the season, resulting in neurological bruising or possible shearing of neurons, which prevented them from benefiting from expected practice effects. No specific area of cognitive functioning appeared to be most affected by these practice effects. The test scores of the players are all in the direction of a diminished level of performance and suggest there is slight impairment in the rugby players' cognitive functioning post-season. Mean scores analysis highlighted that the rugby group performed less well than the control group on the majority of the tasks and in a range of cognitive domains. This decrease in performance compared to the control group highlights the fact that some of the players were likely to have been exposed to some form of MHI, which has had a negative effect on their neuropsychological performance.

These results support the findings of numerous researchers in the field who have also found that MHI, either in the form of severe concussion or mere continual 'knocks' to the head for extended periods of time, have had a negative effect on learning and memory (Barth et al., 1983; Gronwall & Wrightson, 1981; Lovell et al., 1999), speed of processing and attention and concentration (Barth et al., 1989; Barth et al, 1983; Gronwall & Wrightson, 1981), planning, visuo spatial, constructional abilities and verbal fluency (Barth et al., 1983; Lovell et al., 1999). As the first aim hypothesised, it was expected that the players would perform slightly less effectively on the post-season testing session as the results have suggested. The reason for this prediction was that the players are assumed to 'bruise' their brains during a game due to the impact of the

scrums, mauls, and tackles. During bruising the axons are stretched and sheared resulting in decreased transmission between axon and dendrite, hence a lowered capacity for optimal cognitive functioning (Hanlon et al., 1999). Since the players at this level are exposed to continual 'knocks' to their head, face and neck for extended periods of time and from being involved in heavy forms of contact, it is likely for this continual impact to cause some type of acceleration and deceleration force of the brain to occur within the skull. This movement of the brain causes damage to delicate axons in the form of tearing and shearing of neurons, as explained by Edwards (1993) in the Acceleration-Deceleration model. If additional rotational forces of the brain occur, further damage to the deeper structures of the brain could result in more permanent and disabling characteristics (Hovda et al, as cited in Lovell et al, 2004). Due to the results presented in this study we can presume that some of the players were exposed to *diffuse axonal injury (DAI)* during the course of the season, which is known to have negative consequences on cognitive performance (Guskiewicz *et al.*, 2004).

Hypothesis 2

This hypothesis aimed to assess neurocognitive performance of players following a concussive injury.

Neuropsychological testing is generally considered a sensitive and thorough method of detecting and characterising cognitive and behavioural effects after concussion (Collins et al., 1999). Often extensive neuropsychological evaluation by a neuropsychologist or trained professional immediately after injury is not feasible due to the lack of available trained staff, limited resources, financial aid etc. Unfortunately, within this particular study the researcher was unable to assess the immediate side line effects of the concussive injury. Due to reasons of practicality, testing was conducted only a few days

following the concussive injury (+/- 36 hours post injury). There was no person/coach who had been professionally trained to administer any sideline assessment such as the Maddocks Questions, or able to assess the presence of LOC or PTA which potentially limited the researcher's knowledge about the severity and nature of the concussive injury. The researcher had to rely on 'self-reported' symptoms of the injury, which was coupled with its own set of complications such as under reporting of injuries, underestimation of post concussion symptoms etc.

Surprisingly the results in this study showed that no variable, except RAVLT 20 minute delayed recall, appeared significant ($p=<0.05$), indicating that there were no substantial declines in cognitive test performance post concussive injury. Due to the variable RAVLT 20 minute delayed recall occurring in isolation, we are unable to conclude whether this variable is indicative of a neuropsychological deficit or occurred due to chance. Within this study the five concussed players were grouped together, and the group means for pre and post injury were compared for each neuropsychological measure. Due to this grouping it is possible that subtle individual differences went unnoticed, resulting in only one variable presenting with a significant value. As highlighted in the literature review, each player presents with their own unique signs and symptoms of a concussive injury and with differing neuropsychological deficits depending on a wide range of factors. These factors include the nature and severity of the injury, age, education, presence of learning difficulties, ADHD, alcohol consumption, previous concussive injuries, and the nature and severity of the injury etc (Kirkendall et al., 2001). Thus it is advisable for future researchers to analyse and interpret neuropsychological results of concussed players individually, such as in the form of case studies.

The results could be further explained by looking at the 'time-period' that lapsed between the player's injury and the time of the post-concussion assessment (on average 48 hours post injury). From reviews of recent literature, a neuropsychological test battery can show concussion resolution anywhere between 48 hours to 3 months (Grindel et al., 2001). As previous research has indicated, 1) it is possible for the injured player to recover relatively quickly after the injury provided the injury was mild in nature and 2) in order to assess if neuropsychological deficits are present it is advised to assess the injured players as soon as possible following the injury.

In a study by McCrea et al. (2003), on 2385 high school and college football players, the results showed that athletes who experienced LOC or PTA following concussion displayed the most severe neurocognitive impairments, although declines in cognitive functioning were still present in concussive injuries without LOC and PTA. Their results showed that all groups returned to baseline levels of cognitive functioning within 48 hours post injury. The results of this present study are in agreement with McCrea et al., 2003 in that players without LOC or PTA did show subtle declines in cognitive performance and the group appeared to have recovered relatively quickly post injury. Within the present study no player reported experiencing PTA or LOC for more than one minute following their concussive injury. The researcher therefore concluded that all concussions suffered during the season were mild to moderate in nature. It is likely that due to the mild nature of these injuries, neither profound diffuse axonal damage, nor damage to the deeper structures of the brain took place. If this were the case, the results would have been more likely to show more severe impairments in neuropsychological functioning. Due to the players experiencing mild concussion it is likely that they recovered relatively quickly, thus supporting the above-mentioned explanation relating to recovery time and cognitive performance.

Another important explanation for the results to consider is that of small sample size n=5. It is generally not recommended to run statistical analyses on small sample sizes as this decreases the statistical power of the results.

Although post-concussion cognitive performance did not reach levels of significance on the majority of the variables ($p=<0.05$), analysis of mean scores showed a definite trend towards poorer cognitive performance following the concussive injury. There appeared to be a decrease in cognitive performance in 22 out of the 27 neuropsychological measures indicating poorer performance in all areas of cognitive functioning assessed in this research. Only five neuropsychological measures did not appear to be affected by the injury, namely RAVLT B1 distractor list, RAVLT learn, Trail Making A & B and Stroop Words. These neuropsychological measures relate to areas of memory, speed of processing and attention and concentration. These findings are likely to be explained by the fact that the players benefited from 'practice' or 'learning' effects on these particular measures. On RAVLT B1 the concussed players improved their scores from 6.20 to 7.00, RAVLT Learn improved from 6.20 to 6.60, Trail A improved from 26.68sec to 22.31sec, Trail B improved from 51.95sec to 48.11sec, and Stroop Error improved from 16.41sec – 15.14sec. Although alternative test measure forms were used whenever possible, interestingly enough, there were no alternative forms available for Trail Making A and B, nor the Stroop test indicating it is likely that the injured players improved their scores due to being familiar with these tests and knowing what to expect. The researcher is unable to conclude whether scores in the RAVLT B1 distractor list and RAVLT learn improved due to 'learning effects' or chance.

Overall, we can conclude from the analysis of the means scores that the results do indeed support the above hypothesis that concussive injures have a negative impact on cognitive performance.

Hypothesis 3

This hypothesis aimed to assess the relationship between the individually-rated Post Concussion Symptom Scale (PCSS) scores and post-season neuropsychological scores. It was hypothesised the more severe the post-concussion symptoms, the more the post-season test results would be affected, as high scores on this scale are indicative of possible concussive injury.

Research regarding the association of performance on neuropsychological testing with post-concussive symptoms has not yet been firmly established, and the results of various studies conducted in the past have often been contradictory. According to McCrory & Johnston (2002), recent findings of abnormalities in executive function, working memory and attention tasks, as seen by functional magnetic resonance imaging (MRI) scanning and neuropsychological testing, indicate that post-concussive symptoms are likely to be indicative of injury. Collins et al. (2003) concluded that high scores on the PCSS were related to low scores on reaction time and memory tasks. In another study conducted by Collins et al. (2006), they found that in particular the symptom of 'headaches' was associated with lowered choice and reaction times. This present study undertook a similar research strategy to examine whether high scores reported on the PCSS were related to poorer neuropsychological test scores. From the results we were able to conclude that the PCSS did appear to be a sensitive measure in detecting neuropsychological changes resulting from possible MHI or continual neurological impact over one full season of play.

This study showed numerous significant relationships existed between high PCSS scores and poor post-season neuropsychological performance. No particular neuropsychological measure appeared more sensitive to the effects of high PCSS scores. The following variables showed a significant relationship was present: RCF copy,

RAVLT 20 minute delayed recall, RAVLT immediate, RAVLT B1 distractor list, SDMT written and oral version, digits forwards, Stroop Words Error, Stroop Colour Error and Trail B. We can further hypothesise that the following areas of cognition are sensitive to high scores on the PCSS: planning, visuo spatial and constructional ability, attention and concentration, memory and speed of processing. Tasks of attention and concentration appeared to be most affected by high scores on the PCSS, which fall in line with the findings of McCrory & Johnston (2002). Surprisingly, no significant relationship was found between verbal fluency and high PCSS scores, despite this area of cognition proving to be sensitive to the players' participating in rugby over one full season. These findings are in contrast to isolated results presented by Barth et al. (1983) and Lovell et al. (1999), who found significant relationships present between low verbal fluency performance and high post concussion symptoms among their athletes. These, as mentioned are 'isolated reports' and not much further information is provided in the literature to support the fact that verbal fluency is indeed sensitive to post-concussion symptoms, and thus further research in this area is strongly recommended. Although a significant relationship was not found between verbal fluency and high PCSS, on closer analysis of raw mean scores, there did appear to be a trend between decreased verbal fluency performance and high PCS scores, suggesting the possibility of a relationship existing.

The results suggest there was a relatively equal distribution of affected neuropsychological scores between the 'physical, typical and cognitive features' of the PCSS. Interestingly, a stronger relationship was found between cognitive scores and the 'emotional features' of the PCSS. Emotional features included symptoms of increased nerves, irritability, feeling more emotional and increased levels of sadness. The area 'emotional symptoms' raises its own set of questions and complications relating to which

comes first – 'the chicken or the egg?' i.e. do the players perform poorly on neuropsychological testing due to prior existing psychological reasons (such as feeling irritable or despondent due to losing a competitive game), or are emotional symptoms induced by high levels of neurological impact exposed to during the game? Nonetheless, it is interesting to note that the 'emotional features' of the PCSS appears to be most sensitive in detecting poorer cognitive performance and the presence of a possible concussive injury.

Due to the players completing the PCSS after every game throughout the season, their awareness of the symptoms of concussion is likely to have improved, thus becoming more self-reflective about possible injury. This could have affected the manner in which they approached their post-season neuropsychological testing session. For example, the players who became aware that they suffered from regular post-concussive symptoms could have been more motivated and tried even harder in their end of season testing, in fear of their test results decreasing substantially in comparison to their fellow players. Via this regular self-reflection, the players could also have become more aware of the fact that they overexerted themselves in a game, thus affecting the manner in which they approached their next game with regards to speed, determination, and competitiveness. These are potential factors to consider when analysing post-season neuropsychological scores.

In the future, it may be advisable for all players to fill out a similar scale following each game, as a means of self-monitoring and self-regulating. This would serve as a good 'protective and preventative' measure for future, cumulative head injury. Players who are concerned with high levels of post-concussive symptoms could seek further medical attention and receive the appropriate care. This would prevent numerous players from ignoring their symptoms and carrying on with further game play, resulting in the player

being exposed to yet another knock, leading to possible full blown post-concussive syndrome or even worse, SIS.

However effective this strategy would be in theory, it could also pose numerous problems of practicality, such as finding the time and motivation to administer and complete the forms after every game, especially after the players wanting to celebrate a victorious win immediately after play. Other areas of concern relate to the fact that this is a highly 'subjective' measure, and each player may interpret his signs and symptoms differently. Due to the 'macho, underfeatable' image portrayed by numerous rugby players, they may choose to undervalue the severity of symptoms they are experiencing in fear of being frowned upon by coaches or fellow team mates, or worse, being excluded from further play until their symptoms cease. However, I feel this is still an important preventative measure to consider.

Hypothesis 4

The aim of this hypothesis was to assess whether players reporting a history of 3 or more previous concussions had lower scores at baseline compared to the control group, suggestive of the presence of cumulative effects of concussion.

Among many researchers it has been a common assumption in sports medicine that a ***prior history of concussion*** is predictive of a lowered threshold and worse outcome following subsequent concussive injury. However, not all studies report evidence for cumulative effects. From the results presented in this study, we are unable to support the above hypothesis that players who do have a prior history of concussion are suffering from the residual cumulative effects of MHI. These results fall in line with previous research conducted by Macciocchi et al. (2001). These researchers also found no neuropsychological deficits present among football players who had a history of two or

more concussions compared to those athletes with only one previous concussion, on tests such as the PAST, Trails A and B, or the SDMT (Macciocchi et al., 2001). Guskiewicz et al. (2002), also experienced similar findings and found no association between chronic cognitive impairment and a history of mild concussions among collegiate players. As mentioned in the literature review, every concussion presents differently with its own unique set of signs and symptoms in each individual (Guskiewicz et al., 2004). This unique presentation is partly due to the angle, rate and speed at which the player's brain is hit, affecting different areas of the brain responsible for varying neuropsychological processes. It is probable that the players in this particular study had been exposed to differing levels of neurological impact and injury resulting in the dissimilar presentation of cognitive deficits. Following similar research designs that have appeared effective in the past, as well as for reasons of practicality, individuals with a history of 3 or more concussions were analysed as a 'group', and not as individuals. When these individuals were analysed as a 'group' certain deficits may have been undetected and gone unidentified by the researcher. Thus, in the future it may prove more beneficial to analyse the players with a history of concussion individually in order to identify unique patterns of cognitive processing and the possible related neuropsychological deficits.

During the detailed history interview the majority of players reporting previous concussions experienced mild to moderate concussive injuries, and only three players mentioned experiencing LOC and PTA following concussion. From this information we can assume that on impact the acceleration/deceleration forces of the brain within the skull did not cause severe damage to the deeper structures of the brain causing serious and permanent cognitive damage as could occur in grade 3 concussions. The players appear to have fully recovered from their previous injuries and are not suffering any cumulated effects of MHI. If continual damage to the deeper structures of the brain were

to occur via experiencing repeated concussions before full recovery had taken place, further results will likely show more severe deficits in neuropsychological functioning, however this was not the case in the present study.

Another explanation for these findings could relate to the issue of 'time-periods' that had lapsed between each concussive injury. Over time it is possible for these cells to regenerate and repair themselves, provided no further injury takes place before complete recovery has occurred. Within this rugby sample it is likely that adequate time-periods lapsed between the concussive injuries and appropriate recovery times were provided before the player returned to the game. This would have allowed for full regeneration of these cells to take place, thus leaving no permanent neurological damage. Previous research suggests that on average cognitive functioning returns to normal within 5 to 7 days post injury (McCrea et al., 2003). From the individual history interviews, it appears that the majority of players were put out of play for numerous days following injury before being allowed to return-to-play. The teams' coaches also followed a step-wise process, similar to the step-wise process suggested by the CIS Group in Prague (2004). This would have eased the injured player back to fitness and allowed for adequate time-periods to lapse in order for full recovery to take place. Returning to play while the athlete is not clear of post-concussion symptoms may lead to irreversible cognitive deficits or even death, although prolonged cognitive deficits can be seen after a single insult (Grindel et al., 2001). For this reason, Second Impact Syndrome (SIS) is of major concern in the field of contact sport as players and coaches often do not allow adequate time-periods to pass following concussive injuries and the player returns back to the game prematurely.

Hypothesis 5

The aim of this hypothesis was to assess whether there were any differences between the forward and backline players' neuropsychological performance at both or either of the measurement intervals.

The reasoning behind this hypothesis related to the theory that forward players are generally exposed to higher levels of neurological impact, compared to backline players, due to being involved in more scrums, mauls and tackles during play (Edwards, 1993). This high level of impact often results in the bruising, shearing or tearing or neurons and can have a direct negative effect on neuropsychological performance (Guskiewicz et al., 2004). It was therefore hypothesised that the forward players would have lower post-season scores as a result of higher levels of impact, or even show possible cumulative effects of injury in their pre-season test scores.

However, the results found in this research were not in keeping with this hypothesis. During the pre-season testing interval only one variable was found to show a significant difference between the forward and backline players, namely RAVLT recognition. This significant value was presumed to be an anomaly due to occurring in isolation, and no other variable measuring a similar cognitive domain appeared significant. The pre-season results showed no differences between the forwards' and backlines' cognitive performance in any cognitive domain. I can therefore state that no evidence of cumulative effects of concussion was present in the forwards group despite the previously held belief that forwards are exposed to higher levels of neurological impact when compared to backline players. These results suggested that the majority of forwards had fully recovered from any neuropsychological deficits they potentially suffered in the past as a result of injury, prior to the pre-season testing interval.

Neither were any significant values found in the neuropsychological test performance between the forward and backline players on analysis of the post-season testing interval, again suggesting there were no neuropsychological differences present between these two groups of players. We are therefore unable to accept the above hypothesis.

These findings could be further explained by looking at uneven distribution of forward and backline players. In total, there were 14 backline players and 21 forward players. This uneven distribution of player position is likely to have skewed the data, thus not giving us a fair representation of how each group performed. In future studies it would be advisable to ensure there is a larger sample of subjects which would increase the statistical power of the results, as well as selecting equal numbers of different positions if the study wishes to analyse this particular hypothesis in detail.

LIMITATIONS OF THE PRESENT STUDY

Several limitations of this study warrant consideration. First, the issue of too few subjects raises concern. If more subjects had been employed, the statistical power of the results would have been increased resulting in stronger, more reliable findings, thus further aiding the acceptance or disagreement of the presented hypotheses. Secondly, most of the concussive injuries experienced by the players were self reported, which had the possibility of numerous concussions going underreported due to symptoms being unrecognised and underestimated, or in fear of being excluded from the game. All reported concussions were also of mild to moderate severity, and thus clear/severe impairments in neuropsychological performance were not evident. Thirdly, due to this study sample consisting of male adult athletes, it is unclear if this data can be applied to female contact sporting groups as well as younger, school going athletes. Lastly, in this study all players were considered homogenous and analysed within groups. Thus, the average 'group' results were not able to detect whether any subtle 'individual' differences were present among the injured players. Due to the fact that concussive injury presents differently between individuals, including differing signs, symptoms, and recovery times, it is advised to analyse the players with newly acquired injuries, or a history of concussive injuries independently - possibly in the form of individual case studies, with the purpose of identifying unique patterns of cognitive performance. This would help the researcher gain a clearer understanding of how neuropsychological performance is affected by MHI, and could give possible reasons as to why certain areas of cognition are more affected than others, depending on the type of injury as well as the individual themselves.

CONCLUSION

Surprisingly for a country where rugby receives so much media attention, research into rugby injuries in South Africa started only in the 1980's following the tragic death of the Western Province full back, Chris Burger, in a Currie Cup match against the Orange Free State. Many believe it is a gloomy indication that it took the death of a top player to prompt research into rugby injuries in South Africa. However, over the past decade, this area of research has become a popular area to investigate, and numerous interesting and well-grounded pieces of research have been produced and published in South Africa. Recently in South Africa, this area of neuropsychological research has spread beyond the scope of assessing club and university rugby players, and there appears to be a new shift towards researching school-boy rugby – an area that definitely requires attention, and would directly benefit all adolescents playing rugby in South Africa.

This paper demonstrates that concussion can present serious consequences for some athletes and warrants the attention it has received. This present study has given a clear description of the potential negative consequences of playing rugby, which were clearly evident when looking at the change in scores between pre and post-season testing and the general declines in almost all neuropsychological scores following a concussive injury. Although this study dealt mainly with 'normal' players, the results shown here are a cause for concern. What has become evident is that the player need not be exposed to severe concussion in order to experience some form of cognitive impairments. Even if these impairments are minimal, they are however still present and have the potential of accumulating, which could lead to disastrous permanent deficits.

The increased research and attention into concussion has demonstrated the enormous complexity of this field, which to date has produced numerous contrasting and conflicting

findings regarding the understanding, diagnosing, and treatment of concussion. Thus it is imperative that present and future researchers continue their work in this field in order to increase ones' understanding of a potentially devastating phenomenon within this complex field. While research has provided interesting and useful findings, it has raised numerous unanswered questions that require immediate further investigation. We need to enhance our understanding of the pathophysiologies of concussion and the mechanisms of injury so that we may develop treatment models that are evidence based, and prevention strategies to stop these appalling and often disabling injuries from occurring in the first place.

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APPENDICES

Appendix A

Consent Form

I ....., hereby give my voluntary consent to participate within this study researching the consequences of cumulative effects of 'knocks' in rugby on neuropsychological performance of rugby players.

I am fully aware of the purpose of this research, as well as the demands made upon me to participate within the research. I am willing to be honest and truthful when involved in neuropsychological testing.

I understand that all the information I give will be kept within in the strict confidence of the researchers' themselves. No where in the publication or presentation of this study will my name be mentioned nor my personal details elicited. This confidential information will only be used within and for the purpose of this study.

I am aware that I am free to withdraw from this study at any pointing time should I feel un-at-ease or uncomfortable.

I acknowledge that my participation within his research will not benefit myself directly. However, I am pleased to assist the researchers by being involved in this study, thereby helping to provide valuable information to further enhance the understandings of concussion and the effects of cumulative head injury within sporting injuries.

I am aware that I am free to contact both the researcher and the supervisor of this project, should I wish to ask further questions or clarify uncertainties.

Name: _____ Date:_____

Signature:_____

Witness: _____

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Appendix B

Confidential Demographics Form

1. Name: _____

2. Date of Birth: _____

3. Place of Birth: _____

4. Length of Education (number of years)

Schooling: _____

College or University: _____

5. Have you ever been diagnosed with a learning difficulty by a qualified professional? Yes / No

What was the diagnosis: _____

Professional's qualification: _____

6. Have you ever been concussed? Yes / No

How many times: _____

How long ago? _____

Symptoms / Severity of concussion: _____

7. Do you have any medical conditions? E.g. epilepsy, diabetes, hypertension? Yes / No

8. Do you currently take any medication? Yes / No

Name of medication? _____

9. What position do you play? _____

10. How many years have you been playing rugby? _____

Appendix C

Post Concussion Symptom Scale (PCSS)

NAME:_____ **DATE:**_____

SYMPTOM	0	1	2	3	4	5	6
Headache							
Nausea							
Vomiting							
Balance Problem							
Dizziness							
Fatigue							
Trouble falling asleep							
Sleeping more than usual							
Drowsiness							
Sensitivity to light							
Sensitivity to noise							
Irritability							
Sadness							
Nervousness							
Feeling more emotional							
Numbness of tingling							
Feeling slowed down							
Feeling mentally 'foggy'							
Difficulty concentrating							
Difficulty remembering							
Visual problems							

0 - NO SYMPTOMS

1- VERY MILD

2 - MILD

3 - MODERATE SYMPTOMS

4 - SIGNIFICANT

5 - VERY SIGNIFICANT

6 - SEVERE SYMPTOMS

Appendix D

Excel table for Players Group: Demographic details, test scores

No	AGE	EDUC	CONCUSSION	POSITION	YEARSPLAY	RAY	STROOPDOTS	STROOPDOTERRS	STROOPW
1	21	13	0	center	15	33	11	0	11.34
2	22	15	0	full back	9	32	10	0	11
3	20	14	0	prop	16	32	12	0	15
4	22	12	0	flank	13	34	13.05	0	14.85
5	22	13	5	center	10	30	12.23	0	14.19
8	21	16	15	hooker	16	30	16.21	1	12.48
9	23	15	5	prop	16	34	11.38	0	11.17
10	25	14	1	forward	10	34	11.96	0	14.51
11	25	14	1	flank	15	30.5	12	0	12
12	23	16	0	hooker	17	32	10	0	10
13	23	16	1	prop	12	30	14.61	0	15.87
14	26	12	3	center	21	34	12.05	0	13.74
15	26	16	7	fly half	20	31.5	14.99	1	15.69
16	19	13	1	prop	7	34	8	0	10
18	24	12	1	flank	14	30.5	11.81	0	16.27
19	22	12	0	scrum half	11	33	13	0	15
20	20	15	1	fly half	11	35	14	0	12
21	21	15	0	prop	14	32	12.91	0	15.72
22	26	12	6	scrum half	20	34	12.14	0	15.53
23	22	15	1	hooker	18	34	12.1	0	13.6
24	22	12	3	full back	7	32	11.38	0	14.5
25	19	12	0	wing	10	34	11.95	1	12.12
26	20	12	0	forward	13	36	12.18	0	14.94
27	19	13	0	hooker	13	34	16	1	14
29	20	12	1	eigth man	10	32.5	14.97	0	14.28
30	20	12	1	hooker	14	35.5	14	0	20
31	19	12	5	lock	11	32	10.73	0	13.44
32	20	14	0	scrum half	15	32	13.09	0	11.74
34	19	12	1	prop	12	28.5	15.3	0	15.5
35	19	12	3	fly half	12	35	12	0	15.63
38	20	13	7	flank	11	32	14.32	0	21
40	20	12	0	center	9	30	13	0	17
41	19	12	4	lock	14	26	13.7	0	12.5
42	20	13	0	wing	12	35	13.7	0	15.16
43	21	12	0	lock	10	30	15.8	0	22.1

STROOPWRDERR	STROOPCOLOUR	STROOPCOLERRS	RAY2MR	RAVLTT	RAVLTLEARNC	RAVLTRECOG	RAVLTDELAY	RAVLTA1	RAVLTB
0	17	0	24.6	40	6.0	14	13.0	5	14.0
0	22	1	24.5	57	7.0	13	10.0	7	11.0
0	23	1	23	53	5.0	14	11.0	8	8.0
0	19.58	0	27.5	63	8.0	15	11.0	7	11.0
0	17.54	0	22	50	6.0	14	11.0	7	6.0
0	20.38	1	15	54	7.0	15	12.0	7	11.0
0	18.64	0	20	50	9.0	15	12.0	5	12.0
0	22.73	0	22	48	5.0	14	11.0	7	11.0
0	19	0	15.5	65	4.0	15	11.0	10	10.0
0	18	0	23	57	7.0	15	15.0	7	15.0
0	18.49	0	22.5	40	4.0	13	9.0	6	11.0
0	18.05	0	24.5	50	7.0	14	12.0	6	15.0
1	25.88	2	14	56	7.0	13	11.0	8	9.0
0	22	1	23.5	52	7.0	13	9.0	6	10.0
0	22	2	26.5	49	4.0	13	8.0	7	11.0
0	20	1	26	38	5.0	11	6.0	5	8.0
0	18	0	28	63	5.0	13	10.0	10	12.0
0	28.08	2	17.5	40	4.0	12	5.0	6	6.0
1	22.29	0	19.5	61	6.0	14	12.0	9	7.0
0	20.5	1	22.5	50	6.0	15	11.0	6	11.0
0	19.52	0	17.5	46	5.0	12	11.0	6	10.0
1	15.1	4	29	51	4.0	14	9.0	7	10.0
0	24.16	0	25	59	9.0	15	13.0	6	13.0
0	17	0	25	53	9.0	14	14.0	6	14.0
0	20.05	0	27	57	6.0	15	10.0	8	11.0
1	26	0	26	46	4.0	12	9.0	7	12.0
0	17.12	0	33	56	3.0	15	12.0	10	13.0
0	21.01	0	23	60	7.0	14	12.0	8	14.0
1	26	2	18.5	58	8.0	15	15.0	7	15.0
1	22.37	1	26	50	5.0	13	7.0	7	9.0
0	28.3	3	25.5	47	7.0	12	12.0	5	10.0
0	28	2	21	40	0.0	11	6.0	7	7.0
0	19.6	2	25	52	6.0	14	14.0	7	13.0
0	36.53	0	25	49	7.0	14	13.0	5	11.0
0	20	0	24	61	7	15	13	7	13

RAVLTA6	SDMT WRITTN	SDMTOR	DIGITF	DIGITB	F	A	S	FAS	FASAN	TRAILA	TRAILB
7	53.0	57	5.0	5	13.00	9.00	10	32	16.00	35	64
7	60.0	78	5.0	5	18	12.00	16	46	16.00	18	49.00
8	58.0	64	7.0	4	14	10.00	14	38	20.00	19	41.00
6	53.0	63	8.0	6	13	10.00	17	40	19.00	23.7	51.44
13	56.0	65	7.0	6	15	10.00	13	38	19.00	20	49.70
6	52.0	56	7.0	4	12	7.00	13	32	21.00	23.41	57.00
3	58.0	66	7.0	5	12.00	12.00	14.00	38	21.00	25.82	50.84
6	46.0	49	8.0	4	5	5.00	10	20	14.00	24.69	53.68
6	57.0	68	8.0	7	15	13.00	18	46	21.00	21	40.00
15	66.0	69	6.0	6	13	9.00	13	35	19.00	20	51.00
6	41.0	54	7.0	6	12	10.00	16	38	15.00	33	48.00
8	56.0	60	6.0	4	11	13.00	19	43	19.00	20.64	54.37
9	48.0	57	5.0	4	11	11.00	13	35	24.00	36.24	120.00
4	57.0	63	7.0	4	16	13.00	17	46	20.00	37	64.00
6	49.0	59	5.0	3	7	8.00	12	27	15.00	28.46	68.00
8	55.0	49	5.0	7	19	9.00	15	43	14.00	31	56.00
8	54.0	60	7.0	5	13	8.00	10	31	13.00	32	49.00
6	45.0	51	6.0	4	11	6.00	11	28	17.00	38.02	61.78
4	42.0	62	7.0	5	13	11.00	18	42	21.00	31.73	74.58
7	57.0	73	6.0	6	16	11.00	14	41	23.00	27.3	59.00
5	55.0	56	5.0	5	8	11.00	15	34	18.00	21.04	58.01
7	57.0	62	7.0	6	10	10.00	10	30	17.00	33.72	40.01
9	57.0	65	7.0	5	10	5.00	8	23	12.00	22.64	62.34
6	60.0	57	7.0	5	16	14.00	19	49	20.00	42	120.00
7	54.0	62	6.0	5	13	13.00	14	40	17.00	34.55	62.00
10	54.0	57	6.0	4	6	12.00	14	32	15.00	27.57	65.00
7	68.0	61	7.0	7	7	8.00	10	25	19.00	21.54	44.26
7	67.0	62	8.0	6	16	11.00	13	40	20.00	15.39	50.53
7	63.0	78	5.0	4	9	8.00	14	31	19.00	27.2	66.00
4	63.0	62	6.0	6	6	4.00	17	27	18.00	25.67	49.01
9	58.0	59	5.0	4	14	11.00	15	40	18.00	23	65.00
5	34.0	37	6.0	3	12	7.00	10	29	15.00	35	109.00
4	51.0	55	5.0	5	15	18.00	21	54	20.00	25.8	53.00
6	51.0	62	6.0	5	17	12.00	15	34	15.00	29.07	84.48
9	48.00	47	6	5	8	8.00	11	27	17.00	32.9	103.9

RAY	CasePost	AgePost	EdPost	ReyCopyP	StroopDOTSP	StroopErrorsP	StroopWdsP	StroopErrorsP	StroopColourP
20.5	1	21	13	33	11.2	0	11	1	18.2
25.5	2	22	15	34	10.68	0	22.56	0	13.49
23.5	3	20	14	36	13	0	14.2	0	16.2
30	4	22	12	33	10.38	0	12.29	0	18.22
19	5	22	13	30	12.04	0	14	0	17.01
17	8	21	16	30	12.83	0	12.55	0	17.73
21.00	9	23	15	31	11	0	11	0	18.5
17.5	10	25	14	29	12	0	16	1	15
12.5	11	25	14	33	11.51	0	14.46	1	14.76
22.5	12	23	16	30	10	0	11	0	15
22.5	13	23	16	34	16	0	12.96	0	19.66
24.5	14	26	12	36	12.33	0	17.86	2	17.01
16.5	15	26	16	32	13	0	14.3	0	20.15
22.5	16	19	13	30	10	0	23	0	20
29	18	24	12	28	10.86	0	16.09	1	21
19	19	22	12	33	14	0	14	0	16.5
26	20	20	15	34	13	0	18	0	20
18	21	21	15	35	10	0	13	0	25
13.5	22	26	12	34	12.34	0	15.12	0	22.44
23	23	22	15	24	10.03	0	10.44	0	15.12
12	24	22	12	35	14.48	0	14.31	0	19.71
31.5	25	19	12	28.5	12.3	0	13	0	18.1
30	26	20	12	36	20	1	15	0	20
23	27	19	13	23.5	13	0	11	0	14
24	29	20	12	33.5	12.07	0	15.48	0	19.8
26	30	20	12	32	13.7	0	14.8	0	20.8
28	31	19	12	34	9.5	0	9	0	15
24	32	20	14	33	16	0	21	0	20
16	34	19	12	25.5	10.85	0	14.29	0	23
23	35	19	12	34	14.04	0	13.32	0	18.04
26.5	38	20	13	31	14.58	0	21.37	0	37.39
18.5	40	20	12	29	15.12	0	15.07	0	29.25
26	41	19	12	28	13.99	0	18.9	1	21.96
26.5	42	20	13	33	12	0	20	0	22
23	43	21	12	31	13.73	1	18.16	0	31.33

StroopColourErrorP	Rey2MP	RAVLTTP	RAVLT51DP	RAVLTRECOGP	RAVLTDELP	RAVLTA1P	RAVLTB1P	RAVLTA6P	SDMTWP	SDMTOP
1	30	47	5	14	12	7	7	12	55	60
0	31	51	5.0	14	13.0	7	4.0	12	71.0	80
0	29	49	6.0	14	9.0	7	4.0	11	66.0	71
1	31	49	4.0	15	12.0	7	7.0	12	67.0	76
1	22.5	49	6.0	14	13.0	6	5.0	13	59.0	64
0	16	40	7.0	13	10.0	4	4.0	10	51.0	57
0	20	49	7	14	12	6	5	12	63	69
0	19.5	46	3.0	12	10.0	8	7.0	8	46.0	67
0	23	53	6.0	14	12.0	7	6.0	11	62.0	65
0	24	45	12.0	15	15.0	3	3.0	15	73.0	74
0	24.5	44	6.0	9	7.0	5	6.0	7	44.0	52
0	28	48	7.0	8	8.0	5	5.0	8	54.0	57
0	19	57	6.0	13	11.0	8	8.0	10	47.0	58
0	12	57	4.0	13	10.0	9	5.0	8	59.0	75
1	22.5	49	3.0	12	8.0	7	4.0	10	56.0	59
0	29	40	4.0	12	6.0	6	4.0	7	53.0	69
2	30	47	7.0	11	10.0	6	10.0	10	52.0	58
0	28	38	5.0	12	7.0	4	5.0	9	44.0	50
0	21	54	5.0	14	12.0	7	4.0	8	52.0	59
2	27.5	62	6.0	15	14.0	7	9.0	12	53.0	48
0	30.5	44	6.0	13	11.0	5	3.0	9	56.0	56
0	22	49	3.0	15	11.0	8	5.0	11	51.0	57
0	30	54	4.0	14	11.0	8	5.0	12	67.0	67
0	24	49	3.0	12	11.0	8	5.0	12	51.0	68
0	22.5	50	8.0	14	13.0	6	5.0	14	61.0	64
1	26	60	4.0	13	9.0	9	7.0	11	51.0	57
1	34	61	3.0	14	13.0	9	7.0	13	66.0	67
0	30	56	9.0	14	13.0	6	5.0	14	61.0	63
1	18	55	8.0	15	13.0	6	3.0	12	72.0	83
0	27	49	9.0	14	15.0	5	6.0	14	57.0	59
1	28	51	0.0	13	9.0	8	6.0	10	53.0	58
3	21.5	48	7.0	13	9.0	5	4.0	13	45.0	56
0	23	39	2.0	14	8.0	6	5.0	8	50.0	61
0	28	51	8.0	13	13.0	6	5.0	13	57.0	62
2	33	51	9	15	12	4	4	10	46	62

DIGITFP	DIGITBP	CPOST	FPOST	LPOST	CFLPOST	ANIMALSP	TRAILAP	TRAILBP	RCF20P
6	5	10.00	13.00	8	31	18.00	37.5	62.3	22
7.0	5	16	12.00	13	41	18.00	25.65	46.67	30
6.0	5	17	14.00	14	45	15.00	20	32.40	31
8.0	5	9	8.00	7	24	21.00	24.82	49.11	29
7.0	7	12	13.00	14	39	20.00	22	51.20	23
5.0	3	14	9.00	8	31	18.00	21.55	51.67	17
7	5	10.00	12.00	11.00	33	16.00	23.20	48.50	20.00
7.0	4	13	7.00	12	32	16.00	22	75.00	19.5
7.0	6	17	16.00	13	46	23.00	25.75	42.04	24
6.0	6	14	8.00	14	36	17.00	18	40.00	24
6.0	4	13	11.00	11	35	16.00	20.73	45.75	20
5.0	3	14	18.00	15	47	17.00	19.34	44.26	28.5
6.0	5	12	11.00	12	35	20.00	34.13	61.42	19
9.0	6	16	12.00	11	39	18.00	28	79.00	13
5.0	3	7	8.00	5	20	14.00	26.48	56.51	22.5
7.0	6	16	17.00	18	51	19.00	26.5	53.50	31
7.0	4	8	11.00	11	30	10.00	29	61.00	27
4.0	4	9	10.00	9	28	10.00	25	56.00	28
6.0	5	10	13.00	14	37	20.00	26.5	63.23	20
6.0	4	11	15.00	14	40	21.00	30.56	80.41	23.5
5.0	5	7	8.00	9	24	13.00	23.49	53.41	29.5
6.0	6	14	16.00	12	42	16.00	17.37	73.00	22
6.0	4	8	7.00	6	21	15.00	28	57.00	24
6.0	4	20	18.00	18	56	16.00	33	55.00	23
6.0	6	13	12.00	13	38	20.00	27.61	56.52	23
5.0	4	12	13.00	14	39	10.00	34.5	86.00	26
7.0	5	8	9.00	4	21	18.00	24	44.00	30
7.0	7	16	15.00	13	44	16.00	22	49.00	27
4.0	4	8	11.00	9	28	20.00	35.4	66.73	21
6.0	5	13	5.00	12	30	19.00	25.65	75.96	24
6.0	3	12	10.00	6	28	17.00	21.42	50.17	30
4.0	4	8	11.00	9	28	13.00	27.31	76.00	19
6.0	3	13	11.00	7	31	16.00	26.1	69.43	27
6.0	4	11	14.00	13	38	16.00	30	120.00	26
7	5	11	11.00	11	33	15.00	46.3	120	29

Appendix E

Excel table for Controls Group: Demographic details, test scores

No	AGE	EDUC	RAY	STROOPDOTS	STROOPDOTERRS	STROOPW	STROOPWRDERR	STROOPCOLOUR
1	23	13	30	14.68	0	16.24	0	22.27
2	26	18	36	13.8	0	14.23	0	29.66
3	27	12	33	15.28	0	12.13	0	19.69
4	23	14	36	13.68	0	14.79	0	30.99
5	20	13	30	12.84	0	12.98	0	30.88
6	25	15	30	12.5	0	12.3	0	20.52
7	26	13	28	12.73	0	31.52	0	21.2
8	22	14	34	12.03	0	23.05	1	32.55
9	20	12	29	14.9	0	17.69	0	28.62
10	25	12	26	13.23	1	14.55	0	18.15
11	20	14	33	13.48	0	14.02	1	19.11
12	27	13	32	10.74	0	12.37	0	28.26
13	22	13	29	10.02	0	16.07	1	17
14	21	15	33	15.92	2	15.43	0	28.41
15	25	16	34	13.08	0	12.79	0	25.07
16	26	15	35	15.57	1	1.2	0	24.78
17	18	13	30	22.9	3	21.06	0	38.7
18	26	13	31	14.5	0	14.96	0	20.15
19	24	15	35	14.06	1	12.86	0	19.48
20	21	15	34	12.24	0	14.03	0	20.83
21	22	15	33	10.02	0	10.95	0	18.8
22	27	17	34	16	0	15	0	25
23	28	13	32	15	0	21	0	45
24	22	14	32	11.35	0	14.48	1	23.36
25	24	12	34	12.47	0	13.02	0	15.03
26	25	19	36	13.62	0	15.19	0	23.07
27	25	15	26	17.28	1	17.08	2	28.15
28	21	12	34	14.88	0	17.07	0	22.68
29	26	17	35.5	12.43	0	11.17	0	23.48
30	26	14	34	7.57	0	8.15	0	14.79
31	26	14	33	11.64	0	13.5	0	20.03
32	21	14	32	11.29	0	13.15	0	13.68
33	26	12	33	11.35	0	11.32	0	15.29
34	25	16	36	18	0	29	0	26
35	24	17	28	19	0	22	1	30

STROOPCOLERRS	RAY2MR	RAVLTT	RAVLTLEARNC	RAVLTRECOG	RAVLTDELAY	RAVLTA1	RAVLTB	RAVLTA6	SDMT WRITTN	SDMTOR
0	29	55	5.0	14	8.0	8	6.0	9	60.0	65
1	24	38	5.0	13	11.0	5	6.0	10	56.0	59
0	26	48	7.0	13	12.0	5	7.0	11	47.0	63
1	23	59	8.0	15	15.0	7	7.0	13	55.0	62
0	18	49	8.0	14	13.0	5	6.0	12	42.0	46
0	7	43	5.0	9	6.0	6	4.0	8	56.0	65
2	13	51	8.0	14	11.0	6	5.0	11	48.0	53
3	25	46	6.0	15	11.0	6	5.0	10	42.0	53
1	9	48	6.0	14	13.0	6	6.0	12	43.0	53
0	13	44	7.0	14	9.0	5	5.0	11	58.0	66
0	27	65	7.0	14	15.0	8	5.0	10	69.0	69
3	14.5	53	6.0	13	11.0	8	7.0	13	46.0	52
1	10.5	52	4.0	14	9.0	8	8.0	10	56.0	60
1	13	48	3.0	8	13.0	7	2.0	8	48.0	47
3	27	56	7.0	10	13.0	7	7.0	12	56.0	60
3	24	48	5.0	13	8.0	6	7.0	11	42.0	52
3	27	51	6.0	14	12.0	7	7.0	12	53.0	60
3	19	45	4.0	11	6.0	6	5.0	10	51.0	58
0	26.5	50	8.0	14	11.0	5	4.0	14	51.0	60
1	25	62	7.0	12	14.0	8	5.0	11	63.0	61
0	1.5	51	4.0	14	10.0	8	4.0	10	57.0	63
5	19.5	42	4.0	15	5.0	8	5.0	5	42.0	50
4	18	45	9.0	13	9.0	5	5.0	5	40.0	53
0	26.5	66	4.0	15	13.0	10	10.0	14	41.0	63
0	27.5	56	7.0	15	9.0	7	6.0	9	56.0	69
0	29	72	1.0	14	15.0	14	4.0	15	63.0	65
0	20.5	47	5.0	10	12.0	7	5.0	10	50.0	52
0	13.5	64	6.0	14	13.0	7	4.0	13	53.0	48
0	23	52	12.0	15	10.0	4	6.0	12	53.0	61
0	26	48	7.0	15	11.0	6	5.0	12	57.0	55
0	23	44	7.0	10	9.0	5	9.0	7	49.0	54
0	19	59	8.0	12	11.0	7	5.0	13	57.0	60
1	20	48	3.0	12	6.0	8	8.0	7	50.0	61
0	20	57	10.0	15	15.0	5	8.0	15	53.0	48
0	19	47	9.0	14	11.0	5	7.0	13	55.0	55

DIGITF	DIGITB	F	A	S	FAS	FASAN	TRAILA	TRAILB	RAY		CasePost
8.0	5	11	8.00	16	35	14.00	21.29	49.66	30	0	1
7.0	3	4	9.00	9	22	14.00	21.4	40.42	24	0	2
7.0	5	14	12.00	16	42	18.00	28.53	41.31	26	0	3
6.0	4	16	10.00	15	41	16.00	27.93	58.36	20.5	0	4
6.0	6	11	9.00	12	32	18.00	32.23	80.63	17	0	5
7.0	5	16	10.00	15	41	21.00	25.6	120.00	13	0	6
8.0	4	9	7.00	12	28	18.00	28.07	79.00	15	0	7
6.0	4	10	7.00	13	30	20.00	31.3	68.93	23	0	8
8.0	4	9	9.00	11	29	15.00	43.38	85.72	11	0	9
7.0	5	10	5.00	12	27	17.00	24.61	63.85	15	0	10
8.0	5	14	16.00	18	48	18.00	21.58	47.26	29	0	11
6.0	6	16	13.00	10	39	19.00	29.92	63.41	15	0	12
6.0	5	13	10.00	11	34	16.00	20.18	86.86	10	0	13
7.0	3	11	12.00	10	33	18.00	27.8	70.00	15	0	14
8.0	5	15	11.00	17	43	16.00	41.22	52.10	29	0	15
6.0	5	11	10.00	11	32	10.00	23.92	60.46	25	0	16
7.0	7	10	12.00	7	29	15.00	26.46	60.03	24	0	17
6.0	4	6	9.00	14	29	13.00	55.26	84.16	26.5	0	18
6.0	5	11	6.00	17	34	16.00	37.33	69.07	22	0	19
7.0	4	17	13.00	18	48	21.00	21.1	41.35	23	0	20
7.0	3	31.38	61.00	11	103.38	4.00	10	18.00	18.5	0	21
5.0	3	11	7.00	10	28	15.00	27	103.00	22	0	22
5.0	5	12	9.00	11	32	24.00	37	60.32	16	0	23
6.0	5	15	15.00	22	52	21.00	30.45	64.55	26.5	0	24
8.0	7	12	11.00	18	41	24.00	18.77	58.01	20.5	0	25
7.0	4	19	12.00	20	51	24.00	25.12	47.04	25.5	0	26
8.0	6	6	12.00	16	34	16.00	18.27	120.00	16.5	0	27
5.0	5	13	9.00	15	37	20.00	28.53	83.00	14	0	28
6.0	5	11	11.00	9	31	18.00	36.71	70.00	22	0	29
7.0	6	9	5.00	10	24	19.00	44.95	56.55	24.5	0	30
8.0	8	14	16.00	18	48	20.00	23.65	43.11	24	0	31
6.0	4	12	8.00	6	26	17.00	20.7	53.82	15.5	0	32
8.0	5	12	10.00	16	38	20.00	21.35	44.98	19	0	33
7.0	7	12	12.00	20	44	21.00	26	49.00	21.5	0	34
5.0	6	13	11.00	14	38	16.00	35	57.00	14.5	0	35

AgePost	EdPost	ReyCopyP	StroopDOTSP	StroopErrorsP	StroopWdsP	StroopErrorsP	›pColourP	StroopColourErrorP	Rey2MP	RAVLTTP
23	13	35	11.03	0	15.2	1	20.2	0	32	59
26	18	32	15.25	0	15.3	0	19.03	0	22	51
27	12	34	11.03	0	13.45	0	16.6	0	34	50
23	14	35	15.3	1	13.96	0	18.89	0	22	59
20	13	31.5	16.02	0	17.54	0	22.39	0	25	48
25	15	35	13.16	0	15.36	0	18.59	0	23	51
26	13	29.5	14.29	0	16.56	1	28.23	0	20	50
22	14	33	11.29	1	15	0	25	0	20.5	41
20	12	31.5	14.22	0	15.96	0	26.4	0	16	53
25	12	33	11.09	0	13.31	0	13.91	0	23	57
20	14	34	11.31	0	12.53	0	19.07	0	34	57
27	13	28.5	9.92	0	11.17	0	14.2	0	25	59
22	13	31	17.2	0	11.07	0	13.02	0	20	49
21	15	33	16.94	2	15.87	0	26.51	1	19	46
25	16	35	15.36	1	12.2	0	16.71	1	34	42
26	15	32	9.39	0	12.5	0	21.45	0	28	56
18	13	33	13.69	0	13.82	0	7.65	0	28	51
26	13	34	14.62	0	17.56	0	23.16	1	20.5	35
24	15	36	14.12	1	13.3	0	18.56	0	31	52
21	15	33	11.82	0	13.12	0	21.72	1	22.5	50
22	15	33.5	9.74	0	10.06	0	17.05	2	23.5	49
27	17	34	16.28	1	17.99	0	23.71	0	18.5	43
28	13	32.5	17.64	1	23.32	0	17.12	2	28	40
22	14	33	10.09	0	10.53	0	22.1	0	32	63
24	12	34	9.71	0	11.16	0	18.93	0	34	50
25	19	36	11.22	0	13.02	0	17.55	0	28.5	73
25	15	34	14.23	0	13.5	0	20.23	0	25	45
21	12	33	11.19	0	14.78	0	18.73	0	15.5	45
26	17	32	11.79	0	12.29	0	18.21	0	20.5	50
26	14	30.5	7.5	0	9.02	0	13.6	0	26	38
26	14	35	13.84	0	14.12	0	17.96	0	29	50
21	14	35	11.3	0	12	0	13.01	0	24	56
26	12	33	10.41	0	10.1	0	14.86	1	31.5	47
25	16	33	15.43	2	20.9	1	23.4	1	28	46
24	17	33.5	12.51	1	14.37	0	22.97	0	26.5	67

RAVLT51DP	RAVLTRECOGP	RAVLTDELP	RAVLTA1P	RAVLTB1P	RAVLTA6P	SDMTWP	SDMTOP	DIGITFP	DIGITBP	CPOST	FPOST	LPOST
7	14	11	8	6	12	72	78	8	6	15.00	16.00	13.00
6.0	13	13.0	6	6.0	11	54.0	65	8.0	5	12	14.00	10.00
8.0	14	13.0	5	6.0	13	54.0	67	7.0	5	15	18.00	13.00
10.0	12	14.0	5	5.0	14	58.0	69	7.0	4	15	14.00	12.00
5.0	13	9.0	7	7.0	7	54.0	59	8.0	5	14	20.00	18.00
4.0	14	11.0	8	6.0	10	61.0	61	7.0	5	4	9.00	8.00
5.0	15	6.0	6	5.0	9	50.0	47	6.0	4	9	8.00	10.00
6.0	13	7.0	5	4.0	7	46.0	46	7.0	5	11	14.00	16.00
7.0	12	8.0	6	7.0	8	49.0	47	7.0	3	12.00	11.00	10.00
5.0	13	12.0	8	6.0	12	57.0	71	5.0	4	11	10.00	9.00
8.0	15	15.0	7	7.0	14	66.0	64	8.0	6	20	17.00	14.00
7.0	15	10.0	8	10.0	12	51.0	55	7.0	4	21	22.00	24.00
7.0	15	12.0	6	7.0	11	66.0	71	7.0	4	12	10.00	10.00
7.0	14	10.0	6	3.0	12	50.0	52	8.0	4	14	14.00	12.00
6.0	14	8.0	6	4.0	13	63.0	68	8.0	5	15	14.00	12.00
7.0	15	12.0	7	5.0	12	57.0	53	8.0	5	14	9.00	17.00
7.0	14	9.0	6	7.0	9	65.0	78	8.0	7	17	16.00	17.00
3.0	14	6.0	5	4.0	7	50.0	56	5.0	3	11	13.00	9.00
10.0	15	13.0	5	3.0	14	49.0	64	6.0	5	14	9.00	12.00
4.0	13	10.0	7	5.0	9	72.0	69	7.0	6	17	18.00	11.00
6.0	15	9.0	7	5.0	11	71.0	71	7.0	5	12	12.00	13.00
4.0	11	8.0	6	5.0	8	48.0	52	5.0	3	9	13.00	6.00
7.0	13	8.0	4	4.0	9	40.0	51	6.0	5	13	10.00	8.00
7.0	14	11.0	10	4.0	11	50.0	51	6.0	5	20	20.00	13.00
1.0	14	8.0	9	5.0	11	51.0	47	7.0	7	12	17.00	14.00
2.0	14	14.0	13	14.0	15	71.0	71	8.0	6	22	23.00	22.00
5.0	14	11.0	7	5.0	10	59.0	58	8.0	6	12	8.00	10.00
5.0	12	7.0	7	5.0	9	55.0	60	6.0	5	10	10.00	13.00
7.0	13	13.0	6	5.0	12	59.0	59	7.0	5	11	12.00	9.00
5.0	15	9.0	5	4.0	9	54.0	50	7.0	5	14	11.00	15.00
6.0	13	8.0	7	6.0	9	68.0	65	7.0	5	17	14.00	14.00
7.0	13	12.0	7	6.0	13	54.0	60	6.0	5	10	12.00	11.00
4.0	12	9.0	7	6.0	8	66.0	73	8.0	6	16	17.00	18.00
5.0	12	8.0	6	10.0	4	55.0	67	8.0	4	13	13.00	14.00
9.0	15	15.0	6	10.0	10	58.0	52	7.0	5	16	18.00	17.00

CFLPOST	ANIMALSP	TRAILAP	TRAILBP	RCF20P
44	23.00	15.23	36.83	32
36	24.00	21.55	33.29	27
46	21.00	29.70	57.28	30
41	17.00	29.51	91.98	23.5
52	18.00	21.59	58.79	25
21	18.00	42.12	63.55	20
27	16.00	29.16	89.42	23
41	26.00	28.93	60.34	28
33	18.00	19.01	60.03	14.5
30	16.00	27.97	50.22	22.5
51	21.00	20.78	37.87	34
67	14.00	26.76	93.90	25
32	14.00	22.02	60.25	13
40	15.00	40.17	108.15	21.5
41	19.00	23.19	46.73	35
40	19.00	24.36	48.53	28
50	15.00	26.29	60.30	28
33	15.00	26.21	60.03	20
35	17.00	20.62	60.37	30
46	21.00	18.07	48.35	27
37	16.00	23.69	45.13	23.5
28	14.00	33.43	101.18	18.5
31	18.00	40.17	78.06	29
53	18.00	24.35	77.56	30.5
43	18.00	22.63	46.81	31
67	17.00	21.91	33.21	26.5
30	19.00	17.23	48.28	23.5
33	16.00	33.12	60.74	13.5
32	20.00	36.09	67.60	23
40	19.00	45.62	42.11	25.5
45	18.00	23.22	45.94	29
33	18.00	19.23	52.39	20
51	21.00	26.20	49.58	30.5
40	21.00	40.00	54.94	24
51	19.00	20.47	49.54	28

Appendix F

Excel table for concussed players preseason baseline test scores and post-concussion test scores

	PLAYER	RAY	D	E	W	E	C	E	RAY	TOTAL	LEARN	RECOG	DELAY	IMM
POST CONCUSSION TEST SCORES	1	33	12.67	0	12.33	0	19.03	0	22.5	48.0	5	13.0	10	7
	2	34	9.93	0	12.06	0	33.04	0	32	55.0	6	14.0	10	7
	3	28.5	20.4	0	19.86	0	32.16	2	20	50.0	7	14.0	8	6
	4	28.5	14.51	2	15.23	0	26.78	2	21	44.0	7	11.0	7	4
	5	30	15.2	0	16.2	1	24.3	6	10	52.0	8	13.0	10	6
PRE CONCUSSION TEST SCORES	1	34	11.38	0	11.17	0	18.64	0	20	50.0	9	15.0	12	5
	2	32	10.73	0	13.44	0	17.12	0	33	56.0	3	15.0	12	10
	3	34.00	20.20	0.00	22.54	0.00	24.19	0.00	28.00	51.00	6.00	15.00	11.00	8.00
	4	32	14.32	0	21	0	28.3	3	25.5	47	7	12	12	5
	5	30	12.23	0	14.19	0	17.54	0	22	50.0	6	14.0	11	7.0

VI	B1	SDMT WRITTN	SDMT OR	DIGIT F	DIGIT B	C	F	L	CFL	Animal	TRAIL A	TRAIL B	RAY
10.0	6.0	58	62.0	5	4.00	12.00	13.00	5.00	30.00	15.00	23.95	50.54	22.5
11.0	11.0	69	59.0	6	5	5.00	8.00	12.00	25.00	13.00	19.34	36.29	31
10.0	5.0	47	56.0	6	4	16.00	18.00	15.00	49.00	19.00	23.01	63.30	21
10.0	6.0	55	58.0	6	4	9.00	13.00	9.00	31.00	19.00	22.63	37.43	18
14.0	7.0	53	65.0	7	6	16.00	7.00	15.00	38.00	23.00	22.60	53.00	13.5
12.0	3.0	58	66.0	7	5	12.00	12.00	14.00	38.00	21.00	25.82	50.84	21
13.0	7.0	68	61.0	7	7	7.00	8.00	10.00	25.00	19.00	21.54	44.26	28
9.00	6.00	53.00	55.00	6.00	6.00	16.00	17.00	25.00	58.00	22.00	23.06	49.97	25
10	9	58	59	5	4	14	11	15	40	18	23	65	26.5
13	6.0	56	65.0	7	6	15.00	10.00	13.00	38.00	19.00	20.00	49.7	19

Appendix G

Correlation Table – Post Concussion Symptoms Scale (PCS) and post-season test scores

		headache	nausea	vomiting	balance	dizziness	fatigue	diff fall asleep	more sleep	drowsiness	light sensitivity	noise
RCF Copy	Correlation Coefficient	-0.04	-0.14	-0.26	-0.12	0.09	-0.07	-0.19	-0.28	-0.14	-0.09	-0.08
	Sig. (2-tailed)	0.82	0.42	0.13	0.50	0.59	0.69	0.27	0.11	0.42	0.60	0.65
STROOP Dots	Correlation Coefficient	-0.14	0.02	-0.16	-0.09	0.05	-0.15	0.10	0.12	0.12	0.13	0.18
	Sig. (2-tailed)	0.41	0.92	0.36	0.62	0.77	0.39	0.57	0.48	0.48	0.45	0.29
STROOP DOTS Error	Correlation Coefficient	-0.26	-0.05	-0.14	-0.14	-0.19	-0.30	-0.18	-0.19	-0.06	-0.17	-0.13
	Sig. (2-tailed)	0.12	0.79	0.41	0.43	0.27	0.08	0.29	0.27	0.74	0.32	0.45
STROOP Words	Correlation Coefficient	0.01	0.05	-0.23	-0.04	-0.08	-0.20	0.03	0.15	0.16	0.23	0.20
	Sig. (2-tailed)	0.94	0.79	0.18	0.81	0.67	0.24	0.87	0.38	0.36	0.19	0.26
STROOP WORDS Error	Correlation Coefficient	0.25	0.00	-0.11	0.03	-0.05	0.20	-0.06	0.28	0.16	0.19	0.13
	Sig. (2-tailed)	0.14	0.98	0.52	0.87	0.78	0.26	0.74	0.11	0.36	0.28	0.44
STROOP Colour	Correlation Coefficient	-0.16	-0.06	-0.18	-0.04	-0.15	-0.25	0.13	0.06	0.28	0.18	0.15
	Sig. (2-tailed)	0.36	0.74	0.29	0.84	0.37	0.16	0.45	0.71	0.11	0.29	0.38
STROOP COLOUR Error	Correlation Coefficient	0.32	0.26	0.42	0.54	0.23	0.31	0.23	0.48	0.51	0.32	0.32
	Sig. (2-tailed)	0.06	0.13	0.01	0.00	0.18	0.07	0.18	0.00	0.00	0.06	0.06
RCF 2 mln Recall	Correlation Coefficient	0.22	0.16	-0.10	0.15	0.20	0.05	-0.07	0.04	0.14	0.04	0.08
	Sig. (2-tailed)	0.20	0.36	0.57	0.40	0.24	0.79	0.68	0.84	0.44	0.84	0.63
RAVLT Total	Correlation Coefficient	-0.16	0.00	0.13	0.10	0.08	-0.09	-0.18	-0.13	0.05	0.05	-0.10
	Sig. (2-tailed)	0.35	0.98	0.45	0.58	0.64	0.60	0.31	0.44	0.76	0.78	0.58
RAVLT Learn	Correlation Coefficient	-0.04	0.04	-0.06	-0.02	-0.08	0.13	-0.03	-0.22	0.14	-0.29	0.07
	Sig. (2-tailed)	0.82	0.83	0.75	0.93	0.63	0.44	0.86	0.21	0.42	0.10	0.68
RAVLT Recognition	Correlation Coefficient	-0.06	0.01	-0.03	-0.13	-0.14	0.15	-0.09	-0.13	-0.04	-0.24	-0.28
	Sig. (2-tailed)	0.72	0.95	0.85	0.46	0.44	0.38	0.61	0.47	0.80	0.16	0.10
RAVLT 20 min delay recall	Correlation Coefficient	0.10	0.11	0.05	0.07	-0.01	0.21	-0.13	-0.21	-0.03	-0.25	-0.28
	Sig. (2-tailed)	0.55	0.55	0.78	0.68	0.94	0.22	0.45	0.23	0.88	0.15	0.11
RAVLT Immediate	Correlation Coefficient	-0.03	-0.12	0.14	0.09	0.07	-0.05	-0.17	0.11	-0.15	0.17	-0.14
	Sig. (2-tailed)	0.86	0.48	0.43	0.60	0.69	0.76	0.32	0.52	0.38	0.33	0.44
RAVLT B1 - distractor	Correlation Coefficient	0.31	0.09	0.22	0.30	0.23	0.24	0.05	0.37	0.12	0.25	0.17
	Sig. (2-tailed)	0.07	0.61	0.20	0.09	0.18	0.17	0.76	0.03	0.50	0.14	0.32
RAVLT V1 – 2 min recall	Correlation Coefficient	0.10	-0.01	-0.03	-0.05	-0.15	0.37	-0.12	-0.08	0.06	-0.20	-0.11
	Sig. (2-tailed)	0.59	0.97	0.88	0.78	0.39	0.03	0.49	0.65	0.74	0.25	0.52
SDMT Written	Correlation Coefficient	0.05	-0.08	-0.05	-0.08	-0.08	0.16	-0.13	-0.25	-0.21	-0.34	-0.31
	Sig. (2-tailed)	0.76	0.65	0.76	0.64	0.65	0.36	0.44	0.15	0.23	0.05	0.07
SDMT Oral	Correlation Coefficient	-0.06	-0.21	-0.03	-0.19	-0.29	0.06	-0.16	-0.15	-0.31	-0.30	-0.37

	Sig. (2-tailed)	0.74	0.22	0.88	0.28	0.09	0.73	0.37	0.39	0.07	0.08	0.03
Digits Forwards	Correlation Coefficient	0.19	0.04	0.01	-0.06	0.04	0.17	0.09	0.16	-0.20	-0.10	-0.20
	Sig. (2-tailed)	0.28	0.83	0.93	0.75	0.84	0.33	0.63	0.36	0.25	0.56	0.25
Digits backwards	Correlation Coefficient	-0.17	-0.15	-0.14	-0.25	-0.13	0.06	-0.25	-0.11	-0.36	-0.45	-0.39
	Sig. (2-tailed)	0.34	0.40	0.43	0.15	0.45	0.75	0.15	0.53	0.03	0.01	0.02
COWAT C	Correlation Coefficient	-0.14	-0.03	-0.14	-0.31	-0.12	-0.03	-0.11	-0.05	-0.23	-0.18	-0.11
	Sig. (2-tailed)	0.41	0.88	0.43	0.08	0.49	0.86	0.54	0.76	0.19	0.29	0.54
COWAT F	Correlation Coefficient	0.01	-0.01	0.02	0.04	0.01	-0.05	0.20	0.14	0.26	0.07	0.31
	Sig. (2-tailed)	0.94	0.95	0.90	0.84	0.94	0.79	0.25	0.41	0.13	0.68	0.07
COWAT L	Correlation Coefficient	0.00	0.15	0.12	0.04	0.09	0.03	0.06	0.01	-0.04	0.00	0.22
	Sig. (2-tailed)	0.99	0.38	0.50	0.84	0.60	0.87	0.72	0.95	0.82	0.98	0.20
COWAT CFL	Correlation Coefficient	-0.06	0.04	0.00	-0.09	-0.01	-0.06	0.04	0.03	-0.03	-0.07	0.15
	Sig. (2-tailed)	0.73	0.82	1.00	0.63	0.97	0.74	0.82	0.86	0.88	0.70	0.38
COWAT Animals	Correlation Coefficient	0.03	-0.02	0.14	0.01	0.08	0.12	0.17	0.00	-0.06	-0.11	-0.23
	Sig. (2-tailed)	0.87	0.89	0.44	0.94	0.65	0.50	0.34	1.00	0.74	0.53	0.19
Trail A	Correlation Coefficient	-0.21	-0.18	0.17	0.20	-0.18	-0.18	-0.07	0.11	0.26	0.05	0.25
	Sig. (2-tailed)	0.22	0.29	0.32	0.27	0.29	0.30	0.70	0.54	0.14	0.76	0.15
Trail B	Correlation Coefficient	-0.12	-0.01	0.19	0.25	-0.08	-0.04	0.06	0.26	0.29	0.26	0.35
	Sig. (2-tailed)	0.48	0.96	0.29	0.16	0.65	0.83	0.74	0.14	0.09	0.13	0.04
RCF 20 min recall	Correlation Coefficient	0.20	0.28	-0.03	0.15	0.21	0.00	0.08	0.00	0.10	0.16	0.15
	Sig. (2-tailed)	0.26	0.10	0.86	0.41	0.22	0.99	0.66	0.99	0.56	0.37	0.40

		irritability	Sad-ness	Increased Nerves	more emotional	tingle	slow	foggy	Difficulty Concentrate	Difficulty remember	Visual disturbance
RCF Copy	Correlation Coefficient	-0.32	-0.22	-0.46	-0.20	-0.33	-0.21	-0.04	-0.29	-0.27	-0.37
	Sig. (2-tailed)	0.06	0.20	0.00	0.25	0.05	0.23	0.82	0.09	0.12	0.03
STROOP Dots	Correlation Coefficient	0.20	0.12	0.10	0.21	0.07	0.04	0.18	0.12	0.21	0.13
	Sig. (2-tailed)	0.25	0.49	0.58	0.22	0.68	0.83	0.31	0.48	0.22	0.47
STROOP DOTS Error	Correlation Coefficient	-0.19	-0.13	-0.15	-0.17	-0.16	-0.23	-0.21	-0.21	-0.18	-0.12
	Sig. (2-tailed)	0.27	0.45	0.38	0.32	0.35	0.18	0.22	0.23	0.29	0.48
STROOP Words	Correlation Coefficient	0.16	-0.10	0.12	0.05	-0.09	0.14	0.21	0.24	0.23	-0.13
	Sig. (2-tailed)	0.35	0.57	0.49	0.79	0.62	0.42	0.22	0.17	0.18	0.45
STROOP WORDS Error	Correlation Coefficient	0.15	-0.05	0.39	0.15	0.04	0.13	0.21	0.32	0.20	-0.03
	Sig. (2-tailed)	0.40	0.77	0.02	0.40	0.84	0.45	0.22	0.07	0.26	0.85
STROOP Colour	Correlation Coefficient	0.20	0.06	0.03	0.14	-0.06	0.05	-0.01	0.20	0.30	0.09
	Sig. (2-tailed)	0.25	0.73	0.85	0.41	0.71	0.75	0.97	0.27	0.08	0.62
STROOP COLOUR Error	Correlation Coefficient	0.36	0.09	0.28	0.44	0.19	0.37	0.16	0.36	0.42	0.17
	Sig. (2-tailed)	0.04	0.59	0.10	0.01	0.27	0.03	0.34	0.03	0.01	0.34
RCF 2 min Recall	Correlation Coefficient	-0.10	-0.21	-0.27	-0.07	-0.04	0.07	0.03	-0.07	0.03	-0.18
	Sig. (2-tailed)	0.57	0.22	0.11	0.70	0.81	0.69	0.88	0.70	0.86	0.29
RAVLT Total	Correlation Coefficient	-0.06	-0.11	-0.28	-0.13	-0.26	0.05	-0.03	-0.06	-0.18	-0.21
	Sig. (2-tailed)	0.73	0.54	0.11	0.45	0.13	0.78	0.88	0.74	0.29	0.22
RAVLT Learn	Correlation Coefficient	-0.12	0.01	-0.25	-0.13	0.09	-0.26	-0.03	-0.16	-0.02	0.06
	Sig. (2-tailed)	0.51	0.97	0.15	0.47	0.60	0.13	0.84	0.37	0.91	0.71
RAVLT Recognition	Correlation Coefficient	-0.10	0.16	-0.26	-0.14	0.15	-0.10	-0.24	-0.21	-0.16	0.09
	Sig. (2-tailed)	0.56	0.36	0.13	0.43	0.40	0.57	0.17	0.23	0.36	0.62
RAVLT 20 min delay recall	Correlation Coefficient	-0.09	-0.18	-0.37	-0.25	-0.02	-0.08	-0.03	-0.15	-0.29	-0.10
	Sig. (2-tailed)	0.62	0.30	0.03	0.14	0.93	0.66	0.86	0.38	0.09	0.57
RAVLT Immediate	Correlation Coefficient	0.05	-0.14	-0.10	-0.05	-0.34	0.19	-0.02	0.03	-0.22	-0.30
	Sig. (2-tailed)	0.79	0.41	0.57	0.76	0.04	0.29	0.89	0.88	0.20	0.08
RAVLT B1 distractor	Correlation Coefficient	0.23	-0.33	-0.06	0.17	-0.12	0.39	0.34	0.22	-0.10	-0.31

	Sig. (2-tailed)	0.19	0.05	0.72	0.32	0.50	0.02	0.04	0.21	0.57	0.07
RAVLT V1 - 2 min delay recall	Correlation Coefficient	-0.02	-0.17	-0.24	-0.18	0.12	-0.03	-0.05	-0.12	-0.15	-0.14
	Sig. (2-tailed)	0.91	0.33	0.17	0.31	0.48	0.84	0.78	0.49	0.39	0.44
SDMT Written	Correlation Coefficient	-0.38	-0.11	-0.34	-0.36	-0.02	-0.16	-0.24	-0.30	-0.12	-0.16
	Sig. (2-tailed)	0.03	0.53	0.05	0.03	0.92	0.35	0.16	0.09	0.49	0.37
SDMT Oral	Correlation Coefficient	-0.32	-0.03	-0.31	-0.43	-0.10	-0.15	-0.30	-0.38	-0.28	-0.21
	Sig. (2-tailed)	0.06	0.87	0.07	0.01	0.57	0.39	0.08	0.03	0.10	0.22
Digits Forwards	Correlation Coefficient	-0.07	-0.27	-0.24	-0.20	-0.05	0.18	0.00	-0.13	-0.27	-0.38
	Sig. (2-tailed)	0.67	0.12	0.17	0.26	0.79	0.31	1.00	0.46	0.11	0.03
Digits backwards	Correlation Coefficient	-0.26	-0.20	-0.49	-0.33	-0.09	-0.22	-0.37	-0.40	-0.47	-0.29
	Sig. (2-tailed)	0.13	0.25	0.00	0.06	0.60	0.20	0.03	0.02	0.00	0.09
COWAT - C	Correlation Coefficient	-0.10	0.05	-0.05	-0.10	0.13	-0.05	0.00	-0.11	-0.28	-0.15
	Sig. (2-tailed)	0.55	0.75	0.78	0.57	0.45	0.78	0.99	0.54	0.10	0.38
COWAT - F	Correlation Coefficient	0.17	0.19	-0.07	0.25	0.02	0.07	0.01	0.19	-0.07	-0.03
	Sig. (2-tailed)	0.34	0.28	0.71	0.15	0.93	0.70	0.97	0.29	0.67	0.85
COWAT - L	Correlation Coefficient	0.07	0.11	-0.11	0.11	0.06	-0.08	0.10	0.07	-0.28	-0.13
	Sig. (2-tailed)	0.68	0.53	0.52	0.51	0.72	0.66	0.56	0.71	0.10	0.45
COWAT - CFL	Correlation Coefficient	0.03	0.10	-0.10	0.07	0.05	-0.04	0.01	0.03	-0.27	-0.14
	Sig. (2-tailed)	0.85	0.57	0.56	0.68	0.76	0.84	0.97	0.85	0.11	0.42
COWAT - Animals	Correlation Coefficient	-0.05	0.14	0.01	0.02	0.20	0.08	0.02	-0.05	-0.11	0.10
	Sig. (2-tailed)	0.79	0.42	0.98	0.92	0.24	0.64	0.93	0.78	0.52	0.58
Trail A	Correlation Coefficient	0.19	0.08	-0.16	0.15	-0.31	-0.05	-0.13	0.10	-0.01	0.14
	Sig. (2-tailed)	0.26	0.63	0.36	0.40	0.07	0.77	0.45	0.59	0.98	0.42
Trail B	Correlation Coefficient	0.44	0.13	0.12	0.25	-0.15	0.15	0.21	0.30	0.05	0.22
	Sig. (2-tailed)	0.01	0.45	0.51	0.15	0.38	0.40	0.22	0.09	0.77	0.20
RCF 20 min recall	Correlation Coefficient	-0.12	-0.02	-0.12	-0.01	0.08	0.12	0.14	-0.01	0.13	-0.08
	Sig. (2-tailed)	0.49	0.90	0.49	0.97	0.63	0.50	0.43	0.96	0.46	0.66

player	Total games played this season	headache	nausea	vomitting	balance
1	6	14	0	0	2
2	6	4	0	0	0
3	8	0	0	0	0
4	10	8	0	0	0
5	10	15	8	1	5
6	0	5	4	1	1
7	0	8	0	0	0
8	10	13	2	1	5
9	10	0	0	0	0
10	10	10	10	0	0
11	10	0	0	0	0
12	10	15	3	0	1
13	10	0	0	0	0
14	10	0	0	0	0
15	5	0	0	0	0
16	8	1	1	1	1
17	10	15	3	1	6
18	7	0	0	0	0
19	8	1	1	0	0
20	6	9	7	5	7
21	4	2	2	0	3
22	0	0	0	0	0
23	20	0	0	0	0
24	9	0	0	0	0
25	10	0	0	0	0
26	7	6	6	6	6
27	14	12	6	1	4
28	10	6	2	0	0
29	10	0	0	3	5
30	6	0	0	0	0
31	8	13	6	0	3
32	10	3	0	0	0
33	2	10	5	0	0
34	10	9	2	0	3
35	10	0	2	0	

dizziness	fatigue	fall sleep	sleep mor	drowsy	light	noise	irritability	saddness
0	16	0	13	7	0	0	6	0
0	0	0	0	0	0	0	0	0
0	16	0	0	0	0	0	0	9
2	12	1	3	0	0	0	0	0
1	19	5	10	2	0	0	5	0
7	7	3	0	3	0	0	1	1
0	6	7	0	0	0	0	0	0
2	11	0	6	0	3	0	6	0
0	6	0	0	0	0	0	0	0
0	10	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
3	6	1	2	9	3	5	0	0
0	0	0	0	0	0	0	0	0
0	2	0	2	0	0	0	0	0
0	2	0	0	0	0	0	0	0
1	1	12	4	1	1	1	1	4
2	7	3	5	7	2	6	1	0
0	0	0	0	0	0	0	0	0
2	3	2	0	1	3	0	2	1
7	9	8	6	7	3	5	8	5
5	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0
6	6	0	4	4	3	3	6	0
4	12	0	0	6	6	0	0	0
3	7	0	3	3	0	0	1	0
0	0	0	0	3	0	0	0	2
0	13	0	0	0	0	0	0	0
1	1	9	4	2	6	0	3	0
0	43	3	35	16	49	42	24	3
0	7	5	9	9	7	3	7	5
0	8	4	0	8	2	2	6	0
0	0	0	0	2	0	0	0	0

nervous	emotional	tingling	slowed	foggy	concentrate	remember	visual
0	1	0	3	0	3	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	3	4	0	0	1	0
1	3	4	9	7	10	0	0
3	1	5	5	5	3	1	3
0	0	0	0	0	0	0	0
2	0	0	5	14	2	0	0
0	0	0	0	0	0	0	0
0	0	24	0	0	0	0	0
0	0	0	0	0	0	0	0
1	1	1	3	17	2	2	0
0	0	0	0	0	0	0	0
0	0	0	2	0	0	0	0
10	0	0	0	0	3	11	0
1	1	1	3	1	1	1	1
0	2	0	4	8	3	9	0
0	0	0	0	0	0	0	0
0	1	0	1	2	2	0	0
6	6	6	5	8	7	6	4
0	0	0	0	0	0	1	2
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0
0	5	0	6	6	6	0	0
0	0	0	6	8	0	0	0
0	0	1	5	3	2	2	0
0	0	0	0	0	0	2	3
0	0	0	0	10	0	0	0
2	1	1	7	8	5	5	0
8	4	7	3	2	0	15	2
3	6	10	11	11	12	11	3
0	0	0	4	8	6	0	0
0	0	0	0	0	0	0	0

Appendix H

SPSS shortened names and corresponding full names of neuropsychological measures

SPSS VALUES	DATA ANALYSIS AND RESULTS VALUES	EXPLANATION OF EACH VALUE
REY	**RCF Copy**	Rey complex Figure (RCF) Copy the figure as accurately as possible
STROOPDOTS	**STROOP Dots**	STROOP Colour word test – name the colour of the dots presented as quickly as possible
STROOPDOTERRS	**STROOP Dots Error**	STROOP Colour word test – errors made when naming the dots
STROOPW	**STROOP Words**	STROOP Colour word test – name the colour of the words presented as quickly as possible
STROOPWRDERR	**STROOP Words Error**	STROOP Colour word test – errors made when naming the colours of the words
STROOPCOLOUR	**STROOP Colour**	STROOP Colour word test – name the colour of the colour words presented as quickly as possible
STROOPCOLERRS	**STROOP Colour Error**	STROOP Colour word test – errors made when naming the colours of the colour words
RAY2MR	**RCF 2 min recall**	Rey Complex Figure 2 minute recall. Recall the figure previously drawn from memory and re-draw it again as accurately as possible
RAVLTT	**RAVLT Total**	Rey Auditory Verbal Learning Test Total: the total of all words remembered out of all 5 trials
RAVLTLEARNC	**RAVLT Learn**	Rey Auditory Verbal Learning Test : the difference between trail 1 and trail 5 showing the learning curve
RAVLTRECOG	**RAVLT Recognition**	Rey Auditory Verbal Learning Test Recognition: the total real words the subject could remember when given a mixed list of real and nonsense words
RAVLTDELAY	**RAVLT 20 min delay recall**	Rey Auditory Verbal Learning Test Delay: the total number of words the subject could

		remember 20 minute later after first trial
RAVLTA1	**RAVLT Immediate**	Rey Auditory Verbal Learning Test: immediate memory – the first trail of initial words
RAVLTB	**RAVLT B1 distractor list**	Rey Auditory Verbal Learning Test: distractor list – a different list of words called out after the 5th trial of the initial list
RAVLTA6/V1	**RAVLT A1/V1 2 min delay recall**	Rey Auditory Verbal Learning Test: required to remember the first list of words 2 minutes after the 5th trial, and following the distractor list
SDMT WRITTN	**SDMT Written**	Symbol Digit Modality Test: subjects to write down the number of the related symbol as fast as possible – subject given a 60 second time limit
SDMTOR	**SDMT Oral**	Symbol Digit Modality Test: subject to call out the number of the related symbol as fast as possible – subject given a 60 second time limit – examiner writes down number
DIGITF	**Digits forwards**	Digit Forwards: subjects to repeat numbers in same as order called out by examiner
DIGITB	**Digits backwards**	Digit Forwards: subjects to repeat numbers in reverse order to called out by examiner
F / C	**COWAT F**	Controlled Oral Word Association Test (COWAT): subjects to list as many words as possible beginning with the letter F / C in 60 seconds
A / F	**COWAT A**	Controlled Oral Word Association Test (COWAT): subjects to list as many words as possible beginning with the letter A / F in 60 seconds
S / L	**COWAT S**	Controlled Oral Word Association Test (COWAT): subjects to list as many words as possible beginning with the letter S / L in 60 seconds
FAS / CFL	**COWAT FAS**	Controlled Oral Word Association Test (COWAT): the total of all three previous trials
FASAN / ANIMALS	**COWAT Animals**	Controlled Oral Word Association Test (COWAT): subjects to list as many animals as possible in 60 seconds, RULE: no birds nor fish
TRAILA	**Trail Making A**	Trail A: subjects to link letters of the alphabet with a pencil as fast as possible

TRAIL B	**Trail Making B**	Trail B: More complex task – subjects to link letters of the alphabet with corresponding numbers as fast as possible i.i. a-1-b-2-c-3-d-4 etc
RAY	**RCF 20 min delay recall**	Rey Complex Figure 30 minute delayed recall: subjects to remember and re-draw the figure they copied at the beginning of the test