Neurocognitive Effects of Head and Body Collisions on Club Level Rugby Union Players

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ABSTRACT

The objective of the study was to investigate the cumulative neurocognitive effects of repetitive concussive and subconcussive events in club level Rugby Union (hereafter rugby) during the course of one rugby season, in a combined group and individualized case-based approach. Amateur adult club level rugby players (n = 20) were compared with a non-contact control group (n = 22) of equivalent age, years of education and estimated IQ (p = > .05, in all instances), although the two groups were clearly differentiated on the basis of a history of reported concussions (p = < .05). Video analyses documented the tackling maneuvers observed amongst the players during all matches across the rugby season revealing a sobering average of more than a thousand tackles per player, excluding any contact practice sessions. Five rugby players (n = 5) who were observed to have a head jarring event were also isolated for individualized postconcussive follow-up analysis of their neurocognitive profiles. Measures included the ImPACT Verbal and Visual Memory, Visual Motor Speed and Reaction Time composites and the Purdue Pegboard. Independent and dependent statistical analyses were employed to compare the rugby versus control group neurocognitive test profiles at and between the three test intervals. Correlational analyses explored the association between concussion, tackling and neurocognitive test outcomes. Descriptive comparisons of individual neurocognitive test scores with normative data were employed for the case analyses. Taken together, the results implicated vulnerability amongst club rugby players on the motor and speeded tasks, with less robust indications on the memory tasks. While limited in terms of its small sample size, it is considered that the outcome of the study was rendered more robust by virtue of being methodologically multifaceted with heuristic implications for future research studies in the area. The novel inclusion of tackling data as well as fine-tuned case analyses, were of particular relevance in that regard. The results add to a growing body of literature that implicates deleterious neurocognitive effects in participants of a sport such as rugby due to repetitive head jarring incidents that are intrinsic to the game.

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CHAPTER 1
INTRODUCTION

This chapter reviews the underlying motivation for this study with specific reference to club-level Rugby Union (hereafter Rugby) in South Africa and the management of brain injury. This is followed by the rationale and broad research questions of the present study, concluding with the thesis structure. Throughout the thesis the rules of APA Style, detailed in the *Publication Manual of the American Psychological Association*, (6th ed., 2010) have been applied.

1.1 INTRODUCTION

Trauma to the head, in any form, is the most common cause of brain injury and is the inevitable consequence of complex biochemical and neurochemical cascade mechanisms directly and immediately activated by a traumatic insult. It incorporates clinical and pathological constructs, and can be defined as a complex pathophysiological process based on temporal neuronal dysfunction affecting the brain and is induced by biomechanical forces. The mechanical forces of linear and rotational head accelerations/decelerations are hypothesized to be the primary risk factors for Mild Traumatic Brain Injury (MTBI), a phenomenon that is commonly referred to as concussion or concussive brain injury, especially in the sports arena. While it is accepted that MTBI may be associated with causation other than concussive brain injury, for the purposes of this thesis these terms will be used interchangeably. Both direct and inertial loading of the head may result in a concussive brain injury and typically results from a direct or indirect impact to the head, face, neck, or elsewhere on the body with an 'impulsive' force. This frequently causes disruption of brain centres responsible for heart rate, breathing, and consciousness, and typically results in a spectrum of neuropsychological and neurophysiological changes that may be temporary or permanent. (Aubry, Cantu, Dvořák et al., 2002; Barth, Alves, Ryan, Macciocchi, Rimel, Jane & Nelson, 1989; Frencham, Fox & Maybery, 2005; Guskiewicz & Mihalik, 2011; Lezak, Howieson & Loring, 2004; Lovell, Collins, Iverson, Johnston & Bradley, 2004; McCrory et al., 2013, 2009; Signoretti, Vagnozzi, Tavazzi & Lazzarino, 2010).

Trauma to the brain produces alterations at various levels of cognitive functioning (Collins, Lovell & McKeag, 1999; Erlanger, Kutner, Barth & Barnes, 1999), and there has been an increased interest in contact sports into the acute, chronic and cumulative deleterious neurocognitive effects of repeated MTBI (Barth et al., 1989; Grindel, Lovell & Collins, 2001; Lezak et al., 2004; Lovell, Collins, Iverson, Johnston & Bradley, 2004 ; McCrea, Guskiewicz, Marshall et al., 2012; McCrea, Prichep, Powell, Chabot & Barr, 2010; Rabadi & Jordan, 2001). In the acute condition, cognitive sequelae usually improve and/or resolve within three months post-injury, and those effects that persist for longer than three months can be considered chronic (i.e. relatively permanent) (Barth et al., 1989; Bernstein, 2002; Frencham, Fox & Maybery, 2005; Lezak et al., 2004; Lovell, Collins, Iverson, Johnston & Bradley, 2004; Reitan & Wolfson, 1999; Vanderploeg, Curtiss & Belanger, 2005). Therefore, for the purposes of this thesis, the terms acute and chronic will pertain to the time frames of within three months and longer than three months, respectively. In both the acute and chronic conditions, MTBI typically leads to impairments in memory, attention, planning, cognitive flexibility, reaction time and processing speed (Barth et al., 1989; Eckner, Kutcher, Broglio & Richardson, 2013; Lezak et al., 2004; Lovell, Collins, Iverson, Johnston & Bradley, 2004).

There is growing evidence to support the cumulative deleterious neurocognitive effects of repeated MTBI, as seen in contact sport research such as boxing, ice hockey, and a cluster of the football codes including soccer, American football, Rugby League and Rugby (Dawodu, 2009; Field et al., 2003; Gaetz & Bernstein, 2001; Gardner, Shores & Batchelor, 2010; Grindel, Lovell & Collins, 2001; Guskiewicz et al., 2005; Hinton-Bayre & Geffen, 2002; Iverson, Gaetz, Lovell & Collins, 2004; Lovell & Collins, 1998; Matser, Kessels, Jordan, Lezak & Troost, 1998; Matser, Kessels, Lezak, Jordan & Troost, 1999; Pettersen & Skelton, 2000; Shuttleworth-Edwards, Border, Reid & Radloff, 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith & Radloff, 2008; Shuttleworth-Edwards & Whitefield, 2007; Webbe & Ochs, 2003; Witol & Webbe, 2003). In addition, a series of studies demonstrate long-term deleterious neurocognitive deficits in association with increased numbers of MTBIs (Gardner, Shores & Batchelor, 2010; Killiam, Cautin & Santucci, 2005; Moser, Schatz & Jordan, 2005).

There are still limited published studies on the neurocognitive effects of rugby-related MTBI, and it appears that only nine studies have been published (Farace, Ferree, Hollier, Barth & Shaffrey,

2003; Gardner, Shores & Batchelor, 2010; Pettersen & Skelton, 2000; Shuttleworth-Edwards, Border, Reid & Radloff, 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Radloff, Whitefield-Alexander, Smith & Horsman, 2013; Shuttleworth-Edwards, Smith & Radloff, 2008; Shuttleworth-Jordan, Puchert & Balarin, 1993; Thornton, Cox, Whitfield & Fouladi, 2008). Exposure to multiple mild brain injuries is a characteristic feature of rugby at all levels of participation (school, university, club, provincial and national). In accordance with this a body of research supports the notion that the severity and duration of functional impairment is increased with repeated concussive and subconcussive (microtraumatic brain injuries) episodes in conjunction with the postulated cumulative effects resulting in chronic neuropsychological sequelae (Erlanger et al., 1999; Iverson, Gaetz, Lovell & Collins, 2004; Killiam, Cautin & Santucci, 2005; Rutherford, Stephens & Potter, 2003; Macleod, 1993; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007a). Subconcussive injuries are proposed as being "*events similar to those giving rise to concussion but involving smaller impact forces that operate below the threshold necessary to produce symptoms"* (Shuttleworth-Edwards & Whitefield, 2007).

In contrast to the above research, a study on high school and collegiate American football athletes argues that evidence for prolonged deleterious neurocognitive effects of MTBI, although in the absence of controlled demographic variables, is not convincing (Solomon, Ott & Lovell, 2011), and a study of a large sample of high school and collegiate athletes found no evidence of residual impairments on performance-based measures of cognitive functioning (McCrea, Guskiewicz, Randolph et al., 2012). Therefore, based on both relatively robust and a minority of weaker studies, the jury is still out on the issue whether or not there might be long-term deleterious effects arising out of participation in contact sports (Shuttleworth-Edwards & Whitefield, 2007). Consequently, there is a growing consensus of opinion that sustains the need for vigilant identification, assessment and management of MTBI on an individual basis at all levels of play (American Academy of Neurology, 2013).

General assessment issues related to the specific field of neurocognitive assessment are vital in investigating possible cumulative effects of concussive and subconcussive events and include the effect on neurocognitive functions that is pertinent to MTBI. Several well-validated tests, as reviewed by McCrea, Iverson, Echemendia, Makdissi & Raftery (2013), are appropriate for use

in the assessment of acute concussion in the competitive sport environment. Many consider neuropsychological assessment to be a sensitive method for the evaluation of cognitive effects following concussion (Baroff, 1998; McCrea, Kelly, Randolph, Cisler & Berger, 2002).

A number of researchers reviewed the advantages and disadvantages of different neuropsychological assessment modalities and multi-modal assessment paradigms and validated the clinical application of computerised neurocognitive assessment over traditional paper-and-pencil tests (Collie, Makdissi, Maruff, Bennell & McCrory, 2006; Collie, Darby & Maruff, 2001; Collins, Echemendia & Lovell, 2004; Iverson, Lovell & Collins, 2002a; Schatz, Pardini, Lovell, Collins & Podell, 2006; Schatz & Zilmer, 2003). Accordingly, in recent years, a number of computer-based systems have been developed for concussion management, including Automated Neuropsychological Assessment Metrics (ANAM), CogState Sport (previously termed CogSport), Concussion Resolution Index (HeadMinder) and Immediate Post-concussion Assessment and Cognitive Testing (ImPACT) (Aubry et al., 2002; Mayers & Redick, 2012; Pretz, 2007; Schatz & Browndyke, 2002; Schatz & Zilmer, 2003; Shuttleworth-Edwards & Border, 2002). These have been comprehensively reviewed and the authors concluded that there is a need for additional research prior to the consideration of computerised neuropsychological testing as a routine standard in concussion management (Randolph, McCrea & Barr, 2005).

ImPACT was identified for the purpose of the present study, due to it being widely used for evaluating sports concussion in the sports arena, it being neuropsychologically and technically sophisticated, and measuring different aspects of cognitive functioning (Iverson, Lovell & Collins, 2002a; Shuttleworth-Edwards & Whitefield-Alexander, 2013). A recent study on computerized neurocognitive testing for the management of sport-related concussions of high school athletes revealed that the vast majority of the respondent schools (93%) used ImPACT (Meehan III, d'Hemecourt, Collins, Taylor & Comstock, 2012).

Published research studies utilising ImPACT, which was designed to simultaneously evaluate multiple cognitive domains, have shown to be sensitive to the effects of concussion (Collins & Hawn, 2002; Collins, Iverson, et al., 2003; Lovell & Collins, 2002; Lovell et al., 2004; Schatz et al., 2006). These research studies have been largely conducted on contact sports in the United States of America and more particularly on the National Football League, National Hockey

League and other athletes. In South Africa research studies including ImPACT have been conducted on rugby at the school, university and professional level (Clark, 2010; Shuttleworth-Edwards, Smith & Radloff, 2008) but to the author's knowledge, no research has been conducted as yet in respect of adult amateur rugby players at club level. The ImPACT test, being mouse-driven, necessarily calls upon hand-motor dexterity, however the composite scores incorporate several tasks and hand-motor speed *per se* may be 'diluted' and therefore warrants investigation in its own right.

Research on MTBI and hand-motor functioning in the sports context appears to be minimal, and the only research done on the effect of MTBI on motor speed was done on soccer players utilizing the Finger Tapping Test (Baroff, 1998). Measures of hand-motor reaction speed, like the Purdue Pegboard, have been shown to provide accurate indexes of cognitive changes following brain injury (Lezak et al., 2004). Shuttleworth-Jordan et al. (1993) investigated the acute and chronic effects of rugby-related MTBI and incorporated the Purdue Pegboard test. Therefore, it was decided that the present research would investigate the cumulative neurocognitive effects of frequent head and body collisions on club level rugby players using the computer-based assessment instrument (ImPACT) together with the traditional neurocognitive tool that measures hand-motor speed (Purdue Pegboard).

It would appear that there are no studies available in contact sport, with the exception of limited soccer studies, that directly investigate the frequency of player-to-player and player-to-ground collisions, and the consequent neurocognitive outcome. In all the soccer studies, as reviewed in and criticised by Rutherford, Stephens & Potter (2003), the frequency of headings (head-to-ball collisions) on neuropsychological impairment, were limited to self-reported, subjective estimates by the soccer players. From the critique in soccer studies, a more useful approach, than self-estimates alone, would be to combine the self-reported number of collisions, sideline record keeping of collisions with the rating of collisions based on video recordings of the games.

Notational video analysis involves the systematic analysis of game footage and this will assist in the external identification of the frequency of collisions and its relationship to neurocognitive outcome. Gabbett, Jenkins & Abernethy (2011) video recorded and documented the frequency of physical collisions and incidence of contact injury in professional Rugby League, and King,

Hume & Clark (2011) utilised video analyses for the nature of tackle-related injuries for a single team in professional Rugby League. In college football the number of head hits in all players was monitored by movie review (Marchi et al., 2013) and tackle characteristics in school, college and professional rugby were observed and coded from video (McIntosh, Savage, McCrory, Fréchède & Wolfe, 2010). Video-based match analysis has previously been used to assess injury situations in professional soccer (Andersen, Larsen, Tenge, Engebretsen, & Bahr, 2003; Arnason, Tenga, Engebretsen & Bahr, 2004; Hawkins & Fuller, 1998; Rahnama, Reilly & Lees, 2002).

All of these studies did not indicate the number of tackles over one season on club level rugby players *per se*. Therefore, the present study incorporated, in addition to the neurocognitive assessment, video-based game analyses to investigate head and body collisions and the possible contributing cumulative effect on concussive and subconcussive events. It is anticipated that this multiple mode of recording collisions will identify more incidences of MTBI than would normally be reported and/or diagnosed by players, coaches and medical staff.

The apparent neuropsychological recovery observed following brain injury may be explained in terms of the concept of human beings having a functional reserve or a threshold for the effects of brain injury (Blessed, Tomlinson & Roth, 1968; Satz, 1993; Stern, 2002). The notion of having a reserve is used in the medical field as well as a number of rugby-related MTBI studies to evaluate and interpret findings (Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008). Brain Reserve Capacity Theory as explicated by Satz (1993), and further elaborated on by Stern (2003) is a heuristic model to explain individual fortification from or susceptibility to clinical symptoms associated with brain injury. Broadly, the theory indicates that individuals uniquely possess the capability to withstand and compensate for mild, traumatically induced neuronal loss. However, when an individual's cognitive reserve is depleted beyond a certain threshold, such as due to concussive and subconcussive events, certain neurocognitive deficits emerge (Jordan, 1997; Randolph, 2001; Satz, 1993; Stern, 2003; Weight, 1998). Explications of the brain reserve and cognitive reserve concept (Barth et al., 1996; McCrea, 2008; Satz, 1993; Stern, 2003) proposes a hypothetical, multifactoral construct that correlates with unique individual factors such as premorbid health, underlying psychopathology, cognitive ability, age, general intelligence, educational level,

severity of injury, the existence of postconcussive symptoms and psychological reaction to the injury.

Based on the abovementioned empirical rationale, and a cognitive reserve conceptual framework, both an independent cross-sectional and a dependent prospective design were employed for the present study. This incorporated group analyses of club level rugby players and comparative club-level non-contact sports controls (predominantly cricket players). A case study investigation of five individuals, who were identified with possible concussive injury by means of video analysis, was also included. The objective of this study is to address the following two broad research questions:

1) Whether or not rugby players of adult club level rugby suffer chronic neurocognitive sequelae as demonstrated on the ImPACT neurocognitive screening test and the Purdue Pegboard test, as a result of long-term exposure to concussive and subconcussive events associated with playing rugby and/or the additional overlay of undisclosed concussive and subconcussive events occurring over one rugby season;

2) Whether or not the number of tackling collisions for rugby players at adult club level rugby over a single rugby season can be linked to evidence of neurocognitive vulnerability established on the basis of outcome on the ImPACT neurocognitive screening test and the Purdue Pegboard test, as a result of long-term exposure to concussive and subconcussive events associated with playing rugby and/or the additional overlay of undisclosed concussive and subconcussive events occurring over one rugby season.

1.2 THESIS STRUCTURE

The thesis consists of a theoretical framework and literature research, followed by the methodology, results, references and appendixes. For ease of reference, all tables and figures are prefixed with the chapter number in order to simplify their mention in the thesis.

Chapter one (Introduction). This chapter offers the background and introduction to this thesis.

Chapter two (Traumatic Brain Injury). This chapter discusses Traumatic Brain Injury (TBI) in general in terms of definitions, types, mechanisms and biomechanisms, neurophysiology, and classification of TBI in order to put MTBI in context.

Chapter three (Mild Traumatic Brain Injury). This chapter encompasses extensive literature review on the neurophysiology of Mild Traumatic Brain Injury (MTBI) in general, classification, mechanisms and biomechanisms, neuropsychological consequences, neurocognitive recovery and risk factors influencing outcome of MTBI. The influence of Brain Reserve Capacity is also highlighted.

Chapter four (MTBI in contact sports). This chapter focuses on MTBI in contact sports, with reference to the epidemiology, mechanism of MTBI and neurocognitive consequences in four identified contact sports in the football codes, namely soccer, American football, Rugby League and Rugby.

Chapter five (Assessment and management of sports-related MTBI). This chapter focuses briefly on the medical and more intensively on the neurocognitive assessment and management of MTBI.

Chapter six (Methodology). This chapter outlines the methodology of the empirical investigation. This includes the procedure followed by a description of the participants, demographic data, the procedure, the measures and administration, and the data analyses.

Chapter seven (Group Analyses). This chapter presents the results of the group analyses pertaining to the Rugby Group and the comparative Non-Contact Sports Control Group.

Chapter eight (Individual Player Analyses). This chapter presents the results of the Individual analyses pertaining to the individual players identified for case analyses.

Chapter nine (Discussion). This chapter concludes the thesis with a discussion. This is followed by the reference list and appendixes.

CHAPTER 2

TRAUMATIC BRAIN INJURY

This chapter reviews Traumatic Brain Injury in general, including a description of focal and diffuse brain injuries and the mechanisms and biomechanics of injury. This is followed by the classification of the severity of traumatic brain injury, with reference to Loss of Consciousness (LOC) and Posttraumatic Amnesia (PTA).

2.1 TRAUMATIC BRAIN INJURY

Trauma to the head, in any form, is the most common cause of substantial and traumatic physical, motor, cognitive, memory, and psychosocial deficits and/or disability (Bailes & Hudson, 2001; Centers for Disease Control and Prevention, 2003; Rees, 2003; Thurman, Alverson, Dunn, Guerrero, & Sniezek, 1999). Motor vehicle and motorcycle accidents are the most frequent cause of head injuries, followed by falls, occupational injuries, recreational accidents, assaults, sports collisions and/or being accidentally struck by objects or bodies (Asikainen, 2001; Bernstein, 1999; Cassidy, Carroll, Peloso et al., 2004; Evans, 2004; National Centre for Injury Prevention and Control, 2007; Pettersen & Skelton, 2000; Rassovsky, Satz, Alfano, Light, Zaucha, McArthur & Hovda, 2006; Weight, 1998; Zhang, Yang & King, 2004). The extrapolation of incidence figures to the world population is complicated by incongruence in injury reporting and diagnosis but suggest that every year, 54 to 60 million people endure a brain injury and some 2.2 to 3.6 million of these sustain a moderate or severe TBI (Anderson, 2012b).

Traumatic Brain Injury (TBI) incorporates clinical and pathological constructs, and can be defined as a multifaceted pathophysiological process affecting the brain, that is induced by biomechanical forces. *Head Injury* and *Brain Injury* are two distinct entities that are often used interchangeably, but are not necessarily, related. Traumatic Brain Injury (TBI) is a more specific term than Head Injury. A head injury is best defined as an injury that is clinically evident upon physical examination and is recognised by the presence of observable signs of injury, e.g. abrasions, contusions and lacerations (Lezak et al., 2004; Von Holst & Cassidy, 2004). TBI is a

nondegenerative, noncongenital insult to the brain caused by an external mechanical force and can occur without external indications of trauma. TBI is serious and potential devastating, with symptoms that encompass an altered state of consciousness that may lead to temporary or permanent neuropathological, biochemical, neuropsychological and behavioural changes (Dawodu, 2009; Jay, Goka & Arakaki, 1996; Tellier, Malva, Cwinn, Grahovac, Morrish & Brennan-Barnes, 1999). Therefore, for the purpose of this study, the term 'traumatic brain injury' (TBI) is used despite reference to the condition in citing literature as 'head injury'.

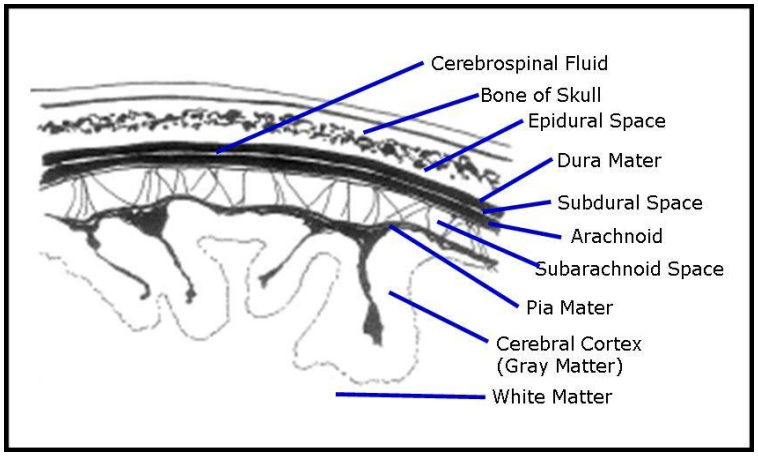
TBI involves ionic and metabolic events, from which damaged cells may recover, or degenerate and/or die and furthermore results in neuronal loss in the cortex, hippocampus, cerebellum and thalamus. Even in the absence of degenerative changes there is a complex pathophysiological reaction that can lead to persistent dysfunction with identifiable cognitive and neurobehavioural deficits following brain injury. Brain injuries transpire when the tensile effects on axons or parenchymal deformations do not exceed the level where structural damage occurs. Biochemical and neurochemical perturbations cause shearing in the axons and in the small vessels of the brain, and appear to be most disparaging to brain tissue (Baker & Patel, 2000; Signoretti et al., 2010). Symptoms appear in different intensities and depend on factors such as location of injury, the intensity and angle of the impact as well as the number of previous injuries (Boden, Kirkendall & Garrett, 1998; Cantu, 1992).

Characteristically the skull sustains and absorbs the greatest impact forces and resultant kinetic energy. However, slow blood accumulation in the epidural space may result in a relatively asymptomatic presentation until the underlying compressed brain leads to neurologic dysfunction, brain herniation and in some instances even death.

The brain is condensed within the rigid and inelastic calvarium (the upper dome-like portion of the skull, excluding the lower jaw) and is buoyant in cerebrospinal fluid (a cushioning and protective shock absorber), with several dural attachments to bony ridges that make up the interior contours of the skull (Figure 2.1). A tough outermost layer, called the dura mater ensures that the delicate brain is protected and can withstand quite substantial translation and deformation (Bigler, 2003; Cantu, 1992; Crippen, 2009; Darby & Walsh, 2005; Guskiewicz, Bruce, Cantu et al., 2004; Noakes & Du Plessis, 1996). This helps prevent the soft, fragile, inherently

inhomogeneous and anisotropic brain tissue and intracranial contents from excessive movement. If not for this protection, uncontrolled movements may induce inertial forces and result in various distinct compressive and tensile stresses, as well as shearing and tearing of the brain substance whenever the head is suddenly accelerated, decelerated or rapidly rotated (Cantu, 1992, 1996; Darby & Walsh, 2005; Gilchrist, 2004; Zhang, Yang & King, 2004).

Figure 2.1 Meninges of the Brain



2.2 NEUROPHYSIOLOGY OF TRAUMATIC BRAIN INJURY (TBI)

Brain injuries can be seen as a clinical syndrome resulting from a combination of predominantly neural or vascular events brought on by mechanical forces. According to Gennarelli & Graham (1998) the spectrum of brain injuries can range from merely focal to diffuse. While neuronal death is associated with focal injuries due to contact forces, death of oligodendrocytes (glial cells that produce an oily substance called myelin that wraps around axons in layers) may be a hallmark of diffuse brain injury. The basic pathophysiology of focal brain injury following a global insult is slightly less complex than the development of diffuse damage to the axons and the dendrites that results in widespread neuronal dysfunction (Bailes & Hudson, 2001; Gaetz, 2004).

2.2.1 Focal Brain Injury

Focal brain injury occurs in the form of scalp injury, skull fracture, and surface contusions. Contusions are areas of focal cortical injury that results from direct external contact forces or

when the brain strikes the intracranial surface of the skull, as well as in the event of frank disruptions of primary localised vascular and neuronal brain tissue. It also includes intracranial haematoma formation in extradural, subarachnoid, subdural and intracerebral areas (Table 2.1), which in severe cases can result in a coma caused by brain shift, herniation and/or brain stem compression.

Depending on the location and degree of impact, several types of primary brain contusions can occur. Contusions typically occur at the apex of gyri (highest part on the ridges of the brain surface) and appear as either multiple punctuate haemorrhages or streaks of haemorrhage, with an eventual progression of bleeding into the adjacent white matter (Gennarelli & Graham, 1998). Cortical-subcortical contusions result from any combination of frontal, parieto-temporal or occipital impacts. The force to the head is applied over a short period and is focally concentrated to the skull and brain surface, not necessarily implicating the reticular pathways, and thus a small focal cortical contusion may not alter consciousness (Rees, 2003). It is the acceleration induced by the impact, and not the head contact itself that result in focal brain injury.

In addition, the brain manifests with a more general response to injury that is generalized brain swelling within the intracranial compartment, but only small increases in volume can be tolerated before pressure rises dramatically (Crippen, 2009). The pressure inside the brain rises even without active bleeding from arteries or veins into the extradural or subdural compartments and results in vasomotor paralysis (pressure rises above a critical value resulting in loss of normal blood flow to the brain). This excessive pressure causes cerebral oedema, which leads to an irreversible and fatal increase in intracranial pressure, followed by fatal herniation of structures in the brainstem (Gaetz, 2004; Fisher & Vaca, 2004; Zhang, Yang & King, 2001a, 2001b).

2.2.2 Diffuse Brain Injury

Diffuse brain injury is characterized by extensive, generalized damage to the white matter of the brain and includes Diffuse Axonal Injury (DAI), hypoxic-ischemic damage and vascular injury (Smith & Meaney, 2003). Although termed ‘diffuse’, it can more accurately be described as ‘multifocal’, appearing throughout the deep and subcortical white matter, and is particularly widespread in midline configurations, including the splenium of the corpus callosum. Diffuse

brain injury and the level of immediate neurologic impairment correlate with the extent and severity of widespread axonal damage, which manifests as strains on nodal and paranodal regions and the distribution of focal lesions in the axonal components. It typically results in widespread mechanical effects and neurological dysfunction associated with swollen, beaded and varicose axonal, neuronal and microvascular fibres that lead to altered membrane potential and even depolarisation (Gilchrist, 2004; Raghupathi, 2004; Zhang et al., 2001a). This induces relatively low-energy damage affecting a multitude of distinct regions of neural tissue. For example, severe deceleration forces related with a high speed motor vehicle accident and with no head impact may produce a pattern of predominantly diffuse injury, with several small traumatic foci related to petechial haemorrhage or the tearing of small blood vessels.

The damage that accompanies diffuse TBI consists of minute lesions and lacerations scattered throughout the brain substance. In addition to the cell damage, there may also be damage to blood vessels resulting in bleeding either into the epidural or subdural spaces or within the brain substance itself (Table 2.1). This can cause several types of intracranial haemorrhages, including the following:

1) Epidural haematoma, which occurs from, impact loading to the skull with associated laceration of the dural arteries and blood collection can cause rapid neurologic deterioration;
2) Subdural haematoma that tend to transpire with injuries to the cortical veins or pial artery with an associated high mortality rate;
3) Intracerebral haemorrhage that occurs within the cerebral parenchyma secondary to lacerations occurring with extensive cortical contusion;
4) Intraventricular haemorrhage that tends to happen in the presence of extremely severe TBI; and
5) Subarachnoid haemorrhage that occur because of lacerations to the superficial micro vessels in the subarachnoid space.

Table 2.1 Focal and Diffuse Brain Injury

Focal	Diffuse
Scalp injury	Diffuse Axonal Injury (DAI)
Skull fracture	Diffuse vascular injury
Contusions	Hypoxic-Ischemic damage
Intracranial Haematoma	Intracranial Haemorrhage
Extradural	Epidural Haematoma
Subarachnoid	Subdural Haematoma
Subdural	Intracerebral Haemorrhage
Intracerebral	Intraventricular Haemorrhage
	Subarachnoid Haemorrhage
Oedema	
Haemorrhage	Haemorrhage

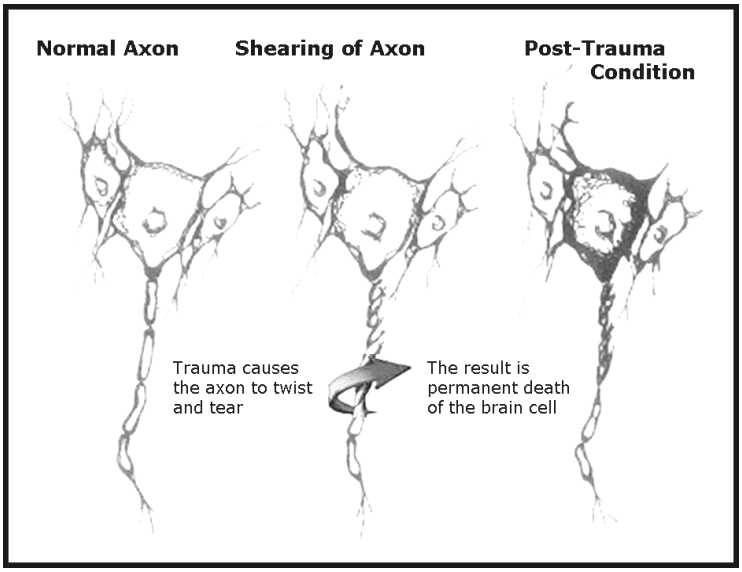
(Asikainen, 2001)

DAI is caused by inertial forces, but it is the contact forces that often cause the necessary levels of acceleration of head movement to create pressure gradients and to induce tentorium (the extension of the dura mater that separates the cerebellum from the inferior portion of the occipital lobes), falx (a strong, arched fold of the dura mater that descends vertically in the longitudinal crevice between the cerebral hemispheres), tensile and compression strains of axonal tissue and the shearing of white-matter fiber tracts.

DAI, therefore, is equally reliant on the magnitude and the rate of strain during TBI and only requires rapid acceleration/deceleration of the head, which results in the rapid flexion-extension movement of the neck. This occurs when the rate of the skull deceleration is exceedingly rapid and when the head both decelerates and rotates. Under these conditions, the protective capabilities of the brain's own protective mechanisms are exceeded, and the movement of the inert brain inside the skull cannot be slowed down sufficiently. This produces irreversible injury to a large number of nerve cells at the site of impact and manifest in sheer strains within the cranial vault (Figure 2.2). Subsequently this may lead to sheering of neurons and blood vessels occurring principally in the brainstem that joins the cerebral cortex to the spinal cord (Gaetz, 2004; Levin, Amparo, Eisenberg, Williams, High, McArdle & Weiner, 1987). The force of the impact (kinetic energy) transmits a rapid acceleration-deceleration to the brain causing it to move in a linear direction either sagittal (front-to-back), lateral (side-to-side), oblique (falling in

between) or, in the severe instances, in a rotational direction (Guskiewicz et al., 2004). The direction of rotation influences the severity of the brain injury (Elson & Ward, 1994; Ommaya, 1996) and the direction of impact is a crucial factor in brain injury tolerance (Gennarelli, Thibault, Tomei, Wiser, Graham & Adams, 1987; Hodgson, Thomas & Khali, 1983). Zhang et al. (2001) demonstrated that the human head has a lower tolerance from a lateral impact in comparison to a frontal impact with the same energy.

Figure 2.2 Mechanism of Axonal Injury



The determination of the acute severity of a brain injury is crucial to the assessment of the individual and a classification system was developed to determine the progressive grades of DAI. Adams, Doyle & Ford *(*1989) described three severity grades of TBI ranking with particular reference to localized injuries. Grade I is characterized by axonal injury in the white matter of the parasagittal cerebral hemispheres, corpus callosum, brain stem and cerebellum; Grade II refers to focal lesions in the corpus callosum, and Grade III by added focal lesions in the dorsolateral quadrants of the rostral brain stem. Gaetz (2004) briefly summarized grades I and II as involving cortical-subcortical disconnection, grades II and III as involving cortical-subcortical and diencephalic disconnection, with grades IV and V involving cortical-subcortical, diencephalic and mesencephalic disconnection (Table 2.2).

Table 2.2 Progressive Grades of Diffuse Brain Injury

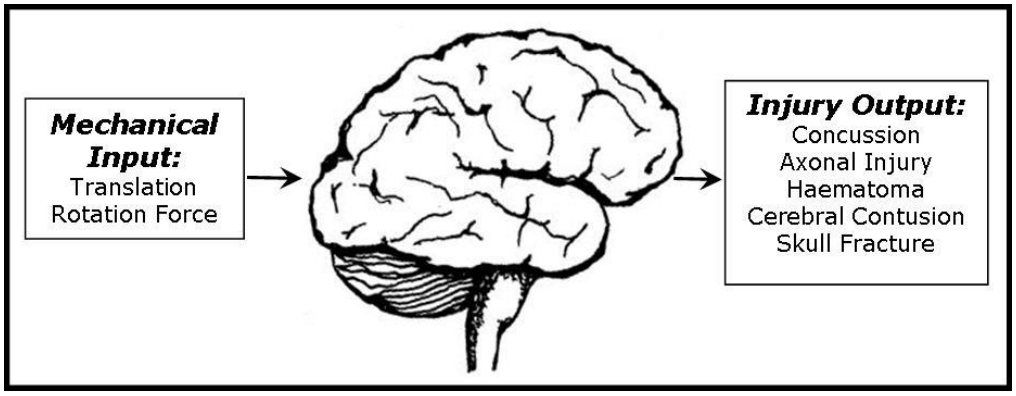
Grade	Lesion	Disconnection
I	Cerebral hemispheres, corpus callosum, brain stem and cerebellun	Cortical-subcortical
II	Corpus callosum	Cortical-subcortical and diencephalic
III	Brain stem	
IV		Cortical-subcortical, Diencephalic and Mesencephalic
V		

(Adams et al., 1989; Gaetz, 2004)

2.3 MECHANISMS AND BIOMECHANICS OF TBI

TBI is divided into two subcategories: (i) a primary injury, which is induced by a mechanical force occurring on impact, and (ii) a secondary injury, which is not mechanically induced and may demonstrate long-term effects. TBI can further be classified based on two principal *mechanisms* of injury, namely: 1) the contact phenomena or "impact loading", and 2) the rotational acceleration force or "impulsive loading" (Dawodu, 2009; Poirier, 2003; Uzzell, 1999) (Figure 2.3). Injury to the orbital frontal cortex is particularly common following TBI and the mechanism of injury typically includes generalized lesions throughout the brain, with or without localized damage such as abrasions, contusions and lacerations to tissue on the inferior aspect of the frontal and anterior temporal lobes (Darby & Walsh, 2005; Lezak et al., 2004; Varney & Menefee, 1993).

Figure 2.3 Mechanism of Traumatic Input-output Injury



In the context of TBI, the term "impact" typically indicates an injurious blow that makes direct contact with the head. Direct impact refers to a local disturbance at the site of impact (fracturing, penetration, perforation) and the initiation and propagation of transient stress waves (dilational, shear, tensile and compressive) in the intracranial tissues and skull. Indirect impact, on the other hand, refers to an impact that sets the head in motion without directly striking it. This type of impact accelerates the skull-brain system and damage appears to be due to a rapid sequence of events beginning with the inward molding of the skull at the point of impact and compensatory out bending followed by rebound effects as a response to the mechanical impact. This can be related to the mechanism of coup and/or contre-coup rebounding of the brain within the cranial vault (Rangel-Castilla, Gasco, Hanbali & Salinas, 2008), and will be discussed in more detail in Chapter 3.

TBIs are described as either an open injury (invasive or penetrative) or a *closed* injury (non-invasive or non-penetrating) and can result from direct and/or indirect impact events (Guskiewicz & Mihalik, 2011; Nicholl & LaFrance, 2009). An *open* invasive head injury, as the name suggests, involves penetration of the skull and/or dura. A *closed* non-invasive head injury, describes an insult to the head that does not penetrate the skull or any of the meninges but has primary consequences such as bleeding or swelling of the brain or damage to the brain's surface following impact (Gilchrist, 2004; Lezak et al., 2004; Martin, 1998).

The actual movement of the brain during an impact is influenced by the magnitude and direction of the applied force – more force means more injury. Basic physic principles explain how significant forces can result in brain injury when the head or body is rapidly accelerated and/or decelerated. The dissipation of mechanical forces often leads to brain injury, and movement of any kind can be based on Newton's Laws of Uniform Motion (Cantu, 1986; Hamill & Knutzen, 1995; McKenzie, Hodge & Sleivert, 2000; Young, 1992), and will be discussed in more detail in Chapter 3.

2.4 CLASSIFICATION OF TRAUMATIC BRAIN INJURY SEVERITY

TBI can be measured on a continuum from mild to severe and ranges from the surface of the brain inward with increasing amounts of damage at each level of depth as the biomechanical forces of impact increase. The range of TBI severity begins with bumps so mild as to leave no behavioural or cognitive traces with cerebral functions altered to varying degrees but with no apparent effects. However, the linear model also predicts increasing grades of neuropathological and neurobehavioural sequelae. At the other end of the severity continuum, there may be Diffuse Axonal Injury (DAI) when shear stress/strain exceeds the tissue injury threshold resulting in prolonged comas or permanent brain damage. Severity criteria, in other words, should be able to indicate a threshold below which no loss of function occurs and a ceiling beyond which irreversible changes in brain function can occur.

The purpose of classification of brain injury severity is:

1) to manage the acute stage of TBI;
2) to determine possible complications;
3) to determine potential for recovery, and
4) to determine the inter-relation of injury and subsequent sequelae.

Templer, Hartlage & Cannon (1992) confirm the consistency of evidence that head trauma can make the individual's brain more sensitive to subsequent trauma. The nature and the pathophysiological processes set in motion by TBI presents with a wide range of severity and clinical and experimental evidence suggests temporal neuronal dysfunction (Dikmen & Levin, 1993; McCrory & Johnston, 2002; Signoretti et al., 2010). Furthermore, the ambiguity in the classification of head injury severity is a likely source of inconsistencies and may be the reason why recovery patterns cannot be predicted. Gale, Johnson, Bigler & Blatter (1995) established a correlation between the amount of damage and injury severity, and Bigler (2003) reiterates that this fact demonstrates that as severity of injury increases, the brain or hippocampal volume decreases and cerebral atrophy increases, indicating that structural lesions should also be considered.

There is a definite need to group differences in the severity of head injury according to the individual's risk level and complication rates. Numerous grading systems were developed to determine the severity of injury and to predict outcome and include the assessment of various TBI parameters incorporating loss of consciousness (LOC), and posttraumatic amnesia (PTA).

2.4.1 Glasgow Coma Scale (GCS) and the Head Injury Severity Scale (HISS)

The need for a universally accepted classification system for TBI based on the presence, degree and duration of altered consciousness led to the development of the Glasgow Coma Scale (GCS) in 1974. This was the first attempt to create a standardized clinical scale that allowed for reliable neurologic assessment and to facilitate inter-observer communication in a clinical setting (Teasdale & Jennett, 1974). The GCS is based on neurological responses and appears to be the most commonly used rating scale for assessing and grading the severity of TBI (Arciniegas, Anderson, Topkoff & McAllister, 2005; Petchprapai & Winkelman, 2007). The GCS uses a single linear measure of injury severity, namely conscious state, which permits 120 possible mathematical combinations of eye, verbal and motor scores. The severity can be classified according to subjective estimates by relatively inexperienced healthcare providers regarding the duration of LOC and PTA, which are both transient sequelae of closed head injury. It is imperative to note that an isolated GCS score is of limited value and that it does not have prognostic value, while the use of serial GCS scores can be of clinical value.

The GCS score, with its associated time conditions for loss of consciousness (LOC), provides structure in decision-making with regard to injury severity (Ingebrigtsen, Romner & Kock-Jensen, 2000; Uzzell, 1999). The Head Injury Severity Scale (HISS), developed by Stein & Spettell (1995), expanded the spectrum of grades of head injury severity, based on the GCS, into four subgroups: Minimal, Mild, Moderate and Severe (Table 2.3).

Table 2.3 Grading Scales for Head Injury Classification (Incorporating GCS)

Head Injury Grade	Clinical Characteristics
Minimal	GCS = 15, no loss of consciousness (LOC)
Mild	GCS = 13–15, brief (< 5 minutes) LOC or post-traumatic amnesia (PTA) within 24 hours of injury or impaired alertness or memory
Moderate	GCS = 9-12, LOC ≥ 5 minutes or PTA within 1 to 6 days of injury or focal neurologic deficit
Severe	GCS = ≤ 8, or PTA 7 or more days from injury

(Anderson, 2012; Asikainen, 2001; Stein & Spettell, 1995)

The AVPU Scale (Alert, Voice, Pain, Unresponsive) is a simplification of the 13 possible outcomes on the Glasgow Coma Scale and assesses a patient's response in three measures - Eyes, Voice and Motor skills (McNarry & Bateman, 2004). Table 2.4 indicates four possible recordable outcomes.

Table 2.4 AVPU Scale

Outcome	Description
Alert	Fully awake but not necessarily orientated. Spontaneously opens eyes, responds to voice (although may be confused) and will have bodily motor function
Voice	Responds when talked to. Response could be as little as a grunt, moan, or slight move of a limb when prompted
Pain	Response (withdrawal from pain or reflex response) to a painful stimulus such as sternal rub or pinching
Unresponsive	No Eye, Voice or Motor response to voice or pain

(McNarry & Bateman, 2004)

2.4.2 Loss of consciousness (LOC)

Consciousness is structurally produced in the cerebral hemispheres, including the pons and the medulla and extends to the midbrain where it forms the reticular activating system. This pathway modulates the perception of events, controls integrated responses and refers to a sense of awareness (Crippen, 2009). Damage to this specialized part of the midbrain results in immediate loss of consciousness (LOC), which may last from a few seconds to minutes and is established by the number of nerve cells damaged, the severity of the damage and the site of impact. Therefore, LOC is defined as an unawareness or inability to respond to the environment. This does not include transient confusion or any other alterations of mental status, such as being dazed, disoriented or confused (Petchprapai & Winkelman, 2007). There are a few theories that propose the occurrence of LOC because of TBI (Table 2.5), but no mechanism or combination of mechanisms can exactly explain this phenomenon.

Table 2.5 Theoretical Postulations of Loss of Consciousness

Theory	Postulation
Centripetal Hypothesis	Mechanically induced forces disrupt brain function
Convulsive Hypothesis	Changes in general neuronal firing
Pontine Cholinergic System Theory	Activated cholinergic neurons suppress responses
Reticular Theory	Brainstem's temporarily paralysed reticular formation

(Mendez, Hurley, Lassonde, Zhang & Taber, 2005; Shaw, 2002)

LOC is often the result of rotational forces exerted at the junction of the upper midbrain and thalamus and the disruption of axons along the neuroaxis that cause transient interference of the functioning of the reticular neurons that maintain alertness, modulates perception of events and integrates responses (Crippen, 2009; Ropper & Gorson, 2007). Levels of consciousness range on a continuum from being fully alert, to drowsiness, lethargy, obtundation, stupor, coma and lastly brain death (Crippen, 2009; Lezak et al., 2004).

In general, TBI severity is indicated by the magnitude and duration of LOC, especially when the duration is hours or days (Cantu, 2001). In a comparative study of three groups of subjects with transient LOC, equivocal LOC and no LOC, Iverson, Lovell & Smith (2000) found no differences in the outcome on a variety of cognitive dimensions. Lovell, Iverson, Collins, McKeag & Maroon (1999) also questioned the paramount importance of LOC in the grading of severity, as they found no significant differences between LOC and neuropsychological measures used in their study.

2.4.3 Posttraumatic amnesia (PTA)

A diagnosis of TBI can be made even in the absence of documented LOC, and the main diagnostic criteria have shifted over time from LOC to Posttraumatic Amnesia (Collins, Iverson, Lovell, McKeag, Norwig & Maroon, 2003; Ruff, 2005). Post Traumatic Amnesia (PTA) generally refers to the subacute phase of recovery immediately following TBI and is supported by its positive relation to acute neurological abnormalities as well as the extent of damage to the brain (Cantu, 2001; Levin, Benton & Grossman, 1982; Levin, Eisenberg & Benton, 1989; Rimel, Giordani, Barth, Boll, & Jane, 1981). PTA appears not to affect visuospatial attention tasks (Ruff, Evans & Marshall, 1986). The clinical use and proven validity of memory impairment as a predictive measure of TBI outcome is a widely accepted indicator in the clinical field (Ahmed, Bierley, Sheikh & Date, 2000; Gronwall & Wrightson, 1980). In the absence of a globally acceptable definition for PTA, two types of amnesia can be considered (Cantu, 2001; Martin, 1998):

1) *Retrograde amnesia* - a difficulty or inability to recall recent events or information preceding the onset of brain trauma. The period tends to be relatively short, i.e. 30 minutes or less and is recorded from the actual time of the most recent recollection of an incident, up until the time of injury;
2) *Anterograde amnesia* – a difficulty or inability to remember events subsequent to the onset of the injury. This tends to result in an inability to form new memories and, therefore, results in defective recent memory and is recorded from the time of injury up until continuous memory returns.

Table 2.6 indicates that PTA is best regarded as a logarithmic scale (Asikainen, 2001; Binder, 1997; Borg, Holm, Cassidy, Peloso, Carroll, von Holst & Ericson, 2004; Cullum & Thompson, 1997; Teasdale & Brooks, 1985). Borgaro, Prigatano, Kwasnica & Rexer (2003) explain the severity of PTA as having positive neuroimaging with space-occupying lesion(s), LOC and the preponderance of cognitive over emotional symptoms.

Table 2.6 Post Traumatic Amnesia (PTA) Duration and Injury Severity

PTA Duration	Severity
< 5 minutes	Very mild
5-60 minutes	Mild
1-24 hours	Moderate
1-7 days	Severe
1-4 weeks	Very severe
> 4 weeks	Extremely severe

(Asikainen, 2001; Binder, 1997; Borg et al., 2004; Cullum & Thompson, 1997; Teasdale & Brooks, 1985).

It is crucial not to be over dependent on a classification category or GCS score, but to use these in conjunction with a relevant neurologic and neuropsychological assessment. Neuropsychological assessment is concerned with, and helpful in the diagnosis of, individuals that fall between classification extremes (Lezak, Howieson & Loring, 2004). The interpretation of these neuropsychological scores depends on the severity of the injury, knowledge of the GCS, results from neuro-imaging findings, the length of LOC (Binder, 1997), as well as the presence of acute cognitive abnormalities that contribute positively to the prediction of the course of the injury and prognosis. TBIs are postulated to occur along a continuum, with MTBI at the mild end of the TBI severity range, and therefore MTBI cannot be understood as a totally distinct entity from TBI in general (Reitan & Wolfson, 2000). Nevertheless, there are specific issues that pertain particularly to MTBI, and these warrant detailed exposition, to be pursued in the following chapter.

CHAPTER 3

MILD TRAUMATIC BRAIN INJURY

This chapter reviews mild traumatic brain injury (MTBI), and the classification and biomechanics thereof, with special reference to Newton's Laws of Uniform Motion. The neurocognitive consequences following MTBI are described, and the neurocognitive functions are grouped and discussed under two broad domains of function, namely Memory and Motor Speed. This is followed by the risk factors influencing MTBI outcome and the chapter concludes with a delineation of the brain and cognitive reserve concept, which forms the theoretical foundation for this study, and is used to conceptualise the individual's capacity to absorb brain injury.

3.1 MILD TRAUMATIC BRAIN INJURY

Varied signs and symptoms characterize MTBI and account for the difficulty in the precise and concise, often ambiguous, definition of the phenomenon of MTBI. In the literature, there is no uniform and universally accepted definition of MTBI (Cantu, 1997, 1996; Lovell, Collins & Bradley, 2004; Powell, 2004; Pretz, 2007; Rutherford, Stephens & Potter, 2003; Satz, 2001). MTBI is the commonly accepted scientific term in contemporary literature, although it is often used interchangeably with the terms 'minor brain injury', 'mild head injury', 'cerebral concussion' and 'concussion' (Anderson, Northam, Hendy & Wrennall, 2001; Barth, Varney, Ruchinskas & Francis, 1999; Guskiewicz et al., 2004; Levin, Eisenberg & Benton, 1989; Lezak et al., 2004; Maroon, Lovell, Norwig, Podell, Powell & Hartl, 2000). Therefore, as indicated in Chapter one, while it is accepted that MTBI may be associated with causation other than concussive brain injury, for the purposes of this thesis the terms MTBI and concussion will be used interchangeably.

In broad terms Hovda, Prins, Becker, Lee, Bergsneider & Martin (1999) describe concussion as "*a neurometabolic cascade of events whereby excitotoxic mechanisms depletes energy stores, accompanied by ionic fluxes and neuronal/axonal dysfunction and injury that has grave implications for cerebral vulnerability, cell death and permanent neurocognitive deficits*".

More recently, also in broad terms, the Third International Conference on Concussion held in Zurich (2008), and the American Academy of Neurology's (AAN) terminology, classified concussion as "*a mild diffuse brain injury that incorporates changes in clinical, neuropathologic and biomechanical constructs. It is defined as a trauma-induced pathophysiological alteration in mental status that causes a graded set of clinical syndromes that may or may not involve loss of consciousness, even with or without traumatic abnormality on standard structural neuro-imaging*" (American Academy of Neurology, 1997; Aubry et al., 2002; Bazarian, Blyth & Cimpello, 2006; Gilchrist, 2004; Kelly & Rosenburg, 1997; Kelly & Rosenburg, 1998; Maroon, Field, Lovell, Collins & Post, 2002; Maroon, Lovell, Norwig, Podell, Powell & Hart, 2000; McCrory et al., 2009; McCrory & Johnston, 2002; Noakes & Du Plessis, 1996; Rees, 2003).

More specifically, in order to create an evidence-based diagnosis of MTBI, Rees (2003) highlights the necessity of four factors to be present: (i) a sufficient plausible mechanical force applied to the brain causing micro structural or molecular injury; (ii) acute clinical effects that are both recognisable and verifiable; (iii) independent partitioning of non-specific or confounding symptoms and findings; and (iv) a discernable endpoint of recovery or disability. Helpful data in support of a clinical diagnosis of MTBI are outlined in Table 3.1 and can include non-specific overlapping symptoms of comorbid musculoskeletal injury, traumatic stress and depression (Rees, 2003).

A clinical diagnosis of a suspected MTBI can, therefore, include one or more of the following: symptoms (e.g. headache, nausea, and vomiting), physical signs (e.g. loss of consciousness, dizziness, and balance problems), behavioural and/or emotional changes (e.g. irritability, nervousness, and sadness), cognitive impairment (e.g. difficulty concentrating, slowed reaction times, and memory problems) and disturbances in sleeping patterns (e.g. drowsiness, sleeping more/less than usual) (McCrory, Meeuwisse, Johnston, Dvořák, Aubry, Mollay & Cantu, 2009; Stewart, McQueen-Borden, Bell, Barr & Juengling, 2012).

Table 3.1 Helpful data in the Clinical Diagnosis of MTBI

Type of Criteria	*Data*
Obligatory criteria	A credible mechanism of injury * Craniofacial impact *
Major Criteria	Loss of Consciousness* Amnesia for blow * Disordered awareness * Finite Post Traumatic Amnesia * GCS score of less than 15 Initial vomiting with headache Vertigo
Non-specific Criteria	Headache, nausea, vomiting, balance problems, dizziness, fatigue, disordered sleep, drowsiness, sensitivity to light and noise, irritability, sadness, nervousness, feeling more emotional, numbness or tingling, feeling slowed down, feeling mentally foggy, difficulty concentrating and visual problems
Neuropsychological assessment results	Deficits in functioning

*Minimum requirements for retrospective diagnosis (Rees, 2003)

3.2 BIOMECHANICS OF MTBI

Mechanics deal with the science of the impact of forces on objects and these principles can be superimposed on the biological human system. The biomechanics of brain injury deals with the study of relationship between applied forces and head movement due to impact injuries and is similar for both MTBI and TBI (see Chapter 2), although individual differences and varied reactions to forces of equivalent intensity need to be taken into account.

3.2.1 Neurophysiology of MTBI

MTBI typically involves acceleration and rapid deceleration forces that may be caused either by a direct or indirect impact to the head. These mechanical forces occur when the head collides with

a solid or non-yielding object at a tangible speed; or when the rate of skull deceleration is exceedingly rapid and the freely mobile head both decelerates and rotates beneath the point of cranial impact as a result of the forceful impact to the anterior or posterior thorax (Alexander, 1995; Petchprapai & Winkelman, 2008; Poirier, 2003).

Under these conditions, the movement of the inert brain inside the skull cannot be slowed down sufficiently, and exceeds the brain's protective capabilities. This produces irreversible injury to a large number of nerve cells at the site of impact and manifests in sheer strains within the cranial vault (Lezak et al., 2004; Rutherford et al., 2003). These in turn lead, to the sheering of neurons and blood vessels that occur principally in the brainstem that joins the cerebral cortex to the spinal cord (Gaetz, 2004; Levin, Amparo, Eisenberg, Williams, High, McArdle & Weiner, 1987).

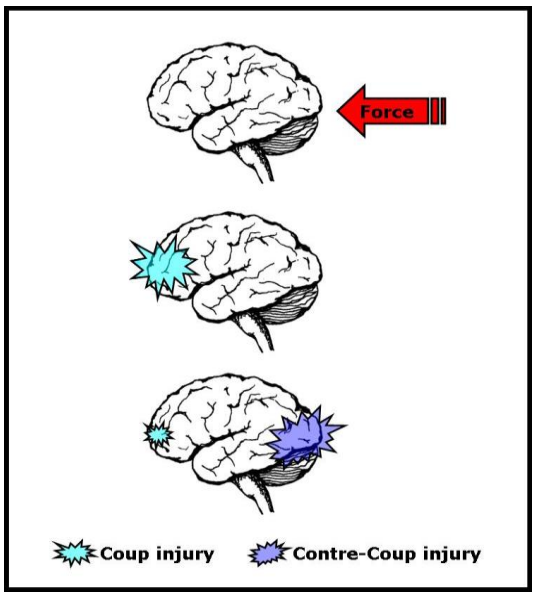
The average adult male brain weighs 1.336 kg, and per centimetre body height brain weight increases by an average of about 3.7 gram (Hartmann, Ramseier, Gudat, Mihatsch & Polasek, 1994). The brain can achieve significant momentum (mass x velocity) when an individual collides with a non-yielding object or solid surface and the resulting combined motion (described by words such as displacement, speed, velocity and acceleration) is in the direction of the body with the larger initial magnitude of momentum. The dissipation of this momentum (mechanical forces) often leads to brain injury, and movement of any kind can be based on Newton's Laws of Uniform Motion (Cantu, 1986; Hamill & Knutzen, 1995; McKenzie, Hodge & Sleivert, 2000; Noakes & Du Plessis, 1996; Young, 1992). Barth, Freeman, Broskek & Varney (2001) suggest that the Newtonian physics approach be applied to the measurement of acceleration-deceleration mechanical forces in order to comprehend the severity of brain injury, and two of Newton's laws were included in this study.

3.2.1.1 Newton's Laws of Uniform Motion

Newton's first law states "***any object that is either moving or stationary will tend to stay that way unless a force acts upon it***". The most obvious mechanical force is *contact force* (force of impact), and it is the predominant cause of injuries. The type and extent of damage directly relates to the speed and direction of movement (linear or rotational) and the time and distance of deceleration as any change in velocity will influence the outcome (Barth, Freeman, et al., 2001).

This phenomenon can also be described in terms of the *contre-coup* effect (Figure 3.1) and refers to the fact that forceful impact to the movable head is likely to produce a contusion ipsilaterally to the site of impact (*coup).* A contusion sustained to an area contralateral to the external injury (*contre-coup*), most frequently occur in the frontal and temporal lobes and produces as much, or more damage to brain tissue as to the original site of impact (Darby & Walsh, 2005; Guskiewicz et al., 2004). There appears to be no scientific evidence suggesting that either of the *coup* or *contre-coup* mechanisms of injury is more serious than the other, due to most movement-induced injuries involving a combination of these mechanisms. *Coup* and *contre-coup* contusions result in discrete impairment of neurocognitive functions interceded by the cortex at the site of the lesion, and are often multiple and frequently associated with other axial haemorrhagic lesions.

Figure 3.1 Contre-coup Injury to the Brain



Newton's second law states "***the force applied to an object is equal to the acceleration of the objects involved multiplied by their mass (force = mass x acceleration)***". Force is a vector quantity and the description is based on the direction as well as the magnitude of the applied force, including linear and rotational head acceleration and/or deceleration (Guskiewicz & Mihalik, 2011; Hamill & Knutzen, 1995; Young, 1992). These forces involve the *rotational acceleration* where the brain rotates around its center of gravity, and there is a disruption of the electrophysiological and subcellular activities of the neurons of the reticular activating system situated in the midbrain and the diencephalic region. Head and neck movement on impact results

in *angular acceleration,* which is a combination of translational and rotational acceleration. Velocity, duration, acceleration-deceleration rate and the direction of head movement are variables that affect the severity of brain injury. Therefore, MTBI does not always require a direct impact to the head, and the rapid angular acceleration in itself is often sufficient to set these forces in motion (Bailes & Hudson, 2001; Barth et al., 2001; Gaetz, Goodman & Weinberg, 2000).

3.2.2 Secondary Effects of MTBI

MTBI may not initially result in extensive neuronal damage, but the neurons remain vulnerable to any changes in cerebral blood flow (Cantu, 2001). Experimental studies indicate that a disturbance in the metabolic auto regulation hub creates injury-induced vulnerability that is characterized by an increased demand for glucose and the inexplicable reduction in cerebral blood flow (Fisher & Vaca, 2004; Wojtys, Hovda, Landry, Boland, Lovell, McCrea & Minkoff, 1999). The consequence is an inability of the neurovascular system to respond to the increasing demand for energy in order to re-establish the normal chemical and ionic atmospheres. This is a potentially dangerous outcome as the altered environment and profound brain damage can result in death within a few minutes, leaving little time for emergency interventions (Fisher & Vaca, 2004; Kelly, Nichols, Filley, Lillehei, Rubinstein & Kleinschmidt-DeMasters, 1991; Wojtys et al., 1999). In certain instances, the manifestation of structural damage falls below the current threshold of neuroimaging detection (Mathias, Beall & Bigler, 2004; McAllister & Arciniegas, 2002; Weinstein, Turner, Kuzma & Feuer, 2013), and this casts doubts on the ostensible *transient* nature of the injury.

There exists a certain amount of controversy regarding the underlying mechanisms, definition and existence of this rare, critical, often fatal secondary brain injury phenomenon described as Second Impact Syndrome (Bernhardt, 2009; Fischer & Vaca, 2004; McCrory, 2001; Mendez, Hurley, Lassonde, Zhang & Taber, 2005). Second Impact Syndrome (SIS), transpires when a second sub-lethal, minor impact follows an initial mild brain injury to an asymptomatic, compliance-compromised brain (Bailes & Cantu, 2001; Bey & Ostick, 2009; Cantu, 2003; Cantu & Voy, 1995; Iverson, Gaetz, Lovell & Collins, 2004; Macciocchi, Barth & Littlefield, 1998; Maroon et al., 2000; McCrory& Berkovic, 1998a; Putukian & Echemendia, 1996; Wojtys et al.,

1999). According to Barth et al. (1999), the first concussive injury impairs the system, and without sufficient time to recover between brain traumas, a second impact that may be remarkably insignificant, further compromises the system and, thus, creates a more serious malfunction.

Vagnozzi, Tavazzi, Sinoretti et al. (2007) used the impact acceleration model of diffuse TBI, confirmed the hypothesis of a metabolically 'vulnerable brain' originally proposed by Giza & Hovda (2004) and demonstrated that a second MTBI may result in catastrophic damage depending on the time lapse between traumatic insults. There is also the likelihood for developing chronic detrimental behavioural or cognitive consequences due to these multiple concussions (Barth et al., 1983; Belanger, Spiegel & Vanderploeg, 2010; Gardner, Shores & Batchelor, 2010; Guskiewicz et al., 2003; Iverson, Gaetz et al., 2004; Kelly & Rosenberg, 1997; Koh, Cassidy & Watkinson, 2003; Macciocchi & Littlefield, 1998; Maroon et al., 2000; McCrory, 2002a; Mrazik, Ferrara, Peterson, Elliott, Courson et al., 2000; Putukian & Echemendia, 1996; Rutherford, Stephens, Potter & Fernie, 2005; Saunders & Harbaugh, 1984; Wilberger, Haag & Maroon, 1991; Wojtys, Hovda, Landry, et al., 1999).

3.3 CLASSIFICATION OF MILD TRAUMATIC BRAIN INJURY

With sensitivity to MTBI in sport seemingly on the rise and the detrimental effect it has on athletes of all ages, there is a heightened interest in the prevention, recognition, treatment and management of MTBI. According to Mihalik, McCaffrey, Rivera, Pardini, Guskiewicz, Collins & Lovell (2007) there have been at least 19 different grading scales that are mostly anecdotal, with a lack of empirical evidence to substantiate any one of these as higher in priority than the other. Grading scales are designed around the presence and duration of concussion signs and symptoms and aim to afford guidelines in the recognition, diagnosis, predicted prognosis and outcome of a concussive injury.

MTBI falls within the mild end of the TBI continuum (Guskiewicz et al., 2004; Mahoney, 2009; McCrea, 2008; Reitan & Wolfson, 2000; Satz, 2001), and is defined by at least one of the following:

1) any period of loss of consciousness (LOC) for less than 30 minutes, with a GCS of 13 to 15;
2) any memory loss for events immediately prior or post accident with Post Traumatic Amnesia (PTA) for less than 24 hours;
3) any alterations in the mental state at the time of the trauma (e.g. dazed, disoriented or confused);
4) any focal neurological deficits (e.g. double vision, loss of balance) that may or may not be transient (Barth, Varney, Ruchinskas & Francis, 1999; Carroll, Cassidy, Holm, Kraus & Coronado, 2004; Collins, Lovell & McKeag, 1999; Johnston, McCrory, Mohtadi & Meeuwisse, 2001; Leclerc, Lassonde, Delaney, Lacroix & Johnston, 2001; Rees, 2003; Ruff, 2005; Satz, Alfano, Light, Morgenstern, Zaucha, Asarnow et al., 1999), with no evidence of skull fracture or intra-cranial pathology (Koh, Cassidy & Watkinson, 2003).

Although there are multiple different versions of grading systems that further delineate the severity of the MTBI itself, for illustrative purposes the AAN guidelines for MTBI grading is tabled here (Table 3.2). From this system it is considered that transient confusion of less than 15 minutes is assessed as a grade I concussion and corresponds to the common "ding" in the contact sports arena. A grade II concussion is characterized by confusion that lasts for more than 15 minutes or in the presence of retrograde amnesia (memory loss for events preceding the impact event). A grade III concussion is typified by the presence of either brief or prolonged traumatic LOC (Guskiewicz et al., 2004; Lovell, Collins & Bradley, 2004; Lovell, Iverson, Collins, McKeag & Maroon, 1999).

Table 3.2 MTBI Grading Guidelines

Grade	Characteristics
I	Transient confusion < 15 minutes
II	Confusion that lasts > 15 minutes or by retrograde amnesia
III	LOC (brief or prolonged)

(Guskiewicz et al., 2004; Lovell, Collins & Bradley, 2004; Lovell et al., 1999).

A recent update on the abovementioned 1997 AAN guidelines presented at the AAN 65th Annual Meeting, emphasizes the movement away from any grading scales and towards an individualized assessment of MTBI, and the individualized management thereof (American Academy of Neurology, 2013).

3.4 NEUROCOGNITIVE CONSEQUENCES OF MTBI

Clinical neuropsychology is an applied science concerned with the behavioural expression of brain dysfunction (Lezak et al., 2004), and includes neurocognitive functions. The most prominent neurocognitive functions typically impaired following MTBI, including sport-related concussion, are diminished attention, impaired memory and learning (Echemendia, Putukian, Mackin, Julian & Shoss, 2001; Erlanger, Feldman et al., 2003; Frencham, Fox & Maybery, 2005; Guskiewicz et al., 2001; Kiliam, Cautin & Santucci, 2005; Lovell, Collins, Iverson et al., 2004, 2003; Matser, Kessels, Lezak & Troost, 2001; Matser, Kessels, Lezak, Jordan & Troost, 1999; Webbe & Ochs, 2003; Witol & Webbe, 2003), reduced visual motor processing speed (Barth et al., 1989; Covassin et al., 2008; Echemendia et al., 2001; Erlanger, Feldman et al., 2003; Hinton-Bayre & Geffen, 2004; Mathias et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Webbe & Ochs, 2003), slowed reaction time (Broshek, Kaushik, Freeman, Erlanger, Webbe, & Barth, 2005; Eckner, Kutcher, Broglio & Richardson, 2013; Erlanger et al., 2003), and/or a reduced ability to plan and switch between tasks (Matser et al., 1999). This is due to the normal processing of information being dependent on intact neural structures and functional pathways that sub serve a particular cognitive ability (Barth, Macciocchi, Giordani, Rimel, Jane & Boll, 1983; Bazarian, Blyth & Cimpello, 2006; Lezak et al., 2004; Mathias, Beall & Bigler, 2004).

Each cognitive function domain differs from one another in their neuro-anatomical organization and their behavioural expression while sharing other basic neuro-anatomical and psychometric relationships within the functional system. Although the separation of cognition and motor function is arbitrary, there is a commonality in the neural underpinnings of cerebellum damage that is known to cause deficits in cognition as well as motor control (Konczak & Timmann, 2007; Pugh & Lipsitz, 2002). The loss of 'processing speed capacity' includes compromised *reaction time*, slowed *decision-making*, impaired *motor speed*, impaired *concentration* and impaired *memory* (Frencham, Fox & Maybery, 2005; Gronwall, 1989, 1987).

The neurocognitive functions that will be targeted for the purposes of this study, are grouped into two broad domains of functioning regularly applied in clinical and research settings namely (i) Memory and (ii) Motor Speed (Lezak et al., 2004; Matzer et al., 1999; Shuttleworth et al., 2008). The grouping within these functional modalities is made in terms of the broad nature of the skill that is called upon to complete a task. Specifically, verbal memory and visual memory, are included in the broad overriding domain of 'Memory'; visual motor processing speed, reaction time and hand-motor skills are all included in the broad overriding domain of 'Motor Speed'.

3.4.1 Memory

Memory refers to the capacity to retain and use information for adaptive reasons, and involves the ability to register, learn and retrieve information (Lezak et al., 2004). Memory is one of the cognitive functions most vulnerable to impairment as a result of brain injury, as damage to the cortex can result in impaired learning and memory (Capruso & Levin, 1992; Catroppa & Anderson, 2009; Pettersen & Skelton, 2000). Shores, Lammel, Hullick et al. (2008) found deficits in learning and memory to be a sound predictor of outcome following MTBI. For the purposes of this thesis, the category Memory is employed to cover any tasks that involve learning and memory and incorporates verbal memory and visual memory.

3.4.1.1 Verbal Memory

Verbal memory incorporates attention, consolidation and retrieval and individuals with MTBI tend to have difficulty with various measures of verbal learning, verbal fluency and verbal memory. Impaired verbal memory performance implies a tendency to retrieve fewer words, with evidence for more errors and less accuracy with retrieval (Kurca, Sivak & Kucera, 2006; Mathias et al., 2004; Pettersen & Skelton, 2000). Mathias et al., (2004) found deficits in initial learning of verbal material, and immediate and delayed verbal memory deficits one month post MTBI, compared with controls. In addition MTBI patients revealed both verbal and visual memory impairment seven days postinjury (40% poorer on verbal memory), with significant improvements at one month and additional gains at three months postinjury (Ruff et al., 1989).

3.4.1.2 Visual Memory

Visual memory includes measures of visual attention, scanning, colour perception, recognition, organization and interference and deficits in visual memory have been found following MTBI (Lezak et al., 2004). The assessment of visual memory employs tests that measure visuospatial functioning (without looking at the constructs involved in visuospatial processing) and assess visual integrity in terms of analysis and synthesis (Jagaroo, 2009). Visual memory can be measured via recall and reproduction of figures. Chronic consequences of deficits in visual memory, visuo-processing, visuospatial and visuo-perceptual functioning is in evidence following MTBI (Matser, Kessels, Lezak & Troost, 2001; Matser, Kessels, Lezak, Jordan & Troost, 1999; Matser et al., 1998).

3.4.2 Motor Speed

Motor Speed refers to the amount of time it takes to produce the response output once an individual receives a specific cue. For the purposes of this thesis the term Motor Speed is employed to cover any tasks that incorporate visual motor processing speed, reaction time and hand-motor skills.

3.4.2.1 Visual Motor Processing Speed

Processing speed, refers to the ability to rapidly and efficiently respond to basic stimuli, and is typically defined as speed of task completion with reasonable accuracy (Rucklidge & Tannock, 2002). Visual motor processing speed calls upon overall problem-solving skills, perceptual ability and higher-order tactual problem-solving abilities (Prigatano, 1986) and relates to the completion rate of cognitive activities (Catroppa & Anderson, 2009). Visual motor processing speed underlies any deficits in cognitive functions and is considered a sensitive indicator of deficits following MTBI (Hinton-Bayre et al., 1997).

A reduction in visual motor processing speed has been described as a sensitive but not specific characteristic frequently seen to occur with TBI (Mathias & Wheaton, 2007) and is regularly also a common consequence following MTBI (Covassin, Stearne & Elbin, 2008; Echemendia et al., 2001; Gaetz & Bernstein, 2001; Hinton-Bayre, Geffen & MacFarland, 1997; Mathias et al., 2004; Pettersen & Skelton, 2000; Ponsford, Wilmott, Rothwell, Cameron, Kelly, Nelms, et al., 2000). Reduced visual motor processing speed typically differentiates MTBI patients from controls in numerous studies (Gronwall, 1989; MacFlynn, Montgomery, Fenton & Rutherford, 1984; Mathias et al., 2004). Measures of visual motor processing speed show significant correlations with the Concussion Resolution Index (CRI) and illustrate the relationship between standard measures of visual motor processing speed and slowed reaction time using a computerized protocol (Erlanger et al., 2003).

3.4.2.2 Reaction Time

Reaction time refers to an individual's 'preparedness' to select and initiate the appropriate response when a stimulus occurs and the actual time it takes to complete this decisional phase. Reaction time has both central (the time taken to select the response) and peripheral (the time taken to initiate the response) components. The time from the firing of the efferent signal centrally to the onset of muscular contraction peripherally is relatively constant for any given response (Kerr, 1982).

Research demonstrates that reaction time is sensitive to the effects of MTBI (Collie, Makdissi et al., 2006; Collins, Grindel et al., 1999; Covassin et al., 2008; Cremona-Meteyard & Geffen, 1994; Eckner, Kutcher, Broglio & Richardson, 2013; Iverson et al., 2004; Macciocchi et al., 1996; Maddocks & Saling, 1996; Makdissi et al., 2010, 2001; Pettersen & Skelton, 2000; Sosnoff, Broglio, Hillman & Ferrara, 2007). Slowed reaction time, in the absence of a specific motor disability, represents overall mental slowing and is one of the most meaningful features of MTBI. A slowing in reaction time is evident as task complexity increases (Lezak et al., 2004), and slowed reaction time is of a longer duration among symptomatic individuals compared to asymptomatic individuals (Collie, Makdissi et al., 2006). Slowed reaction time has been used to differentiate concussed from nonconcussed individuals (Bleiberg, Kane, Reeves, Garmoe & Halpern, 2000), although Cremona-Meteyard & Geffen (1994) found no slowing in simple reaction time following MTBI. Simple reaction time can recover as early as five to ten days following MTBI (Bleiberg et al., 2004), and choice reaction time, with reference to correct versus incorrect responses, remained slowed one month following MTBI (Halterman et al., 2005).

3.4.2.3 Hand-Motor Skill

Hand-motor skill refers to the process of interaction between the perceptual systems, the brain and the individual's reaction to such perceptual stimuli. Hand-motor skill implies some level of conscious control rather than simply reflexive activity, which may be guided and determined by feedback received from various sensory receptors (Kerr, 1982). Disturbances of purposeful motor innervations and adequate sensorimotor co-ordination (impaired motor speed and hand-eye co-ordination) may appear when there is a breakdown in motor integration and executive functioning, as these are integral to the performance of complex learned tasks (Lezak et al., 2004). MTBI leads to problems in the intuitive performance of these tasks and the known detrimental effects include slowed motor execution (De Beaumont, Mongeon, Tremblay, Messier, Prince, Leclerc, Lassonde & Théoret, 2011).

3.5 NEUROCOGNITIVE RECOVERY FOLLOWING MTBI

Neurocognitive recovery following MTBI depends on inter-individual differences that impact on both neurocognitive and symptom recovery duration (McCrory, Johnston et al., 2005). Previous research supports a pattern of cognitive recovery following an exponential course of initial rapid recovery with indications of deceleration over time. The pattern appears the same for both general and sports-related MTBI (Bleiberg, Cernich, Cameron, Sun, Peck, Ecklund et al., 2004; Hinton-Bayre & Geffen, 2004; Lovell, Collins, Iverson et al., 2003; Schretlen & Shapiro, 2003; Shuttleworth-Edwards, Border et al., 2004; Stephens, Rutherford, Potter & Fernie, 2005). Recovery following brain injury comprises of two stages, and the acute and chronic stages following MTBI will be discussed next, with reference to the cumulative deleterious effect of repeat MTBI.

3.5.1 Acute and Chronic stages of Neurocognitive Recovery

There is an increased interest into both the acute and chronic neurocognitive effects of MTBI due to the fact that trauma to the brain produces alterations at various levels of cognitive and executive functioning (Collins, Lovell & McKeag, 1999; Erlanger, Kutner, Barth & Barnes, 1999; Frencham, Fox & Maybery, 2005; Grindel, Lovell & Collins, 2001; Rabadi & Jordan, 2001), with some authors suggesting minimal persistent neuropsychological deficits following MTBI (Binder, 1997; Binder, Rohling & Larrabee, 1997; Satz, 2001). In both the acute and chronic stages, MTBI typically leads to impairments in memory, attention, planning, cognitive flexibility, reaction time and processing speed (Barth et al., 1989; Eckner, Kutcher, Broglio & Richardson, 2013; Lezak et al., 2004; Lovell, Collins, Iverson, Johnston & Bradley, 2004).

Cognitive sequelae usually improve and/or resolve within three months post-injury, and those effects that persist for longer than three months can be considered chronic (i.e. relatively permanent) (Barth et al., 1989; Bernstein, 2002; Frencham, Fox & Maybery, 2005; Lezak et al., 2004; Lovell, Collins, Iverson, Johnston & Bradley, 2004; Reitan & Wolfson, 1999; Vanderploeg, Curtiss & Belanger, 2005). It appears that most individuals recover within three months, and few cases experience chronic neurocognitive effects persisting beyond three months following MTBI (Barth et al., 1983; Belanger & Vanderploeg, 2005; Bernstein, 2002; Collins,

Grindel et al., 1999; Echemendia et al., 2001; Levin, Mattis et al., 1987; Ponsford et al., 2000; Shuttleworth-Edwards & Whitefield, 2007; Vanderploeg, Curtiss & Belanger, 2005).

In the *acute stage*, the brain recovers from the effects of metabolic and membrane failure, neurotransmission impairments, haemorrhage and oedema (swelling of tissue following injury). A certain degree of axonal regeneration occurs immediately after the injury and therefore, during the first three weeks of recovery, higher intellectual functions, including the ability to process, to classify and integrate information, memory and learning, may be compromised (Noakes & Du Plessis, 1996). There are relatively few evidence-based studies reporting on the length of time for both cognitive and symptom recovery following MTBI (McCrea et al., 2003, 2010; McCrory, 2002). Some authors report that the neurocognitive effects of MTBI resolve between two to seven days (Bernhardt, 2009; Ellemberg, Henry, Macciocchi, Guskiewicz & Broglio, 2009; Iverson, 2007; McCrea, Barr et al., 2005; Pellman, Lovell, Viano, Casson & Tucker, 2004; Pellman, Lovell, Viano, & Casson, 2006), while other authors report the resolution of neurocognitive effects within ten days (Bailes & Cantu, 2001; Barth et al., 1989; Belanger et al., 2005; Bleiberg et al., 2004; Collie, Makdissi, Maruff, Bennell & McCrory, 2006; Collins, Grindel et al., 1999; Echemendia et al., 2001; Field, Collins, Lovell & Maroon, 2003; Hinton-Bayre et al., 1997; Iverson, Gaetz, Lovell & Collins, 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008). Overall, it is presumed that up to 90% of MTBI will resolve within seven to ten days, although children and adolescents may take longer to recover (Halstead & Walter, 2010; McCrory et al., 2009).

In the *chronic stage* of recovery, the brain reorganizes itself: axons and new collaterals sprout, and connected subcortical structures and other regions help to compensate for the loss. It is this stage that reflects the individual's functional recovery (Lezak et al., 2004; Martin, 1998) and the greatest recovery is in the first few months, with little or no significant recovery after a period of six months, and no spontaneous recovery occurring after one year.

Cumulative and more permanent neurocognitive impairment arise from multiple incidents of MTBI that are often below the threshold of symptom presentation. The National Football League recently acknowledged the potential risk for chronic adverse effects following MTBI (American

Academy of Neurology, 1997; Bailes & Cantu, 2001; Bohnen, Jolles & Twijnstra, 1992; Rutherford et al., 2003; Schwartz, 2010, 2009).

3.5.2 Cumulative Effects of MTBI

Multiple concussive and subconcussive (microtraumatic brain injury) events, have additive negative neurocognitive effects, and following an apparent full recovery, residual sequelae increase vulnerability towards central nervous system (CNS) stressors (e.g. alcohol, fatigue or hypoxia), and towards sustaining a further MTBI (Gronwall & Wrightson, 1975; Shuttleworth-Jordan, Puchert & Balarin, 1993). One sustained concussion is a significant risk factor for a future concussion, and it is postulated that successive concussive events may leave the individual with prolonged recovery or even with cumulative, chronic, negative neurocognitive consequences (Bender, Barth & Irby, 2004; Bernhardt, 2009; Cantu, 2001; Guskiewicz, Marshall et al., 2007; Guskiewicz, McCrea, Marshall et al., 2003; Guskiewicz, Mihalik et al., 2007; McCrory & Berkovic, 1998a).

The ever-increasing substantiation that successive episodes of concussion can cause cumulative damage to the neurocognitive functioning of the brain is amid the possible development of symptoms/complications later in life (Baugh, Stamm, Riley et al., 2012; Collins, Lovell, Iverson, Cantu, Maroon & Field, 2002; Turner, Lucke-Wold, Robson, Omalu, Petraglia & Bailes, 2013). These cumulative concussive and subconcussive injuries may slow the recovery of neurological functioning (Mendez, Hurley, Lassonde, Zhang & Taber, 2005). There is an increased possibility to perform worse on cognitive testing, indices of memory, hand-motor dexterity, with the likelihood of slowed recovery and ongoing post-concussive symptoms (Collins et al., 1999; Iverson, Brooks, Lovell & Collins, 2006; Iverson, Gaetz, Lovell & Collins, 2004a; Iverson, Gaetz, et al., 2003; Makdissi, Darby, Maruff, Ugoni, Brukner & McCrory, 2010; Matser, Kessels, Jordan, Lezak & Troost, 1998; Shuttleworth-Edwards, Border, Reid & Radloff, 2004; Shuttleworth-Jordan, Puchert & Balarin, 1993). Several studies attest to the delayed recovery or cumulative, chronic neurocognitive consequences among athletes with a history of prior concussions, in comparison with athletes sustaining one MTBI (Covassin et al., 2008; Gaetz, Goodman & Weinberg, 2000; Guskiewicz et al., 2003; Iverson et al., 2004; Lovell, Collins,

Iverson et al., 2004; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007).

A history of one MTBI event has been associated with slower neurocognitive recovery among collegiate football athletes (Guskiewicz, McCrea, Marshall et al., 2003). A history of two or more MTBI events has been associated with significantly slower performance in processing speed, increased duration of deficits on verbal memory and reaction time, slower recovery and significantly suppressed P3 amplitude event-related potentials (Collins, Grindel et al., 1999; Covassin et al., 2008; Iverson et al., 2004). A history of three or more MTBI events has been associated with diminished memory performance and slower processing speed (Gaetz et al., 2000; Gardner et al., 2010; Guskiewicz et al., 2000; Iverson et al., 2004). In contrast, some studies found no association between poorer neurocognitive test performance on computerized or traditional neurocognitive tests for athletes with a history of MTBI events, compared with athletes without a prior history of MTBI (Bruce & Echemendia, 2009; Collie, McCrory & Makdissi, 2006; Iverson Brooks, Lovell & Collins, 2006).

Iverson, Brooks, Collins & Lovell (2006) found that reaction time was not sensitive to the chronic or cumulative effects of MTBI between athletes with no, one or two prior MTBIs at baseline assessment. In contrast, Covassin et al. (2008) found reaction time sensitive to the cumulative effects of MTBI, in athletes with a prior history of MTBI than when assessed five days post MTBI. Persistent mild cognitive deficits suggestive of cumulative MTBI was evident in older, more senior level athletes, and can be indicative of the additive effects of concussive and subconcussive events as due to participation in contact sport (Baroff, 1998; Cremona-Meteyard & Geffen, 1994; Matser et al., 1998; Rutherford et al., 2005; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards & Whitefield, 2007; Spear, 1995; Tysvaer & Lochen, 1991).

As indicated, the literature confirming the cumulative effects of multiple concussions is mixed, and Iverson, Echemendia, LaMarre, Brooks & Gaetz (2012) found provocative but not persuasive results that multiple concussions could have a lingering deficit on memory. Over the years, the Concussion in Sport Group (CISG) tended to see concussive brain injury as a functional rather than a structural disruption, thereby negating the presence of permanent effects (Aubry et al.,

2002; McCrory et al., 2009). However, in a later consensus paper from this group, held in Zurich (2008), there was some acknowledgement of the possible presence of chronic deleterious consequences in some cases.

3.6 RISK FACTORS INFLUENCING MTBI OUTCOME

Individuals with higher intelligence and/or higher education, an active lifestyle in a favourable environment, good health and genetics and emotional status may contribute to inter-individual variability on neurocognitive measures, and therefore, the prognosis following MTBI cannot be generalized. A variety of identified factors contribute to the prognosis following brain injury and potential neurocognitive risk factors include, but are not limited to age, genetic factors, a history of prior brain injury and/or the under-reporting of MTBI due to the non-recognition of signs and symptoms, and the pre-existence of neurologic and psychiatric conditions (McCrory, Collie, Anderson & Davis, 2004; Mushkudiani, Engel, Steyerberg et al., 2007; Sherrill-Parrison, Donders & Thompson, 2000; Vanderploeg, Belanger & Curtiss, 2006).

3.6.1 Age

Adults, adolescents and children respond differently to MTBI, with children being more susceptible and vulnerable to MTBI events (Anderson et al., 2001; Giza & Hovda, 2004; Halstead & Walker, 2010). Empirical studies reveal that school football players take longer to recover than older professional or university athletes (Collins, Lovell, Iverson, Ide & Maroon, 2006; Field et al., 2003; Pellman, Lovell, Viano & Casson, 2006). Gronwall & Wrightson (1974) demonstrated age-related outcome with evidence for slowed processing speed and persistent memory deficits. A slowing of central information processing speed, with associated diminution of channel capacity, occurs in many types of cerebral pathology and may have enduring effects on neural pathway development, experience-dependent plasticity, neurotransmission and metabolism (Giza & Hovda, 2004). The gradual age-related loss of brain functioning coupled with a sustained brain injury earlier in life can accelerate the time at which a critical brain reserve threshold (discussion to follow) is reached (Mortimer, 1997; Mortimer, French, Hutton, & Schuman, 1985; Mortimer, Van Duijn, Fratglioni et al., 1991).

The cumulative risk of experiencing some form of brain injury increases with chronological age and MTBI can accentuate the effect of normal biological aging and age-related decline in cognitive functioning (Klein, Houx & Jolles, 1996). Older adults demonstrate decreased motor function with a gradual increase in reaction time, indicating slower response times as one gets older (MacFlynn, Montgomery, Fenton & Rutherford, 1984; Nesselroade & Salthouse, 2004). Due to the variety of older individuals afflicted by Chronic Traumatic Encephalopathy (CTE), emerging evidence indicates a conservative estimate of lifetime prevalence of CTE in retired American football players to be at least 3.7% (Saulle & Greenwald, 2012) and in retired professional boxers as high as 20% (Lakhan & Kirchgessner, 2012).

In the football codes (soccer, American football, Rugby League and Rugby), persistent mild cognitive deficits suggestive of cumulative brain injury, are evident among older athletes. This is indicative of the additive effects of concussive and subconcussive injuries resulting from years of participation in contact sports (Matser et al., 1999; 1998; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards & Whitefield, 2007; Spear, 1995). A study by Downs & Abwender (2002) demonstrated a dose-response relationship between lengthy soccer careers and poorer neuropsychological performance. Thus, the risk for deficits in concentration, reaction time and conceptual thinking increased in frequency as the individual player gets older, and there exists a positive association with a history of prior MTBIs that enhance the vulnerability to protracted symptomatology (Binder, 1986; Guskiewicz, Marshall, Bailes et al., 2007; Tucker, 1997).

3.6.2 Genetic factors

Cumulative TBI and Alzheimer's Dementia (AD) show similar histopathology abnormalities, particularly that of amyloid deposition, cholinergic activity changes and in some instances neurofibrillary tangles, and suggest a genetic predisposition towards the adverse effects of TBI (Jordan, 2004). The influence of the Apolipoprotein E (APOE) genotype is evident in these abnormalities and is a cholesterol transporting molecule in the central nervous system that exists in three isoforms ε2, ε3 and ε4. Genetic studies identified the ε4 allele of the APOE gene on chromosome 19 as a genetic predisposition to trauma vulnerability, impaired cognitive functions and a risk factor for developing AD (Jordan, 2004; Teasdale, Nicoll, Murray, & Fiddes, 1997).

There is evidence of a positive correlation between lower cognitive performance, increased chronic neurological deficits and possession of the APOE ϵ4 genotype in older players and/or players with a number of years of cumulative exposure to contact sports (Jordan, 1997; Kutner, Erlanger, Tsai, Jordan & Relkin 2000; Lishman, 1997). A prospective study found no associations between outcome, the APOE ϵ4 genotype and scores on the Glasgow Coma Scale (GCS) six months following TBI, although a significant reaction between APOE ϵ4 and age was revealed (Teasdale, Murray & Nicoll, 2005).

3.6.3 Pre-existing Neurologic and Psychiatric Conditions

The presence of a comorbid neurological or psychiatric condition contributes to the potential risk of persisting neuropsychological deficits following MTBI (Collins, Grindel et al., 1999; McCrory et al., 2004; Shuttleworth-Edwards, Border et al., 2004). Reviews of Shuttleworth-Edwards & Whitefield (2007) indicate the presence of a learning disability combined with a history of two or more concussions lead to poorer performance on tests of executive functioning and mental processing speed. The presence of depression does not clearly contribute to acute cognitive sequelae of MTBI, nor indicates significant poorer performance than those without depression. The only suggested interaction was with word recognition within 24 hours of sustaining a MTBI (Preece & Geffen, 2007).

3.6.4 History of Prior MTBI

The cumulative secondary effects of MTBI, discussed in detail above, attest that athletes with a history of prior MTBI lowers the threshold for sustaining a subsequent MTBI, and they may experience poorer neurocognitive outcomes when compared with athletes with no prior history of MTBI (Collins, Grindel et al., 1999; Shuttleworth-Edwards, Border et al., 2004). A history of MTBI has been associated with lowered baseline performance on visual motor processing speed among American football players (Collins, Grindel et al., 1999).

3.6.5 Under-reporting or Non-recognition of MTBI

An alarming factor affecting prognosis is the tendency for underreporting or non-recognition of concussion amongst athletes, and this poses a risk for repeat concussions (Erlanger, Feldman et al., 2003; Field et al., 2003; MacLeod, 1993; McCrea et al., 2004; Shuttleworth-Edwards, Noakes, et al. 2008; Shuttleworth-Edwards & Whitefield, 2007; Sturmi, Smith & Lombardo, 1998; Susco, 2003). A study from Columbia University identified 70 concussions among 436 college football players, despite subjective self-reports of recovery, and nearly 40% of these concussed athletes still had significant deficits on neurocognitive testing (Helwick, 2013). The high incidence of underreporting or non-recognition by both coaches and individuals remain significantly problematic because of a variety of reasons of which the following are prevalent:

1) the pressure to perform and the loss of objectivity by fellow players, coaches, parents and spectators in order for players to continue with the game (Kushner, 2001; McCrea et al., 2005), and the
2) the lack of education and knowledge to recognize the immediate dangers and long-term consequences of continuing to play under these circumstances (Cantu, 1998; Geberich, Priest, Boen, Straub & Maxwell, 1983; McCrea, Hammeke, Olsen, Leo & Guskiewicz, 2004).

The reported concussion rates in incidence studies likely constitute a significant underestimation due to the lack of knowledge and/or ignorance of what constitutes concussion (Bernhardt, 2009; Boffano, Boffano, Gallesio, Roccia, Cignetti & Piana, 2011; Lovell, Collins, Iverson et al., 2004; McCrea, Hammeke, Olsen, Leo & Guskiewicz, 2005; Pretz, 2007; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards, Noakes, Radloff et al., 2008; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards & Whitefield, 2007). Delaney (2005) found 88.6% of concussed patients visiting an Emergency Department did not recognize concussion signs and symptoms, and 28.2% of these were involved in activities posing a high risk for repeat concussion.

Essentially, all the above risk factors alone or in various combinations will cause injury outcome to differ from one individual to another (Macciocchi, Barth & Littlefield, 1998; Mortimer, 1997).

The theory most widely used to conceptualize individualized brain capacity to absorb injury will be discussed in more detail in the following subsection.

3.7 THE CONCEPT OF BRAIN AND COGNITIVE RESERVE

There is evidence that an inherent redundancy and flexibility in brain functioning will permit resilience in any situation where the brain sustains an injury, and therefore, the concept of brain or cognitive reserve is relevant in individuals exposed to the cumulative effects of frequent exposures to head and body collisions.

The apparent neuropsychological recovery observed following brain injury is explained by the concept of human beings having a functional reserve or a threshold in order to modulate the relationship between brain pathology and outcome (Barnett & Sahakian, 2008; Blessed, Tomllinson & Roth, 1968; Satz, 1993; Stern, 2002, 2003, 2006,2009). The medical field uses the notion of having a reserve to explain individual fortification from, or susceptibility to clinical symptoms associated with brain injury that may result in different levels of neurocognitive impairment and rates of recovery (Stern, 2002, 2003, 2006,2009). The theory developed, and most widely used, to conceptualise the brain's capacity to absorb pathology (injury or disease), individual differences, individual physiological reactions to injury, resilience and capacity for recovery, has been presented in a variety of terms that include *Brain Reserve*, *Brain Reserve Capacity*, *Cognitive Reserve* and *Neural Reserve* (Barnett, Salmond, Jones & Sahakian, 2006; Mortimer et al., 1991; Stern, 2002, 2003, 2006,2009). Literature, to some extent interchangeably refers to the various reserve theories that are not mutually inclusive and tend to be overlapping, with cognitive reserve currently tending to be the preferred term implying more than merely functional impairment (Stern, 2009).

Reviews of the cognitive reserve concept (McCrea, 2008; Satz, 1993) infer the consideration thereof as a hypothetical, multifactorial construct that correlates with unique individual factors such as premorbid health, underlying psychopathology, cognitive ability, age, general intelligence, educational level, severity of injury, the existence of postconcussive symptoms and psychological reaction to the injury. Broadly, the concept of cognitive reserve capacity indicates that individuals uniquely possess the capacity to withstand and compensate for mild,

traumatically induced neuronal loss until an individual threshold is met due to inherent redundancies in brain structures and systems (Barnett & Sahakian, 2008; Randolph, 2001; Satz, 1993; Stern, 2002, 2003, 2006,2009; Weight, 1998). Neuropsychological data from Binder (1986) support the hypothesis of selective vulnerability and individual response differences to a reduction in reserve capacity. The recovery from TBI is possible even as the individual may continue to suffer from a reduction in cognitive reserve, or may temporary lower the neurocognitive threshold due to the interaction of injury and pre-injury variables, although subsequent damage beyond an individual's threshold causes rapid cognitive decline and possible permanent impairment.

A higher level of education and intelligence (related to functional independence and cognitive test performance) may preserve functional capacity and may compensate for cognitive inefficiency regardless of injury severity. This is consistent with prevailing clinical assumptions that greater premorbid intellectual functioning may decrease vulnerability to cognitive deficits and may lead to improved post-injury functioning and recovery (Adams, Parsons, Culbertson & Nixon, 1996; Coffey, Saxton, Ratcliff, Bryan & Lucke, 1999; Kesler, Adams, Blasey & Bigler, 2003; Lezak et al., 2004; Mortimer, 1997; Mortimer & Graves, 1993; Reitan & Wolfson, 1999). Education, life experiences and cultural aspects may impart cognitive reserve over and above innate intelligence and it is a valuable prognostic factor regardless of injury severity (Echemendia, 2004; Jeon, Kim, Kim, Chang & Bai, 2008; Ostrosky-Solis, 2004).

Where the margin of brain reserve is less, vulnerability and susceptibility to the deleterious outcomes of MTBI and the risk of impairment are greater (Shuttleworth-Edwards & Whitefield, 2007). In the case of an individual with reduced cerebral capacity, a brain injury is more likely to result in neurocognitive impairment. It is further possible that in particularly stressful situations or physically stressful competitive conditions, cognitive deficits become more pronounced (Bailes & Cantu, 2001; Dixon et al., 1994; Killam et al., 2005), due to the rate of reserve activation and the limits in the activation process (Baltes, Kühl, Gutzmann & Sowarka, 1995). Support for the concept of cognitive reserve is found in studies on the cumulative effects of rugby-related MTBI (Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007). These studies incorporated controls and found deficits in visual motor processing speed

and memory among older, cognitively vulnerable rugby players who have been exposed to the game for a number of years.

It can be hypothesized that individuals with lowered cognitive reserve and slower processing speed may be more at risk, with increased symptom presentation and changes in neurocognitive function due to former biological insults. In the event of multiple lesions or combination of vulnerability factors, it can be concluded that

1) the aggregate effect may lower the threshold and cognitive reserve capacity level (2002; Satz, 1993; Stern, 2009); and
2) the effect of frequent and cumulative brain insults (as in the event of lengthy exposure to contact sports) may increase vulnerability to symptom presentation and the reduction of cognitive functioning (Collins et al., 1999; Leibovici, Ritchie, Ledesert & Touchon, 1996; Shuttleworth-Edwards & Whitefield, 2007; Sosnoff, Broglio & Ferrara, 2008).

CHAPTER 4

MILD TRAUMATIC BRAIN INJURY IN CONTACT SPORTS

This chapter reviews four identified contact sports in the football codes, namely soccer, American football, Rugby League and Rugby. A brief description of the mechanisms of MTBI for each sport provides the background to the neurocognitive consequences of that specific sport. Rugby is dealt with more extensively in this chapter and in this thesis and includes the tackling phenomenon and a description of the prominent types of tackles found in rugby.

4.1 EPIDEMIOLOGY OF MTBI IN SPORT

Participation in sporting activities in the United States results in up to 3,8 million mild brain injuries annually (Langlois, Rutland-Brown & Wald, 2006; Sosin, Sniezek & Thurman, 1996; Terrell, 2004), and worldwide up to 19% of athletes are annually at risk of sustaining a MTBI (Anderson, Schnor, Schroll & Hein, 2000; Matser et al., 2004; McManus, 2006; Pretz, 2007). Participation in a contact sport, therefore, is recognized worldwide as the most common cause of injury and constitutes a primary public health concern due to the likelihood of high-speed contact i) with the ground, ii) with another player (head and body), and/or iii) with equipment and objects (Aubry et al., 2002; Bailes & Cantu, 2001; Collins et al., 1999; Dvořák, McCrory, Aubry, Molloy & Engebretsen, 2009; Guskiewiecs et al., 2000; Poirier & Wadsworth, 2000).

There is an increase in MTBI resulting from recreational, amateur and professional sports, even when protective devices are in use (Zhang et al., 2004). There is also growing evidence to support the devastating potential of repetitive minor head injury in contact sports (Anderson, 2012a; Matser et al., 1999, 1998; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards & Whitefield, 2007; Spear, 1995).

The acquisition of MBTI is a paramount concern in all sports that involves head impacts or collisions which can cause the brain to rapidly accelerate or decelerate (Bailes & Cantu, 2001;

Bailes & Hudson, 2001). The intensity of contact sports manifest with frequent and intense impacts as elucidated by Broglio, Sosnoff, Shin, He, Alcaraz & Zimmerman, (2009). Data from the National Collegiate Athletic Association Injury Surveillance System reveal that the most frequently scrutinized mechanism of injury, as observed in football and soccer, is the physical contact phenomenon and accompanying rotational acceleration forces that may cause a concussive injury (Dick, 2003). In essence, it is the combination of the intensity of the activity and the years of exposure to the game, that ultimately determines the risk of MTBI in contact sports. Based on the prevalence of concussion in contact sports (Tommasone & McLeod, 2006), it is evident that boxing, ice hockey, and a cluster of the football codes, including soccer, American football, Rugby League and Rugby, all carry a high risk for head injury with the potential for significant adverse neurocognitive sequelae. The current study will briefly focus on the football codes that form a distinct entity (soccer, American football, Rugby League) and will deal with Rugby more extensively.

4.2 MECHANISM OF MTBI IN CONTACT SPORTS

4.2.1 Soccer

Soccer, commonly referred to as football, involves accidental collisions, player-to-ground contact, head-to-head or other head-related collisions, including the purposeful use of the head for controlling and advancing the ball by means of heading (propelling the ball with one's head) in order to score more goals than the opposing team. Heading the ball is used in both defensive and offensive play, with approximately 12 to 32 headers occurring per game (Rutherford et al., 2003). There are globally an estimated 265 million soccer players at risk for MTBI, due to these game tactics (Bailes & Cantu, 2001; Fédération Internationale de Football Association, 2007).

Headings relate to potential cumulative effects of numerous subconcussive blows (Baroff, 1998; Kelly & Rosenburg, 1997; Roberts, 2011), due to the angular acceleration caused by frontal and lateral heading impacts with medium velocities while bracing the neck muscles to minimise head acceleration (Bailes & Cantu, 2001; Rutherford et al., 2003). A study by Withnall, Shewchenko, Gittens & Dvořák (2005) provides biomechanical insight into the risks and high injury potential of head and neck injury associated with upper extremities and head-to-head collisions.

Accidental head impacts and head-to-head collisions can generate enough forces to cause brain injury (Kirkendall, Jordan & Garrett, 2001); although McCrory (2003) found it to be both uncommon and unlikely to result in cumulative brain injury, due to the low frequency of this phenomenon. In contrast, Frenguelli, Ruscito, Bicciolo, Rizzo & Masserelli (1991), found inter-player collisions as the major source of head injuries, and Matser et al. (2004) found head to head, head to the ground, and head to the body collisions the most frequent cause of MTBI.

A study utilising neuroimaging techniques found that soccer players are more likely to have EEG abnormalities, cortical atrophy and more mild neurological abnormalities than controls (Spear, 1995).

4.2.1.1 Neurocognitive Consequences

The frequency of headings (head-to-ball collisions) on neurocognitive impairment, are mostly limited to self-reported, subjective estimates of the soccer players (Baroff, 1998; Rutherford, Stephens, and Potter, 2003). Neurocognitive deficits are evident in players who accumulate many subconcussive blows over years of participation in the game (Matser et al., 1998; Witol & Webbe, 200) and are evident in players following the resolution of neurological symptoms (Maddocks & Saling, 1996). Research suggests the presence of cumulative effects associated with a concussion in amateur soccer players, with discernable deficits in memory, planning and attention (Killam, Cautin & Santucci, 2005; Matser, Kessels & Lovell, 2004; Matser, Kessels, Lezak, Jordan & Troost, 1999; Tysvaer & Einar, 1991). Rutherford, Stephens & Potter (2003) suggest the need to investigate the distinction between the neuropsychological effects of concussive and sub-concussive head trauma.

The effect of the measured quantity-response relationship (the frequency of headers and the number of soccer-related concussions) on cognitive functioning indicates a lowered performance on focused attention and visual motor processing tasks. Weaker neurocognitive performance is in evidence in the event of the player utilizing the heading technique in moderate to high frequency (Abreau, Templer, Schuyler & Hutchinson, 1990; Matser et al., 1998; 1999; 2004; Matser, Kessels, Lezak & Troost, 2001; Tysvaer & Lochen, 1991; Webbe & Ochs, 2003). There is a significantly negative correlation between the number of games played and rapid, complex

visual motor processing tasks (Witol & Webbe, 1994). The length of participation in soccer positively correlates with, and is more predictive of, cognitive deficits than the frequency of headers alone (Abreau et al., 1990; Downs & Abwender, 2002; Webbe & Ochs, 2003; Witol & Webbe, 1994; 2003). Evidence supporting the cumulative effects of MTBI on attention measures (scores reduced by 1.5% for each previous MTBI incident), was found among soccer players, aged 13 to 16 years, who had not sustained a MTBI within three months (Stephens, Rutherford, Potter & Fernie, 2010). In contrast, some studies found no indications of poorer performance post MTBI (Matser et al., 2001). A number of researchers found no adverse effects or evidence of neuropsychological impairment due to heading or the existence of a history of prior concussions (Guskiewicz, Marshall, Broglio, Cantu, & Kirkendall, 2002; Straumer-Naesheim, Andersen, Dvořák & Bahr, 2005).

4.2.2 American Football

American football involves an extremely large number of body contacts between opposing players (player-to-player collisions) with many blows either indirectly or directly to the head, causing the head to accelerate and rapidly decelerate. Based on multiple season data surveys, the majority of MTBI injuries occur due to linear head impacts with another helmeted player, and translational acceleration resulting from considerable head impact velocity and velocity changes (Brolinson, Manoogian, McNeely, Goforth, Greenwald & Duma, 2006; Pellman et al., 2004; Pellman, Viano, Tucker, Casson & Waeckerle, 2003; Viano, Casson & Pellman, 2007; Zhang et al., 2004).

This reiterates the phenomenon of possible over-reliance on rigid protective equipment. The protective equipment (helmets and padding) can cause more aggressive and severe forces as the player is struck purposefully with significantly higher velocity, higher acceleration impacts which adds to the kinetic energy. The two primary mechanisms of severe head injuries in American football result from (i) the acceleration force of the striking player's head and torso load through his neck that occurrs with helmet-to-helmet impacts and (ii) during the tackling maneuvers of both the ball carrier and the tackler (Barth et al., 1989; Fick, 1995; Maroon, Steele & Berlin, 1980; Viano & Pellman, 2005).

Studies of American football players utilising neuroimaging techniques found evidence of brain atrophy and cavum septum pellucidum along with amyloid β, tau, and TDP-43 pathologies (McAllister, Flashman, Maerlender et al., 2013). Tremblay, De Beaumont, Henry, van Boulanger et al. (2012) investigated the effects of sports concussion and aging on American football and ice hockey players using multimodal neuroimaging in conjunction with cognitive assessment and found a significant enlargement of the lateral ventricles that correlates with episodic memory decrements and a combined effect of age and concussion on cortical thickness that correlates with episodic memory decline. Concern regarding Chronic Traumatic Encephalopathy (CTE) prompted the National Football League to ban the most dangerous helmet-on-helmet hits (Malone, 2012). The emergence of CTE is evident in data on American football players and indicates that the stage of CTE correlates with increased exposure and duration of football play (McKee, Stein, Nowinski et al., 2013).

Recent studies (Barr, Prichep, Chabot, Powell & McCrea, 2011; McCrea, Prichep, Powell & Barr, 2010; O'Neill, Naunheim, Prichep & Chabot, 2011) showed abnormal features of brain electrical activity at injury and persisting beyond observed clinical symptomatic recovery. Event-related potentials (ERP) appear more sensitive than neuropsychological testing alone and revealed significant differences between athletes with and those without a history of prior MTBI, whereas ImPACT revealed no significant differences between these groups (Broglio, Pontifex, O'Connor & Hillman, 2009). Studies of sports-related concussions adapting neuropsychological measures to the Functional MRI (*f*MRI) assessment environment, found a more prolonged clinical recovery following hyper activation on *f*MRI scans and have been particularly illuminative regarding the effects of MTBI from the initial injury to recovery (Chen, Johnston, Frey, Petrides, Worsley & Ptito, 2004; Lovell, Pardini, Welling, Collins, Bakal, et al., 2007).

A Study of sports-related concussions utilizing blood-brain barrier disruption (BBBD) and the accompanying surge of the astrocytic protein S100B in association with Diffusion Tensor MRI (DT-MRI) found a supportive relation between repeated BBBD and potential risk for cognitive changes (Marchi, Bazarian, Puvenna, Janigro, Ghosh, et al., 2013). Using Positron emission tomography (PET) imaging with FDDNP, a novel tracer molecule that binds to tau and amyloid in the brain, researchers found that compared with controls, tau protein deposits were higher in all subcortical regions and in the amygdala of retired National Football League (NFL) players

(Cassels, 2013). A study exploring the chronic stages of repetitive sports-related brain injury in 100 retired American Football players, using Single photon emission computed tomography (SPECT), revealed hypoperfusion in the prefrontal and temporal poles, occipital lobes, anterior posterior cingulate gyri, cerebellum and hippocampus (Amen et al., 2011).

4.2.2.1 Neurocognitive Consequences

Participation in American football is associated with significantly lower cognitive scores on measures of general cognitive functioning, visual motor processing speed, accuracy, reaction time, memory and attention (Iverson et al., 2004; Kutner et al., 2000; Lovell, Collins, Iverson, Field, Maroon, Cantu et al., 2003; Lovell, Collins, Iverson et al., 2004). Macciochi, Barth, Rimel & Jane (1996) involved 2300 college football athletes in their study and 183 sustained MTBIs that resulted in impaired cognitive performance for sustained auditory attention, visual motor speed, attention, concentration and memory.

There are significant impairment in performance on measures of working memory and verbal learning two hours, and 48 hours post injury on working memory, verbal learning and verbal memory, among male and female college athletes compared with controls (Echemendia et al., 2001). No significant differences between the groups were found one week post injury and Echemendia et al. (2001) pointed out the equivalent *pre*-season scores on the HVLT learning index, with the controls benefiting from practice effects at the 48 hour assessment interval, while the MTBI group did not. Lovell, Collins, Iverson et al. (2004) found deficits on the ImPACT memory composite with increased symptoms reported within 36 hours of a MTBI. Among high school athletes, Lovell, Collins, Iverson et al. (2003) found deficits on the ImPACT memory composite up to a week post injury, despite symptom resolution within four days. Verbal memory appears sensitive to the cumulative effects of MTBI and athletes with a prior history of MTBI performed significantly worse, when assessed five days post MTBI (Covassin et al., 2008).

Visual memory deficits are in evidence more than five days post MTBI with significantly poorer scores at 24 hours and three days post injury on total figures and delayed recall on the Benton Visual Spatial Memory Test-Revised (Field et al., 2003). These findings are supported by

significantly poorer performance on immediate and delayed recall and deficits in visual memory within 48 hours of injury (Pellman, Lovell et al., 2004). De Beaumont et al. (2009) found chronic cognitive deficits in episodic memory (in addition to slowed motor execution on a diadochokinesia task) among former hockey and American football players who sustained their last sport-related MTBI more than three decades earlier. This emphasises the potential for cognitive and motor aberrations in late adulthood even after only one or two MTBIs (De Beaumont et al., 2009).

Macciocchi et al. (1996) found no improvement on visual motor processing tasks within five days of MTBI. Barth et al. (1989) and McCrea et al. (2003) found subtle differences in visual motor processing speed and reported a return to *pre*-season levels within five to ten days post MTBI. Visual motor processing speed typically returns to normal within one to six months, although the severity and duration of this functional impairment is aggravated by the cumulative effect of repeat incidents of MTBI (Cantu, 2001). Among high school athletes, MTBI resulted in impaired visual motor processing speed within 24 hours of injury (75% of athletes), with slower visual reaction times at one month post injury (61% of athletes), and at three months post MTBI (55% of athletes) (Wilberger et al., 1991). Macciocchi et al. (1996) found college level athletes failed to show improvement on visual motor processing tasks within five days of MTBI compared to controls. The latter finding concur with those of Barth et al. (1989) and McCrea et al. (2003), who found subtle differences for visual motor processing speed among athletes that returned to baseline level within five to ten days post MTBI. In contrast, Echemendia et al. (2001) reported a faster recovery on processing speed 48 hours post MTBI among male and female college athletes, compared with controls. Lovell, Collins, Iverson, Johnston & Bradley (2004) found high school athletes' visual motor speed on ImPACT, slowed slightly within 36 hours post MTBI, and then improved significantly on baseline performance six days post MTBI.

Sosnoff et al. (2008) found impaired reaction times on the CRI computerised test, within 48 hours of MTBI, compared with controls. Makdissi et al. (2001) found reduced simple reaction time within 72 hours of MTBI, compared with controls who improved on this measure. Maddocks & Saling (1996) found reduced choice reaction time within five days of MTBI compared to *pre*-season and controls. Covassin et al. (2008) found reaction time sensitive to the cumulative effects of MTBI on athletes with a prior history of MTBI, five days post MTBI.

Collie, Makdissi et al. (2006) found reaction time deficits more prevalent among symptomatic versus asymptomatic athletes within 11 days post MTBI. The presence of both migraine or headache have been associated with slowed reaction time within a week post MTBI, and have been found predictive of clinical recovery (Iverson et al., 2004; Mihalik et al., 2005). A study by Gaetz et al. (2000) found junior athletes with a history of three or more MTBIs, performed worse on visual stimuli reaction time tasks at least six months post MTBI. In contrast, Lovell, Collins, Iverson et al. (2004) found only a slight slowing in reaction time within 36 hours of MTBI that improved significantly on *pre*-season performance six days post MTBI. Lovell (2006), however, found reaction time not sensitive to the chronic or cumulative effects of MTBI at *pre*-season for athletes with none, one or two prior MTBI events.

Data from a small sample of retired professional American football players suggest an increased risk and earlier onset of memory impairment, mild cognitive impairment and Alzheimer's dementia (Amen, Newberg, Thatcher, Yin, Wu, Keator et al., 2011; De Beaumont, Théoret, Mongeon, Messier, Leclerc, Tremblay et al., 2009). Players with a history of more than one concussion are associated with long-term deficits in visual motor processing speed, reaction time and executive functioning with a trend towards significant lower memory scores (Collins, Grindel, Lovell, Dede, Moser, Phalin, Nogle et al., 1999; Iverson et al., 2002a; Maddocks & Saling, 1996). A study by Guskiewicz, Marshall, Bailes, McCrea, Cantu, Randolph et al. (2005) indicated a threefold prevalence of reported significant memory deficits with a history of prior concussions. Tremblay, De Beaumont, Henry, van Boulanger et al. (2012) found episodic memory decline in former athletes with concussion and a significant decline on measures of semantic verbal fluency.

4.2.3 Rugby League

Rugby League is a physical body-contact sport where the players require a combination of speed, stamina, strength and agility and produces the highest relative frequency of concussion in contact sports in Australia (Hinton-Bayre & Geffen, 2004). The literature on rugby league injury is small but growing, thereby causing variability in the nature and incidence/prevalence of injury (Hoskins, Pollard, Hough & Tully, 2006). There is a high incidence of head and neck injuries, and concussion has been reported as the most frequent injury in a survey of 24 rugby league

teams (Seward, Orchard, Hazard & Collinson, 1993). Gabbett (2003; 2000) suggests that the intensity of rugby league impacts significantly on brain injury rates.

In both amateur and professional rugby league, the tackle manoeuvre is identified as the most common cause of brain injury (Gabbett, 2003; Gissane, Jennings & Standing, 1993; Gissane, Jennings & White, 1998; Stephenson, Gissane & Jennings, 1996). Players playing in the "forward" position are more likely to be injured than players in the backline positions (Seward et al., 1993), although Hinton-Bayre, Geffen & Friis (2004) found all playing positions to be equally vulnerable.

4.2.3.1 Neurocognitive Consequences

Post Traumatic Amnesia (PTA) appears to be associated with impaired visual motor processing speed at day two postinjury (Hinton-Bayre & Geffen, 2002). Hinton-Bayre & Geffen (2004) found reduced performance on complex attention, visual motor speed, and visual motor co-ordination upon re-testing 24-48 hours following a concussive incident. An earlier study found visual motor processing speed as sensitive to impairment within 48 hours post MTBI (Hinton-Bayre, Geffen & McFarland, 1997). Hinton-Bayre & Geffen (2002) found impairment in visual motor processing speed the most reliable cognitive indicator of MTBI, on day two and day ten post MTBI, among 175 concussed rugby league athletes. Hinton-Bayre et al. (1999) found that 80% of athletes improved on visual motor processing speed at one to three days, and 35% of athletes improved one to two weeks post MTBI, with recovery to *pre*-season levels taking three to five weeks.

Cremona-Meteyard & Geffen (1994) found no differences on reaction time between players with or without a MTBI within two weeks of injury, with the MTBI group showing little benefit on reaction time to cued targets. One year later the deficit on reaction time to cued targets remained. A prospective three-season study (Hinton-Bayre, Geffen & Friis, 2004) showed that tackles targeted at shoulder level and higher accounted for a significant number of concussions rarely with Loss of Consciousness (LOC), and amnesia. Headaches and postural unsteadiness were the most common indicators of concussive injury.

4.2.4 Rugby

Rugby is an exciting popular full-body contact sport that is played in South Africa at school (children starting to play the game from as young as eight to ten years of age) and at adult level (club, provincial and national). Rugby draws large crowds with games televised across the globe and involves frequent and high-speed collisions between players, and players making contact with the surface, and concussive type injuries account for 11 to 35.9% of rugby injuries (Kohler, 2004; McIntosh, McCrory, Finch & Wolfe, 2010; Nicol, Pollock, Kirkwood, Parekh, & Robson, 2010; Shuttleworth-Edwards, Noakes et al., 2008; Shuttleworth-Edwards, Smith & Radloff, 2008).

When a player executes a tackle and strikes his head against an immovable object, usually either the ground surface or part of his opponent's body, his head is instantaneously decelerated, but the body continues to move forward (Aubry et al., 2002). The acceleration-deceleration mode of injury, as described by Barth et al. (2001), incorporates direction and momentum, and recognises that no single individual concussive injury may fall exactly within a singular category. Diffuse axonal injury, therefore, only requires rapid acceleration/deceleration of the head, which results in the rapid flexion-extension movement of the neck. This occurs when the rate of skull deceleration is extremely rapid, and (1) the head of the tackled player, who has been running at speed, strikes the ground surface, or (2) when the head both decelerates and rotates, such as in the event of an oblique/side tackle in which the head is not directly involved.

Head and body collisions are classified as a mechanism of brain injury, and two types of injury may occur – extrinsic and intrinsic injuries. According to Noakes & Du Plessis (1996) the extrinsic type of injury manifests with a directly applied external force, by means of head-to-head, head-to-body or contact with a solid surface such as the ground or the goalposts. The majority of these types of injuries result from collisions with other players. Intrinsic injuries result from repetitive exposure to cumulative effects of frequent head and body collisions, with the associated potential for pronounced effects as the current study will emphasise. Viano et al. (2007) determined that head displacement, head rotation and neck loads contributed to maximum strains in the midbrain after high impact forces. A player's technique, his *pre*-season neck strength and his ability to tense his neck muscles may reduce the potential for serious injury as it

contributed in decreasing the angular acceleration of the head (Sturmi, Smith & Lombardo, 1998; Tysvaer, 1992; Viano et al., 2007).

4.2.4.1 Tackling In Rugby

The high incidence of head and neck injuries for rugby varies between 25 to 52% and represents a substantially higher incidence rate than found in rugby league, American football and/or soccer (Junge, Cheung, Edwards & Dvořák, 2004; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards, Noakes et al., 2008; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007). A six-year prospective study of injuries to elite Australian rugby players, seasonal recordings of nine rugby teams and incidence studies confirm the head and neck as the most commonly injured body site (Bathgate, Best, Craig & Jamieson, 2002; MacLeod, 1993; Micheli & Riseborough, 1974; Myers, 1980; Seward et al., 1993).

The tackling maneuver is synonymous with high frequency, high speed, high-velocity collisions amongst players, and is considered to be potentially the most perilous activity on the rugby field and was identified as a significant risk factor inherent to rugby (Fuller et al., 2010). Tackling is the result of abruptly stopping another player's body from travelling in the direction in which it was headed, and it is the phase of play with the highest frequency of MTBI (Bathgate et al., 2002; Fuller, Brooks, Cancea, Hall & Kemp, 2007; Garraway & Macleod, 1995; Jakoet & Noakes, 1998; Kemp, Hudson, Brooks & Fuller, 2008; Kerr, Curtis et al., 2008; McIntosh & McCrory, 2005; Schneiders, Takemura & Wassinger, 2009; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Whitefield, 2007). Tackling has the potential for dual trauma, and the impact of the tackle is the most frequent cause of injury as players are often struck in midair and tackled backward, or from the side and consequently also hit their head against the ground (Quarrie & Hopkins, 2008). This means that one player gains the momentum the other loses, resulting in the mutual cancellation of momentum as both players come to a stop (Hamill & Knutzen, 1995; McKenzie et al., 2000; Young, 1992).

Player position has an effect on the site and the type of the tackle-related injury, and there is an increased risk for concussion because of these tackles. Players in the forward position are exposed to considerably more head, face and neck injuries (Gissane, Jennings, & White, 1997;

Jakoet & Noakes, 1998; King, Hume & Clark, 2011; Noakes & Du Plessis, 1996), with players in the backline position significantly more prone to tackle-related injuries (Fuller et al., 2010; King et al., 2011). Numerous studies confirm that the player making a tackle is more likely to suffer a concussion, or sustain more serious injuries, than the player being tackled (Barth et al., 1989; Noakes & Du Plessis, 1996; Williams, 1984). A two-season prospective cohort study that included video analysis, by Fuller, Ashton, Brooks, Cancea, Hall & Kemp (2010) indicated an equal injury risk for both types of player position, and Gabbett, Jenkins & Abernethy (2011) found playing position and the type of tackle received have a greater influence than the number of physical tackles made. Quarrie & Hopkins (2008) reported that most injuries are due to high or above the waistline tackles, made from the front or the side. Furthermore, they found ball carriers are at a higher risk from tackles to the head-neck region, whereas tacklers were most at risk when making lower (below the waistline) tackles.

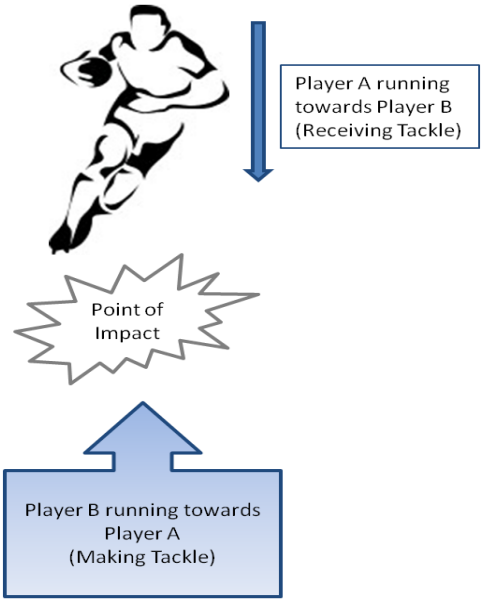
The physical demands associated with tackles, suggest that playing position and the type of tackle have a greater effect than the actual number of physical tackles performed (Gabbett, Jenkins & Abernethy, 2011). Players are often moving at high speeds and are struck by more than one opposing player in aforementioned high-velocity, high-acceleration tackles. The two tackling phases, tackling or being tackled, account for 50 to 55% of all non-catastrophic head and concussive injuries (Garraway, Lee, Macleod, Telfer, Deary & Murray, 2000; Kemp et al., 2008; Noakes & Du Plessis, 1996; Scher, 1987). There is a similar incidence of MTBI for both ball carriers and tacklers (Fuller et al., 2010; Garraway, Lee & Macleod, 1999; Wilson et al., 1999). Fuller et al., (2010; 2008) identified playing position, the player's speed, impact force, head position, head/neck flexion, body region struck, as well as the sequence of the events, direction and type of tackle as injury risks associated with tackling in rugby.

4.2.4.1.1 Prominent Types of Tackles in Rugby

Tackling and being tackled head-on are the most common mechanisms of injury (Kemp, Hudson, Brooks & Fuller, 2008). Linear deceleration tackles occur head-on within the tackled player's range of vision (Figure 4.1) and occur much more frequently (Garraway, Lee, Macleod, Telfer, Deary & Murray, 1999; Wilson, Quarrie, Milburn & Chalmers, 1999). A linear head-on tackle occurs when *Player A* moves directly towards *Player B*, and in the event of both players running

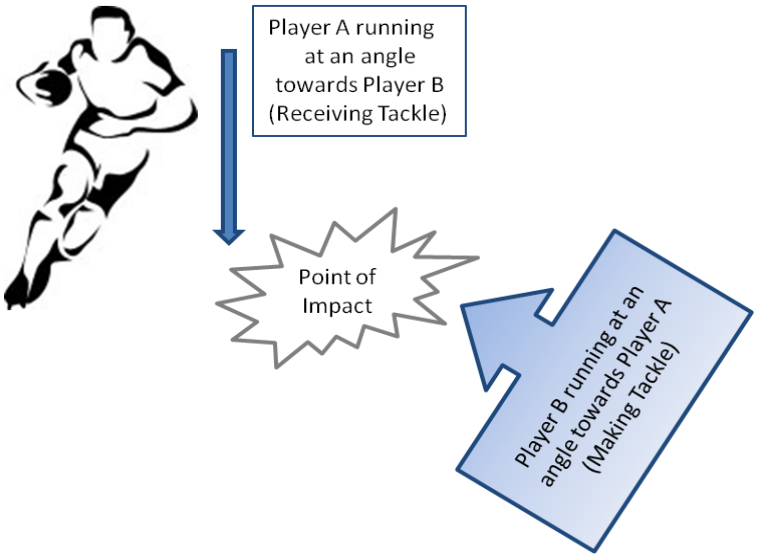
at the same speed, both players will quickly experience deceleration on impact. *Player A* usually expects the tackle and braces himself by aligning his body and tensing his neck muscles. If *Player B* hits *Player A* head-to-head or shoulder-to-shoulder in a linear fashion, they are likely to decelerate rapidly and as a greater force is applied there is a definite likelihood of MTBI (Barth et al., 2001). In the event of *Player B* making a tackle below the waistline, *Player A* will probably have a longer deceleration distance and time, and this may reduce the applied forces to the brain.

Figure 4.1 A linear Head-on Tackle



In the event of the two players hitting one another at an angle, also called an oblique/side tackle, the probability for the players' heads to collide are decreased, although shoulder-to-shoulder impacts also result in acceleration/decelerations due to the mechanical forces applied. In this situation, the distance and time prior to hitting the surface, are usually longer, and the injury severity is likely to be less (Barth et al., 2001). Should *Player A* (Figure 4.2) not expect the tackle; it is more likely for him (*Player A*) not to align his body and encounter a whiplash-type force at an oblique angle. It is essential to note that angular impacts can cause rotational forces to the brain due to the lesser flexibility of the neck and the creation of torque by the rotation of the head either in or out of its original plane. The potential of neurologic injury is substantially increased because of rapid changes in velocity (directional speed) over short distances, times, or both and this will have a considerable influence on the brain's functional ability subsequent to the injury.

Figure 4.2 An Oblique/Side Tackle



Players have a tendency to tackle above the waistline in an attempt to minimize the risk of being struck by the flailing body parts of the other player, as is evident in the commonly applied high knee action in order to avoid being tackled. In the event of a player being ankle tapped or when he trips while running at full speed, the player will hit the ground surface with the full velocity of his forward motion. The type and severity of brain injury resulting from this motion will depend on which body part hits the ground surface first and whether the head comes to an abrupt halt or not.

Taking the acceleration/deceleration biomechanics of head and body collisions into consideration, and the documented direct causative link with concussive and subconcussive events that are in turn linked with compromised neurocognitive function, rugby players are likely to incur neurocognitive deficits in association with repeated exposure to the multitude of possible tackling situations reviewed above and from the contact sports literature. Many studies investigate the biomechanisms of concussive injury, player position, and injury type and injury site and the causative link with a higher risk of concussive injury, with or without video analysis (Gabbett et al., 2011; Gabbett, Jenkins & Abernethy, 2011; Gissone et al., 1997; Guskiewicz & Mihalik, 2011; King et al., 2011; Sharp et al., 2001).

Specifically, from a methodological standpoint a number of these studies use prospective observational epidemiology analyses for tackle-related injuries and analyze video recordings to investigate the mechanism of injury and the nature of the tackles (Fuller et al., 2010; Longo, Huijsmans, Maffulli, Denaro, & De Beer, 2011; Quarrie & Hopkins, 2008; Withnall et al., 2005). However, to the author's knowledge there are no studies that yield exact incidence figures on the number of tackles taken and received by rugby players over a season at any level of play, and nor has this detailed tackling occurrence been explored in association with the player incidence of reported prior concussions and/or investigated as a contributory factor in enhanced risk of neurocognitive dysfunction.

4.2.4.2 Neurocognitive Consequences

Despite the high incidence of MTBI, there appears to be a limited number of published studies on the neurocognitive effects of MTBI in rugby. Of these, there are five South African studies (Shuttleworth-Edwards, Border, Reid & Radloff, 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Radloff, Whitefield-Alexander, Smith, & Horsman, 2013; Shuttleworth-Edwards, Smith & Radloff, 2008; Shuttleworth-Jordan, Puchert & Balarin, 1993), one American study on female rugby players (Farace, Ferree, Hollier, Barth & Shaffrey, 2003), two Canadian studies (Pettersen & Skelton, 2000; Thornton, Cox, Whitfield & Fouladi, 2008), and one Australian study (Gardner, Shores & Bachelor, 2010). The rugby-related MTBI studies provide support for relatively poorer neurocognitive performance by rugby athletes in the acute and chronic phases following MTBI (Farace et al., 2003; Gardner et al., 2010; Pettersen & Skelton, 2000; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Radloff, Whitefield-Alexander, Smith, & Horsman, 2013; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Jordan et al., 1993; Thornton, Cox, Whitfield & Fouladi, 2008). The acute, chronic and cumulative deleterious neurocognitive effects of repeated MTBI were discussed in detail in Chapter 3.

A decline in attention and memory specific tasks is indicative of the presence of compromised cognitive performance, with deficits reported in the cognitive domains of visual motor processing speed and hand-motor function (Shuttleworth-Edwards et al., 2013, 2004). Subtle deficits are in evidence for chronic declarative memory, working memory and divided and selective attention

(Pettersen & Skelton, 2000). An earlier study involved the *pre*- versus *post*-season assessment of cognitive functions and neurocognitive vulnerability was evident in attention, working memory and hand-motor function (Shuttleworth-Jordan, Puchert & Balarin, 1993).

The conclusion to a three-phase study by Shuttleworth-Edwards et al. (2004) demonstrates consistent clinically relevant neurocognitive vulnerability on tests of visual motor speed following MBTI. There is evidence for persistent neurocognitive deficits in visual motor processing speed (measured on DSST, TMT A and B, and ImPACT Visual Motor Speed composite) from high school through to adult and national levels of play (Farace et al., 2003; Gardner et al., 2010; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Jordan et al., 1993). Rugby players at school level through to adult national level showed significantly poorer performance than controls on tests of visual motor processing speed (Shuttleworth-Edwards & Radloff, 2008). Various studies consistently differentiate concussed players from controls on visual motor processing speed tasks (Farace, Ferree, Hollier, Barth & Shaffrey, 2003; Gardner, Shores, & Batchelor, 2010; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Radloff et al., 2013; Shuttleworth-Edwards, Smith et al., 2008). A study by Gardner, Shores & Batchelor (2010) indicate that rugby players with a history of multiple concussions perform significantly lower on two processing speed measures from both traditional and computerized tests. Compared with controls, visual motor processing speed and composite balance measures remain impaired up to ten days following MTBI (Cripps & Livingston, 2013; Peterson et al., 2003). Overall, studies indicate discernable chronic neurocognitive deficits in visual motor processing speed and hand-motor function, with sub-acute deficits in evidence for attention and memory.

CHAPTER 5

ASSESSMENT AND MANAGEMENT OF SPORTS-RELATED MTBI

This chapter briefly focuses on the medical and more intensively on the neurocognitive assessment and management of MTBI. The medical assessment of MTBI includes the utilization of sideline evaluation, postural stability testing with brief reference to neuroimaging and the detection of structural, functional and metabolic changes in the brain. The neurocognitive assessment of MTBI and the computer-based tests commonly used in sport-related MTBI are discussed including the Immediate Post-concussion Assessment and Cognitive Testing (ImPACT) program and the Purdue Pegboard used in this study.

5.1 ASSESSMENT AND MANAGEMENT OF MTBI

An individualized approach to MTBI assessment and management is now the standard, as there is appreciation for the variability in MTBI sequelae and recovery among individuals and the realization that MTBI can present with or without apparent cognitive deficits (American Academy of Neurology, 2013; Echemendia et al., 2001; Guskiewicz et al., 2004).

Based on research, the multidisciplinary approach to the individual assessment and management of MTBI, integrates clinical/medical analysis that incorporates sideline and postural stability assessment, neurocognitive assessment and neuroimaging (American Academy of Neurology, 2013; Aubrey et al., 2001; Echemendia & Cantu, 2003; Iverson, 2007; McCrea, Barr et al., 2005; McCrea, Guskiewicz et al., 2003; Peterson et al., 2003). Levin & Benton (1986) emphasize the value of neuropsychological assessment in order to identify the presence, and type of, deficit in cognitive functioning and to assist in the individual recovery/management process.

5.1.1 Sideline Assessment of MTBI

The sideline assessment of MTBI is challenging, given the elusiveness and transparency of injury, as well as the sensitivity and specificity of the sideline assessment tools (Putukian, Raftery, Guskiewicz, Herring, Aubry, Cantu & Molloy, 2013). Self-report concussion-related symptom checklists are the most commonly used instruments in the management of concussion. A recent study, however, indicates that nearly 40% of identified concussed athletes still had significant deficits on neurocognitive testing, despite subjective self-reports of recovery (Helwick, 2013). The considerable variation that exists in the content of these checklists, led neuropsychologists to devise and give input into the development of these instruments in an attempt to incorporate time-efficiency in the brief evaluation of cognitive function, for use by sports coaches, medical personnel, including physiotherapists. These brief cognitive screening tests are useful in distinguishing concussed from non-concussed players. Therefore, the need for a sideline intervention is two-fold in order to 1) protect the player from further injury by quantifying the severity of the impairment during the acute post-injury phase; and 2) determine the eligibility of return-to-play in the same match or practice session (McCrory et al., 2009; Randolph, McCrea & Barr, 2005).

The sideline evaluation of cognitive function provides a tool for assessing mental status immediately following MTBI, are based on, and correlate with the Mini Mental State Examination (Table 5.1). Simple orientation questions prove unreliable in the sports arena, especially when compared with memory questions (a component of cognitive function that may be preserved) (Bruno, Gennarelli & Torg, 1987; Kohler, 2004; McCrory et al., 2009; McCrory, 2002; McCrea, 2001), and should include more than the stereotypical association of disorientation to time, place, or situation. In addition to impaired orientation and memory, a range of subtle and mild neurocognitive deficits include (i) reduced planning and mental flexibility; (ii) reduced attention and visual motor processing speed; and (iii) slowed reaction times.

Table 5.1 Example Items as seen on the Mini Mental State Examination

Function Assessed	Item
Orientation	What is the year? What is the date? What is the day of the week? What is the month?
Repetition of 3 objects	
Attention	Subtraction of 7 from 100 and successive subtraction from the number remaining
Recall	Name the 3 objects mentioned earlier
Language	Name objects pointed at Repetition of phrases Follow a simple written command

(Trzepacz & Baker, 1993)

5.1.1.1 Sideline Measures of Cognitive Function

Although not the primary focus of this study, for completion the most widely applied sideline assessment of MTBI will be briefly reviewed in that, it may have promising future clinical application in sport-related MTBI. Prior to recent developments in sideline measures, there were two validated neurocognitive tests to make a rapid sideline diagnosis of concussion, the Maddocks questions and the Standardized Assessment of Concussion (SAC) (incorporated into Table 5.2). The Maddocks questions combine scientific validity with a quick simple and practical tool administered either on the field or on the sideline, with any incorrect response indicative of a possible concussion that requires the removal of the player from the playing field for further medical evaluation (Maddocks, Dicker & Saling, 1995).

Table 5.2 Sideline Assessment of Cognitive Function

Function tested	Item
Orientation	Name, date, age Month, year, time Field, opponents (today/last week) Which half is it? Which side scored last? Did we win last week?
Immediate memory	Repeat words/ Hopkins Verbal Learning Test
Concentration	Reverse digits Reciting information backwards (months, serial 7s/3s) Spell words backwards
Delayed memory recall	Recall word list/ Hopkins Verbal Learning Test

(Incorporating SAC, Maddocks Questions and Hopkins Verbal Learning Test)

The Standardised Assessment of Concussion (SAC) was developed to document the possible presence and severity of neurocognitive deficits and mental status capacities associated with a sports-related concussion (Broglio, Macciocchi & Ferrara, 2007; Hinton-Bayre, Geffen, Geffen & McFarland, 1999; McCrea, Kelly, Randolph, Kluge, Bartolie, Finn, & Baxter, 1998; McCrea, Kelly, Kluge, Ackley & Randolph, 1997; Wojtys et al., 1999). The SAC includes measures of orientation (day, month, year, and time), immediate memory (five-word list), concentration (reciting information backward – numbers, letters, and months) and delayed memory recall (retrieving the original five words).

A quick screening for the presence of neurologic signs is embedded in the SAC and includes an assessment of strength, sensation and coordination. Any disturbance in postural stability may also be recorded. The SAC is more sensitive in detecting mental status abnormalities and to differentiate among players, when a player is compared with his own *pre*-season assessment results and it is a valid instrument for the detection of immediate effects of concussion (Barr & McCrea, 2001; McCrea et al., 2002; 1998; 1997). The overall score shows a significant decline in performance when assessed immediately following a suspected concussion. Brandt & Benedict (2001) replaced the SAC recall test of five words with the Hopkins Verbal Learning Test (a relatively brief 12-word list consisting of three different semantically clustered groups, with six

equivalent versions that allow for multiple assessments) and found it to be more sensitive to concussion, as five words are within the average person's memory capacity.

The 2001 Concussion in Sport Group (CISG) Consensus Meeting defined concussion and recommended individualised clinical and cognitive post-injury management strategies. The 2004 CISG Consensus Meeting produced a standardised Sport Concussion Assessment Tool (SCAT) to aid the diagnoses, assessment and management of concussion. In 2008, the SCAT was modified and included balance assessment and consisted of both subjective and evaluative components, which consisted of a post-concussion symptom scale, modified Maddock's questions, cognitive assessment and neurological screening. A brief sideline version of the SCAT2 was developed to help on-site concussion identification (PocketSCAT2) (Finch, McCrory, Ewing & Sullivan, 2013; King, Brughelli, Hume & Gissane, 2013).

The Sideline ImPACT (distinct from the ImPACT computerised program) is a touch screen palm-held device for on-field assessment. This device contains details of the athlete's MTBI history, previous ImPACT assessment results as recorded across different injury events. The Sideline ImPACT takes about five minutes to administer and provides a brief mental status examination. The device also evaluates observed signs and reported symptoms, and records concussion details such as the point of impact and additional details from the protective equipment used (ImPACT, 2004).

5.1.1.2 Recent developments in Sideline Assessment Protocols

For the past four years the Sport Concussion Assessment Tool 2 (SCAT2), has been widely used internationally as a practical and moderately effective instrument to manage concussion. The SCAT2 has face validity, but reliability and change scores have not been reported to date (Alla, Sullivan, Hale & McCrory, 2009; King, Brughelli, Hume & Gissane, 2013). The 2012 CISG Consensus Meeting provided the opportunity to identify the most sensitive and reliable concussion components for inclusion in a revised version – the SCAT3, and decided the test battery should include an initial injury severity assessment using the Glasgow Coma Scale, followed by observed and documented concussion signs, assessment of neurocognitive function and balance function (Guskiewicz, Mihalik, McCrory, McCrea, Johnston, Makdissi, Dvořák,

Davis & Meeuwisse, 2013). The International Rugby Board (IRB) Pitch Side Concussion Assessment Working Group developed the Pitch Side Concussion Assessment (PSCA) as a tool to optimize the management of the player with a suspected concussion, and to assist in differentiating between a subconcussive and concussive event (All Blacks, 2012). The PSCA (Table 5.3) draws on a number of different elements that have been used in concussion assessment for several years and incorporates the Maddocks questions, a 20-second tandem balance test, and concussive signs and symptoms. The team doctor or referee can request a PSCA, in the presence of any of the following, (i) suspected loss of consciousness; (ii) ataxia (unsteady on feet); (iii) disorientation or confusion, and/or (iv) other symptoms or signs suggesting a suspected concussion.

Table 5.3 Pitch Side Concussion Assessment (PSCA)

Assessment	Description
On the Pitch	Confirmed LOC[l], Tonic posturing, Convulsions
Pitch Side Assessment 1	Maddocks Questions
Pitch Side Assessment 2	Tandem Balance Test
Pitch Side Assessment 3	Symptom-related Questions to player
Pitch Side Assessment 4	Symptom-related Observations by Team Doctor

[l]Note: Confirmed LOC - not responding to orders, not moving apart from reflex movement (All Blacks, 2012)

The cognitive function of answering general orientation questions remains relatively efficient in a sports-related concussion, but questions measuring short-term memory have been shown to be more sensitive and, therefore, it is regarded as a good indicator of concussive injury. Such abbreviated testing paradigms are designed for rapid concussion diagnosis on the sideline of the relevant sports field, and are not able to discern the delayed onset of subtle deficits typically found at 48 hours post-injury (McCrea, Kelly, et al., 2002). Sideline evaluations of cognitive function are not meant to replace conventional comprehensive neurological and neurocognitive testing, which might reveal subtle deficits that could persist beyond the acute phase of MTBI (Shuttleworth-Edwards, 2008). Furthermore, neuropsychological assessment is useful in overcoming the limitations of subjective questioning, where an athlete may underreport or be

unaware of his cognitive deficits or concussive symptoms (Erlanger, Feldman, Kutner, Kaushik, et al., 2003).

Therefore, a proactive approach to MTBI monitoring and management should include appropriate on-field assessment, medical follow-up, the presence of medical personnel at all matches and also incorporate the administration of pre- and post- neuropsychological assessment (Shuttleworth-Edwards, Noakes et al., 2008).

5.1.2 Medical Assessment of MTBI

Any neurologic emergency, systemic trauma or spinal injury requires the assessment of multiple areas of functioning, and any neurological changes (altered consciousness, seizures, weakness or numbness, slurred speech, worsening headaches, disorientation, double vision) or progressive deterioration on a neurological examination necessitates an immediate specialist referral (Anderson & Murata, 2009; Crippen, 2009; Department of Veteran Affairs, 2009; Hinton-Bayre & Geffen, 2004; Johnston, McCrory, Mohtadi & Meeuwisse, 2001; McCrory et al., 2009). Certain circumstances require specific medical assessments that include posturography as measured through clinical postural stability assessment and the use of neuroimaging and electroencephalography may have promising clinical application in sport-related MTBI (Davis, Iverson, Guskiewicz, Ptito & Johnston, 2009; Lovell, Collins & Fu, 2003).

5.1.2.1 Postural Stability Testing

Postural stability testing is a component of the physical examination and is important to include at *pre*-season and during postinjury evaluations to identify and monitor underlying postural instability arising from concussion (Cripps & Livingston, 2013). Postural stability as measured through clinical balance testing allows for the assessment of physical abilities and cortical neuronal functioning at rest and during tasks, and gives an indication of pre-injury levels of functioning (Iverson, 2007; McCrea, Barr et al., 2005; Peterson et al., 2003; Thompson, Sebastianelli & Slobounov, 2005). Athletes with cerebral concussion demonstrate acute balance deficits, which are likely the result of not using information from the vestibular and visual systems effectively (Guskiewicz, Ross & Marshall, 2001). Therefore, postural stability testing is

proposed for diagnosis and return-to-play decisions following MTBI. Multiple studies, as reviewed by Guskiewicz (2003), used both sophisticated force plate technology, as well as less sophisticated clinical balance tests, and identified postural stability deficits lasting several days following sport-related concussion.

It appears that postural stability testing offers a functional tool for objectively assessing the motor domain of neurologic functioning, and should be regarded a reliable and valid adjunct to the assessment of concussion (Cripps & Livingston, 2013). A variety of postural stability testing options are available including the Sensory Organization Test on the NeuroCom Smart Balance Master System as well as the Balance Error Scoring System (BESS). More recently the modified BESS was included as part of a sideline tool for concussion. The BESS is a brief clinical measure of postural stability and demonstrated good concurrent validity and test reliability (Davis et al., 2009; Guskiewicz, 2004).

5.1.2.2 Neuroimaging in the assessment of MTBI

Although not routinely used for sport-related MTBI, non-evasive neuroimaging techniques offers highly sensitive and reliable mapping of MTBI through the use of static 2-D and reconstructed 3-D images in order to obtain structural, functional and metabolic information concerning the brain (Aubry et al., 2002; Bigler & Orrison, 2004; McCrory et al., 2009). There are indications of *structural* disruption from studies involving brain tissue pathology, Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) scanning. Evidence for *functional* disruption is derived from studies examining cognitive and balance tests, functional MRI (*f*MRI), Positron Emission Tomography (PET), Single Photon Emission Computed Tomography (SPECT) scanning and Functional Transcranial Doppler (*f*TCD) (Bazarian, Blyth & Cimpello, 2006; Bigler & Orrison, 2004; Kaplan, Sadock & Grebb, 1994). Functional imaging techniques provide some evidence of cerebral dysfunction that does not show up on structural imaging (Bigler, 2001), and is valuable in detecting cognitive impairments of working memory and information processing that is typical to MTBI (Davis, Iverson, Guskiewicz, Ptito & Johnston, 2009; Gaetz & Bernstein, 2001).

Although not the primary focus of this study, for completion the most commonly used neuroimaging techniques will be briefly reviewed in that they may have promising future clinical

application in sport-related MTBI (Davis et al., 2009; Gaetz & Bernstein, 2001; Lovell, Collins & Fu, 2003). The neuroimaging techniques discussed in this section are listed in Table 5.4 and highlight how integrally linked and not easily classified, as being merely structural or functional, they are with specific reference to MTBI.

Table 5.4 Neuroimaging Techniques in MTBI

Technique	Structural	Functional
Electrophysiological Techniques	Varying degrees of structural information	Primarily functional
Computed Tomography (CT)	Structural information	
Magnetic Resonance Imaging	Primarily structural information	Some functional information
Positron Emission Tomography (PET)	Significant structural information	Primarily functional information
Single Photon Emission Computed Tomography (SPECT)	Significant structural information	Primarily functional

(Kaplan, Sadock & Grebb, 1994)

5.1.2.2.1 Electroencephalograms (EEG) and Evoked and Event-related Potentials

Electroencephalography (EEG) records spontaneous electromagnetic fluctuations in different parts of the brain, using multiple non-invasive *micro*electrodes placed on the scalp that indicates activity levels. EEG, known for its use as a first-line method to determine gross brain activity and function, still provides valuable information regarding fluctuations in brain electrical activity and information regarding the relationship with function (Davidson, 1988). The use as the only criterion for brain activity has decreased with the advent of anatomical imaging techniques such as Computed Tomography (CT) and Magnetic Resonance Imaging (MRI), especially in light of most EEG studies of MTBI that did not show significant abnormalities (Wrightson & Gronwall, 1999). An index of brain dysfunction (TBI Index) used in conjunction with EEG found increased symptoms and decreased cognitive performance only at the time of injury (Prichep, McCrea, Barr, Powell & Chabot, 2012).

Derivatives of the EEG technique include evoked potentials (EP), which involves averaging the EEG activity time-locked to the presentation of a visual or auditory stimulus or while processing a particular task or responding to a particular stimulus. Event-related potentials (ERP) are large, slow brainwaves that appear due to complex sensory or cognitive stimulation utilized in the study of a number of cognitive processes including memory, language, and attention. It also provides useful information regarding deficits in cognitive processing and sensory pathway processing (Baker & Hutchinson, 2008; Mendez et al., 2005; Martin, 1998). Reaction time measures following minor head injury show significant slowing of the EEG frequency spectra and prolonged auditory brainstem evoked responses latencies, providing evidence of central slowing (MacFlynn et al., 1984).

5.1.2.2.2 Computed Tomography (CT) Scanning

A Computed Tomography (CT) scan is the diagnostic study of choice because it has a rapid acquisition time, is universally available, is easy to interpret and is reliable. It is perfect in the delineation of bone and in the detection of skull fracture. Different tissues absorb differing amounts of x-ray energy, and this leads to the detection of structural anomalies in the brain (trauma and lesions) and is also sensitive to indications of haemorrhage and oedema (Kaplan, Sadock & Grebb, 1994). It is typically the first scan performed in a sustained TBI, including MTBI with the loss of consciousness (LOC) and/or Post-traumatic Amnesia (PTA) (Bigler, 2010; Gonzalez & Walker, 2011).

CT scanning is generally not indicated in head injury individuals with a GCS score of 15 and with no LOC or PTA. In individuals with LOC or PTA, CT scanning is not indicated if there is no headache or vomiting, if aged above 60, if intoxicated, if there are deficits in short-term memory or with the occurrence of seizures (Jagoda, Bazarian, Bruns, Cantrill, Gean, Howard et al., 2008). The detection of blood by CT scanning is one possible indicator of diffuse axonal injury (DAI), and when confirmed by MRI, it acts as a marker of damaged axons (Lipton, Gulko, Zimmerman, Friedman, Kim, & Gellella, 2009).

5.1.2.2.3 Magnetic Resonance Imaging (MRI), Functional MRI (fMRI) and Diffusion Tensor MRI (DT-MRI)

Magnetic resonance imaging (MRI) has higher sensitivity in the assessment of the overall structural integrity and subtle pathology of TBI. MRI is the radiological method of choice to reveal the detailed inner structure and restricted function of the brain when postconcussive symptoms are present weeks to months post injury without a previous or with a normal CT scan (Gonzalez & Walker, 2011). MRI has superior resolution, can distinguish between the different, typically small, and at times subtle soft tissue lesions of the brain, and is capable of taking thinner slices through the brain; therefore, it provides much greater contrast than computed tomography (CT) (Kaplan, Sadock & Grebb, 1994).

Quantitative MRI analyzed a month or more post-injury provides additional information regarding atrophic brain changes and areas of hemosiderin deposit, and is definitely the method of choice, with 96 to 98% accuracy in differentiating MTBI from non-MTBI groups (Holli, Harrison, Dastidar, Waljas, Ohman, Soimakallio et al., 2009). The most common structural deficits in MTBI are the presence of intraparenchymal signal abnormalities and atrophy (Bigler, 2001; Bigler & Orrison, 2004). Advances in MRI methods made the extension from structure imaging toward function inferences possible and enhanced the sensitivity in the detection of pathophysiological effects.

Functional MRI (*f*MRI) is still in the early stages of utilization in the sports arena, but already emphasizes the identification of underlying pathology by simultaneously assessing structure *and* function. This opens the door for direct observation of functionally induced neural or cognitive changes as a magnetic field passes through the head and measures blood oxygenation levels (Martin, 1998; Van Boven, Harrington, Hackney, Ebel, Gauger, Bremner et al., 2009). *f*MRI is noninvasive, does not require the injection of a radioisotope into the bloodstream, and is, therefore, appropriate for repeated studies. The reverberations produced by the resonance of hydrogen molecules are detected and produces excellent anatomical images that reflect which regions of the brain are working, how much, and for how long during certain tasks. *f*MRI displays visual images, sounds and kinetic stimuli and can be used to reveal brain processes associated with perception, thought and action. *f*MRI has been increasingly used for

investigating mechanisms of brain function after MTBI as well as changes that occur during recovery.

Diffusion Tensor MRI (DT-MRI) is an advanced, non-invasive in vivo diagnostic method that can determine the macroscopic axonal organization in nervous system tissue in order to provide neural tract images as an alternative to using this information solely for the purpose of allocating contrast or colours to pixels in a cross sectional image. It also provides valuable structural information as the molecular diffusion in tissues reflect interactions with macromolecules, fibers, and membranes. DT-MRI measures the bulk motion of water molecular diffusion patterns and reveals microscopic minutiae either about tissue architecture, normal or in a diseased state. It is rapidly becoming a standard for diffuse brain injuries, and can reveal abnormalities in white matter fiber structure, and DTI also provides models of brain connectivity (Benson, Gattu, Sewick, Kou, Zakariah, Cavanaugh & Haacke, 2012; Gonzalez & Walker, 2011; Jones & Leemans, 2011).

5.1.2.2.4 Positron Emission Tomography (PET)

Positron emission tomography (PET) is a nuclear medicine imaging technique that requires the injection of manufactured radioactive compounds that moves through the bloodstream and accumulates in different locations and concentrations in the brain. The increase and decrease in brain activity via the measurement of brain oxygen consumption, blood flow and glucose metabolism produces a 3-D image of functional activity (Gonzalez & Walker, 2011; Kaplan, Sadock & Grebb, 1994; Martin, 1998). The system detects pairs of gamma rays emitted indirectly by a positron-emitting radionuclide (tracer), introduced into the body on a biologically active molecule while the head of the patient is in the PET camera. The degree of gamma rays is transformed into 3-D colour-coded images or 4-D space (the fourth dimension being time), which indicates regions, that are high or low in metabolic activity or where there is increased blood flow. If the biologically active molecule chosen for PET is FDG (an analogue of glucose), the metabolic activity will produce more gamma rays because they take up more glucose. Although the use of this tracer results in the most common type of PET scan, other tracer molecules indicate the tissue concentration of many other types of molecules of interest.

The greatest benefit of PET is that blood flow and oxygen and glucose metabolism reflects the amount of brain activity in various brain regions, although it is limited to monitoring short tasks. PET is most useful in diffuse brain damage where small changes in brain volume and gross structure exist for reliable differentiation on CT and standard MRI images.

5.1.2.2.5 Single Photon Emission Computed Tomography (SPECT)

Single photon emission computed tomography (SPECT) is an alternative functional imaging modality to PET and uses gamma rays to obtain 2-D images from multiple angles and a tomographic reconstruction algorithm yields a 3-D view of the cortical surface of the brain. Any irregularities in the surface of the brain represent decreases in perfusion that may relate to decreases in neuronal activity. The basic technique requires injection of a rapidly absorbed gamma-emitting radioisotope (radioactive tracer) that can be seen by a gamma-camera while the head of the individual is in the camera tube.

SPECT represents a more commonly available technology, provides information regarding glucose utilization and other metabolic processes and generally correlates with the persistence of cognitive deficits and chronic postconcussive symptoms (Bigler & Orrison, 2004; Jacobs, Put, Ingels, & Bossuyt, 1996; 1994). Both PET and SPECT have revealed hypometabolism in the frontal and temporal lobes at rest and during working memory tasks and correlated with decreased memory function following MTBI (Mendez et al., 2005).

5.1.2.2.6 Transcranial Doppler (TCD) and Functional Transcranial Doppler (fTCD)

Transcranial Doppler (TCD) offers an excellent temporal resolution in comparison to other neuroimaging techniques, and measures the velocity of blood flow through the brain's blood vessels and is a relatively quick, inexpensive, and portable test. It is often used in conjunction with other tests such as MRI, carotid duplex ultrasound and CT scans. The technique contributes substantially to the elucidation of the hemispheric organization of cognitive, motor, and sensory functions in adults and children. Preliminary data from an ongoing study on sport-related MTBI indicates neurocognitive impairment improved over time in association with altered cerebrovascular functioning (Tegeler, Kim, Collins, Steelman, Westwood et al., 2009).

Functional Transcranial Doppler sonography (*f*TCD) is a neuroimaging tool for measuring changes in cerebral blood flow velocity due to neural activation during cognitive tasks. *f*TCD utilizes pulse-wave Doppler technology to document blood flow velocities in the anterior, middle, and posterior cerebral arteries. Similar to other neuroimaging techniques such as *f*MRI or PET, *f*TCD is based on a close coupling between regional cerebral blood flow changes and neural activation. *f*TCD is particularly useful in the study of major brain functions such as language, facial processing, color processing, and intelligence processing.

5.1.2.3 Multimodal Use of Neuroimaging Techniques

The use of multimodal techniques enhance the detection and characterization of structural, functional and metabolic changes in brain functioning and provide complimentary information regarding neural, vascular and network conditions that sub serve cognitive and behavioral states. The ultimate goal of including neuroimaging findings is to add on to treatment regimens and outcome. Data from complementary high-time-resolution techniques, such as PET and SPECT, are increasingly superimposed and read in combination with CT or MRI scans. This is to achieve a more precise anatomical and metabolic location of the functional information and to correct for variable attenuations caused by differences in individuals' head sizes (Kaplan, Sadock & Grebb, 1994).

EEG has several valid points as a tool for exploring brain activity as it detects changes within a millisecond timeframe, considering an action potential takes approximately 0.5-130 milliseconds to propagate across a single neuron, depending on the neuron type. EEG measures the brain's electrical activity directly while other methods record changes in blood flow (e.g., SPECT, *f*MRI) or metabolic activity (e.g., PET), which are indirect markers of brain electrical activity. EEG used simultaneously with *f*MRI produces and records high-temporal-resolution data with high-spatial-resolution data.

During a multimodal neuroimaging approach, neuropsychological probes stimulate particular regions of brain activity, and when compared with *pre*-season, conclusions can be made regarding the functional correspondence to particular brain deficits following MTBI, with the

consideration of certain risk factors. Overall, if MTBI investigation warrants the use of neuroimaging, it is recommended that multiple neuroimaging measures be used. This is because different measures have differing sensitivity in detecting residual injuries (Hofman et al., 2001; Kesler, Adams & Bigler, 2000).

5.2 NEUROCOGNITIVE ASSESSMENT FOR SPORT-RELATED MTBI

Neuropsychology focuses on the relationship between the brain and behaviour, and neurocognitive assessment can be broadly described as a procedure that involves the quantification of changes in brain function following brain injury and involves the identification of preserved cognitive functions (Echemendia et al., 2009; Kozora & Gerber, 2004; Levin et al., 1987; Shuttleworth-Edwards, 2008). According to Levin & Benton (1986), neurocognitive assessment aims to (i) identify the presence and deficit type in cognitive functioning; (ii) differentiate between brain injury and other factors causing cognitive impairment; (iii) evaluate deficits and preserved functions; (iv) assist in recovery, and (v) provide objective data for research.

While medical assessment can detect signs of neurological dysfunction and neuroimaging assessment can detect neurological structural damage, neither form of assessment can detect subtle neurocognitive deficits arising from MTBI (Collins & Hawn, 2002). In the absence of demonstrable neuroimaging abnormalities, neurocognitive assessment may provide the most sensitive guide for investigating subtle neurocognitive changes following MTBI, and it is more frequently used as a key component towards the multi-layered management of sport-related MTBI (Aubry et al., 2002; Collins & Hawn, 2002; Cremona-Meteyard & Geffen, 1994; Guskiewicz et al., 2004; Lovell & Collins, 2002; McCrory, Johnston et al., 2005; Mendez et al., 2005; Podell, 2004).

Neurocognitive assessment (as distinct from the brief sideline measures of cognitive functioning discussed earlier) has been found to be particularly useful in the detection of neurocognitive impairment and to chart and monitor recovery (Barr & McCrea, 2001). This is especially relevant considering that neurocognitive recovery can precede or follow symptom recovery, or in some instances an asymptomatic athlete can experience either a delayed onset of symptoms or a

delayed resolution of neurocognitive deficits (Aubry et al., 2002; Barr & McCrea, 2001; Field et al., 2003; Lovell, Collins, Iverson et al., 2004; McCrory, Johnston et al., 2005; Mendez et al., 2005; Shuttleworth-Edwards & Whitefield, 2007).

Neurocognitive assessment for sport-related MTBI has been developed for use with athletes during the past two decades following a series of concussive injuries in high-profile National Football League (NFL) athletes during the 1990s. Thereafter the directive to employ baseline assessments for all athletes was initiated by the National Hockey League (NHL), and this escalated the use of neurocognitive assessments in sports that provide objective data for analysis of cognitive function. Over the past ten years neurocognitive assessment has become a vital part of both the assessment and management of sport-related MTBI in numerous sports (Pretz, 2007).

Overall criticism of neurocognitive assessment for sport-related MTBI is that subtle MTBI cognitive deficits are not always identified (Baker & Hutchinson, 2008). Randolph et al. (2005) found that none of the reviewed traditional and computerised neurocognitive tests, met all the psychometric criteria to warrant their inclusion in the management of sport-related MTBI and require further sensitivity, reliability and validity studies. Furthermore, some authors are not in favour of neurocognitive testing while the athlete is symptomatic, and questions whether neurocognitive recovery follows symptom recovery as neurocognitive impairment in the absence of symptoms one week post MTBI, has not been demonstrated in a significant number of concussed athletes (Belanger & Vanderploeg, 2005; McCrea et al., 2003; McCrory, Johnston et al., 2005; Randolph et al., 2005).

In contrast, Lovell (2006) argues that athletes should not be asymptomatic prior to neurocognitive assessment, as it contributes to a 26% improved diagnostic yield compared with the evaluation of symptoms alone in differentiating concussed athletes from nonconcussed athletes, and adds towards the management of athletes during the early stage of recovery (Lovell, 2006; Van Kampen et al., 2006).

The usefulness of sport-related neurocognitive assessments increase with the utilisation of *pre*-season baseline levels of functioning, against which postinjury deficits can be quantitatively and objectively compared (McCrory, Makdissi, Davis & Collie, 2005; Schatz & Browndyke, 2002).

Neurocognitive assessment in sport-related MTBI involves *pre*-season baseline assessment and postconcussive follow-up assessment and, pertaining to this study, serial assessments (at *mid*- and *post*-season).

5.2.1 Pre-Season Baseline Assessment

The concept of cognitive impairments presupposes a baseline level of cognitive functioning obtained prior to the commencement of the rugby season. The baseline cognitive assessment of individual players is paramount in the neuropsychological assessment of players at *pre*-season, and provides a basis for the direct comparison in the event of a concussive injury during the season, and needs to be compared to provide quantitative neuropsychological data (Echemendia et al., 2009). Therefore, the comparison standard, may be *normative* (derived from an appropriate population) or *individual* (derived from the individual's history and/or present characteristics), depending on the purpose of the assessment (Hinton-Bayre, Geffen & McFarland, 1997; Kelly & Rosenburg, 1997; Lovell & Collins, 1998; Martin, 1998).

*Pr*e-season baseline assessments are important for the following reasons:

1) Individual players differ in terms of performance on tests of memory, attention, and visual motor processing speed,
2) Individual players may suffer from learning disabilities, attention deficit disorder, or other psychological factors such as anxiety or depression
3) Individual players differ with regard to their history of prior concussions.

Similar patterns of cognitive difficulties may be observed as a result of a concussion or unrelated factors can be secondary to a recent or previous event. The benefit of knowing how the player performed prior to a concussive injury allows for informed decisions regarding the presence or absence of subtle aspects of MTBI and changes in neurocognitive functioning can then be analysed and managed accordingly. A critical review of the literature, however, did not find sufficient evidence to recommend the widespread routine use of baseline neuropsychological assessment (Echemendia, Iverson, McCrea, Macciocchi, Giola, Putukian & Comper, 2013). Randolph (2011) cautions on the over-reliance on baseline neuropsychological testing for the

classification of a player's neurocognitive status as the use of these measures may even increase the risk in some cases.

5.2.2 Serial and Postconcussive Assessment

Successive neuropsychological assessments, repeated at regular intervals, provide a reliable indication of fluctuations in neurocognitive functioning. The use of serial (repeated) assessments tracks an individual's neurocognitive performance/recovery over time (Duff, Beglinger et al., 2007).

There are currently two methods of serial and/or postconcussive assessment: the first is to follow up at prescribed intervals post-injury while the second method is to begin the assessment once the athlete is asymptomatic (Guskiewicz et al., 2004; Johnston et al., 2005; McCrory et al., 2009). It may seem unpractical to assess a symptomatic player that is being withheld from play, but serial assessment at this delicate stage can be crucial in detecting postconcussive complications (Guskiewicz et al., 2004). Follow-up assessment is indicated when a player displays any deficits on neurocognitive performance and should be undertaken within 24 to 72 hours, but not immediately after a practice session or game as fatigue can affect the results of neurocognitive assessment (Covassin, Weiss, Powell & Womack, 2007) . Lovell & Collins (1998) found that an interval of five days allowed for the practical re-assessment prior to the next scheduled game.

In order to avoid possible cumulative injury in the vulnerable post-injury recovery period, it is considered standard practice that a return to *pre*-season baseline (hereon referred to as *pre*-season) scores or better is necessary before considering further participation in contact sports. It is further recommended that an individual is symptom-free and cognitively intact at both rest and following exertion activity before active participation (Lovell & Collins, 2002). The return to the athlete's own *pre*-season scores and/or obtaining results within normative limits (compared with gender and age stratified normative scores) may be indicative of a positive return-to-play decision. Echemendia et al. (2001) cautioned that a return to *pre*-season levels might not be a reliable indicator of "normal" functioning, and based on their research they recommend the exceeding of *pre*-season scores, particularly on measures with known practice effects.

Two types of neurocognitive assessment measures commonly used in sport-related MTBI research and in the clinical setting will now be discussed.

5.2.3 Traditional Neurocognitive Assessment

The use of traditional paper-and- pencil neurocognitive assessment measures in the sports arena resulted in a rapid expansion of knowledge regarding sport-related concussive injuries. This in itself led to the development of a number of assessment batteries that included measures of cognitive abilities most susceptible to subtle neurocognitive changes and have demonstrated their effectiveness as sensitive indicators of detecting any deficits following MTBI, such as attention and concentration, memory, visual motor processing speed, and reaction time (Collie, Maruff, Makdissi et al., 2003; Guskiewicz et al., 2004; MacFlynn et al., 1984; Mathias et al., 2004). Numerous studies attest to the need for more demanding measures that can detect *subtle* neurocognitive deficits (Bernstein, 1999).

Traditional neurocognitive assessment measures have certain limitations in that it is relatively costly, time consuming, and there is in general a shortage of trained neuropsychologists to oversee the administration and interpretation of results (Lovell & Collins, 2002; Lovell, Collins & Bradley, 2004; Lovell, Collins, Pardini, Parodi & Yates, 2005). Traditional paper-and- pencil assessment batteries are originally designed for the detection of gross brain injury deficits and lack sufficient sensitivity to discern the very mild and often subtle cognitive deficits on repeated assessments following concussion (Collie, Darby & Maruff, 2001; Lovell, 2002). Traditional tests are also limiting in their restricted range of possible scores, floor and ceiling effects (a ceiling effect exists when there is a maximum performance score for a test) and poor test-retest reliability (Collie et al., 2001).

Traditional neurocognitive assessment batteries were not designed for repeated testing paradigms or extended baseline studies, but are more sensitive than EEG or CT at uncovering subtle damage. Traditional tests of visual motor processing speed (Symbol Digit, DSST and Speed of Comprehension Test) are sensitive to MTBI effects, however, practice effects on second assessment occasions needs to be considered in order not to assume recovery erroneously (De Monte, Geffen & Massavelli, 2006; De Monte, Geffen & Kwapil, 2005).

Gardner et al. (2010) reiterates the discrepancies between neurocognitive testing formats where studies utilising traditional tests tend to support the notion of detrimental cognitive effects and studies with computerised tests tend to demonstrate no effect. The recent development and ongoing studies of computerised diagnostic tools reflect the interest in the application of sophisticated technology in order to provide more accurate diagnoses. These aspects highlight the advantages and variability of computer-based assessments in the initial and chronic stages of MTBI.

5.2.4 Computerized Sports-Related Neurocognitive Assessment

The development of computerized neurocognitive assessment in the sports arena has grown and is occupying a dominant place in the neurocognitive assessment of sports related concussions. Furthermore, assessments need to be portable, have a brief self-administration time, be cost-effective, allow for the evaluation of large numbers of athletes and facilitate the randomization of stimuli. Standardized, self-administrated computerized neurocognitive assessment measures have many advantages including (i) a normative comparison standard that presents information in a standardized and consistent manner; (ii) the accurate recording of responses; and (iii) centralized data analysis and scoring that allow for almost immediate availability and reporting of results following assessment. The automation of response recording and stimulus presentation in computer-based assessments allow for the direct measurement of cognitive changes associated with MTBI at a fraction of a second. However, computerized neurocognitive assessment measures have certain limitations including, being less flexible and interactive than one-on-one assessment; and not being able to measure verbal functioning or auditory memory (Schatz & Browndyke, 2002; Schatz & Zilmer, 2003). Accordingly, Schatz & Zilmer (2003) view computerized neurocognitive assessment as a sophisticated screening tool in the evaluation of cognitive abilities.

The recent development of computerized neurocognitive assessment programs that measure variability in performance use infinitely randomized test paradigms that promote the efficient and accurate clinical evaluation of reaction time and visual motor processing speed (Bleiberg, Garmoe, Halpern, Reeves, & Nadler, 1997; Collie, Darby & Maruff, 2001; Lovell et al., 2005;

Lovell & Collins, 2002). Recent studies included computerized neurocognitive assessment in the clinical evaluation of the athlete and highlighted the increased diagnostic accuracy of sports-related concussion (Broglio, Macciocchi & Ferrarra, 2007; Fazio, Lovell, Pardini & Collins, 2007; Van Kampen, Lovell, Pardini, Collins & Fu, 2006). The screening for conditions such as depression are not typically employed as players are motivated to return to play, and the assessment typically involves a 15 to 30 minute battery of tests measuring specific neurocognitive domains memory, attention, visual motor processing speed and reaction time.

The efficacy of computerized reaction time measures in identifying these cognitive changes has been documented (MacFlynn, Montgomery, Fenton & Rutherford, 1984; Makdissi, Collie, Maruff, Garby, Bush, McCrory & Bennell, 2001; Stuss, Stethem, Hugenholtz et al., 1989; Warden, Bleiberg, Cameron, Ecklund, Walter, Sparling et al., 2001). In contrast, the traditional neurocognitive assessments of reaction time are inferred measures using a single integer. Computerized assessment allows for the evaluation and recording of reaction times accurately in milliseconds, and tests of simple reaction time are repeatable as they do not suffer greatly from practice effects and ensures better test-retest reliability (Bleiberg, Garmoe, Halpern, Reeves, & Nadler, 1997). However, Erlanger et al. (2003) found evidence of the statistical phenomenon known as regression to the mean, where athletes performed fast at the first test of reaction time and slowed towards the group mean at the second test regardless of sustaining a MTBI or not, and they used the multiple regression statistical technique to overcome this obstacle.

Practice effects, as a particularly important methodological problem in sports-related MTBI, need always be considered in concurrence with the use of an appropriate control group that allow for the measurement of error. Another method of reducing the magnitude of practice effects is by using alternate forms of a test or test battery (Barth et al., 1999; Collie, Darby & Maruff, 2001). Despite practice effects being minimized by randomized test items allowing for several alternate forms, practice effects are still a threat in that the athlete becomes familiar with a test format and procedure (Collie et al., 2004). A possible solution is to evaluate athletes twice at baseline, and use the second test as the optimum baseline (Collie et al., 2004; Makdissi et al., 2001).

Computerized neurocognitive tests usually employ Reliable Change Indices (RCIs) which denote statistical differences between an individual's score on different assessment intervals (Collie,

Maruff et al., 2003). Adjusted RCIs are calculated to control for practice effects, whereby the predicted postinjury score equates the baseline score and the mean practice effect demonstrated by the normative sample and allows for meaningful interpretations of change (Parsons, Notebaert, Shields & Guskiewicz, 2009). Collie, Maruff, McStephen & Darby (2003) point out that alterations in neurocognitive assessment scores following MTBI, are indicative of cognitive change due to the injury and not as a result of the normal fluctuation in performance or measurement error. The ability to detect subtle changes in an athlete's neurocognitive test performance is, therefore, largely an issue of test reliability. *Reliability* is defined as the ability of a test to consistently measure a certain cognitive domain over a number of assessment periods, without being affected by practice effects (ImPACT, 2005). *Validity* is defined as the ability of a test to be sensitive to what it set out to test, i.e. to be able to distinguish concussed athletes from non-concussed athletes (ImPACT, 2005; Lezak et al., 2004). The validation of a neurocognitive test is a gradual process that incorporates results from numerous studies over extended periods, and one key aspect of validity is to correlate computerized test scores with traditional neuropsychological test scores to understand the presumed underlying measured constructs (Iverson, Lovell & Collins, 2002a).

Several versions of computerized neuropsychological programs exist and are based on neurocognitive functions that are most sensitive to impairment following MTBI, such as attention, memory, reaction time and processing speed (Podell, 2004; Sosnoff et al., 2007). Four computerized neurocognitive programs, currently detailed in the scientific literature and summarized in Table 5.5, are commercially marketed and available to athletic programs. They will be briefly described and include: the Automated Neuropsychological Assessment Metric (ANAM), the Concussion Resolution Index (Headminder CRI), CogState Sport and the Immediate Post-concussion Assessment and Cognitive Test (ImPACT), used in this study (Guskiewicz et al., 2004; Randolph, McCrea & Barr, 2005; Sosnoff et al., 2007).

Table 5.5 Computerized Neuropsychological Tests: Neurocognitive Components

Test	Subtests	Administering time
ANAM	Simple Reaction Time Visual Working Memory Sustained Attention Processing Speed and Working Memory Visual Matching Verbal Working Memory	15-20 minutes
CRI	Simple Reaction Time Complex Reaction Time Visual Recognition Processing Speed Memory	20-25 minutes
CogState Sport	Reaction Time Sustained Attention Divided Attention New Learning Short-Term Memory Working Memory Incidental Memory Adaptive Problem Solving Spatial Abilities Decision Making	15-20 minutes
ImPACT	Verbal Memory Visual Memory Visual Motor Speed Reaction Time Impulse Control	20-25 minutes

(Randolph, McCrea & Barr, 2005)

5.2.4.1 Automated Neuropsychological Assessment Metric (ANAM)

ANAM is the result of 30 years of computerized psychological test development to meet the requirement for consecutive testing and precision measurement of cognitive processing in a diversity of contexts, including sports medicine (Reeves, Winter, Bleiberg & Kane, 2007). ANAM includes 31 test modules and several companion functions designed for documenting demographic information and extracting summary information for research purposes. The pseudo-randomization procedures permit the design of multiple alternative forms from item sets and the use for performance monitoring and in repeated measures designs. The battery includes

measures of attention and concentration, simple reaction time, memory, cognitive processing efficiency, continuous performance (fatigue level), mental flexibility, spatial processing, and psychomotor performance (Cernich, Reeves, Sun & Bleiberg, 2007). The test does not provide the user with an index score and administration time will depend on the amount of subtests used, but will usually take about 15 to 20 minutes to administer. McCaffrey, Mihalik, Crowell, Shields & Guskiewicz (2007), found repeat testing attributed to a learning effect in a study.

The ANAM Sports Medicine Battery (ASMB) is a specialized subset intended for *pre*-season assessment of athletes and monitoring of concussion recovery. Tests in the ASMB evaluate sustained attention, mental flexibility, cognitive-processing efficiency, arousal/fatigue level, learning, recall, and working memory. The battery is able to accommodate repeated-measures testing and a pseudo-randomization procedure minimizes practice effects that can result because of repeat testing (Reeves et al., 2007). The ASMB reveals adequate concurrent validity in measuring similar traditional test constructs, including the COWAT, Digit Symbol, PASAT, Stroop Color Word Test and TMT A and B (Bleiberg et al., 2000; Woodard et al., 2002). Use of the RCIs, developed for the test, revealed high specificity to MTBI, but low sensitivity, although the mathematical processing subtest revealed 100% sensitivity to MTBI (Cernich et al., 2007; Parsons et al., 2009). Studies on high school athletes revealed impairments on reaction time and processing speed for up to six days, and memory deficits up to ten days following MTBI (Sim, Terryberry-Spohr & Wilson, 2008). Bleiberg et al. (2004) conducted a prospective study and found cognitive deficits following boxing-related MTBI compared to controls, with recovery occurring between three to seven days post MTBI.

5.2.4.2 Concussion Resolution Index (CRI)

The Concussion Resolution Index (CRI) is an online assessment tool, comprising multiple alternate forms to assess cognitive functions and track symptom resolution following sport-related MTBI (Headminder, 2003). It consists of six subtests measuring reaction time, visual recognition and visual motor processing speed (Barth, Broshek, Erlanger, Feldman, Freeman, Kaushik, et al., 2000; Erlanger, Feldman et al., 2003). Three factors are derived from these subtests: Simple Reaction Time (i.e., speed of motor response to a visual cue), Complex Reaction Time (i.e., speed of decision-making), and Visual Scanning/Processing Speed. A self-report

symptom checklist is included to track symptom resolution, as well as a short questionnaire to gather demographic information, concussion details and medical history.

Research (Echemendia et al., 2001; Erlanger et al., 2003) suggest that a reduction in visual motor processing speed may account for decreased test performance across a range of cognitive function such as memory, psychomotor speed and reaction time. Multiple alternate forms within subtests afford reliable assessment of change, relative to a completed *pre*-season assessment. Research studies showed good test-retest reliability as a measure of cognitive performance (Erlanger, Feldman et al., 2003), and measured similar neuropsychological constructs as in traditional tests, namely the Symbol Digit Modalities Test, WAIS-III Digit Symbol, Grooved Pegboard and the Trail Making Test.

The concurrent validity was examined during the test development phase, and CRI indices show moderate correlations with abovementioned traditional neurocognitive tests (Erlanger et al., 2003). The developers report 88% sensitivity in the identification of post-concussion symptoms (Erlanger, Saliba, Barth, Almquist, Webright & Freeman, 2001). The CRI correlates with traditional face-to-face tests that assess visual motor processing speed and is sensitive to post-concussion symptoms. The CRI measures response time more accurately than face-to-face tests, and it has the ability to statistically account for known practice effects over multiple test administrations. In addition, errors are tracked across multiple subtests, providing valuable speed versus accuracy data. Multiple equivalent alternate forms afford simple, reliable, serial assessment of change, relative to a baseline test completed by the athlete (Headminder, 2003).

5.2.4.3 CogState Sport

CogState Sport (previously termed CogSport) is a computerized web-based test battery consisting of playing cards, used as a visual test stimulus to evaluate changes in cognitive function, and includes a symptom checklist (CogState Sport, 2010; Schatz & Zilmer, 2003). It includes measures of reaction time, sustained and divided attention, new learning, short-term memory, working and incidental memory, adaptive problem solving, spatial abilities and decision-making (CogState Sport, 2010; Schatz & Zilmer, 2003). CogState Sport furnishes a report that provides scores for four cognitive domains: psychomotor processing speed, visual attention, visual

learning and memory, and verbal learning and memory (CogState Sport, 2010). A simple reaction time test from the CogState Sport battery (compared to the Digit Symbol Substitution and Trail making tests) showed sensitivity to sport-related concussive injuries (Makdissi et al., 2001). Measures of psychomotor function, working memory and learning were highly reliable and correlated with conventional neuropsychological tests of visual motor processing speed and attention (Collie, Maruff, Makdissi, McStephen, Darby & McCrory, 2003). Evidence of practice effects, were found over brief test intervals, with a further possibility of playing cards affecting outcome (Collie, Maruff, McStephen & Darby, 2003). Limitations related to the use of CogState Sport as an assessment tool included the scoring and analyzing of results via e-mail.

Developers of CogState Sport reported good test-retest reliability (Collie et al., 2003). Concurrent validation studies only found high correlations with the Digit Symbol Substitution Test for working memory and decision-making speed (Collie, Makdissi, Maruff, Bennell & McCrory, 2006; Grindel, Lovell & Collins, 2001). No studies on the specificity or sensitivity are available. No correlations were found between cognitive impairment on CogState Sport and self-reported concussion history, or with exposure to heading in a soccer study (Straumer-Naesheim, Andersen, Dvorak & Bahr, 2005).

5.2.4.4 Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT).

The Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) is a standardised computer based programme (Lovell, Collins, Podell, Powell, & Maroon, 2000) and is described in more detail in Chapter 6. ImPACT randomises test item presentation and includes five neurocognitive test modules providing composite scores for the neurocognitive functions typically affected by MTBI and include verbal and visual memory, visual motor speed and reaction time, with impulse control as a validity measure. The separate modules tap into similar neural mechanisms, and ImPACT shows 79.2 to 81.9% sensitivity to MTBI in terms of neurocognitive impairment or increased symptom reports (Broglio & Puetz, 2008; Broglio, Macciocchi et al., 2007; Schatz et al., 2006). A post concussive 21-item self-report symptom scale is included with percentile scores and the facility to incorporate RCIs as a statistical method to measure meaningful test score changes independent of practice effects (ImPACT, 2005; Iverson, Lovell & Collins, 2010).

ImPACT is designed to yield multiple types of information and to reflect the reality of individuals presenting with different neurocognitive deficits depending on a variety of factors including age and the biomechanics of the injury. ImPACT provides for the use of individual comparison standards that exemplified rate of change, which depended on intra-individual comparisons based on the administering of the same set of tests at spaced intervals (at least three times for the purposes of the current study). ImPACT is designed with multiple repeat testing situations in mind, especially to minimize practice effects and consists of near infinite random forms of alternating stimuli (Maroon et al., 2002). The test is administered within a brief period and is used clinically in the United States of America with the National Football League, the Major Baseball League and the National Hockey League (Iverson, Lovell & Collins, 2002b). The recently released online version of ImPACT shows high levels of sensitivity and specificity (Schatz & Sandel, 2013).

5.2.4.5 Synthesis of Neurocognitive Assessment in MTBI

It would appear that the cognitive functions of visual motor processing speed, reaction time and memory seem to be the most sensitive to the effects of diffuse brain injury (Binder et al., 1997; Frenchman et al., 2005). The tests isolated for use in the present study include the computerized neurocognitive test battery, ImPACT, which evaluates visual and verbal memory, reaction time and visual motor processing speed; and the traditional neurocognitive measure, the Purdue Pegboard Test that evaluates processing speed. ImPACT was identified for the purpose of the present study, as it appears to be the only computerized program that has been continuously revised (now in its fourth edition), and used extensively world-wide, particularly in the USA, for evaluating concussion in the sports arena (Field et al., 2003; Iverson et al., 2002a, 2002b; Lovell et al., 2003, 2004; McClincy et al., 2006; Mihalik et al., 2005; Pellman et al., 2006). A recent study on computerized neurocognitive testing for the management of sport-related concussions of high school athletes revealed that the vast majority of the respondent schools (93%) used ImPACT (Meehan III, d'Hemecourt, Collins, Taylor & Comstock, 2012). The Purdue Pegboard was identified for the purposes of the present study, as it appears to be sensitive to the effects of MTBI (Lezak et al., 2004; Strauss, Sherman & Spreen, 2006).

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Satz (1993) postulated that cognitive deficit would remain undetected (sub-threshold) due to the protector factor of a greater brain reserve capacity (for example, high IQ, high level of education and large brain size), until such time that a sufficiently *challenging* task presents itself. In line with the brain and cognitive reserve theory, neurophysiological studies illustrate that following MTBI there is an increase in the glucose metabolic activity rate in the brain (Giza & Hovda, 2004; Haier et al., 1988). As discussed previously, *f*MRI studies showed larger activation in MTBI patients being scanned whilst simultaneously taking sensitive neuropsychological tests due to a reduction in the brain's ability to process information efficiently, thereby placing additional demands (challenges) on the neural networks involved in successful task completion (Lovell & Collins, 2002; McAllister et al., 1999). Thus, cognitive deficit would not be detected were the neuropsychological measures not sufficiently robust to allow the individual to fall below the cognitive threshold level and present with symptoms.

CHAPTER 6

METHODOLOGY

This chapter reports on the methodological procedures of the present study. It begins with a review of the participants involved in respect of the selection criteria and the comparison groups selected for the purpose of the analysis. This is followed by a description of the demographic data including age, education level, estimated IQ and concussion history for each comparative group. The assessment procedures, measures and administration in respect of the biographical questionnaire, pre-morbid IQ estimate, and neurocognitive measures are subsequently addressed. Finally, attention is given to the data processing and analysis, followed by the statistical hypotheses for this study. All tables appear at the end of each relevant subsection.

6.1 PARTICIPANTS

6.1.1 Rugby Group

For the purposes of this research, the rugby group consisted of players of the first and second teams of a prominent rugby club participating at the Premier League Club level (designated the Rugby Group). Generally, it can be assumed that club rugby players in South Africa have participated in the sport since their early primary school years, through their high school years, totaling around eight to ten years *prior* to competitive adult level participation. Many of these Premier League Club rugby players may have additional years of exposure varying between one to ten years. Therefore, it can be assumed that they are a group that are vulnerable to concussive and subconcussive events during their years of participation in the sport.

Approval and co-operation was obtained from the rugby director and head coach of a South African rugby premier league club (see Appendix A, page 317). Rugby players of the first and second rugby teams were approached with a view to *pre-*, *mid-* and *post*-season neurocognitive evaluation, as some of these players often alternated between the two teams during the season.

The rugby players were briefed about the study and they provided written consent (see Appendix B, page 318).

The rugby players participating in the study included players ranging from 21 to 32 years of age and provided an initial sample of 33 (n = 33). Early in the season nine of the rugby players changed clubs, were drafted into provincial teams or left due to work related demands resulting in a reduction in the rugby sample available for *mid*-season assessment to 24 players (n = 24). Following the *mid*-season assessment interval the sample was further reduced for similar reasons, resulting in a rugby sample of 20 players (n = 20), who were included to participate in the *pre*-, *mid*- and *post*-season assessments for analyses.

During the season there were no individuals formally diagnosed with a concussion. However, under close observation and scrupulous perusal of video-taped footage by the researcher, five players were suspected of sustaining a concussion in that they were observed to have a head or body collision accompanied by concussion-related symptoms (three prior to *mid*-season and two following the *mid*-season assessment). These players were targeted for follow-up assessment with the neurocognitive measures. Four out of the five players returned to their *pre*-season baseline levels at the first post-concussion assessment follow-up. One of the five players, who was suspected of having a concussion prior to the *mid*-season, did not return to his *pre*-season level at the first post-concussion assessment follow-up, and therefore was followed up for a second time. Due to the possible confounding consequence of re-testing that may produce practice effects, this player was excluded from the cross-sectional analysis, in that he would have had the advantage of *two* additional post-concussion assessments at both the *mid*- and *post*-season assessment intervals. It was decided to leave the other four players in the sample despite the added advantage over the Non-Contact Sports controls of one additional assessment prior to the *mid*-season for one player and prior to the *post*-season for three players. This decision was made in the interest of not losing the full impact of the effects of reported and unreported mild concussive events over the season and further reducing the already small sample size. It was anticipated that while each of these players might give the Rugby Group a marginal practice advantage over the Non-Contact Sports Control Group (one rugby player at *mid*-season and three at *post*-season), any possible confounding effects of retaining them in the sample would not be substantive, and if present at all would be in the direction of obscuring deleterious outcome for

rugby players rather than any inflation of such outcome. It is important to note that there were no formally diagnosed concussions and the concussions were only suspected based on the researcher's sideline observation and video analyses.

In summary, for the independent cross-sectional (between group) and the dependent prospective (within group) analyses all players who completed all three assessment intervals at *pre*-, *mid*- and *post*-season were included, such that the following sample number applies: $n = 20$.

6.1.2 Non-Contact Sports Control Group

For the purposes of this research, the non-contact sports controls included a mixture of cricket and cycling athletes, for whom there is a relatively low incidence of concussion relative to rugby (designated the Non-Contact Sports Control Group). A literature search revealed no studies on cricket and concussion or on cycling and concussion. In a prospective study on Australian Cricket at first class level from 1995/1996 to 2000/2001 the rate of injury tended to be low when expressed per hour of play, with 1.9 injuries per 1000 player hours compared to rugby with 69 injuries per 1000 hours (Orchard, James, Alcott, Carter & Farhart, 2002). A ten year incidence study on professional club cricket players revealed that only 5.7% of the overall injury rate of 57.4 injuries per 1000 days of cricket play was to the head and neck (Leary & White, 2000). For inclusion in this study the non-contact sports controls were not to have participated in a contact sport since leaving high school and were furthermore not involved in any contact sport prior to that for more than three years. In the South African context, it was difficult to exclude players based on high school sport participation as rugby and cricket are played in different seasons and athletes often participate in both during their schooling.

It was methodological ideal to have a matched control, with the same age and the closest possible proximation to IQ, as well as demographically correct in order to gauge variation in performance across the *pre*-, *mid*- and *post*-season assessment intervals. In order to acquire an equivalent Non-Contact Sports Control Group the Chief Executive Officer (CEO) and Head Coach of a cricket team of a South African Cricket Union First League Club were approached and informed of the nature of the study. They in turn approached the players who volunteered their willingness to participate in the study. Following this the CEO was given a list containing biographical

requirements of athletes to participate in the study that were based on the overall biographical profile of the Rugby Group (for example six white English speaking 24 year olds, two with Grade 12 and four with tertiary level education). The CEO provided the researcher with a comprehensive list with contact details of 32 players that fitted the various profiles. The researcher contacted all of these cricket players. Of these, 24 confirmed their willingness to participate, and made appointments for assessment. Following this, in order to make up the deficit in number of participants required for the Non-Contact Sports Control Group, the researcher approached individual competitive cyclists. The Non-Contact Sports Control Group were briefed about the study and provided written consent (see Appendix B, page 318).

The total number of cricket players and cyclists participating in the study included players ranging from 21 to 32 years of age and provided an initial Non-Contact Sports controls sample of $n = 32$. Early in the season eight of the cricket players left for overseas clubs, were drafted into provincial teams or left due to work and private reasons resulting in the Non-Contact Sports controls sample available for repeat measures analysis of $n = 24$. Following the *mid*-season assessment the sample was further reduced for similar reasons, resulting in a sample $n = 22$, who were included to participate in the *pre*-, *mid*- and *post*-season assessments for analyses.

In summary, for the independent cross-sectional (between group) and the dependent prospective (within group) analyses all players who completed all three assessment intervals at *pre*-, *mid*- and *post*-season were included, such that the following sample number applies: $n = 22$.

6.1.3 Sampling Details of Comparative Groups

In this subsection the sampling details that apply to the independent cross-sectional and dependent prospective group analyses are described.

6.1.3.1 Pre-, Mid- and Post-Season Independent Cross-Sectional Analysis Comparative Groups

(i) The *Rugby* Group (*n* = 20) was made up of individuals participating in the first and second team of a premier league rugby club.

(ii) The *Non-Contact* Sports Control Group (*n* = 22) was made up of individuals participating in club level cricket (*n* = 15) and cycling (*n* = 7).

6.1.3.2 Pre- Versus Mid- Versus Post-Season Dependent Propspective Analyses Comparative Groups

(i) The *Rugby* Group (*n* = 20) in this *pre*- versus *mid*- versus *post*-season analysis was made up of individuals participating in the first and second team of a premier league rugby club.

(ii) The Non-Contact Sports Control Group (*n* = 22) in this *pre*- versus *mid*- versus *post*-season analysis was made up of individuals participating in cricket (*n* = 15) and cycling (*n* = 7).

6.2 DEMOGRAPHIC DATA

In this subsection the demographic data that apply to the independent cross-sectional and dependent prospective group analyses are described. In order to establish between-group homogeneity, group mean comparisons were calculated for the variables known to have an effect on cognitive test performances, and include age, educational level, estimate IQ and concussion history (Lezak et al., 2004).

6.2.1 Language, Race, Age and Educational level

There is a relatively even distribution across the two groups as these variables were broadly controlled for by virtue of the Non-Contact Sports Control Group being as closely as possible matched to the Rugby Group. The two groups were made up almost exclusively of white English first language or Afrikaans first language athletes who were fluent in English. In addition there were two relatively educationally advantaged black participants in each of the Rugby and Non-Contact Sports Control groups, who had a background of attendance at one of the traditionally white South African schools rather than a township school and are therefore proficient in English. From South African research (Shuttleworth-Edwards, Kemp, Rust, Muirhead, Hartman & Radloff, 2004), it can be extrapolated that this marginal difference in racial composition was of no significance. Consistently across research studies broadly equivalent performance have been demonstrated on cognitive tests in both the verbal and non-verbal areas (including tests of visual motor processing speed), for black African, first language, and white English, first language, groups with relatively advantaged South African education (Shuttleworth-Edwards, Kemp et al., 2004; Shuttleworth-Jordan, 1996).

In all instances the data in respect of the demographic variables were calculated at the time of the *pre*-season assessment. The *age* of each participant was documented in years. The *educational level* of each participant was calculated in years according to the number of successfully completed grades at school (grade 12 being the maximum) and additional years of successfully completed tertiary education. These age and education characteristics of the Rugby and the Non-Contact Sports Control Groups are summarised in Table 6.1. There are no significant differences between the means for any of the comparative sports groups for the variables of age ($p = 0.636$), and years of education ($p = 0.656$), suggesting that all comparative groups are equivalent for both age and years of education at each of these assessment intervals. The age range is 21 to 32 years for all groups.

6.2.2 IQ Index

The Rugby and Non-Contact Sports Control Groups were further measured against a variable that is considered to be influential on cognitive performance, estimate of IQ. An estimate of a *Full*

Scale Intelligence Quotient, reported in IQ points (standardization mean = 100), is based on the Wechsler Adult Intelligence Scale –III (WAIS-III) Picture Completion and Matrix Reasoning Scaled Scores in conjunction with use of the Oklahoma Pre-morbid Intelligence Estimate. Krull, Scott & Sherer (1995) devised the Oklahoma Premorbid Intelligence Estimation (OPIE) formula that uses the two mentioned WAIS-III scores along with demographic data. The OPIE-3P formula is considered to be highly significant and accurate (Schoenberg, Scott, Duff & Adams, 2002), and is described in more detail under the section on measures to follow below. Table 6.1 reveals that there is no significant difference in estimated IQ between the Rugby and Non-Contact Sports Control Groups ($p = 0.181$). The Estimated IQ score for the total sample ranged from 90 to 118.

6.2.3 Concussion History

A retrospective *concussion history* was obtained from each participant, as to whether or not they were previously formally diagnosed with a concussion. Each participant recorded the number of prior concussions on the ImPACT biographical questionnaire and additional concussion history information was elicited and documented on a pencil and paper biographical questionnaire. The information regarding the concussion history was included for descriptive analyses in the individual player profiles, without focusing on severity, and not for inclusion in the comparative group analyses. Given the confusion regarding concussion grades, no differentiation was made in terms of severity of the self-reported sports-related concussions, as there is a poor relationship between subjective complaint and objective measures of impairment (Bernstein, 1999). Therefore, mention is only made of the number of reported concussions. Details of these are tabled in Table 6.1 below. The rugby players in the total sample group reported no concussions other than sports related concussions and so this factor as an exclusion criterion was deemed unnecessary.

The group mean comparisons of the prior concussion history were analysed and independent two-sampled *t*-tests were used for the Rugby versus Non-Contact Sports Control Group comparisons. The Rugby Group revealed a long-term history of significantly more reported concussions than the Non-Contact Sports Control Group, with an effect size that is of clinical relevance (i.e. CI does not contain zero) at *post*-season, all in the direction of the Rugby Group

sustaining more concussions (Table 6.1) ($p = 0.005$, $d = 1.01$). The implication is that the Rugby Group is characterised by a substantial number of rugby players with the critical occurrence of a history of multiple concussions of clinical relevance, whereas with the Non-Contact Sports Control Group this is not an observable trend.

Perusal of Table 6.1 reveals that on average the Rugby Group reported in excess of one concussion with a standard deviation approaching two concussions at ($M = 1.85$, $SD = 2.25$), implying that a substantial number of individuals had in excess of three concussions. In comparison with the Non-Contact Sports Control Group who reported averages of less than one concussion ($M = 0.13$, $SD = 0.34$), implying that there was a significant number of the Non-Contact Sports Control Group that had not sustained even one concussion, and virtually none with more than one concussion. Specifically, 45% of the Rugby Group reported 2+ concussions versus 9% of the Non-Contact Sports Control Group. 18% of the Rugby group reported 2+ concussions, 12% of the Rugby Group reported 3+ concussions, 9% of the Rugby Group reported 4+ concussions and 6% of the Rugby Group reported 6+ concussions.

Table 6.1 Demographic Data and History of Prior Concussions: Rugby versus Non-Contact Sports Control Groups

	Rugby (n = 20)		**Non-Contact (n = 22)**		***t-value***	***Effect size d (95% CI)***	***p-value***
	Mean	**(SD)**	**Mean**	**(SD)**			
Age	26.40	(3.19)	25.86	(4.00)	0.477	0.15 (-0.46, 0.75)	0.636
Years Education	13.55	(1.70)	13.18	(3.29)	0.449	0.14 (-0.47, 0.74)	0.656
Estimated IQ[1]	103.95	(8.49)	107.18	(6.84)	-1.363	-0.42 (-1.03, 0.19)	0.181
No of concussions	1.85	(2.25)	0.13	(0.34)	3.025	1.01 (0.31, 1.71)	0.005**

[1]*Note.* Control for estimated Full Scale IQ established on the basis of WAIS-III Picture Completion and Matrix Reasoning Scaled Scores using the OPIE-3 Estimation Formula.
***** $p \leq .05$, ****** $p \leq .0.01$, *two-tailed*

6.2.4 Individual Player Profiles

The possibility of any player having sustained or being suspected of having a concussive injury provided the basis for individual follow-up. There were no players formally diagnosed with a

concussion over the season, but five players (n = 5) were suspected of having a concussion on the basis of the researcher's sideline and video observations. These five players provided the sample for a set of individual case analyses. More detailed profiles of the demographic data that apply to these five individual player case analyses were integrated into the introductory sections of each of the case analyses in Chapter 8, and are not replicated here.

6.3 PROCEDURE

6.3.1 Rugby Group Procedural Aspects

The study took place over a period of one rugby season during 2005 (a period of approximately seven months) with three assessment intervals, starting at February/March (*pre*-season), June/July (*mid*-season) and October (*post*-season). It was considered that *pre*-season baseline assessments prior to contact training would ensure at least a four months period during which the Rugby Group was not involved in any contact sport, and this approach provided the comparison standard should a player be injured during the season (Lovell et al., 2004). This was done to target persistent cognitive compromise amongst the Rugby Group relative to the Non-Contact Sports Control Group due to both the reported and unreported concussive and sub-concussive events sustained during ten to twenty years of exposure to contact sport.

Mid-season assessments were conducted in the middle of the season, as evenly spaced as possible between the *pre*- and *post*-season assessment intervals (providing an approximate four month test-retest interval period). *Post*-season assessments were conducted at the end of the season with a view to identify residual deficits of previous, and/or any newly acquired acute or sub-acute effects of often unreported concussive or sub-concussive events sustained over the rugby season. The critical issue was the chronicity of these deficits (Binder et al., 1997), and in order to address this, the three assessment intervals were incorporated to determine if the Rugby Group was significantly different from the Non-Contact Sports Control Group across one competitive season.

The researcher conducted all the assessments to ensure standardized test instructions. Participants were tested individually at the rugby club, and the allocated venue was out of bounds

during the assessments in order to control for any possible environmental distracters. Assessments were done at the end of a working day, but prior to practice sessions in the evenings, and 45 minutes were allocated for each of the individual assessments. It is recommended that ImPACT (or any neurocognitive assessment) is not administered following any exertion activity (match or practice session), as maximal exercise prior to administration affects immediate and delayed verbal memory scores negatively (Covassin, Weiss, Powell & Womack, 2007; Lovell & Collins, 2002). Prior to assessment, all participants were provided with information regarding the purpose of the assessment, and confidentiality of all test results was emphasized. At the end of the ImPACT test the participants were given the opportunity to report on the presence of any possible confounding external distractions that could have negatively affected their performances. These factors related to the clarity of the instructions given on the ImPACT test, technical or computer problems, and environmental problems which included possible distracters such as environmental noise.

6.3.2 Non-Contact Sports Control Group Procedural Aspects

The study took place over a period of one cricket season during 2005 and 2006 (a period of approximately seven months) with three assessment intervals, starting at August/September (*pre*-season), January/February (*mid*-season) and May/June (*post*-season). *Mid*-season assessments were conducted in the middle of the season, as evenly spaced as possible between the *pre*- and *post*-season assessment intervals (providing an approximate four month test-retest interval period). The researcher conducted all the assessments to ensure standardized test instructions. Participants were tested individually at the cricket club (cricketers) or at the researcher's office (two cricketers and the cyclists), and venues were out of bounds during assessments to control for any possible environmental distracters. As with the rugby players, assessments were done prior to sports practise in the evenings, as players had work commitments during day time, and 45 minutes were allocated for the assessment. It is recommended that the neurocognitive assessment is not administered following any exertion activity (match or practice session), as maximal exercise prior to administration may have a negative effect on scores. Prior to assessment, all participants were provided with information regarding the purpose of the assessment, and confidentiality of all test results was emphasized. At the end of the ImPACT test the participants were given the opportunity to report on the presence of any possible confounding external

distractions that could have negatively affected their performances. These factors related to the clarity of the instructions given on the ImPACT test, technical or computer problems, and environmental problems which included possible distracters such as environmental noise.

6.3.3 Tackling Procedural Aspects

The study took place over a period of one rugby season during 2005 (a period of approximately seven months), starting in February and concluding in October. The Premier Club Rugby League season consists of three *pre*-season 'friendly' games, 19 'league' games; with two additional games should the team reach the semi-finals and finals, averaging a total of 24 games played during the season per team, and these 24 games played were video recorded for analyses. During the season, the researcher attended all the games played by the first and second team of the participating rugby club and videotaped a total of 48 games (24 games for each of the two teams respectively), excluding the two weekly practice session (not videotaped). The researcher reviewed these videotapes within 24 hours of the game played, and players with a possible concussion-risk were identified for follow-up the following day and these individual incidents were prospectively recorded on a separate spreadsheet for further analyses. The video recordings of these games enabled the researcher to create an events list (made up of different types of tackles) using the Dartfish TeamPro (Dartfish, 2005), one of the more advanced computerized notation systems (discussed in more detail under 6.4.4).

6.3.4 Individual Player Procedural Aspects

As indicated above, the researcher was present at all the games (matches and weekly practices). During the season, five players were observed to have a head or body collision with suspected concussion-related symptoms (three prior to *mid*-season and two following the *mid*-season assessment). The players were identified from sideline observations and/or video recordings of the games played. They were targeted for follow-up assessment with the neurocognitive battery within 72 hours following the suspected concussion. All of the individual players were compared with each player's own *pre*-season baseline assessment scores, the normative scores, and the Non-Contact Sports control mean score for each assessed measure. Four of the five players returned to *pre*-season levels after one follow-up assessment. The fifth player, who was

suspected of a concussion prior to the *mid*-season, did not return to *pre*-season and he was scheduled for follow-up assessments on post-injury days 9 and 16.

6.4 MEASURES AND ADMINISTRATION

Measures for this study consisted of (i) a biographical questionnaire, (ii) a measure of estimated pre-morbid intellectual functioning (WAIS-III Picture Completion and Matrix Reasoning subtests); (iii) neurocognitive measures commonly used for the assessment of concussion and MTBI (the standardised ImPACT computerised neurocognitive test battery with four cognitive modalities: Verbal Memory, Visual Memory, Visual Motor Speed and Reaction Time); a traditional neuropsychological motor co-ordination test, the Purdue Pegboard (Preferred, Non-Preferred, Both and Assembly); and (iv) a software programme (Dartfish Pro) to analyze tackling based on the video recordings. The researcher administered all measures. The biographical questionnaire and tests of general intellectual ability were administered at one sitting at the beginning of the season. The neurocognitive measures (ImPACT and Purdue Pegboard) were administered at one sitting at each of the three assessment intervals. The video recordings were taped continuously throughout the season at all the games played. Each of these measures will be discussed in more detail below.

6.4.1 Biographical Questionnaire

(Administered once at *pre*-season only)

The biographical questionnaire (see Appendix C, page 319) was administered to all participants *prior* to the *pre*-season assessment interval. It was designed to provide the researcher with (i) the biographical information of age, language, level of education, occupation and estimate IQ, (ii) medical and psychiatric history, and (iii) concussion history.

6.4.2 Test of General Intellectual Ability

(Administered once at *pre*-season only)

Two subtests of the Wechsler Adult Intelligence Scale were administered individually *prior* to the *pre*-season assessment interval. Research suggests that intellectual functioning affects

neuropsychological performance, and that to only control for level of education does not control for the variability in intellectual capacity (Lezak et al., 2004; Macchiocci & Barth, 2004). Therefore, when athletes with low pre-morbid intellectual functioning are assigned to control groups this may obscure the overall assessment results between the different groups, with the Non-Contact Sports Control Group appearing unimpaired by comparison. Testing for pre-morbid intellectual ability enabled the researcher to tap into the performance of the Rugby and Non-Contact Sports Control Groups on the parameters of acquired knowledge, thereby providing a comparative IQ potential index between the two groups as a *control* variable. In that estimate IQ is a control variable, it was considered sufficient to administer the test at *pre*-season only. However, to be an adequate test of pre-morbid ability, it must be reliable, correlate highly with IQ in the normal population and be largely resistant to the effects of neurological deficits (Martin, 1998).

The efforts to improve estimates of premorbid cognitive ability yielded various formulas that combine raw assessment scores with demographic variables. Krull et al. (1995) devised the Oklahoma Premorbid Intelligence Estimation (OPIE) formula that uses WAIS-R scores along with age, education, race and regional data and generated formulas for predicting VSIQ, PSIQ and FSIQ. For this reason the OPIE-3P formula was used for estimating pre-morbid FSIQ as it was considered to be noteworthy and exact (Schoenberg, Scott, Duff & Adams, 2002), and the correlations between predicted FSIQ and the actual FSIQ of the WAIS-R standardization population were high ($r = 0.87$). Lezak et al. (2004) reported on the high predicted and actual correlations ($r = 0.87$, $r = 0.78$, and $r = 0.87$ for Verbal-, Performance-, and Full Scale IQ scales respectively). The OPIE-3P uses data from the WAIS-III standardization population to develop regression algorithms that include demographic variables and the Matrix Reasoning and Picture Completion raw scores. The advantage of using demographic variables is that they are independent of the individual's current cognitive state (Martin, 1998). Table 6.2 present details of the OPIE-3P formula.

Table 6.2 OPIE-3P Estimation formula using Matrix Reasoning and Picture Completion. SEest = 7.93

FSIQ =	29.280 + 1.469(MR raw score) + 1.242(PC raw score) + 0.332(Age) + 3.04(Education) + 1.025 (Race) + 0.557 (Region) – 1.278 (Gender)

Coding	**Variables**
Age:	In years
Race;	1 = African, 4 = Caucasian
Education:	1 = 0-8 years; 2 = 9-11 years; 3 = 12 years; 4 = 13-15 years; 5 = 16+ years
Gender:	1 = male
Region:	2 = Gauteng (Northern region of South Africa)[l]

(Schoenberg, Scott, Duff & Adams, 2002)
[l]Note: All the players were from the same region

The Wechsler Adult Intelligence Scale (WAIS) – III Picture Completion (which measures visual organization and reasoning abilities, as well as remote memory and general information) and the Matrix Reasoning (which measures classification, analogy and serial reasoning abilities) subtests, were included in the analysis because of their demonstrated reliability and resistance to neurological insult (Wechsler, 1997). The Picture Completion subtest of the WAIS-III is designated as one of the most resilient and sturdiest indicators of pre-morbid ability in the Wechsler scales (Krull, Scott & Sherer, 1995) and seem to be relatively resistant to brain damage (Lezak et al., 2004). A description of each subtest and its application is cited from the Wechsler Manual (1997).

Picture Completion.

The WAIS-III Picture Completion subtest is a measure of acquired knowledge, visual organisation and reasoning abilities and consists of a set of colour pictures of common objects and settings, each missing an important part that the participant must identify. In accordance with the instruction manual, the participant is instructed to indicate or verbalise the missing part on each picture in an assessment time of approximately 10 to 15 minutes (Wechsler, 1997). The discontinue criterion is five consecutive scores of zero. The Picture Completion subtest is scored according to the WAIS-III manual with scores of either 1 or 0 points for either a correct or incorrect answer.

Matrix Reasoning.

The WAIS-III Matrix Reasoning subtest is a measure of visual motor processing speed and abstract reasoning skills (pattern completion, classification, analogy and serial reasoning) and consists of a series of incomplete grid patterns. In accordance with the instruction manual, the participant is instructed to complete the series by indicating or verbalizing the number of the correct response from five possible options (Wechsler, 1997). This test has no time limit but takes approximately 15 to 20 minutes, but the discontinue criterion is four consecutive scores of zero or four scores of zero on five consecutive items. The Matrix Reasoning subtest is scored according to the WAIS-III manual with scores of either 1 or 0 points for either a correct or incorrect answer.

6.4.3 Neurocognitive measures

(Administered at all three assessment intervals and individually post-concussive)

For the purpose of this study two neurocognitive tests, Immediate Post-Concussion Assessment and Cognitive Test (ImPACT) and the Purdue Pegboard, were included and administered in that order (ImPACT first and followed by the Purdue Pegboard), at each of the three assessment intervals, *pre*-, *mid*- and *post*-season.

6.4.3.1 Immediate Post-Concussion Assessment and Cognitive Test (ImPACT)

ImPACT is a standardised computerised programme developed in the 1990s by Drs. Mark Lovell and Joseph Maroon and became web-based in 2006. For this study the third version, ImPACT 3.0a, was used. The test was completed individually in the standardized automated manner on the researcher's laptop using the baseline version at *pre*-season, the first follow-up test version at *mid*-season and the second follow-up test version at *post*-season (in the event of a concussion the third and fourth follow-up test versions were used). The ImPACT test consists of three parts: (i) Sport and health history, (ii) Symptom Scale and (iii) Neurocognitive test battery. For this study, the symptom scale was not included for interpretation.

Sport and health history

The first section of ImPACT requires the individual to supply basic demographic and descriptive information through a series of easy to follow instructional screens. The individual performs this task via the keyboard and utilizes an external mouse to select responses on the screen. This section asks the individual to answer questions regarding height, weight, sport, position, concussion history, history of learning disabilities and other important descriptive information.

Symptom Scale

This section of ImPACT requires the individual to rate his current symptom status by means of a 7-point Likert-scale. 22 concussive symptoms are included: headache, nausea, vomiting, balance problems, dizziness, fatigue, trouble falling asleep, sleeping more than usual, sleeping less than usual, drowsiness, sensitivity to light, sensitivity to noise, irritability, sadness, nervousness, feeling more emotional, numbness or tingling, feeling slowed down, feeling mentally foggy, difficulty concentrating, difficulty remembering and visual problems (Lovell & Collins, 2002). The individual performs this task by using the external mouse. Individual scores are provided as well as a graphic representation of the symptom total score. For this study, the symptom scale was not included for interpretation.

Neurocognitive test battery

ImPACT consists of six modules designed to simultaneously evaluate multiple cognitive domains that have been shown to be sensitive to the effects of concussion in prior research (Collins, Iverson, et al., 2003; Lovell et al., 2004; Lovell & Collins, 2002; Schatz et al., 2006). These include memory (verbal and visual), attention span (sustained and selective), reaction time to one-hundredth of a second across individual test modules and visual motor processing speed. The test battery (consisting of five different and alternating forms for the Word and Design Memory stimuli) is designed to allow for the automatically randomisation of stimuli each time the test is administered, thereby improving reliability across multiple administrations and circumventing typical practice effects (Lovell et al., 2004; Maroon et al., 2002; Mihalik, McCaffrey, Rivera, Pardini, Guskiewicz, Collins & Lovell, 2007; Withnall et al., 2005). A

description of each module (Table 6.3) and the neurocognitive domain measured is cited from the Complete Handbook for Concussion Management and Clinical Interpretation Manual for ImPACT (2007).

Table 6.3 ImPACT Neurocognitive Test Modules

Test Module	Neurocognitive domain measured
Word Memory	Verbal Recognition and Delayed Memory
Design Memory	Visual Recognition and Delayed Memory
X's and O's	Visual Working Memory and Visual Motor Speed
Symbol Match	Visual Processing Speed, Learning and Memory
Color Match	Reaction Time, Impulse Control/Inhibition and Visual Motor Speed
Three Letters	Working Memory and Visual Motor Speed

(ImPACT Manual, 2007)

Module 1: Word Memory.

This module evaluates attention processes or verbal recognition memory and utilizes a word discrimination paradigm. ImPACT presents twelve target words from a word list (here are five different forms of the word list) twice in order to facilitate learning of the list. On completion of the second presentation of the list, the individual recalls the 24-word list that includes twelve target words and twelve non-target words (chosen from the same semantic category as the target word). The individual responds by clicking the yes or no buttons on the screen and scores are provided both for correct "yes" and "no" responses. Delay Condition**:** Following the administration of all other test modules (approximately 20 minutes), the individual is re-tested for recall via the same described method and scoring procedure.

Module 2: Design Memory.

This module evaluates attention processes and visual recognition memory and utilizes a design discrimination paradigm. Twelve target designs (there are five different forms of this task) appear twice to facilitate learning. At the end of the second presentation of the list, the individual is re-tested for recognition via the presentation of 24-designs consisting of twelve target designs and twelve non-target designs (target designs that had been rotated in space). Similar to the word recognition task, the individual responds by mouse-clicking the yes or no buttons on the screen and individual scores are provided both for correct "yes" and "no" responses. In addition, a total percent correct score is provided. Delay Condition: Following the administration of all other test modules (approximately 20 minutes), the individual is re-tested for recall via the same described method and scoring procedure.

Module 3: X's and O's.

This module measures visual working memory as well as visual processing speed and consists of a visual memory paradigm with a distracter task. The individual practices the distracter task prior to presentation of the memory task. The distracter is a choice Reaction Time test during which the individual clicks the left mouse button if a **blue** square appears and the right mouse button when a **red** circle appears. On completion of the task, the memory task is presented. For each of the trials of the memory task, a screen is displayed for 1.5 seconds that has a computer generated random assortment of illuminated X's and O's. Immediately after the presentation of the three X's or O's, the distracter task re-appears on the screen. Following the distracter task, the memory screen (X's and O's) re-appears and the individual is asked to click on the previously illuminated X's and O's. Scores are provided for correct identification of the X's and O's (memory), Reaction Time for the distracter task, and number of errors on the distracter task. For each administration of ImPACT, the individual completes four trials.

Module 4: Symbol Matching.

This module evaluates visual processing speed, learning and memory. A screen with nine well-known symbols (triangle, square, arrow, etc.) is presented with a number button from 1 to 9

below this grid. The individual clicks the matching number as quickly as possible and remembers the symbol/number pairings. Following the completion of 27 trials, the symbols disappear from the top grid. The symbols again appear below the grid and the individual recalls the correct symbol or number pairing by clicking the appropriate number button. This module provides an average reaction time score and a score for memory.

Module 5: Color Match.

This module represents a choice Reaction Time task and measures impulse control or response inhibition. First, the individual responds by clicking the presented red, blue or green button, to ensure that color blindness does not affect subsequent trials. Next, a word is displayed on the screen in the same color ink as the word (e.g. **RED**), or in a different color ink (**GREEN** or **BLUE**). The individual clicks in the box as quickly as possible *only* if the word matches the color ink. In addition to providing a reaction time score, this task also provides an error score.

Module 6: Three letters.

This module measures working memory and visual motor response speed. First, the individual practices with a distracter task, which consists of 25 numbered buttons on a 5 x 5 grid. The subject clicks as quickly as possible on the numbered buttons backwards starting with 25 and ending with 1. On completion of the initial practice task, three consonant letters are displayed on the screen. Immediately following the display of the three letters, the numbered grid re-appears and the individual clicks the numbered buttons backwards (25 to 1) as quickly as possible. After a period of 18 seconds, the numbered grid disappears and the individual recalls and types the three letters. Both the number placement on the grid and letters displayed are randomized for each trial. This module produces a memory score (total number of correctly identified letters) and a score for the average number of correctly clicked numbers per trial from the distracter test on the five trials.

The automatically generated data for the four relevant composite scores (Verbal Memory, Visual Memory, Visual Motor Speed and Reaction Time) are extracted for the purpose of this research

(Table 6.4). An Impulse Control composite score serves as an indicator of test validity, but was not included in this study.

The Verbal Memory composite score represents the average percentage correct for word recognition (immediate and delayed), a symbol number match task, and a letter memory task (with an interference task). The Visual Memory composite score comprises the average percentage correct for immediate and delayed visual design memory and short-term spatial memory (with an interference task). The Visual Motor Speed composite score represents the weighted average of three tasks performed as interference tasks for the memory paradigms. The Reaction Time composite score represents the average response time on a choice reaction time task, symbol match task and a colour match task.

Table 6.4 Computation of ImPACT Composite Scores

Composite Scores	Contributing scores
Verbal Memory	Word Memory (immediate and delayed), Symbol Match memory score Three Letters memory score
Visual Memory	Design Memory (immediate and delayed) X's and O's
Visual Motor Speed	X's and O's (mean correct distracters), Symbol Match (mean correct responses) Three letters (number of correct numbers correctly counted)
Reaction Time	X's and O's (mean counted correct reaction time), Symbol Match (mean weighted reaction time for correct responses) Colour Match (mean reaction time for correct responses)

(Iverson, et al. (2002b)

6.4.3.1.1 Reliability And Validity Of Impact

The assertion that ImPACT is a reliable and valid tool in concussion assessment has been controversially questioned and discussed in the literature (Kirkwood, Randolph & Yeates, 2009; Randolph, 2011; Randolph et al., 2005). There is adequate test-retest reliability for the ImPACT Verbal memory, Visual Memory and Visual Motor Speed composites that appear sensitive to

MTBI (Iverson, Lovell & Collins, 2003, 2005; Schatz, Pardini, Lovell & Collins, 2006). Higher intra-class correlation (ICC) values, than those provided by other concussion assessment measures, are found for the Reaction Time and Processing Speed composites and a relatively high value for Visual Memory (Pardini & Lovell, 2005). The cumulative damage that may result from repetitive cerebral concussions have been documented, along with more recent research, utilising ImPACT's original version, suggesting that the Memory composite score is very sensitive to cumulative effects (Collins et al., 1999; Gronwall & Wrightson, 1975; Gronwall & Wrightson, 1974; Iverson, Lovell & Collins, 2002). In a study by Schatz (2010), ImPACT 3.0 shows adequate test-retest reliability for *pre*-season assessment two years apart with ICCs for Visual Memory (0.65), Visual Motor Speed (0.74) and Reaction Time (0.68). In a more recent study by Elbin, Schatz & Covassin (2011), ICCs for the ImPACT online version indicated that Visual Motor Processing Speed (0.85) was the most stable composite score, followed by Reaction Time (0.76), Visual Memory (0.70), and Verbal Memory (0.62). Therefore, the online ImPACT baseline is a stable measure of neurocognitive performance across a one-year time for high school athletes, and these reliability data for online ImPACT are higher than the 2-year ICCs previously reported from the desktop version.

A few studies failed to reveal sensitivity for chronic neuropsychological effects of MTBI (Collie, McCrory & Makdissi, 2006; Iverson, Brooks, Lovell & Collins, 2006; Pontifex et al., 2009). Randolph *et al.* (2005) challenged the test-retest reliability of computerised neuropsychological tests for test intervals exceeding months, and Broglio, Ferrara, Macciocchi, Baumgartner & Elliot (2007) found no computerised neuropsychological test, including ImPACT, that exceeded a good test-retest reliability (0.75) for a test interval of 45 days. ImPACT might be more sensitive to neurocognitive changes immediately following MTBI, and cognitive tests that requires executive or cognitive control appears more sensitive than ImPACT in eliciting chronic neurocognitive impairment (Pontifex et al.. 2009).

The *validity* of ImPACT, therefore, refers to this neurocognitive test battery being a valid measure of neurocognitive and neurobehavioral effects of sport-related concussion. The Memory scale shows validity in terms of distinguishing concussed players from non-concussed controls, with an established correlation between Visuospatial Memory Test, the ImPACT Visual Motor Speed and Reaction Time composites, and Trail Making Test (TMT) A and B and a traditional

test used routinely in sport concussion research, the Symbol Digit Modalities Test (SDMT), (Iverson, Lovell & Collins, 2002b; Iverson, Gaetz, Lovell & Collins, 2005; Lovell et al., 2003; Schatz, Pardini, Lovell, Collins & Podell, 2006). Reliable Change Indices (RCIs) have been calculated from test-retest studies for the .80 confidence level to account for measurement error and therefore adjusted each score for practice effects secondary to multiple exposures to the specific test, although further varying time interval studies are needed (Iverson, Lovell & Collins, 2003; Van Kampen et al., 2006).

The American neurocognitive normative data for ImPACT have been empirically validated for English speaking males in South Africa, including non-white males with an advantaged education that is proficient in English (Shuttleworth-Edwards & Whitefield-Alexander, 2013). This cross-cultural norming study is supported by another study that found no significant difference between African Americans and White Americans on the neurocognitive and symptom composites at *pre*-season (Kontos, Elbin, Covassin & Larson, 2010). ImPACT offers sound construct validity with cultural equivalence for *pre*-season assessments, but Kontos et al. (2010) reiterate the need for further research following MTBI.

Divergent validity of ImPACT is demonstrated by an analysis of the relationship between different test components, which show non-significant correlations and overall demonstrate a sensitivity and specificity of approximately 82% and 89% respectively (Schatz et al., 2006). In contrast, a few studies failed to reveal this sensitivity of ImPACT to the chronic neuropsychological effects of MTBI, suggesting ImPACT may be more sensitive to neurocognitive changes immediately following MTBI, rather than subtle chronic deficits that may persist (Broglio, Ferrara, Piland, Anderson, & Collie, 2006; Collie, McCrory, & Makadissi, 2006; Iverson, Brooks, Lovell, & Collins, 2006; Pontifex et al., 2009).

Possible limitations of ImPACT are (i) the Visual Memory test designs have abstract rather than geometric lines found in traditional neuropsychological tests, (ii) the Verbal Memory composite is not a true verbal memory measure in that items are visually and not orally presented, and (iii) the Verbal Memory tasks are based on recognition which is less sensitive to brain damage than memory recall (Lezak et al., 2004). Therefore, traditional neurocognitive tests that incorporate these crucial aspects might reveal greater sensitivity to MTBI than ImPACT alone.

6.4.3.2 Purdue Pegboard

Measures of hand-motor reaction speed, as the Purdue Pegboard provides one of the most sensitive indexes of cognitive changes following brain injury, and the Grooved Pegboard correlates strongly with almost all timed assessments on the Concussion Resolution Index (Collie et al., 2003; Erlanger et al., 2003; Lezak et al., 2004). The Purdue Pegboard (Purdue Pegboard, 2002; Tiffin, 1968) is a standardised measure of dexterity for three types of activity: (i) gross movement of arms, hands, and fingers; (ii) fingertip dexterity, and (iii) speed performance, where the individual places pegs into board holes with rapid, skilful, controlled manipulative movements (Asikainen, 2001). Purdue Pegboard has been reported to be sensitive to the effects of cognitive impairment and is therefore a good measure of diffuse brain injury following concussion (Lezak et al., 2004; Strauss et al., 2006). The Purdue Pegboard differentiated between good recovery and moderate disability (Asikainen, Nybo, Müller, Sarna & Kaste, 1999). The test was completed individually in the standardized manner at the *pre-*, *mid-* and *post*-season assessment intervals.

The Purdue Pegboard consists of four tasks (Preferred, Non-Preferred, Both, and Assembly) performed on the same board equipped with pins, collars, and washers which are located at the top of the board. The attained scores for the four relevant tasks (Preferred, Non-Preferred, Both and Assembly) were extracted for the purpose of this research. A description of each task and its application is cited from the Purdue Pegboard Manual (2002).

Task 1: Preferred.

The participant is instructed to work as quickly and accurately as possible and is given the opportunity to practice. After completion of a practice trial, and following the standard instructions, the participant is instructed to place as many pins possible with the Preferred hand, starting at the top. The time limit for Preferred is 30 seconds and the total score is the sum total of the number of correctly placed pins within the time limit (Lezak, et al., 2004; Purdue, 2002; Tiffin, 1968).

Task 2: Non-Preferred.

The participant is instructed to work as quickly and accurately as possible and is given the opportunity to practice. After completion of a practice trial, and following the standard instructions, the participant is instructed to place as many pins possible with the Non-Preferred hand, starting at the top. The time limit for Non-Preferred is 30 seconds and the total score is the sum total of the number of correctly placed pins within the time limit (Lezak, et al., 2004; Purdue, 2002; Tiffin, 1968).

Task 3: Both.

The participant is instructed to work as quickly and accurately as possible and is given the opportunity to practice. After completion of a practice trial, and following the standard instructions, the participant is instructed to place as many pins possible with Both hands working together, starting at the top. The time limit for Both is 30 seconds and the total score is the sum total of the number of correctly placed pins within the time limit (Lezak, et al., 2004; Purdue, 2002; Tiffin, 1968).

Task 4: Assembly.

The participant is instructed to work as quickly and accurately as possible and is given the opportunity to practice assembling pins, collars and washers. After completion of a practice trial, and following the standard instructions the participant is instructed to complete pin-washer-collar-washer assemblies, with both hands moving and alternating at the same time, starting at the top. The time limit for Assembly is 60 seconds and the total score is the sum total of the number of complete pin-washer-collar-washer assemblies and correctly placed additional parts within the time limit (Lezak, et al., 2004; Purdue, 2002; Tiffin, 1968). The Assembly task appears to also load on a manual dexterity factor that can be defined as the ability to manipulate small objects with skilful and controlled arm-hand movements.

6.4.3.2.1 Reliability and Validity of Purdue Pegboard

Tiffen & Asher (1948) used a large sample of college students, veterans and industrial job applicants (n = 7814) to establish reference values, retest reliability and validity and found Pearson r = 0.60 to 0.79 for one trial administrations and r = 0.82 to 0.91 for three trial scores. A study on the test-retest reliability coefficients of the Purdue Pegboard ranged from r = 0.85 to 0.90 for one trial administrations and from r = 0.92 to 0.96 for the sum of three trials, involving participants with multiple sclerosis (Gallus & Mathiowetz, 2003). Findings further suggest that any changes in Purdue Pegboard scores using one-trial administration may reflect actual change in dexterity, as no practice effect was demonstrated in this study. Buddenberg & Davis (2000) found test-retest reliability coefficients from r = 0.37 to 0.70 for one trial administrations and from r = 0.81 to 0.89 for the sum of three trials, involving college students. Maiden & Dyson (1997) found a positive correlation (0.95) between healthy and injured participants. Studies that examined retest reliability found better results with the three-trial administrations (Yancosek & Howell, 2009).

6.4.4 Video Notational Measures

(Conducted continuously at every game throughout the season)

In order to gather detailed information relating to the collisions encountered during one rugby season and the possible link to concussive brain injury, video footage of each game was captured and entered into a computerized notation system. All the players in the Rugby Group were annotated, which enabled the researcher to define the type and number of tackles made and the type and number of tackles received during the rugby season. The Dartfish TeamPro (Dartfish, 2005), used in this study, is one of the more advanced computerized notation system, and was used in order to enable the researcher to define an events list made up of different types of tackles. The notation system incorporates careful information management and systematic techniques of observation. The researcher utilized video recordings of each game, continuously throughout the season, in order to identify possible concussive events and to monitor different aspects of tackling. The events list was used for tagging individual players in the following categories: (i) ankle tap, (ii) dangerous high tackle, (iii) double tackle, (iv) head-on tackle, (v) grab tackle, (vi) side tackle, and (vii) tackle from behind. These were identified, in collaboration

with the coaches and the notational expert, as the most prominent types of tackles executed during a rugby game.

Video notational analysis within the sport context refers to the methodical collection, analysis, and communication of detailed information relating to a specific sport. Video notational analysis provides accurate information in quantifiable terms that permits accurate and specifically defined feedback. Biomechanics and notational analysis both make extensive use of video analysis and technology. Video analysis has been developed and applied in sport over the last couple of years in a range of applications, such as the assessment of player tackles and injury mechanisms (Wilson et al., 1999; Withnall et al., 2005). Video analysis of collisions yields information about injury functions that relate to linear and angular head accelerations with the risk of concussion.

The accurate notational analysis of specific maneuvers is fundamental in the gathering, analysis and communication of detailed information relating to the collisions encountered during one rugby season. The introduction of computerized notation systems enables post-event analysis in conjunction with video recordings that enable easy access to data (Hughes & Franks, 2004). The program enabled the researcher to define an events list (type and number of tackles) for tagging individual players as it incorporated careful information management and systematic techniques of observation.

6.5 DATA ANALYSIS

For the purpose of this study two neuropsychological measures were chosen for administration, including ImPACT (Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time) and Purdue Pegboard (Preferred, Non-Preferred, Both, Assembly) at the *pre*-, *mid*- and *post*-season assessment intervals. These measures were grouped into two broad domains of functioning that were regularly applied in clinical and research settings (Lezak et al., 2004; Matzer et al., 1999; Shuttleworth et al., 2008) with a focus on the type of tasks that they call upon, namely (i) Memory and (ii) Motor Speed. Verbal and Visual memory (two of the composite scores from ImPACT), were included in the domain of 'Memory' and Visual Motor Speed and Reaction Time (another two of the composite scores) along with the four Purdue Pegboard tasks were included in the domain of 'Motor Speed' (see Table 6.5).

Table 6.5 Conceptual Division of ImPACT Neurocognitive Measures

Domain	Cognitive Tasks
Memory	ImPACT Verbal Memory
	ImPACT Visual Memory
Motor Speed	ImPACT Visual Motor Speed
	ImPACT Reaction Time
	Purdue Preferred Hand
	Purdue Non-preferred Hand
	Purdue Both hands
	Purdue Assembly

Data analysis involved independent cross-sectional analyses, dependent prospective analyses, tackling and correlational analyses and individual player analyses.

6.5.1 Independent Cross-Sectional Analyses

Independent *t*-test analyses were conducted on the data derived from the Rugby Group (n = 20) and the Non-Contact Sports Control Group (n = 22) at the *pre*-, *mid*- and *post*-season assessment intervals to investigate differences in neuropsychological effects in respect of the four ImPACT cognitive composite scores (Verbal Memory, Visual Memory, Reaction Time and Visual Motor Speed), and the four Purdue Pegboard tasks (Preferred, Non-Preferred, Both and Assembly) between the Rugby and Non-Contact Sports Control Groups. Effect sizes with 95% confidence intervals (CI) were calculated for each test to provide additional information on the magnitude of the effect. Effect sizes were evaluated according to the proposed behavioural sciences framework where 0.2 is small, 0.5 medium and 0.8 large. Effect sizes will be interpreted as being of clinical significance (relevance) if the CI does not contain zero.

6.5.2 Dependent Prospective Analysis

Dependent *t*-test analyses were conducted on the data derived from the Rugby Group (n = 20) and the Non-Contact Sports Control Group (n = 22) at the *pre*- versus *mid*- versus *post*-season assessment intervals to investigate differences in neuropsychological effects in respect of the four ImPACT cognitive composite scores (Verbal Memory, Visual Memory, Reaction Time and Visual Motor Speed), and the four Purdue Pegboard tasks (Preferred, Non-Preferred, Both and Assembly) for the Rugby and Non-Contact Sports Control Groups.

6.5.2.1 Significance Level

The level of statistical significance or *p*-value is the criterion used to assess the reliability of the relationship between independent and dependent variables. Effect sizes measure the probability of obtaining a statistically significant result and to assess the strength of the relationship between variables (Tapia & Marsh, 2002; Trusty, Thompson & Petrocelli, 2004). If the overall variability in scores (standard error of the difference in means) is minimal then only a small difference between means of the two groups may reflect a consistent and significant difference (Peers, 1996; Trusty, Thompson & Petrocelli, 2004).

Some researchers in the sports MTBI literature consider the 5% level of significance (a 5% probability that any observed differences in mean scores could have occurred by chance) to be too lenient and there had been an attempt to eliminate Type I error, exemplified by the discussions raised in Rutherford et al. (2005), while others maintain that a test at the 1% level is more likely to enhance the chance of a Type II error (the probability of not finding a significant difference when one exists). There is a risk of making a Type I error (attaining statistical significance falsely) when a statistical test of a null hypothesis is conducted (Howell, 1989; Peers, 1996). Research point out the importance of using discretion in group MTBI research and that the results of significance tests can be misleading because of failing to notice the subtle, although clinically significant effects, and thus being subject to Type II errors (Demakis, 2006; Frencham et al., 2005; Reitan & Wolfson, 1999; Ruff, 2005; Trusty, Thompson & Petrocelli, 2004; Woods, Rippeth, Conover, Carey, Parsons & Troster, 2006). Therefore it is important to

evaluate this particular study, involving neurocognitive assessment, for its susceptibility to Type II *as well as* Type I error and to incorporate the use of effect sizes with confidence intervals.

Based on prior research (Shuttleworth-Edwards et al., 2004) a directional hypothesis is assumed for the independent cross-sectional comparison between Rugby versus Non-Contact Sports Control Groups since it was expected that the Rugby Group would perform worse than the Non-Contact Sports Control Group due to repeated concussive and subconcussive exposure and possible injury sustained due to long-term participation in a contact sport.

In that it was possible to make this directional prediction, one-tailed tests were employed which permit the division of the *p*-values by two. For the dependent prospective comparisons for the Rugby Group at *pre*- versus *mid*- versus *post*-season and for the Non-Contact Sports Control Group at *pre*- versus *mid*- versus *post*-season, it was uncertain whether there would be poorer performances on cognitive assessment due to cumulative sub-concussive effects sustained during the season or because of an overlay on persistent effects in neurocognitive functioning, or whether improvements due to practice effects would obscure poorer performance. Therefore, since no specific differences could be assumed between the groups, a two-tailed test (non-directional test) was used for the dependent analyses which did not permit the division of the *p*-values by two.

6.5.2.2 Alpha Adjustments

The Bonferroni correction is a statistical adjustment to compensate for multiple comparisons made simultaneously on the same data set (Hsu, 1996; Perneger, 1998), and provide a more stringent level of statistical significance according to the number of times an analysis takes place on the same data set, and may help to guard against committing a Type I error (attaining statistical significance falsely) when multiple measures are used. Criticism in the literature has been levelled in the sports MTBI literature at researchers such as Matser et al. (1998; 1999) for the use of multiple measures and using the same control group in different studies whilst not controlling for Type I error by making Bonferroni adjustments. Rutherford et al. (2005) reiterated that the use of multiple measures called for statistical stringency in order to guard against Type I error. If a too stringent statistical adjustment is made to compensate for the use of

multiple measures and where there is the expectation of relatively subtle findings, there may be an inappropriate loss of statistical sensitivity and the danger of Type II error (the probability of not finding a significant difference when one exists). In other words, making appropriate statistical adjustments with a level of statistical *leniency* in order to avoid missing clinically relevant effects and getting the correct balance of statistical power by neither under or over-correction that would thereby result in committing Type I or Type II error, respectively (Johnson & Wichern, 2002; Peers, 1996).

In an attempt to dispel criticisms about the possibility of incurring Type 1 error and to protect against chance effects, Rutherford et al. (2005) and Matser et al. (1999) divided measures (16 and 15 respectively) into functional modalities and used the number of functional modalities to make their alpha adjustment towards stringency. Shuttleworth-Edwards, Smith & Radloff (2008), in relation to concussive effects in adult level rugby, divided a more focused battery of seven measures into two functional modalities, and applied the alpha adjustment towards stringency according to these two modalities. This approach is more focused than that applied in the two soccer studies of Rutherford et al. (2005) and Matser et al. (1999), due to the drastically limited measures under investigation. Furthermore, the targeting of a few cognitive functions, that show sensitivity to MTBI, reduce the possibility of chance effects and increase the statistical power of the analysis.

The present research employed a relatively focused battery of eight neurocognitive subtests that fall within the two neurocognitive functional modalities of Memory and Motor Speed, as indicated in Table 6.5, and correspond with the research of Shuttleworth-Edwards et al. (2008). For the purposes of this study, the Bonferroni's adjustment to the significance level was applied to the neurocognitive assessment comparisons, according to the number of functional modalities (i.e. two modalities) investigated, rather than the number of measures employed (i.e. eight measures). This application demanded the division of the probability level by two rather than eight, and it was considered that this route would provide the appropriate balance of making an adjustment towards stringency to guard against Type 1 error, whilst at the same time not neglecting to take account of the potential for Type II error. Accordingly, significance at $p = 0.05$ was adjusted to $p = 0.025$; significance at $p = 0.01$ was adjusted to $p = 0.005$; and approaching significance at $p = 0.075$ was adjusted to $p = 0.0375$. In all tables significance (one-

tailed with Bonferroni's adjustment) is represented by: ** $p < .01$, * $p < .05$, and approaching significance is represented by: † $p \leq 0.075$. This adjustment was done with the following reasons in mind:

1) only a proportion of rugby players would sustain multiple concussions over their rugby playing careers and consequently significant individual effects were likely to be diluted in the analyses of group effects,
2) the nature of the concussive brain injury was mild and therefore any effects were likely to be relatively subtle, and
3) the sample numbers were relatively small which reduced the likelihood of identifying significant differences between groups.

6.5.3 Tackling Analyses

Tackling data derived from the video-taped footage of each game, were analysed after defining an events list made up of different types of and number of tackles (see 6.3.3). The researcher reviewed the videos of the games and the program enabled the researcher to tag individual players in order to assess tackles and injury mechanisms that relate to linear and angular head accelerations with the risk of concussion. The software program allows the video to play in slow motion while the researcher notes each tackle to an individual player simultaneously on the same computer screen. This method allows for scrupulous counting as the video can be slowed to a speed that is comfortable for the researcher to count and assess tackles. The data was tabulated in a spreadsheet and descriptive statistics were calculated for the rugby players, who participated throughout the season and completed all assessments ($n = 20$), in terms of the means and *SD*s for (i) Tackles Made, (ii) Tackles Received, and (iii) Total Tackles.

6.5.4 Correlational Analyses

Despite the small numbers of participants in the sample, it was decided to conduct a series of exploratory Spearman's Correlational analyses to investigate the following relationships:

1) *concussions* reported on the biographical questionnaire for the Total Group (including both Rugby and Non-Contact Sports controls; *n* = 59 at *pre-*, *n* = 42 at *mid-* and *n* = 36 at *post*-season) in relation to the neurocognitive assessment measures (ImPACT and Purdue Pegboard) at the three assessment intervals;

2) *concussions* reported on the biographical questionnaire for the Rugby Group (*n* = 20) in relation to the neurocognitive assessment measures (ImPACT and Purdue Pegboard) at the three assessment intervals (*pre-*, *mid-* and *post*-season);

3) *concussions* reported on the biographical questionnaire for the Rugby Group (*n* = 20) in relation to the tackling data (Tackles Made, Tackles Received and Total Tackles); and

4) *tackling data* (Tackles Made, Tackles Received and Total Tackles) for the Rugby Group (*n* = 20) in relation to the neurocognitive measures (ImPACT and Purdue Pegboard) at the three assessment intervals (*pre-*, *mid-* and *post*-season).

More specifically, this translated into Spearman's correlation analyses being carried out on the number of concussions reported, as follows: (i) number of *concussions* reported for the Total Group in relation to the neurocognitive measures (ImPACT Verbal Memory, ImPACT Visual Memory, ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both, Purdue Assembly) at each of the three assessment intervals (*pre-*, *mid-* and *post*-season), (ii) number of *concussions* reported for the Rugby Group (*n* = 20) in relation to the neurocognitive measures (ImPACT Verbal Memory, ImPACT Visual Memory, ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both, Purdue Assembly) at each of the three assessment intervals (*pre-*, *mid-* and *post*-season), (iii) number of *concussions* reported on the biographical questionnaire for the Rugby Group (*n* = 20) in relation to the tackling data (Tackles Made, Tackles Received and Total Tackles).

Furthermore, Spearman's correlation analyses were carried out on the tackling data, as follows: (i) tackling data for the Rugby Group (*n* = 20) in the three tackling categories (Tackles Made, Tackles Received and Total Tackles) in relation to the neurocognitive measures (ImPACT Verbal

Memory, ImPACT Visual Memory, ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both, Purdue Assembly) at each of the three assessment intervals (*pre*-, *mid*- and *post*-season).

6.5.5 Individual Player Analyses

It is held in cognitive neuropsychology that the individual case study is much more likely to produce strong evidence for discriminating among theories of normal function and can result in the refinement, confirmation or questioning of such theories (Shallice, 1988). In order to investigate the individual differences in neuropsychological effects at the *pre*-, *mid*- and *post*-season assessment intervals, each perceived concussed player is descriptively compared with his own *pre*-season scores, normative scores as well as with the Non-contact Sports Control Group mean scores in respect of each of the neurocognitive measures (ImPACT Verbal Memory, ImPACT Visual Memory, ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both, Purdue Assembly). Furthermore, the individual player profiles also incorporated additional tackling information to investigate whether the tackling data (i.e., the effects of frequent and repetitive exposure to head and body collisions) had relevance in the overall clinical picture.

6.6 STATISTICAL HYPOTHESES FOR THIS STUDY

Research regarding the possible subconcussive effects of frequent head and body collisions in rugby is limited. In the light of research indicating that the risk of concussion is directly proportional to the amount of games played (Witol & Webbe, 1994), the present research investigated the cumulative neurocognitive effects of frequent head and body collisions on club level rugby players and used a computer-based assessment instrument (ImPACT) together with a traditional neurocognitive instrument that measures hand-motor speed (Purdue Pegboard). The only published study comparing *pre*-, *mid*-, and *post*-season neurocognitive scores included uninjured collegiate football players, with no control group, and found ImPACT and SAC neurocognitive test scores were not significantly altered by a season of repetitive contact in collegiate football athletes who have not sustained a concussion (Miller, Adamson, Pink & Sweet, 2007). It would appear that there are no studies available in contact sport, that both

investigate the frequency of player-to-player collisions and the consequent neurocognitive outcome at *pre-*, *mid-*, and *post*-season with the inclusion of a demographically matched control group.

Pre-season assessments were conducted with the objective of analysing persistent cognitive deficits amongst rugby players compared with Non-Contact Sports controls, as a result of concussive and subconcussive events sustained during many years of previous exposure to the game. *Mid-* and *post*-season assessments were conducted with the objective of analysing cognitive deficits amongst rugby players compared with Non-Contact Sports controls, as a result of years of previous exposure to the game plus any additional concussive and subconcussive events sustained during the 2005 rugby season. It is understood that concussive events may be purposely unreported or unrecognised and it was expected that outcomes for the Rugby Group would be worse than for the Non-Contact Sports Control Group at the *pre*-season assessment interval. It was further expected that adverse outcomes would be more apparent for the Rugby Group at the *mid-* and *post*-season assessment intervals than at the *pre*-season assessment interval due to the possibility of added effects of unreported concussive and subconcussive events.

Differentiating seasonal effects from previous effects was not considered possible as these would operate synergistically with past exposure effects. It was expected that the Rugby Group would show less improvement and evidence of practice effects than the Non-Contact Sports Control Group between the three assessment intervals (*pre-*, *mid-*, and *post*-season) due to their neurocognitive vulnerability. Consequently, and in view of MTBI research findings, and the theoretical underpinnings previously discussed, the following specific hypotheses were formulated for (i) the independent cross-sectional comparisons, (ii) the dependent prospective comparisons; (iii) the correlational analyses, and (iv) the individual player analyses.

6.6.1.1 Independent Cross-Sectional Comparisons

(i) It was hypothesized *on the basis of independent t-test analyses* that there would be significant differences between the mean scores of the Rugby Group relative to the Non-Contact Sports Control Group on the neurocognitive measures at each of the assessment intervals (*pre-*, *mid-*, and *post*-season), in the direction of the Rugby Group performing

worse than the Non-Contact Sports Control Group. This was in support of deleterious neuropsychological sequelae for the Rugby Group relative to the Non-Contact Sports Control Group, due to postulated exposure to the cumulative effects of head and body collisions during many preceding years of rugby participation (all intervals), including concussive and sub-concussive events (*mid*-and *post*-season intervals only), during participation in the 2005 rugby season.

6.6.1.2 Dependent Prospective Comparison

(i) It was hypothesized *on the basis of dependent t-test analyses* that *either* there would be significant differences in the mean scores on the *neurocognitive measures* for the Rugby Group at the *pre*- versus *mid*- versus *post*-season assessment intervals in the direction of worsening performance for the Rugby Group in contrast to no deterioration in scores for the Non-Contact Sports Control Group, due to the deleterious neuropsychological effects of unreported concussive and sub-concussive events sustained by the rugby players during participation in the 2005 rugby season, on top of the long-term effects of concussive and subconcussive events sustained over many prior years of playing rugby, *or* that for the same reason there would be no significant differences in the mean scores on the neurocognitive measures for the Rugby Group at *pre*- versus *mid*- versus *post*-season assessment intervals, in contrast to significant improvement for the Non-Contact Sports Control Group in the mean scores on the neurocognitive measures.

6.6.1.3 Correlational Analyses

(i) It was hypothesized *on the basis of a series of exploratory Spearman's correlational analyses* that *more* concussions would be associated with *poorer* neurocognitive performance across the three assessment intervals (*pre*-, *mid*- and *post*-season) for both the Total Group (including both the Rugby and Non-Contact Sports Control Groups) and the Rugby Group.

(ii) It was hypothesized that on the basis of a series of exploratory Spearman's correlational analyses that *more* concussions would be associated with *higher* number of tackles, in all three tackling categories (i) Tackles Made, (ii) Tackles Received, and (iii) Total Tackles.

6.6.1.4 Individual Player Analyses

(i) It was hypothesized that in terms of BRC theory that the *individual player analyses* would descriptively be comparable with adverse neuropsychological effects that have been reported in the literature for adult athletes who have sustained a concussion on top of a long history of participation in contact sport, and that there would be indications of cognitive deficits established on the neurocognitive measures.

CHAPTER 7

RESULTS: GROUP ANALYSES

The results of this study are presented in this chapter. The results pertaining to the independent cross-sectional (*between* group) comparisons for the Rugby versus Non-Contact Sports Control Groups at the *pre-*, *mid-* and *post*-season assessment intervals are presented in the first section, followed by the results pertaining to the dependent prospective (*within* group) comparisons for the Rugby and Non-Contact Sports Control Groups at the *pre-* versus *mid-* versus *post*-season assessment intervals. Significant results and the general trends pertaining to each analysis are highlighted in the text. A synthesis of the findings for all comparative group analyses is made at the end of each section. Tables detailing the means, standard deviations, *t*-statistics, significant effects (*p*-values) and effect size (*d*-values) for each comparison are provided. Tables for *all data,* appear at the end of each relevant subsection.

7.1 RUGBY AND NON-CONTACT SPORTS CONTROL GROUPS

The first section includes reports on the *independent t*-test comparisons (Table 7.1) between the Rugby Group and the Non-Contact Sports Control Group across all neurocognitive measures at the *pre-*, *mid-* and *post*-season assessment intervals. The composite scores on the ImPACT computerized program reported are incorporated with the Purdue Pegboard into the two modalities of Memory (ImPACT Verbal Memory and ImPACT Visual Memory) and Motor Speed (ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both and Purdue Assembly).

The second section includes reports on the *dependent t*-test comparisons (Table 7.2) for the Rugby Group and the Non-Contact Sports Control Group at the *pre-* versus *mid-* versus *post*-season assessment intervals. The composite scores on the ImPACT computerised program are incorporated with the Purdue Pegboard into the two modalities of Memory (ImPACT Verbal Memory and ImPACT Visual Memory) and Motor Speed (ImPACT Visual Motor Speed,

ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both and Purdue Assembly).

7.1.1 Independent Cross-sectional *t*-test (*between group)* Comparisons

Memory. In the comparison between the Rugby versus Non-Contact Sports Control Groups at the *pre*- and *post*-season assessment intervals (Table 7.1) there was one result that was approaching significance in the direction of the Rugby Group performing worse than the Non-Contact Sports Control Group, with an effect size that is not of clinical relevance (i.e. CI contains zero), namely ImPACT Visual Memory ($p = 0.068$, $d = -0.45$ and $p = 0.064$, $d = -0.49$ respectively). At the *mid*-season assessment interval there were no significant differences, or differences approaching significance for the two memory measures. Overall there was a predominant trend in the direction of the Rugby Group performing worse than the Non-Contact Sports Control Group on ImPACT Visual Memory at the *pre*-, *mid*- and *post*-season intervals. There was a trend of minimal differences in the direction of the Rugby Group performing marginally better than the Non-Contact Sports Control Group at the *mid*- and *post*-season assessment interval on Verbal Memory (score = 0.86 versus 0.85, and 0.88 versus 0.87 respectively).

Motor Speed. In the comparison between the Rugby versus Non-Contact Sports Control Groups at the *pre*-season assessment interval (Table 7.1) there were five results that were significant in the direction of the Rugby Group performing worse than the Non-Contact Sports Control Group with effect sizes that is not of clinical relevance (i.e. CI contains zero), namely (i) ImPACT Visual Motor Speed ($p = 0.001$, $d = -0.98$); (ii) ImPACT Reaction Time ($p = 0.000$, $d = 1.23$); (iii) Purdue Preferred ($p = 0.006$, $d = -0.82$); (iv) Purdue Both ($p = 0.001$, $d = -1.06$); and (v) Purdue Assembly ($p = 0.007$, $d = -0.80$). There was one result that was approaching significance in the direction of the Rugby Group performing worse than the Non-Contact Sports Control Group, with an effect size that is not of clinical relevance (i.e. CI contains zero), namely Purdue Non-Preferred ($p = 0.059$, $d = -0.48$). Overall there was a predominant trend in the direction of the Rugby Group performing worse than the Non-Contact Sports Control Group on all the measures.

In the comparison between the Rugby versus Non-Contact Sports Control Groups at the *mid*-season assessment interval (Table 7.1) all of the Motor Speed results were significant in the direction of the Rugby Group performing worse than the Non-Contact Sports Control Group with effect sizes that was not of clinical relevance (i.e. CI contains zero), namely (i) ImPACT Visual Motor Speed (p = 0.009, d = -0.76); (ii) ImPACT Reaction Time (p = 0.000, d = 1.17); (iii) Purdue Preferred (p = 0.009, d = -0.76); (iv) Purdue Non-Preferred (p = 0.002, d = -0.93); (v) Purdue Both (p = 0.004, d = -0.88); and (vi) Purdue Assembly (p = 0.038, d = -0.56). Overall there was a predominant trend in the direction of the Rugby Group performing worse than the Non-Contact Sports Control Group on all the measures.

In the comparison between the Rugby versus Non-Contact Sports Control Groups at the *post*-season assessment interval there were two significant results in the direction of the Rugby Group performing worse than the Non-Contact Sports Control Group with an effect size that was not of clinical relevance (i.e. CI contains zero), namely (i) ImPACT Visual Motor Speed (p = 0.050, d = -0.52); and (ii) ImPACT Reaction Time (p = 0.005, d = 0.87). Two results were approaching significance in the direction of the Rugby Group performing worse than the Non-Contact Sports Control Group with effect sizes that was not of clinical relevance (i.e. CI contains zero), namely (i) Purdue Preferred (p = 0.053, d = -0.51); and (ii) Purdue Assembly (p = 0.054, d = -0.51). Overall there was a predominant trend in the direction of the Rugby Group performing worse than the Non-Contact Sports Control Group on all the measures.

Table 7.1 Independent Cross-sectional Pre-, Mid- and Post-season comparisons of all Memory and Motor Speed Scores between the Rugby and Non-Contact Sports Control Groups

Pre-season	**Rugby (n = 20) Mean**	**(SD)**	**Non-Contact (n = 22) Mean**	**(SD)**	***t-value***	***Effect size d* (95% CI)**	***p-value***
MEMORY							
ImPACT Verbal Memory	0.84	(0.11)	0.84	(0.09)	-0.251	0.10 (-0.51, 0.70)	0.401
ImPACT Visual Memory	0.69	(0.11)	0.74	(0.11)	-1.517	-0.45 (-1.06, 0.17)	0.068†
MOTOR SPEED							
ImPACT VMS[l]	31.64	(5.74)	37.47	(6.10)	-3.181	-0.98 (-1.60,-0.32)	0.001**
ImPACT Reaction Time	0.651	(0.11)	0.548	(0.05)	4.038	1.23 (1.86, 0.54)	0.000**
Purdue Preferred	14.40	(1.76)	16.05	(2.21)	-2.648	-0.82 (-1.44,-0.18)	0.006**
Purdue Non-Preferred	14.25	(1.52)	15.09	(1.85)	-1.601	-0.48 (-1.10, 0.13)	0.059†
Purdue Both	11.38	(1.42)	12.82	(1.31)	-3.418	-1.06 (-1.68,-0.39)	0.001**
Purdue Assembly	32.35	(5.95)	36.41	(4.13)	-2.589	-0.80 (-1.41,-0.16)	0.007**
Mid-season	**Rugby (n = 20) Mean**	**(SD)**	**Non-Contact (n = 22) Mean**	**(SD)**	***t-value***	***Effect size d* (95% CI)**	***p-value***
MEMORY							
ImPACT Verbal Memory	0.86	(0.84)	0.85	(0.09)	0.546	0.02 (-0.59, 0.62)	0.294
ImPACT Visual Memory	0.75	(0.15)	0.76	(0.10)	-0.220	-0.08 (-0.68, 0.53)	0.413
MOTOR SPEED							
ImPACT VMS[l]	34.60	(7.28)	39.47	(5.42)	-2.476	-0.76 (-1.38,-0.12)	0.009**
ImPACT Reaction Time	0.602	(0.11)	0.504	(0.05)	3.836	1.17 (0.49, 1.80)	0.000**
Purdue Preferred	15.85	(1.31)	17.00	(1.69)	-2.447	-0.76 (-1.37,-0.12)	0.009**
Purdue Non-Preferred	14.80	(1.28)	16.05	(1.40)	-3.001	-0.93 (-1.55,-0.28)	0.002**
Purdue Both	11.88	(1.28)	13.07	(1.43)	-2.838	-0.88 (-1.48,-0.24)	0.004**
Purdue Assembly	32.55	(6.09)	35.55	(4.51)	-1.822	-0.56 (-1.17, 0.06)	0.038*
Post-season	**Rugby (n = 20) Mean**	**(SD)**	**Non-Contact (n = 22) Mean**	**(SD)**	***t-value***	***Effect size d* (95% CI)**	***p-value***
MEMORY							
ImPACT Verbal Memory	0.88	(0.09)	0.87	(0.08)	0.190	0.12 (-0.49, 0.73)	0.425
ImPACT Visual Memory	0.75	(0.16)	0.82	(0.10)	-1.565	-0.49 (-1.11, 0.12)	0.064†
MOTOR SPEED							
ImPACT VMS[l]	35.11	(7.22)	38.98	(7.59)	-1.691	-0.52 (-1.14, 0.09)	0.050*
ImPACT Reaction Time	0.586	(0.09)	0.520	(0.05)	2.746	0.87 (0.23, 1.50)	0.005**
Purdue Preferred	16.25	(1.41)	17.09	(1.82)	-1.660	-0.51 (-1.12, 0.11)	0.053†
Purdue Non-Preferred	15.60	(1.42)	16.23	(1.60)	-1.334	-0.42 (-1.03, 0.20)	0.095
Purdue Both	12.95	(1.27)	13.36	(1.06)	-1.147	-0.35 (-0.96, 0.26)	0.129
Purdue Assembly	33.95	(6.10)	36.45	(3.50)	-1.651	-0.51 (-1.12, 0.11)	0.054†

Significant: * $p \leq 0.05$; ** $p \leq 0.01$, one-tailed with Bonferroni's adjustment.
Approaching significant: † $p \leq 0.075$, one-tailed with Bonferroni's adjustment.
[l]Note: Visual Motor Speed (VMS)

7.1.2 Dependent Prospective (*within* group) Comparisons

7.1.2.1 Rugby Group

Memory. The repeated measures ANOVAs for the Rugby Group (Table 7.2 top section) on the ImPACT Verbal Memory measure at *pre*- versus *mid*- versus *post*-season assessment intervals revealed no significant season effect (Wilks' Lambda = .889, $F = 1.121$, $df = 2,18$, $p = 0.348$). The repeated measures ANOVAs for the Rugby Group on the ImPACT Visual Memory measure at *pre*- versus *mid*- versus *post-season* assessment intervals revealed no significant season effect (Wilks' Lambda = .842, $F = 1.695$, $df = 2,18$, $p = 0.212$).

Motor Speed. The repeated measures ANOVAs for the Rugby Group (Table 7.2 top section) on the ImPACT and Purdue Pegboard neurocognitive measures at *pre*- versus *mid*- versus *post*-season assessment intervals revealed significant results on five of the six measures with the exception of Purdue Assembly, namely (i) ImPACT Visual Motor Speed (Wilks' Lambda = .546, $F = 7.480$, $df = 2,18$, $p = 0.004$); (ii) ImPACT Reaction Time (Wilks' Lambda = .324, $F = 18.753$, $df = 2,18$, $p < .001$); (iii) Purdue Preferred (Wilks' Lambda = .528, $F = 8.054$, $df = 2,18$, $p = 0.003$); (iv) Purdue Non-Preferred (Wilks' Lambda = .608, $F = 5.801$, $df = 2,18$, $p = 0.011$) and (v) Purdue Both (Wilks' Lambda = .423, $F = 12.294$, $df = 2,18$, $p < .001$).

Bonferroni multiple Pairwise comparisons for the Rugby Group revealed that there were significant differences in means for Motor Speed, all in the direction of improved performance, namely (i) ImPACT Visual Motor Speed from *pre*- to *mid*-season and *pre*- to *post*-season ($p = 0.026$ and $p = 0.005$, respectively); (ii) ImPACT Reaction Time from *pre*- to *mid*-season and *pre*- to *post*-season ($p = 0.005$ and $p < .001$, respectively); (iii) Purdue Preferred from *pre*- to *mid*-season and *pre*- to *post*-season ($p = 0.005$ and $p = 0.003$, respectively); (iv) Purdue Non-Preferred from *pre*- to *post*-season ($p = 0.007$); and Purdue Both from *pre*- to *mid*-season and *pre*- to *post*-season ($p = 0.022$ and $p < .001$, respectively).

Overall (Table 7.2), there was a consistent trend for the Rugby Group to improve gradually from the *pre*- to *mid*-season assessment intervals with a sharper improvement at the *post*-season assessment interval.

7.1.2.2 Non-Contact Sports Control Group

Memory. The repeated measures ANOVAs for the Non-Contact Sports Control Group (Table 7.2 lower section) on the ImPACT Verbal Memory measure at *pre*- versus *mid*- versus *post*-season assessment intervals revealed no significant season effect (Wilks' Lambda = .754, F = 3.268, df = 2,20, p = 0.059). The repeated measures ANOVAs for the Non-Contact Sports Control Group on the ImPACT Visual Memory measure at *pre*- versus *mid*- versus *post*-season assessment intervals revealed a significant season effect, namely (Wilks' Lambda = .691, F = 4.474, df = 2,20, p = 0.025).

Motor Speed. The repeated measures ANOVAs for the Non-Contact Sports Control Group (Table 7.2 lower section) on the ImPACT and Purdue Pegboard neurocognitive measures at *pre*- versus *mid*- versus *post*-season assessment intervals revealed significant results on four of the six measures with the exception of ImPACT Visual Motor Speed, and Purdue Assembly, namely (i) ImPACT Reaction Time (Wilks' Lambda = .586, F = 7.067, df = 2,20, p = 0.005); (ii) Purdue Preferred (Wilks' Lambda = .651, F = 5.353, df = 2,20, p = 0.014); (iii) Purdue Non-Preferred (Wilks' Lambda = .582, F = 7.187, df = 2,20, p = 0.004) and (iv) Purdue Both (Wilks' Lambda = .630, F = 5.869, df = 2,20, p = 0.010).

Bonferroni multiple Pairwise comparisons for the Non-Contact Sports Control Group revealed that there were significant differences in means for Motor Speed, all in the direction of improved performance, namely (i) ImPACT Reaction Time from *pre*- to *mid*-season (p = 0.005); (ii) Purdue Preferred from *pre*- to *post*-season (p = 0.009); (iii) Purdue Non-Preferred from *pre*- to *mid*-season and *pre*- to *post*-season (p = 0.005 and p = 0.004, respectively); and (iv) Purdue Both from *pre*- to *post*-season (p = 0.010).

Overall (Table 7.2), there was a consistent trend for the Non-Contact Sports Control Group to improve from the *pre*- to *mid*-season assessment intervals with a more gradual improvement at the *post*-season assessment interval.

7.1.2.3 Interaction Effects between Season and Group

Memory. The repeated measures ANOVAs of group means (Table 7.3) on the ImPACT Verbal Memory measure at *pre*- versus *mid*- versus *post*-season assessment intervals revealed no significant interaction effects or interaction effects that were approaching significance. The repeated measures ANOVAs on the ImPACT Visual Memory measure at *pre*- versus *mid*- versus *post*-season assessment intervals revealed no significant interaction effects or interaction effects that were approaching significance.

Motor Speed. The repeated measures ANOVAs of group means (Table 7.3) for the ImPACT and Purdue Pegboard neurocognitive measures at *pre*- versus *mid*- versus *post*-season assessment intervals revealed significant interaction effects between season and group on (i) ImPACT Reaction Time (Wilks' Lambda = .840, $F = 3.706$, $df = 2,39$, $p = 0.034$), and (ii) Purdue Both (Wilks' Lambda = .829, $F = 4.016$, $df = 2,39$, $p = 0.026$).

Table 7.2. Dependent Prospective comparisons of Memory and Motor Speed for the Rugby and Non-Contact Sports Control Groups at the Pre- versus Mid- versus Post-season Assessment Intervals

	Rugby (*n* = 20)						
	Pre-Season		**Mid-Season**		**Post-Season**		
	Mean	**(SD)**	**Mean**	**(SD)**	**Mean**	**(SD)**	***p*-value**
MEMORY							
ImPACT Verbal Memory	0.84	-0.11	0.86	-0.08	0.88	-0.09	0.348
ImPACT Visual Memory	0.69	-0.11	0.75	-0.15	0.75	0.16)	0.212
MOTOR SPEED							
ImPACT VMS[l]	31.64	-5.74	34.60	-7.28	35.11	-7.22	0.004**
ImPACT Reaction Time	0.65	-0.11	0.60	-0.11	0.59	-0.09	0.000**
Purdue Preferred	14.40	-1.76	15.85	-1.31	16.25	-1.41	0.003**
Purdue Non-preferred	14.25	-1.52	14.80	-1.28	15.60	-1.43	0.011*
Purdue Both	11.38	-1.42	11.88	-1.28	12.95	-1.28	0.000**
Purdue Assembly	32.35	-5.95	32.55	-6.09	33.95	-6.10	0.283
	Non-Contact Sports Control (*n* = 22)						
	Pre-Season		**Mid-Season**		**Post-Season**		
	Mean	**(SD)**	**Mean**	**(SD)**	**Mean**	**(SD)**	***p*-value**
MEMORY							
ImPACT Verbal Memory	0.84	-0.10	0.85	-0.09	0.87	-0.08	0.059
ImPACT Visual Memory	0.74	-0.10	0.76	-0.10	0.82	-0.10	0.025*
MOTOR SPEED							
ImPACT VMS[l]	37.47	-6.10	39.47	-5.42	38.98	-7.59	0.141
ImPACT Reaction Time	0.55	-0.10	0.50	-0.05	0.52	-0.05	0.005**
Purdue Preferred	16.05	-2.20	17.00	-1.69	17.09	-1.82	0.014*
Purdue Non-preferred	15.09	-1.90	16.05	-1.40	16.23	-1.60	0.004**
Purdue Both	12.82	-1.30	13.07	-1.43	13.36	-1.06	0.010**
Purdue Assembly	36.41	-4.10	35.55	-4.51	36.45	-3.50	0.519

Significant: * $p \leq 0.05$; ** $p \leq 0.01$, two-tailed with Bonferroni's adjustment.
Approaching significant: † $p \leq 0.075$, two-tailed with Bonferroni's adjustment.
[l]Note: Visual Motor Speed (VMS)

Table 7.3 Pre- versus Mid- versus Post-season Comparisons on Memory and Motor Speed for Rugby and Non-Contact Sports Control Groups

	Rugby (*n* = 20)							**Non-Contact Sports Control (*n* = 22)**							
	Pre-Season		**Mid-Season**		**Post-Season**			**Pre-Season**		**Mid-Season**		**Post-Season**			
	Mean	**(SD)**	**Mean**	**(SD)**	**Mean**	**(SD)**	***p*-value**	**Mean**	**(SD)**	**Mean**	**(SD)**	**Mean**	**(SD)**	***p*-value**	**Interaction *p*-value**
MEMORY															
ImPACT Verbal Memory	0.84	-0.11	0.86	-0.08	0.88	-0.09	0.074†	0.84	-0.10	0.85	-0.09	0.87	-0.08	0.033*	0.740
ImPACT Visual Memory	0.69	-0.11	0.75	-0.15	0.75	0.16)	0.068†	0.74	-0.10	0.76	-0.10	0.82	-0.10	0.005**	0.054†
MOTOR SPEED															
ImPACT VMS[l]	31.64	-5.74	34.6	-7.28	35.11	-7.22	0.001**	37.47	-6.10	39.47	-5.42	38.98	-7.59	0.168	0.529
ImPACT Reaction Time	0.65	-0.11	0.60	-0.11	0.59	-0.09	0.000**	0.55	-0.10	0.50	-0.05	0.52	-0.05	0.008**	0.034*‡
Purdue Preferred	14.40	-1.76	15.85	-1.31	16.25	-1.41	0.001**	16.05	-2.20	17.00	-1.69	17.09	-1.82	0.002**	0.366
Purdue Non-preferred	14.25	-1.52	14.80	-1.28	15.60	-1.43	0.001**	15.09	-1.90	16.05	-1.40	16.23	-1.60	0.001**	0.265
Purdue Both	11.38	-1.42	11.88	-1.28	12.95	-1.28	0.000**	12.82	-1.30	13.07	-1.43	13.36	-1.06	0.002**	0.026*‡
Purdue Assembly	32.35	-5.95	32.55	-6.09	33.95	-6.10	0.087	36.41	-4.10	35.55	-4.51	36.45	-3.50	0.482	0.587

Significant: * $p \leq 0.05$; ** $p \leq 0.01$, two-tailed with Bonferroni's adjustment
Approaching significant: † $p \leq 0.075$, two-tailed with Bonferroni's adjustment
For interaction values: ‡ $p < .05$, two-tailed
[l]Note: Visual Motor Speed (VMS)

7.2 SYNTHESIS FOR ALL COMPARATIVE GROUP ANALYSES

For the Independent (*between* groups) cross-sectional analyses of the Rugby versus Non-Contact Sports Control Group at the *pre-*, *mid-* and *post*-season assessment intervals, all significant results and most of the overall trends were in the direction of poorer performance for the Rugby Group at all the assessment intervals (*pre-*, *mid-*, and *post*-season). Tests of Motor Speed and specifically ImPACT Visual Motor Speed and ImPACT Reaction Time were consistently significantly depressed for the Rugby Group relative to controls across all the assessment intervals (*pre-*, *mid-*, and *post*-season). Tests of Motor Speed and specifically ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred, Purdue Both and Purdue Assembly were significantly depressed for the Rugby Group relative to controls at the *pre*-season assessment interval, with Purdue Non-Preferred approaching significance. Tests of Motor Speed and specifically ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both and Purdue Assembly were significantly depressed for the Rugby Group at the *mid*-season assessment interval. Tests of Motor Speed and specifically ImPACT Visual Motor Speed and ImPACT Reaction Time were significantly depressed for the Rugby Group at the *post*-season assessment interval, and Purdue Preferred and Purdue Assembly approaching significance.

For the Dependent (*within* group) prospective analyses of the Rugby and Non-Contact Sports Control Groups at *pre-* versus *mid-* versus *post*-season assessment intervals, all significant results and most of the overall trends were in the direction of improved performance for both the Rugby and Non-Contact Sports Control Groups at the *post*-season assessment interval. Significant results were on tests of Motor Speed and were consistently significant on ImPACT Reaction Time and Purdue Preferred, Purdue Non-Preferred, Purdue Both for both the Rugby and Non-Contact Sports Control Groups. Interaction effects for ImPACT Reaction Time and Purdue Both suggest that the Rugby Group started much lower at *pre*-season than the Non-Contact Sports Control Group, but improved significantly more than the Non-Contact Sports Control Group after two more assessments and consequently got closer to the Non-Contact Sports Control Group at *post*-season.

In summary, taking into account all significant results as well as overall trends, both the independent cross-sectional and dependent prospective analyses for the ImPACT and Purdue Pegboard neurocognitive measures revealed a general (even though not entirely consistent) trend in the direction of the Rugby Group attaining comparatively poorer scores than the Non-Contact Sports Control Group at each of the assessment intervals that was consistently more in evidence in the Motor Speed rather than the Memory modality.

7.3 SEASONAL TACKLING DATA

The tackling data were subjected to descriptive statistical analyses, including the calculation of standard deviations and means, and tabulated in relation to detailed tackling analyses for the Rugby Group (n = 20).

Table 7.4 reflected the Rugby Group with descriptive statistics calculated on the basis of 24 games played by each player and videotaped and analysed in respect of (i) Tackles made Above the waist, (ii) Tackles made Below the waist; (iii) Tackles received Above the waist, (iv) Tackles received Below the waist, and (v) Total tackles.

Perusal of Table 7.4 revealed that on average the Rugby Group was involved in a Total of **103.45** tackles (*SD* = 59.98). On average the Tackles Made were **60.7** (*SD* = 36.16), and of those on average **39.85** (*SD* = 23.32) were made above the waist, and on average **20.85** (*SD* = 14.75) were made below the waist. On average the Tackles Received were **42.75** (*SD* = 26.22), and of those on average **29.5** (*SD* = 18.04) were made above the waist, and on average **13.25** (*SD* = 11.09) were made below the waist.

Table 7.4 Individual and Group Mean Analysis of Type of Tackles Made and Received during 24 games played over one rugby season

Player Number[1]	Tackles Made			Tackles Received			Total Tackles
	Above waist	Below waist	Total	Above waist	Below waist	Total	
1	24	5	29	24	3	27	56
2	87	35	122	86	22	108	230
3	26	20	46	4	2	6	52
4	45	12	57	43	20	63	120
5	18	11	29	16	8	24	53
6	8	8	16	7	2	9	25
7	48	24	72	28	8	36	108
8	53	49	102	40	34	74	176
9	30	11	41	15	3	18	59
10	30	27	57	25	18	43	100
11	90	60	150	48	43	91	241
12	6	3	9	6	2	8	17
13	66	35	101	28	17	45	146
14	38	15	53	18	8	26	99
15	46	33	79	32	25	57	136
16	53	21	74	39	9	48	122
17	59	13	72	36	6	42	114
18	8	6	14	19	5	24	38
19	22	11	33	38	19	57	90
20	40	18	58	38	11	49	107
Mean	39.85	20.85	**60.70**	29.50	13.25	**42.75**	**103.45**
Std Dev	23.32	14.75	36.16	18.04	11.09	26.22	59.98

[1]*Note:* Player Number refer to the rugby players included in the group analyses that participated throughout the season

7.4 CORRELATIONS

A series of exploratory Spearman's correlational analyses were run of which the results are tabled below, including

1) *concussions* reported on the biographical questionnaire for the Total Group (including both the Rugby Group and Non-Contact Sports Control Group, n = 59, 42 and 36 respectively) in relation to the neurocognitive assessment measures (ImPACT and Purdue Pegboard) at the three assessment intervals (*pre-*, *mid-* and *post*-season) (Table 7.5),

2) *concussions* reported on the biographical questionnaire for the Rugby Group (n = 20) used in relation to the neurocognitive assessment measures (ImPACT and Purdue Pegboard) at the three assessment intervals (*pre-*, *mid-* and *post*-season) (Table 7.6),

3) *concussions* reported on the biographical questionnaire for the Rugby Group (n = 20) in relation to the tackling data (Table 7.7), and

4) *tackling data* for the Rugby Group in relation to the neurocognitive measures (ImPACT and Purdue Pegboard) at the three assessment intervals (*pre-*, *mid-* and *post*-season) (Table 7.8, Table 7.9 and Table 7.10).

Exploratory Spearman's correlational analyses that were run on the neurocognitive measures (ImPACT and Purdue Pegboard) were reported on in terms of significances in the modalities of ***Memory*** and ***Motor Speed***. In addition, for the purposes of highly tentative exploration of consistent trends, when the correlations for a particular neurocognitive test across the three assessment intervals (*pre-*, *mid-* and *post*-season) were all in the hypothesized direction of *more* concussions associated with *poorer* neurocognitive performance, these findings were highlighted in yellow. Similarly, for the purposes of tentative and speculative exploration of consistent trends, when the correlations for a particular neurocognitive test across the three assessment intervals (*pre-*, *mid-* and *post*-season) were all in the opposite direction of *more*

concussions associated with *improved* neurocognitive performance, these findings were highlighted in blue.

Table 7.5 details the analyses of the *concussions* reported on the biographical questionnaire for the Total Group (including both the Rugby Group and Non-Contact Sports Control Group) in relation to the neurocognitive measures (ImPACT and Purdue Pegboard) at the three assessment intervals (*pre-*, *mid-* and *post*-season). There were no significant correlations in respect of the number of concussions reported in relation to the neurocognitive test data in the modality of Memory, but there were a number of significant correlations in the direction of *more* concussions being associated with *poorer* neurocognitive performance in the modality of Motor Speed: (i) Purdue Preferred (r = -0.295, p = 0.029 at the *mid*-season assessment interval), (ii) Purdue Non-Preferred (r = -0.225, p = 0.043; and r = -0.259, p = 0.049 at the *pre-* and *mid*-season assessment intervals, respectively). In addition, there were consistent trends, in the direction of *more* concussions being associated with *poorer* neurocognitive performance, for the modality of ***Memory***: (i) ImPACT Verbal Memory (r = -0.104, p = 0.217; r = -0.061, p = 0.350; r = -0.149, p = 0.192, at each of the assessment intervals, respectively) and for the modality of ***Motor Speed*** (i) ImPACT Visual Motor Speed (r = -0.151, p = 0.127; r = -0.109, p = 0.246; r = -0.185, p = 0.140, at each of the assessment intervals, respectively), (ii) ImPACT Reaction Time (r = 0.114, p = 0.195; r = 0.155, p = 0.164; r = 0.225, p = 0.094, at each of the assessment intervals, respectively), (iii) Purdue Preferred (r = -0.148, p = 0.132; r = -0.241, p = 0.079, at the *pre-* and *post-* assessment intervals, respectively), (iv) Purdue Non-Preferred (r = -0.090, p = 0.302, at the *post*-season assessment interval), and (v) Purdue Both (r = -0.088, p = 0.253; r = -0.160, p = 0.155; r = -0.155, p = 0.183, at each of the assessment intervals, respectively).

Table 7.6 detailed the analyses of the *concussions* reported on the biographical questionnaire for the Rugby Group in relation to the neurocognitive assessment measures (ImPACT and Purdue Pegboard) at the three assessment intervals (*pre-*, *mid-* and *post*-season). There were no significant correlations in respect of the number of concussions reported in relation to the neurocognitive test data in the modalities of Memory or Motor Speed. A consistent trend, in the direction of *more* concussions being associated with *poorer* neurocognitive performance, was in evidence for the modality of *Memory* (i) ImPACT Verbal Memory (r = -0.104, p =

0.217; r = -0.137, p = 0.861; r = -0.229, p = 0.805, at each of the assessment intervals, respectively).

Table 7.7 detailed the analyses on the *concussions* reported on the biographical questionnaire for the Rugby Group in relation to the tackling data, in respect of the number of concussions reported and the different tackling categories (Tackles Made, Tackles Received and Total Tackles). A consistent trend, in the direction of *more* concussions being associated with *higher* number of tackles, was in evidence for all three tackling categories (i) Tackles Made (r = 0.146, p = 0.270), (ii) Tackles Received (r = 0.045, p = 0.426), and (iii) Total Tackles (r = 0.0017, p = 0.383).

Table 7.8 detailed the analyses on the *tackling data* (Tackles Made) for the Rugby Group in relation to the neurocognitive measures (ImPACT and Purdue Pegboard) at the three assessment intervals (*pre-*, *mid-*, and *post*-season) and there were no significant results in the modalities of Memory or Motor Speed. In the modality of ***Motor Speed*** the following were in evidence in the direction of a *higher* number of tackles being associated with *poorer* neurocognitive performance: (i) Purdue Non-Preferred (r = -0.235, p = 0.320; r = -0.210, p = 0.375; r = -0.118, p = 0.621, at each of the assessment intervals, respectively), (ii) Purdue Both (r = -0.274, p = 0.242; r = -0.136, p = 0.568; r = -0.214, p = 0.365, at each of the assessment intervals, respectively). It is of note that there were consistent tendencies in the opposite direction of a higher number of tackles being associated with improved neurocognitive scores (highlighted in blue) in the modality of ***Motor Speed*** on (i) ImPACT Visual Motor Speed (r = 0.229, p = 0.332; r = 0.195, p = 0.409; r = 0.338, p = 0.145 at each of the assessment intervals, respectively), and (ii) ImPACT Reaction Time (r = -0.308, p = 0.186; r = -0.310, p = 0.184; r = -0.303, p = 0.193, at each of the assessment intervals, respectively).

Table 7.9 detailed the analyses on the *tackling data* (Tackles Received) for the Rugby Group in relation to the neurocognitive measures (ImPACT and Purdue Pegboard) at the three assessment intervals (*pre-*, *mid-*, and *post*-season) and there were no significant results in the modalities of Memory or Motor Speed. In the modality of ***Motor Speed*** the following were in evidence in the direction of a *higher* number of tackles being associated with *poorer*

neurocognitive performance: (i) Purdue Non-Preferred (r = -0.117, p = 0.623; r = -0.164, p = 0.491; r = -0.180, p = 0.446, respectively at each of the assessment intervals), (ii) Purdue Both (r = -0.412, p = 0.071; r = -0.142, p = 0.550; r = -0.389, p = 0.090, respectively at each of the assessment intervals). It is of note that there were consistent tendencies in the opposite direction of a higher number of tackles being associated with improved neurocognitive scores (highlighted in blue) in the modality of ***Memory*** on (i) ImPACT Visual Memory (r = 0.133, p = 0.576; r = 0.160, p = 0.501; r = 0.021, p = 0.931) at each of the assessment intervals, respectively), and in the modality of ***Motor Speed*** on (i) ImPACT Visual Motor Speed (r = 0.348, p = 0.132; r = 0.317, p = 0.174; r = 0.390, p = 0.089 at each of the assessment intervals, respectively), and (ii) ImPACT Reaction Time (r = -0.344, p = 0.137; r = -0.380, p = 0.098; r = -0.298, p = 0.202, at each of the assessment intervals, respectively).

Table 7.10 detailed the analyses on the *tackling data* (Total Tackles) for the Rugby Group in relation to the neurocognitive measures (ImPACT and Purdue Pegboard) at the three assessment intervals (*pre-*, *mid-*, and *post*-season) and there were no significant results in the modalities of Memory or Motor Speed. In the modality of ***Motor Speed*** the following were in evidence in the direction of a *higher* number of tackles being associated with *poorer* neurocognitive performance: (i) Purdue Non-Preferred (r = -0.175, p = 0.460; r = -0.174, p = 0.462; r = -0.166, p = 0.483, respectively at each of the assessment intervals), (ii) Purdue Both (r = -0.317, p = 0.173; r = -0.089, p = 0.708; r = -0.310, p = 0.183, respectively at each of the assessment intervals). It is of note that there were consistent tendencies in the opposite direction of a higher number of tackles being associated with improved neurocognitive scores (highlighted in blue) in the modality of ***Memory*** on (i) ImPACT Visual Memory (r = 0.169, p = 0.476; r = 0.204, p = 0.389; r = 0.047, p = 0.843 at each of the assessment intervals, respectively), and in the modality of ***Motor Speed*** on (i) ImPACT Visual Motor Speed (r = 0.334, p = 0.150; r = 0.272, p = 0.246; r = 0.388, p = 0.091 at each of the assessment intervals, respectively), and (ii) ImPACT Reaction Time (r = -0.345, p = 0.137; r = -0.380, p = 0.099; r = -0.351, p = 0.129, at each of the assessment intervals, respectively).

Table 7.5 Correlation Analysis for Reported Concussions in relation to Neurocognitive Assessment Results for the Total Group (Rugby and Non-Contact Sports controls) at the Three Assessment Intervals

Concussion Data Correlations						
Neurocognitive Measures	**Pre-season (*n* = 59)**		**Mid-season (*n* = 42)**		**Post-season (*n* = 36)**	
	Correlation Statistic (*r*)	***p*-value**	**Correlation Statistic (*r*)**	***p*-value**	**Correlation Statistic (*r*)**	***p*-value**
MEMORY						
ImPACT Verbal Memory	-0.104	0.217	-0.061	0.350	-0.149	0.192
ImPACT Visual Memory	-0.078	0.278	0.104	0.255	-0.065	0.345
MOTOR SPEED						
ImPACT VMS[l]	-0.151	0.127	-0.109	0.246	-0.185	0.140
ImPACT Reaction Time	0.114	0.195	0.155	0.164	0.225	0.094
Purdue Preferred	-0.148	0.132	-0.295	0.029*	-0.241	0.079
Purdue Non-Preferred	-0.225	0.043*	-0.259	0.049*	-0.090	0.302
Purdue Both	-0.088	0.253	-0.160	0.155	-0.155	0.183
Purdue Assembly	0.015	0.454	0.020	0.450	-0.062	0.359

Not Significant unless otherwise specified
* Correlation is significant at the 0.05 level (1-tailed)
[l]Note: Visual Motor Speed (VMS)

Table 7.6 Correlation Analysis for Reported Concussions in relation to Neurocognitive Assessment Results for the Rugby Group at the Three Assessment Intervals

Concussion Data Correlations (n = 20)						
Neurocognitive Measures	**Pre-season**		**Mid-season**		**Post-season**	
	(*r*)	***p*-value**	**(*r*)**	***p*-value**	**(*r*)**	***p*-value**
MEMORY						
ImPACT Verbal Memory	-0.104	0.217	-0.137	0.861	-0.229	0.805
ImPACT Visual Memory	-0.078	0.278	0.271	0.439	0.119	0.320
MOTOR SPEED						
ImPACT VMS[1]	-0.151	0.127	0.055	0.574	0.038	0.811
ImPACT Reaction Time	0.114	0.195	-0.113	0.197	0.006	0.538
Purdue Preferred	-0.148	0.426	0.116	0.473	0.156	0.299
Purdue Non-Preferred	-0.225	0.466	0.298	0.264	0.221	0.076
Purdue Both	-0.088	0.552	0.216	0.308	0.079	0.653
Purdue Assembly	0.015	0.934	0.247	0.255	0.186	0.348

Not Significant unless otherwise specified
(*r*) = Correlation Statistic
[1]Note: Visual Motor Speed (VMS)

Table 7.7 Correlation Analysis for Reported Concussions in relation to Tackling Data for the Rugby Group

Concussion Data Correlations (n = 20)		
	Correlation Statistic (*r*)	***p*-value**
Tackles Made	0.146	0.270
Tackles Received	0.045	0.426
Total Tackles	0.017	0.383

Not Significant unless otherwise specified

Table 7.8 Correlation Analysis for Tackles Made in relation to Neurocognitive Assessment Results for the Rugby Group at the Three Assessment Intervals

Tackling Data Correlations – Tackles Made (*n* = 20)

Neurocognitive Measures	Pre-season		Mid-season		Post-season	
	(*r*)	*p*-value	(*r*)	*p*-value	(*r*)	*p*-value
MEMORY						
ImPACT Verbal Memory	-0.017	0.945	0.296	0.205	-0.149	0.530
ImPACT Visual Memory	0.221	0.349	0.182	0.443	-0.085	0.721
MOTOR SPEED						
ImPACT VMS[1]	0.229	0.332	0.195	0.409	0.338	0.145
ImPACT Reaction Time	-0.308	0.186	-0.310	0.184	-0.303	0.193
Purdue Preferred	-0.056	0.813	-0.010	0.966	0.130	0.584
Purdue Non-Preferred	-0.235	0.320	-0.210	0.375	-0.118	0.621
Purdue Both	-0.274	0.242	-0.136	0.568	-0.214	0.365
Purdue Assembly	-0.056	0.815	0.072	0.762	-0.074	0.757

Not Significant unless otherwise specified
(*r*) = Correlation Statistic
[1]Note: Visual Motor Speed (VMS)

Table 7.9 Correlation Analysis for Tackles Received in relation to Neurocognitive Assessment Results for the Rugby Group at the Three Assessment Intervals

Tackling Data Correlations – Tackles Received (*n* = 20)

Neurocognitive Measures	Pre-season		Mid-season		Post-season	
	(*r*)	*p*-value	(*r*)	*p*-value	(*r*)	*p*-value
MEMORY						
ImPACT Verbal Memory	-0.059	0.806	0.375	0.104	0.026	0.915
ImPACT Visual Memory	0.133	0.576	0.160	0.501	0.021	0.931
MOTOR SPEED						
ImPACT VMS[1]	0.348	0.132	0.317	0.174	0.390	0.089
ImPACT Reaction Time	-0.344	0.137	-0.380	0.098	-0.298	0.202
Purdue Preferred	-0.199	0.401	-0.124	0.602	0.041	0.865
Purdue Non-Preferred	-0.117	0.623	-0.164	0.491	-0.180	0.446
Purdue Both	-0.412	0.071	-0.142	0.550	-0.389	0.090
Purdue Assembly	-0.202	0.393	0.111	0.643	-0.046	0.847

Not Significant unless otherwise specified
(*r*) = Correlation Statistic
[1]Note: Visual Motor Speed (VMS)

Table 7.10 Correlation Analysis for Total Tackles in relation to Neurocognitive Assessment Results for the Rugby Group at the Three Assessment Intervals

Tackling Data Correlations – Total Tackles (n = 20)						
Neurocognitive Measures	**Pre-season**		**Mid-season**		**Post-season**	
	(*r*)	***p*-value**	**(*r*)**	***p*-value**	**(*r*)**	***p*-value**
MEMORY						
ImPACT Verbal Memory	-0.012	0.960	0.406	0.076	0.006	0.980
ImPACT Visual Memory	0.169	0.476	0.204	0.389	0.047	0.843
MOTOR SPEED						
ImPACT VMS[1]	0.334	0.150	0.272	0.246	0.388	0.091
ImPACT Reaction Time	-0.345	0.137	-0.380	0.099	-0.351	0.129
Purdue Preferred	-0.147	0.538	-0.041	0.863	0.123	0.605
Purdue Non-Preferred	-0.175	0.460	-0.174	0.462	-0.166	0.483
Purdue Both	-0.317	0.173	-0.089	0.708	-0.310	0.183
Purdue Assembly	-0.119	0.618	0.146	0.538	0.011	0.962

Not Significant unless otherwise specified
(*r*) = Correlation Statistic
[1]Note: Visual Motor Speed (VMS)

7.5 SYNTHESIS FOR ALL TACKLING AND CORRELATIONAL ANALYSES

Perusal of the tackling data reveal that on average the Rugby Group was involved in a total of 103.45 tackles over one rugby season and on average there were 60.7 Tackles Made and 42.75 Tackles Received. It would be feasible, taking the average club level rugby player and multiplying the average of tackles over one rugby season by the years of exposure to the game, and this translates into more than a thousand tackles per individual, excluding any contact practice sessions.

The overall findings on the tackling data support the hypotheses in that there was a consistent trend, in the direction of *more* concussions being associated with *higher* number of tackles. There was no definitive evidence that the exposure to the amount of tackles made or received contributed directly to the likelihood of sustaining a possible concussive or subconcussive injury during the season.

In terms of this study a number of tentative and speculative exploratory Spearman's Correlational analyses were run but found no significant results and not all of the results were going in the hypothesized direction. However, correlations with regards to the number of reported concussions in relation to the tackling data were consistently in the hypothesized direction of *more* concussions being associated with a *higher* number of tackles.

CHAPTER 8

RESULTS: INDIVIDUAL PLAYER ANALYSES

This chapter presents the individual profiles of the five rugby players who underwent *pre*-season assessments and who were perceived to have sustained a possible concussive injury during the rugby season. The chapter first depicts the overall demographic and clinical features of the individual players and includes the procedural aspects. This is followed by the detailed individual analysis of each of the rugby players in turn, taking into consideration within-subject clinical and demographic details in conjunction with overall tackling and neurocognitive assessment outcomes from *pre*-season through each of the follow-up assessment intervals.

8.1 OVERALL DEMOGRAPHIC AND CLINICAL FEATURES AND PROCEDURAL ASPECTS

No players during the season were formally diagnosed with a concussion, and the players for analysis in this chapter would have gone unnoticed even for a suspected concussive incident if not for the retrospective video analyses of the games. Because concussive and subconcussive injuries occur in a split second, it was difficult to provide exact information on injury mechanisms and play situations leading up to possible injuries at the time of play. Accordingly, a number of players with subtle changes in their performance during a game implicating a possible concussive event were identified on the video analyses following each game, and were approached for follow-up examination on that basis. In that none of them were formally diagnosed with a concussion or sustained an identifiable loss of consciousness, these individual players' results represent the relatively mild spectrum of concussive and subconcussive injury. For this reason, these players are of investigative interest from a neuropsychological perspective in their own right.

For the purpose of this study, all of these individual players underwent *pre*-season assessments as well as follow-up assessments within 72 hours following the suspected concussion (one player was followed up for the second time on post-injury day nine). A summary table of core demographic, concussion history and assessment data in respect of each of the five rugby players and the number of follow-up assessments conducted in each case is indicated in Table 8.1.

Table 8.1 Demographic, Concussion, and Assessment Data in respect of the Five Rugby Players with Suspected Concussion identified for Follow-up Evaluation

	Age	Estimated IQ[1]	Prior Concussions	Follow-up Assessments
Player A	23	113	4	2 Follow-ups Prior to Mid-season
Player B	31	103	None reported	1 Follow-up Prior to Mid-season
Player C	27	116	1	1 Follow-up Prior to Mid-season
Player D	28	101	10	1 Follow-up Prior to Mid-season
Player E	26	104	1	1 Follow-up Prior to Post-season

[1]*Note:* Control for estimated Full Scale IQ established on the basis of WAIS-III Picture Completion and Matrix Reasoning Scaled Scores using the OPIE-3 Estimation Formula.

In order to enhance the detailed analysis of the five rugby players with a suspected concussion, these individual players' tackling data were compared with the Rugby Group means for each tackling category. As tabled in the previous section on the seasonal tackling data (Table 7.4, page 140) the Rugby Group was involved as follows, (i) Total Tackles (M = 103.45, SD = 59.98), (ii) Tackles Made (M = 60.70, SD = 36.16), (iii) Tackles Received (M = 42.75, SD = 26.22). For the purposes of the individual analyses, in each tackling category the rugby players were sorted and ranked from the highest to the lowest number of tackles and the individual players' were plotted against the group performance (player A to E) (Table 8.2).

Table 8.2 Individual Players' position in relation to the Rugby Group with reference to Tackles Made, Tackles Received and Total Tackles

Player	Tackles Made	Player	Tackles Received	Player	Total Tackles
2	108	10	151	10	241
10	90	2	122	2	230
7	74	7	102	7	176
B	**63**	12	101	12	146
AVG	**60.70**	13	79	13	136
15	57	**D**	**74**	**D**	**122**
13	57	6	72	**B**	**120**
16	49	**E**	**71**	**E**	**114**
D	**48**	16	58	6	108
12	45	**B**	**57**	16	107
9	44	9	56	**AVG**	**103.45**
E	**43**	**C**	**53**	9	100
6	36	**A**	**52**	15	90
A	**32**	3	46	**A**	**84**
1	27	**AVG**	**42.75**	**C**	**79**
C	**26**	8	41	8	59
4	24	15	33	1	56
14	24	1	29	3	52
8	18	4	28	4	52
5	9	5	14	14	38
11	8	14	14	5	25
3	6	11	9	11	17

Note: The Rugby Group Mean for each tackling category is indicated in bold as AVG
The Letters refer to the 5 corresponding players included in the Individual Analyses
The Numbers refer to the remaining 16 players not included in the Individual Analyses

8.2 DETAILED INDIVIDUAL RUGBY PLAYER ANALYSES

Each of the individual players is introduced with his biographical information (general information, educational history with an IQ estimate, medical and psychiatric history, and history of prior concussions). This is followed by the individual player's neurocognitive assessment results (ImPACT and Purdue Pegboard). Specifically, the neurocognitive assessment results were divided into the two modalities of Memory (ImPACT Verbal Memory, ImPACT Visual Memory) and Motor Speed (ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both and Purdue Assembly). The neurocognitive assessment results will be presented at the *pre-*, *mid-*, *post-*season, and post-concussion follow-up assessment intervals.

For comparative purposes, each individual player's assessment results for each of the neurocognitive measures were followed by the Non-Contact Sports control mean score for that assessed measure (derived from the group analysis, Table 7.2), at the three primary assessment intervals (*pre-*, *mid-*, and *post*-season). In order to facilitate the comparisons between derived scores and normative data available for the various measures, descriptive data will be converted into comparable statistical formats. Scores for ImPACT Verbal Memory and ImPACT Visual Memory will be multiplied by 100 in order to be statistically compatible with the integer statistic used for the US normative ranges. Scores for ImPACT Visual Motor Speed will be rounded off to one decimal point as per the US norms; scores for ImPACT Reaction time will be retained as is (i.e., rounded off to two decimal points) as per the US norms; and scores for each of the Purdue Pegboard measures will be retained as is (i.e., rounded off to two decimal points) as per the US norms. The individualised neurocognitive assessment score table and the figures for each neurocognitive measure will appear at the end of each relevant player's analysis.

In order to monitor for a significant decline and for the speed of recovery over the whole assessment series, the individual assessment results on ImPACT will be interpreted in relation to the US normative categories (Table 8.3) (ImPACT, 2004) as well as the player's own *pre*-season scores. The Purdue Pegboard assessment results will be interpreted in

relation to the corresponding normative data for adults, stratified on the basis of age (Table 8.4), the Purdue Pegboard normative categories (Table 8.5) (Strauss et al., 2006; Yeudall, Fromm, Reddon & Steffanyk, 1986), and the player's own *pre*-season scores.

This is followed by the individual player's Tackling data (number and type of tackles) across one rugby season. For this purpose, the two main tackling categories (Tackles Made and Tackles Received) were each broken down further, into the two subcategories of *above* and *below* the waist. This was done to assess the possible link to the cumulative aspect of frequent head and body collisions. The respective tackling categories were perused in relation to the overall detailed tackling data of the whole Rugby Group. The relevant Tables and Figures of the tackling data will appear collectively to highlight and reflect the descriptive statistics calculated on the basis of the number of games played by each player.

Table 8.3 ImPACT Normative Categories

	Verbal Memory	Visual Memory	Visual Motor Speed	Reaction Time
Impaired	< 71	< 51	< 23.8	> 0.75
Borderline	72-77	52-60	23.9-28.3	0.74-0.67
Low Average	78-82	61-68	28.4-32.4	0.66-0.61
Average	83-94	69-94	32.5-42.0	0.60-0.51
High Average	95-97	95-97	42.1-46.0	0.50-0.48
Superior	98-99	98-99	46.1-50.0	0.47-0.45
Very Superior	100	100	> 50.0	< 0.44

(ImPACT, 2004)

Table 8.4 Purdue Pegboard Normative Data stratified on the basis of Age

	Age Groups					
	21-25	**(SD)**	**26-30**	**(SD)**	**31-40**	**(SD)**
Preferred	15.44	(1.71)	16.22	(1.81)	15.35	(1.72)
Non-Preferred	15.08	(1.98)	15.41	(2.08)	15.12	(1.77)
Both	12.97	(1.18)	12.94	(1.29)	12.42	(1.65)
Assembly	38.89	(6.6)	39.13	(3.58)	37.50	(3.64)

(Strauss et al., 2006; Yeudall et al., 1986)

Table 8.5 Purdue Pegboard Normative Categories

	Age Group	**Poor**	**Low Average**	**Average**		**High Average**	**Excellent**
		-2 SD	-1 SD	Mean	(SD)	+ 1 SD	+2 SD
Preferred	I	12.02	13.73	15.44	(1.71)	17.15	18.86
	II	12.60	14.41	16.22	(1.81)	18.03	19.84
	III	11.91	13.63	15.35	(1.72)	17.07	18.79
Non-Preferred	I	11.12	13.10	15.08	(1.98)	17.06	19.04
	II	11.25	13.33	15.41	(2.08)	17.49	19.57
	III	11.58	13.35	15.12	(1.77)	16.89	18.66
Both	I	10.61	11.79	12.97	(1.18)	14.15	15.33
	II	10.36	11.65	12.94	(1.29)	14.23	15.52
	III	9.12	10.77	12.42	(1.65)	14.07	15.72
Assembly	I	25.69	32.29	38.89	(6.60)	45.49	52.09
	II	31.97	35.55	39.13	(3.58)	42.71	46.29
	III	30.22	33.86	37.50	(3.64)	41.14	44.78

Note: I = Age Group 21-25; II = Age Group 26-30; III = Age Group 31-40
(Strauss et al., 2006; Yeudall et al., 1986)

8.2.1 Demographic and Clinical History of Player A

GENERAL INFORMATION					
Subject		*Player A*		**Age**	23
Home language	English	**Other**		**Race:**	White
EDUCATION, OCCUPATION AND ESTIMATE IQ					
Level of Education completed			Grade 12, Tertiary Diploma		
Current study, if any			None		
Occupation			Sales Executive		
Estimate IQ (Established on the basis of WAIS-III Picture Completion and Matrix Reasoning Scaled Scores using the OPIE-3 Formula)			113 (Above Average)		
MEDICAL AND PSYCHIATRIC HISTORY					
None					
CONCUSSION HISTORY					
Prior Concussions			4		

8.2.1.1 Player A: Neurocognitive Assessment Results

For *Player A*, the neurocognitive assessment results across all assessment intervals (*pre-*, *mid-*, and *post*-season as well as post-concussion) for each measure are tabulated together with the Non-Contact Sports control mean and the US average ranges (Table 8.6), and illustrated further by means of a figure in respect of each separate measure (Figures 8.1 – 8.8). For discussion purposes the results are interpreted in terms of the normative categories for both ImPACT and the Purdue Pegboard, as found earlier in Tables 8.3 and 8.5. The tables and figures for *Player A* appear together at the end of this subsection (page 161-165).

8.2.1.1.1 Memory

ImPACT Verbal Memory (Table 8.6; Figure 8.1). At the *pre*-season assessment interval *Player A*'s Verbal Memory score fell at the ceiling of the test, revealing a "very superior" performance relative to the US normative categories. At the *P-c1* assessment interval there was a decrease in evidence relative to the *pre*-season score although it was still a "superior" performance relative to the US normative categories. At the *P-c2* assessment interval the score that showed some lowered performance at *P-c1* improved back to the *pre*-season

ceiling level of a "very superior" performance relative to the US normative categories. At the *mid*- and *post*-season assessment intervals the scores continued to reveal the same ceiling level of a "very superior" performance relative to the US normative categories. Compared with the mean scores of the Non-Contact Sports Control Group, *Player A* was consistently performing at a higher level than the controls. The controls remained relatively consistent over the first two assessments and improved at the *post*-season assessment interval, whereas in contrast *Player A* declined in performance immediately post injury, only subsequently returning to his pre-injury level. Given that *Player A* was performing at the ceiling on all assessments except at *Pc-1,* the improvement shown by the controls at the *post*-season interval was not possible for *Player A* in that he was already at the ceiling.

ImPACT Visual Memory (Table 8.6; Figure 8.2). At the *pre*-season assessment interval *Player A*'s Visual Memory score denoted a performance in the upper limits of the "average" range relative to the US normative categories. At the *P-c1* assessment interval there was an improvement in evidence relative to the *pre*-season score albeit still in the upper limits of the "average" range relative to the US normative categories. The *P-c2* assessment interval, the score that showed some improved performance at *P-c1* improved further and denoted a "high average" performance relative to the US normative categories. At the *mid*-season assessment interval, however, there was a worsening in evidence relative to the *P-c1* and *P-c2* scores although it was still an "average" performance relative to the US normative categories. At the *post*-season assessment interval the score showed a substantial improvement on the previous fluctuating scores, and denoted a "superior" performance relative to the US normative categories. Compared with the mean scores of the Non-Contact Sports Control Group, *Player A* was consistently performing at a higher level than the controls. The controls revealed a steady improvement from *pre*- to *mid*-season with a sharp improvement at the *post*-season assessment interval, whereas in contrast *Player A* revealed intermittent performance, and only by *post*-season he scored close to the ceiling of the test.

8.2.1.1.2 Motor Speed

ImPACT Visual Motor Speed (Table 8.6; Figure 8.3). At the *pre*-season assessment interval *Player A*'s Visual Motor Speed score was at a "very superior" level relative to the US normative categories. At the *P-c1* assessment interval there was an improvement in performance relative to the *pre*-season level, denoting a "very superior" performance relative to the US normative categories. At the *P-c2* assessment interval there was a marginal worsening in evidence relative to the *P-c1* assessment score, albeit still being a higher score than achieved at the *pre*-season assessment interval and still denoting a "superior" performance relative to the US normative categories. At the *mid*- and *post*-season assessment intervals the scores were similar to the *P-c1* level, and still denoting a "very superior" performance relative to the US normative categories. In comparison with the Non-Contact Sports Control Group, *Player A* was consistently performing at a superior level across all assessment intervals. The controls revealed a steady improvement from the *pre*- to *mid*-season and the *mid*-season to *post*-season assessment intervals, whereas in contrast *Player A* revealed some intermittent decline in performance due to a lowering at the *P-c2* assessment, although ultimately after multiple assessments he did reveal the ability to improve even further with scores exceeding those of his *pre*-season level of performance.

ImPACT Reaction Time (Table 8.6; Figure 8.4). At the *pre*-season assessment interval *Player A*'s Reaction Time score denoted a "very superior" performance relative to the US normative categories. At the *P-c1* assessment interval there was a marginal increase in Reaction Time, (i.e. a worsening in performance) denoting a "superior" performance relative to the US normative categories. At the *P-c2* and *mid*-season assessment intervals the Reaction Time scores that showed some lowered performance at the *P-c 1* assessment interval revealed a decrease in Reaction Time (i.e. improved performance), denoting a "superior" performance relative to the US normative categories. At the *post*-season assessment interval Reaction Time showed good recovery, and was similar to the *pre*-season level, denoting a "very superior" performance relative to the US normative categories. In comparison with the Non-Contact Sports Control Group, *Player A* was consistently performing at a higher level across all intervals. The controls showed an improved performance due to a decreased Reaction Time score from *pre*- to *mid*-season and stabilised

by the *post*-season assessment interval, whereas in contrast *Player A* revealed some intermittent decline in performance, although ultimately after multiple assessments he regained his *pre*-season scores at the *post*-season assessment interval.

Purdue Preferred (Table 8.6; Figure 8.5). At the *pre*-season assessment interval *Player A*'s Preferred score denoted an "average" performance relative to the Purdue normative categories. At the *P-c1, P-c2* and *mid*-season assessment intervals the scores remained the same, denoting an "average" performance relative to the Purdue normative categories. At the *post*-season assessment interval there was a marginal improvement on all the previous assessments, albeit still denoting an "average" performance relative to the Purdue normative categories. In comparison with the Non-Contact Sports Control Group, *Player A* was consistently performing at a similar level across all intervals. The controls revealed an improvement at the *mid*-season assessment interval, whereas in contrast *Player A* remained consistent in his performance, and ultimately after multiple assessments he only improved to the Non-Contact Sports Control Group level at the *post*-season assessment interval.

Purdue Non-Preferred (Table 8.6; Figure 8.6). At the *pre*-season assessment interval *Player A*'s Non-Preferred score fell in the direction that denoted an "average" performance relative to the Purdue normative categories. At the *P-c1, P-c2* and *mid*-season assessment intervals the scores remained the same denoting an "average" performance relative to the Purdue normative categories. At the *post*-season assessment interval there was a marginal improvement on all the previous assessments, albeit still denoting an "average" performance relative to the Purdue normative categories. In comparison with the Non-Contact Sports Control Group, *Player A* was consistently performing at a similar level across all intervals. The controls revealed an improvement at the *mid*-season assessment interval, whereas in contrast *Player A* remained consistent in his performance, and ultimately after multiple assessments he only improved to the Non-Contact Sports Control Group level at the *post*-season assessment interval.

Purdue Both (Table 8.6; Figure 8.7). At the *pre*-season assessment interval *Player A*'s Both score denoted an "average" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was improvement in evidence compared to *pre*-season and

denoting a "high average" performance relative to the Purdue normative categories. At the *P-c2* assessment interval there was a decrease in evidence compared to the *P-c1* scores denoting an "average" performance relative to the Purdue normative categories and was similar to his *pre*-season levels. At the *mid*-season assessment interval there was an improvement in evidence, similar to the *P-c2* score, but at the *post*-season assessment interval the fluctuations in performance was again evident in the score being similar to the *pre*-season level, albeit still denoting an "average" performance relative to the Purdue normative categories. In comparison with the Non-Contact Sports Control Group, *Player A* was fluctuating in performance across all assessment intervals. The controls showed an improvement by *mid*-season and were consistent at *post*-season, whereas in contrast *Player A* revealed some intermittent fluctuation in performance due to a lowering at the *P-c2* and *post*-season assessment intervals, and ultimately after multiple assessments he struggled to maintain his alternating improved performances and fell back to his *pre*-season score at the *post*-season assessment interval.

Purdue Assembly (Table 8.6; Figure 8.8)**.** At the *pre*-season assessment interval *Player A*'s Assembly score denoted a "high average" performance relative to the Purdue normative categories. At the *P-c1* assessment interval the scores remained the same, denoting a "high average" performance relative to the Purdue normative categories. At the *P-c2* assessment interval there was a marginal decrease in evidence compared to the *P-c1* scores, albeit still denoting a "high average" performance relative to the Purdue normative categories. At the *mid*-season assessment interval there was a marginal improvement on the previous assessment score, not back to the *pre*-season level, but still denoting an "average" performance relative to the Purdue normative categories. At the *post*-season assessment interval the fluctuations in performance was again evident with a substantial improvement on all the previous assessments, denoting an "above average" performance relative to the Purdue normative categories. In comparison with the Non-Contact Sports Control Group, *Player A* was consistently performing at a higher level across all intervals. The controls revealed a slight fluctuation on the *mid*-season assessment interval, implicating that the controls possible started off closer to their ceiling, whereas in comparison *Player A* revealed some intermittent decline in performance due to a lowering at the *P-c2* and *mid*-season assessment intervals, although ultimately after multiple assessments he did reveal the ability to benefit

from practice with scores exceeding those of his *pre*-season scores at the *post*-season assessment interval.

Table 8.6 ***Player A's*** **ImPACT and Purdue Pegboard Repeat Assessment Scores vs the Non-Contact Sports Control Mean Score and the US Average Range**

	Pre-season 2005/02/19	**P-c1 2005/06/06**	**P-c2 2005/06/14**	**Mid-season 2005/06/23**	**Post-season 2005/09/20**	**US Average[1]**
MEMORY						
ImPACT Verbal Memory						
Player A	100	96	100	100	100	83 – 94
Control Mean Score	84	-	-	85	87	
ImPACT Visual Memory						
Player A	90	94	96	90	98	69 – 94
Control Mean Score	74	-	-	76	82	
MOTOR SPEED						
ImPACT VMS[2]						
Player A	47.3	53.7	51.0	53.4	53.3	32.5 -
Control Mean Score	37.5	-	-	39.5	39.0	42.0
ImPACT Reaction Time						
Player A	0.44	0.48	0.46	0.46	0.44	0.60 -
Control Mean Score	0.55	-	-	0.50	0.52	0.51
Purdue Preferred						
Player A	16.00	16.00	16.00	16.00	17.00	15.44
Control Mean Score	16.05	-	-	17.00	17.09	(1.71)
Purdue Non-Preferred						
Player A	15.00	15.00	15.00	15.00	16.00	15.08
Control Mean Score	15.09	-	-	16.05	16.23	(1.98)
Purdue Both						
Player A	12.00	14.00	12.00	14.00	12.00	12.97
Control Mean Score	12.82	-	-	13.07	13.36	(1.18)
Purdue Assembly						
Player A	43.00	43.00	41.00	42.00	47.00	38.89
Control Mean Score	36.41	-	-	35.55	36.45	(6.60)

[1]Note: ImPACT delineated by range; Purdue Pegboard in mean score and Standard Deviation
Bold print represent scores that fall below the lower limit of the average ranges of both ImPACT and the Purdue Pegboard
[2]Note: Visual Motor Speed (VMS)

Figure 8.1 ***Player A's*** **ImPACT Verbal Memory Composite Scores at the Pre-season, Post-concussion 1, Post-concussion 2, Mid- and Post-season Assessment Intervals**

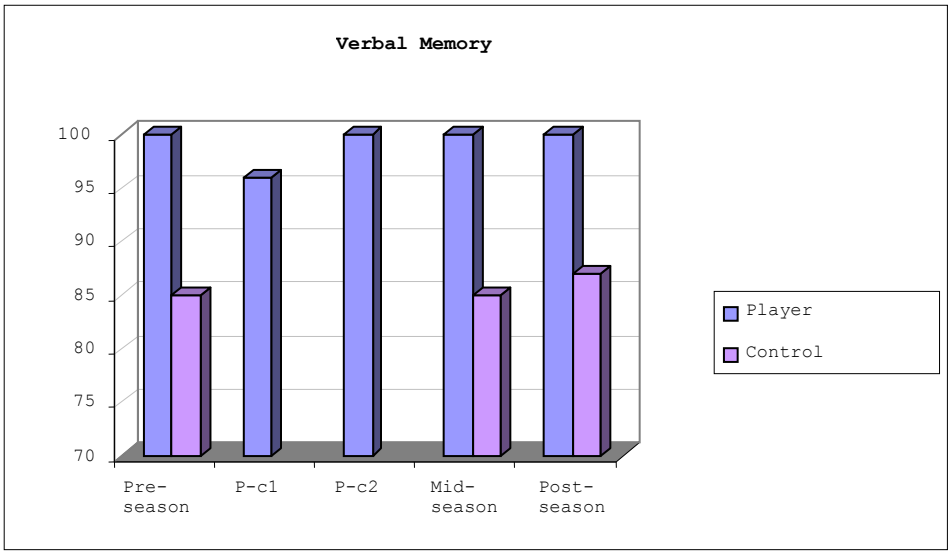


Figure 8.2 ***Player A's*** **ImPACT Visual Memory Composite Scores at the Pre-season, Post-concussion 1, Post-concussion 2, Mid- and Post-season Assessment Intervals**

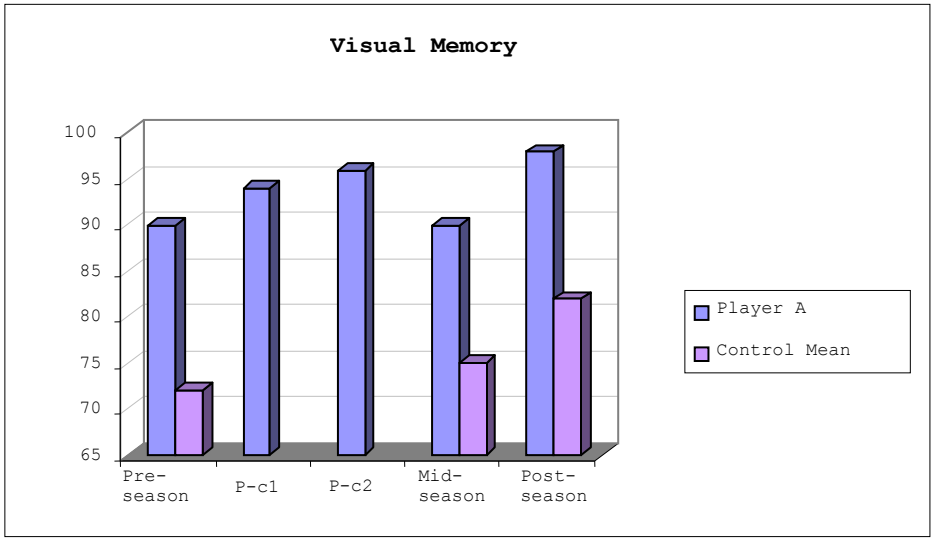


Figure 8.3 Player A's ImPACT Visual Motor Speed Composite Scores at the Pre-season, Post-concussion 1, Post-concussion 2, Mid- and Post-season Assessment Intervals

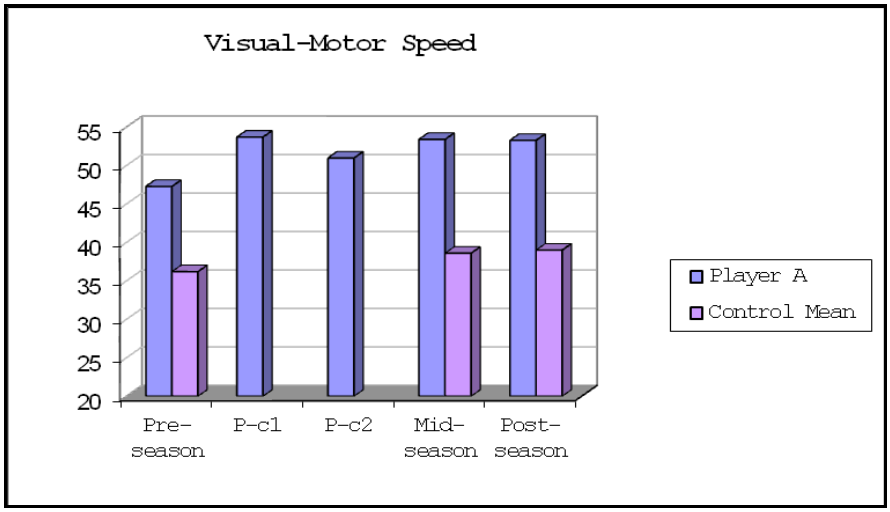


Figure 8.4 *Player A's* ImPACT Reaction Time Composite Scores at the Pre-season, Post-concussion 1, Post-concussion 2, Mid- and Post-season Assessment Intervals

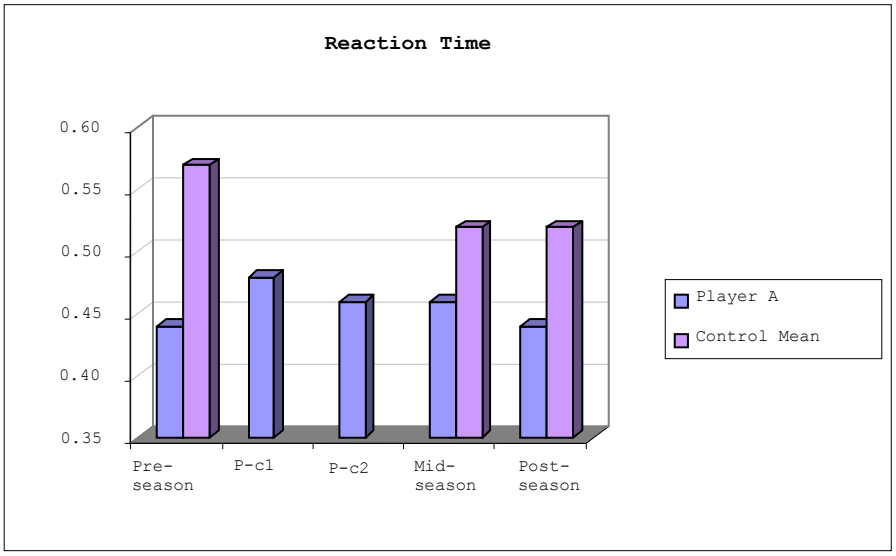


Figure 8.5 ***Player A's*** **Purdue Preferred Scores at the Pre-season, Post-concussion 1, Post-concussion 2, Mid- and Post-season Assessment Intervals**

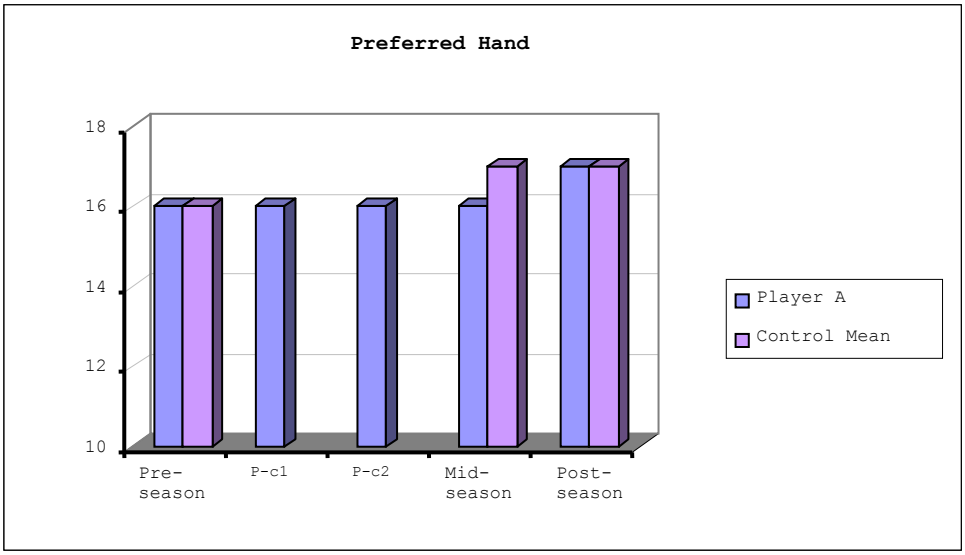


Figure 8.6 ***Player A's*** **Purdue Non-Preferred Scores at the Pre-season, Post-concussion 1, Post-concussion 2, Mid- and Post-season Assessment Intervals**

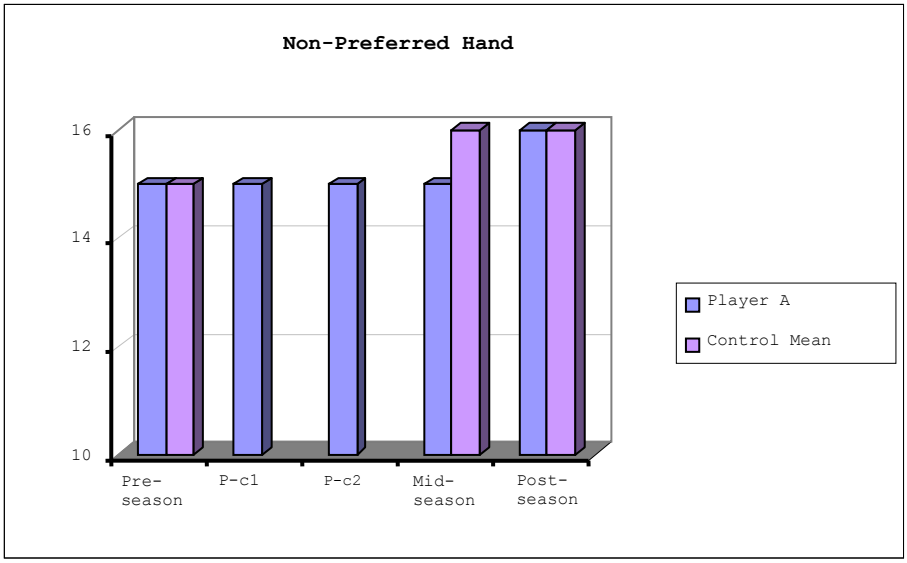


Figure 8.7 ***Player A's*** **Purdue Both Scores at the Pre-season, Post-concussion 1, Post-concussion 2, Mid- and Post-season Assessment Intervals**

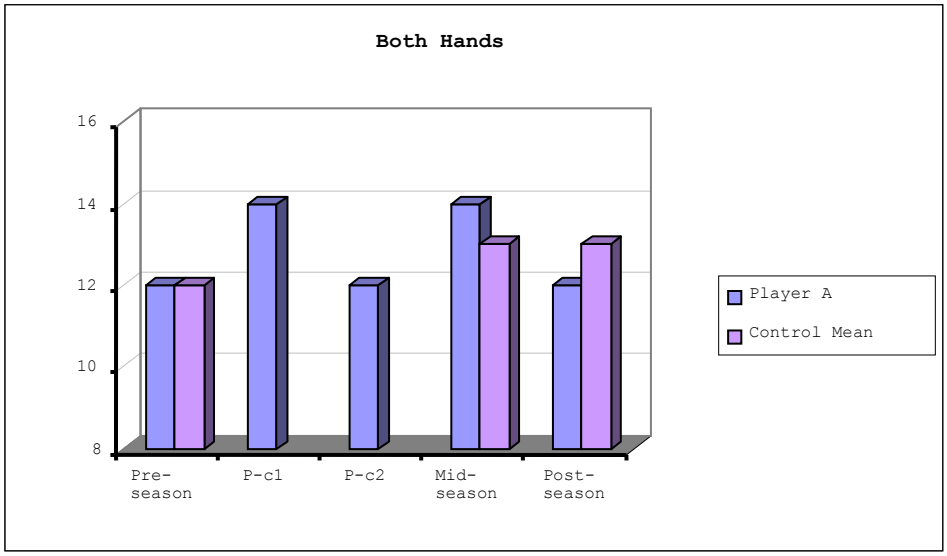
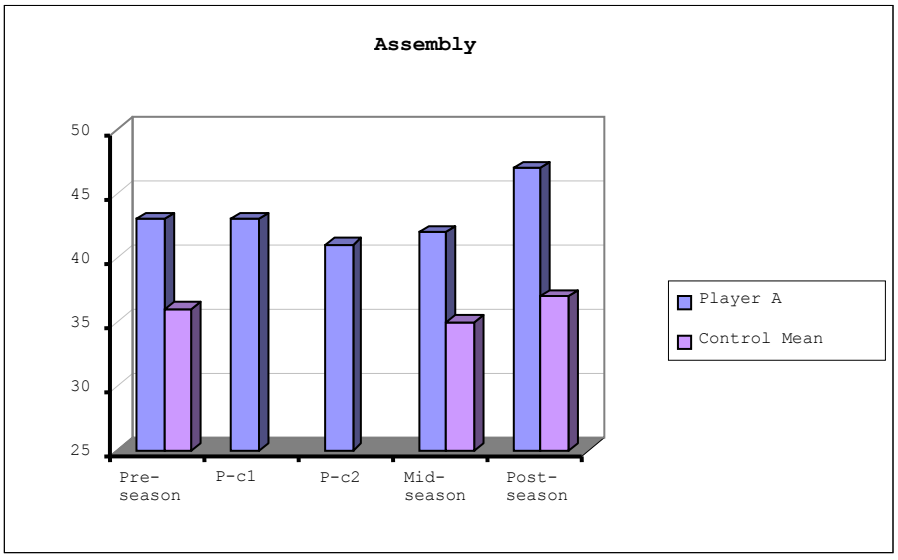


Figure 8.8 ***Player A's*** **Purdue Assembly Scores at the Pre-season, Post-concussion 1, Post-concussion 2, Mid- and Post-season Assessment Intervals**



8.2.1.2 Player A: Tackling

As tabled in the previous subsection (Table 8.2, page 152), *Player A* was involved in a total of **84** tackles, a number which was lower than the team average of 103.45. A further analysis of *Player A*'s tackling data for the purposes of the present subsection, revealed that he *made* a total of **32** tackles, (versus the substantially higher rugby mean of 60.70) of which 66% were made above the waist and 34% were made below the waist (Table 8.7). He *received* a total of **52** tackles (versus the marginally lower rugby mean of 42.75), of which 48% were made above the waist and 52% were made below the waist (Table 8.8). For *Player A*, Grab tackles were the predominant means of making tackles (Figure 8.9), and Side tackles and Head-on tackles were the predominant means of receiving tackles (Figure 8.10).

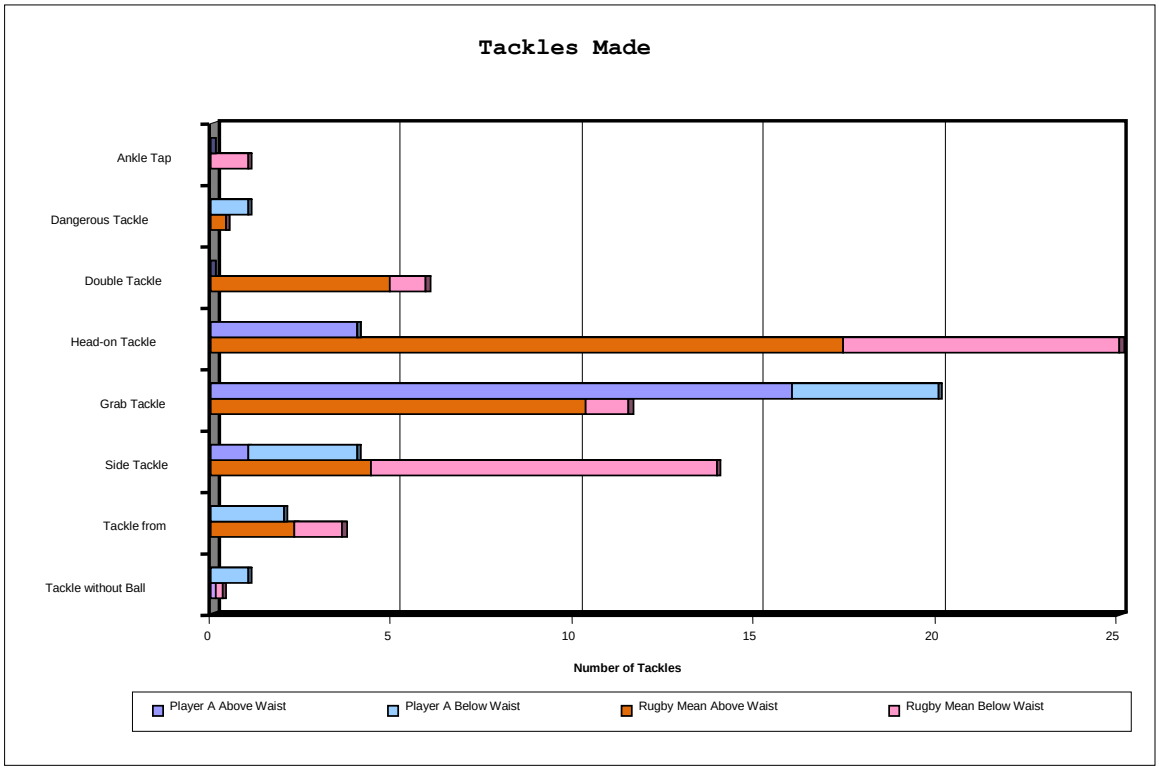
Table 8.7 ***Player A's*** **Number and Type of Tackles Made with Rugby Mean**

	Above Waist		**Below Waist**		**Total**	
	Player A	**Rugby Mean**	**Player A**	**Rugby Mean**	**Player A**	**Rugby Mean**
Ankle Tap	0	0.00	0	1.00	0	1.00
Dangerous Tackle	0	0.38	1	0.00	1	0.38
Double Tackle	0	4.91	0	0.95	0	5.86
Head-on Tackle	4	17.38	0	6.66	4	24.04
Grab Tackle	16	10.33	4	1.24	20	11.57
Side Tackle	1	4.38	3	9.52	4	13.90
Tackle from Behind	0	2.33	2	1.29	2	3.62
Tackle without Ball	0	0.14	1	0.19	1	0.33
Total Tackles	**21**	**39.85**	**11**	**20.85**	**32**	**60.70**
Percentage	**66**	**65**	**34**	**35**	**100**	**100**

Table 8.8 ***Player A's*** **Number and Type of Tackles Received with Rugby Mean**

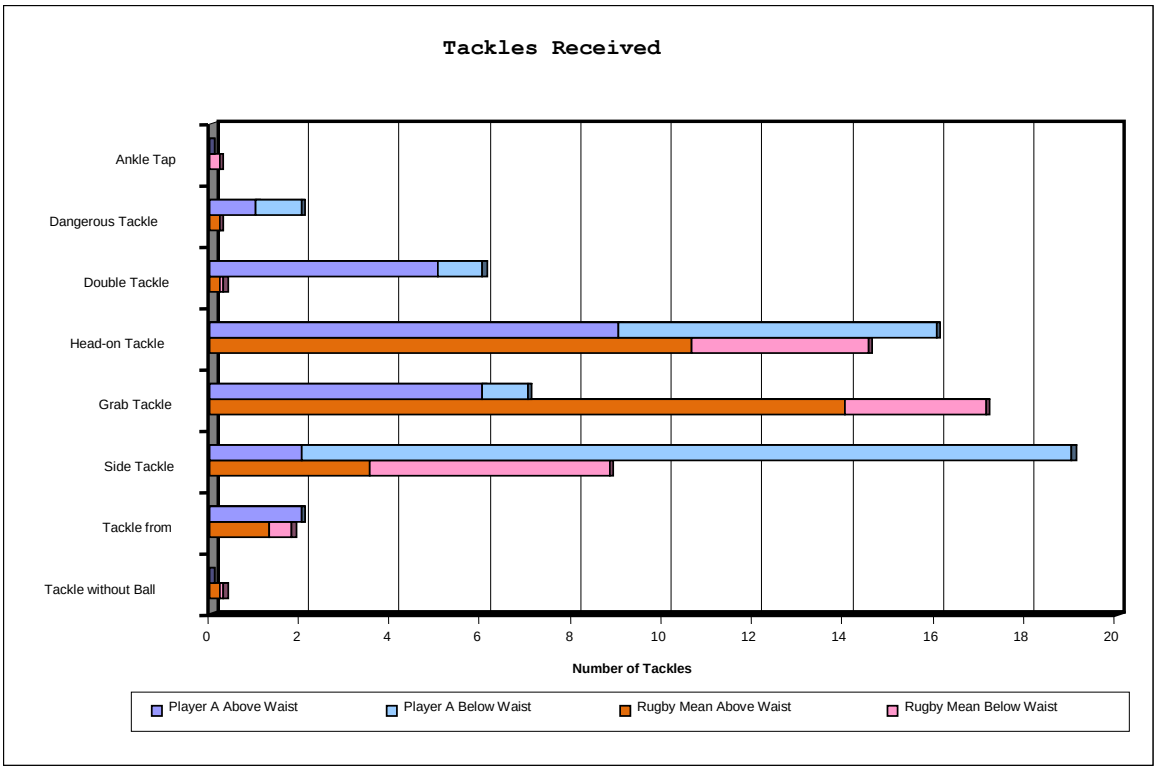
	Above Waist		Below Waist		Total	
	Player A	Rugby Mean	Player A	Rugby Mean	Player A	Rugby Mean
Ankle Tap	0	0.00	0	0.19	0	0.19
Dangerous Tackle	1	0.19	1	0.00	2	0.19
Double Tackle	5	0.19	1	0.09	6	0.28
Head-on Tackle	9	10.61	7	3.90	16	14.51
Grab Tackle	6	13.51	1	3.10	7	16.61
Side Tackle	2	3.52	17	5.31	19	8.82
Tackle from Behind	2	1.29	0	0.52	2	1.78
Tackle without Ball	0	0.24	0	0.14	0	0.37
Total Tackles	**25**	**29.50**	**27**	**13.25**	**52**	**42.75**
Percentage	**48**	**69**	**52**	**31**	**100**	**100**

Figure 8.9 ***Player A's*** **Number and Type of Tackles Made with Rugby Mean**



Note: Rugby Mean (orange and pink) indicated below Player A's performance (dark blue and light blue)

Figure 8.10 ***Player A's*** **Number and Type of Tackles Received with Rugby Mean**



Note: Rugby Mean (orange and pink) indicated below Player A's performance (dark blue and light blue)

8.2.1.3 Player A: Overview

In sum, this was a 23 year old rugby player with estimated above average IQ, who reported a history of four prior concussions excluding the most recent, suspected and not formally diagnosed concussion. His tackling count over the season overall was substantially less than the rugby team's averages on Tackles Made, Tackles Received and Total Tackles. At the *pre*-season assessment interval, his scores were commensurate with an estimated above average IQ denoting a superior performance especially on ImPACT Verbal Memory and ImPACT Reaction Time. His performance on ImPACT Visual Motor Speed at *pre*-season was already at a superior level, improved at the *P-c1* assessment interval and remained in the very superior range across the remaining assessment intervals. At the *P-c1* assessment interval, compared with the *pre*-season assessment interval, he showed signs of decreased cognitive performance on ImPACT Verbal Memory and ImPACT Reaction Time. There were subtle signs of decreased cognitive performance on ImPACT Visual Motor Speed, Purdue Both and Purdue Assembly at the *P-c2* assessment interval and on ImPACT Visual

Memory at the *mid*-season assessment interval. *Player A* only returned to his *pre*-season levels on ImPACT Reaction Time after the fourth assessment, whereas the Non-Contact Sports controls improved over *pre*-season levels quite substantially on the second assessment and sustained it over two additional repeat assessments. Overall for *Player A*, ImPACT Verbal Memory, ImPACT Reaction Time and Purdue Assembly appeared to be the most sensitive and discriminatory indicators of a suspected concussive event.

8.2.2 Demographic and Clinical History of Player B

GENERAL INFORMATION					
Subject		*Player B*		**Age**	31
Home language	English	**Other**		**Race:**	White
EDUCATION, OCCUPATION AND ESTIMATE IQ					
Level of Education completed			Grade 12		
Current study, if any			None		
Occupation			Sales		
Estimate IQ (Established on the basis of WAIS-III Picture Completion and Matrix Reasoning Scaled Scores using the OPIE-3 Formula)			103 (Average)		
MEDICAL AND PSYCHIATRIC HISTORY					
Fractures: Face, Arm and Hand on separate occasions					
CONCUSSION HISTORY					
Prior Concussions			None reported		

8.2.2.1 Player B: Neurocognitive Assessment Results

For *Player B,* the neurocognitive assessment results across all assessment intervals (*pre*-, *mid*-, and *post*-season as well as post-concussion) for each measure are tabulated together with the Non-Contact Sports control mean and the US average ranges (Table 8.9), and illustrated further by means of a figure in respect of each separate measure (Figures 8.11 – 8.18). For discussion purposes the results are interpreted in terms of the normative categories for both ImPACT and the Purdue Pegboard, as found earlier in Tables 8.3 and 8.5. The tables and figures for *Player B* appear together at the end of this subsection (page 174-178).

8.2.2.1.1 Memory

ImPACT Verbal Memory (Table 8.9; Figure 8.11). At the *pre*-season assessment interval *Player* B's Verbal Memory score denoted an "average" performance relative to the US normative categories. At the *P-c1* assessment interval there was a substantial decrease in evidence relative to the *pre*-season score with a "borderline" performance relative to the US normative categories. At the *mid*- and *post*-season assessment intervals the scores revealed the same level of an "average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player B* was consistently performing at a higher level than the controls across all the assessment intervals. The controls remained consistent over the first two assessments and improved at the *post*-season assessment interval, whereas in contrast *Player B* declined in performance immediately post injury, only subsequently returning to his pre-injury level.

ImPACT Visual Memory (Table 8.9; Figure 8.12)**.** At the *pre*-season assessment interval *Player B*'s Visual Memory score denoted a "low average" performance relative to the US normative categories. At the *P-c1* assessment interval there was an improvement on the *pre*-season score denoting an "average" performance relative to the US normative categories. At the *mid*-season assessment interval there was a further improvement on the two previous assessments, denoting an "average" performance relative to the US normative categories. At the *post*-season assessment interval the score showed a considerable improvement on the *pre*-season score, albeit still denoting an "average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player B* was initially performing at a lower level than the controls. The controls showed a gradual improvement from *pre*- to *mid*-season with a sharp improvement at the *post*-season assessment interval, whereas in contrast *Player B* started off low and ultimately by the *post*-season assessment interval he did reveal the ability to benefit from practice.

8.2.2.1.2 Motor Speed

ImPACT Visual Motor Speed (Table 8.9; Figure 8.13)**.** At the *pre*-season assessment interval *Player B*'s Visual Motor Speed score denoted a "borderline" performance relative to

the US normative categories. At the *P-c1* assessment interval there was a decrease in evidence relative to the *pre*-season score, denoting an "impaired" performance relative to the US normative categories. At the *mid*-season assessment interval there was an improvement on the two previous assessment scores, albeit still denoting a "borderline" performance relative to the US normative categories. At the *post*-season assessment interval the score showed a substantial improvement on previous scores, and better than the *pre*-season score, denoting a "low average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player B* was consistently performing at a lower level than the controls. The controls showed a sharp improvement from *pre*- to *mid*-season with a more gradual and steady improvement from the *mid*- to *post*-season assessment intervals, whereas in contrast *Player B* started off considerably lower than the controls, declined in performance immediately post injury, and ultimately by the *post*-season assessment interval he did reveal the ability to benefit from practice.

ImPACT Reaction Time (Table 8.9; Figure 8.14)**.** At the *pre*-season assessment interval *Player B*'s Reaction Time score denoted an "impaired" performance relative to the US normative categories. At the *P-c1* assessment interval there was an increase in Reaction Time scores (i.e. a worsening in performance) denoting an "impaired" performance relative to the US normative categories. At the *mid*-season assessment interval there was an indication of improved performance due to a decreased Reaction Time on the *pre*-season and *P-c1* scores, albeit still denoting a "borderline" performance relative to the US normative categories. At the *post*-season assessment interval the score improved on the previous scores, denoting a "low average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player B* was consistently performing at a lower level than the controls. The controls showed an improved performance due to a decreased Reaction Time score from *pre*- to *mid*-season and stabilised by the *post*-season assessment interval, whereas in contrast *Player B* was initially not performing as well as he could and started off much lower than the controls, declined in performance immediately post injury, and ultimately by the *post*-season assessment interval he did reveal the ability to benefit from practice.

Purdue Preferred (Table 8.9; Figure 8.15)**.** At the *pre*-season assessment interval *Player B*'s Preferred score revealed a "poor" performance relative to the Purdue normative categories. At the *P-c1* assessment interval the score showed no change compared to the *pre*-season assessment, denoting a "poor" performance relative to the Purdue normative categories. At the *mid*-season assessment interval there was an improved performance compared to the two previous assessments, denoting an "average" performance relative to the Purdue normative categories. At the *post*-season assessment interval there was a substantial improvement on the *pre*-season score, denoting performance in the upper limits of the "high average" range relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player B* was initially performing at a lower level than the controls. The controls showed an improvement at the *mid*-season assessment interval, whereas in contrast *Player B* started off much lower than the controls, remained the same immediately post injury, and ultimately by the *post*-season assessment interval he improved considerably and may have benefited from practice.

Purdue Non-Preferred (Table 8.9; Figure 8.16)**.** At the *pre*-season assessment interval *Player B*'s Non-Preferred score revealed a "low average" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was an indication of lowered performance over the *pre*-season score, denoting a "poor" performance relative to the Purdue normative categories. At the *mid*-season assessment interval there was an improvement compared to the two previous assessments, denoting an "average" performance relative to the Purdue normative categories. At the *post*-season assessment interval the score showed a substantial improvement, denoting an "above average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player B* was initially performing at a lower level than the controls. The controls showed an improvement on the *mid*-season assessment interval, whereas in contrast *Player B* started off much lower than the controls, declined in performance immediately post injury, and ultimately by the *post*-season assessment interval he improved considerably on his *pre*-season score.

Purdue Both (Table 8.9; Figure 8.17)**.** At the *pre*-season assessment interval *Player B*'s Both score revealed a "low average" performance relative to the Purdue normative

categories. At the *P-c1* assessment interval there was a worsening of performance, in the direction of a "poor" performance relative to the Purdue normative categories. At the *mid*-season assessment interval the score showed no change compared to the *P-c1* assessment, and finally at the *post*-season assessment interval the score showed an improvement denoting an "average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player B* was performing at a lower level across the first two assessment intervals. The controls showed an improvement by *mid*-season and were consistent at *post*-season, whereas in contrast *Player B* started off lower than the controls, declined in performance immediately post injury, and only improved at the *post*-season assessment interval.

Purdue Assembly (Table 8.9; Figure 8.18)**.** At the *pre*-season assessment interval *Player B*'s Assembly score denoted performance in the lower limits of the "low average" range relative to the Purdue normative categories. At the *P-c1* assessment interval the score decreased marginally, denoting a "poor" performance relative to the Purdue normative categories. At the *mid*-season assessment interval there was a considerable improvement compared to the two previous assessments, denoting an "average" performance relative to the Purdue normative categories. At the *post*-season assessment interval there was a further improvement, albeit still denoting an "average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player B* was initially performing at a lower level than the controls. The controls showed a slight fluctuation in performance on the *mid*-season assessment interval, implicating that the controls possibly started off closer to their ceiling, whereas in contrast *Player B* started off much lower than the controls, declined in performance immediately post injury, improved by *mid*-season and stabilised by the *post*-season assessment interval.

Table 8.9 ***Player B's*** **ImPACT and Purdue Pegboard Repeat Assessment Scores vs the Non-Contact Sports Control Mean Score and the US Average Range**

	Pre-season 2005/02/08	**P-c1 2005/07/07**	**Mid-season 2005/07/28**	**Post-season 2005/09/26**	**US Average[1]**
MEMORY					
ImPACT Verbal Memory					
Player B	93	**74**	92	92	83 – 94
Control Mean Score	84	-	85	87	
ImPACT Visual Memory					
Player B	69	78	82	83	69 – 94
Control Mean Score	74	-	76	82	
MOTOR SPEED					
ImPACT VMS[2]					
Player B	**24.7**	**22.3**	**26.3**	**28.8**	32.5 - 42.0
Control Mean Score	37.5	-	39.5	39.0	
ImPACT Reaction Time					
Player B	**0.77**	**0.78**	**0.70**	0.63	0.60 - 0.51
Control Mean Score	0.55	-	0.50	0.52	
Purdue Preferred					
Player B	**12.00**	**12.00**	16.00	18.00	15.35 (1.72)
Control Mean Score	16.05	-	17.00	17.09	
Purdue Non-Preferred					
Player B	**13.00**	**12.00**	14.00	17.00	15.12 (1.77)
Control Mean Score	15.09	-	16.05	16.23	
Purdue Both					
Player B	11.00	**10.00**	**10.00**	14.00	12.42 (1.65)
Control Mean Score	12.82	-	13.07	13.36	
Purdue Assembly					
Player B	**32.00**	**31.00**	36.00	37.00	37.50 (3.64)
Control Mean Score	36.41	-	35.55	36.45	

[1]Note: ImPACT delineated by range; Purdue Pegboard in mean score and Standard Deviation
Bold print represents scores that fall below the lower limit of the average ranges of both ImPACT and the Purdue Pegboard
[2]Note: Visual Motor Speed (VMS)

Figure 8.11 ***Player B's*** **ImPACT Verbal Memory Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

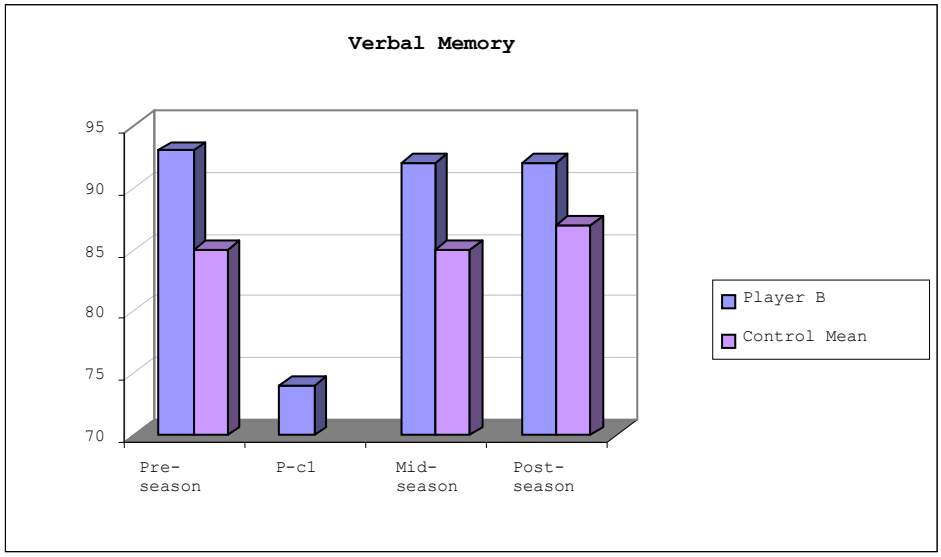


Figure 8.12 ***Player B's*** **ImPACT Visual Memory Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

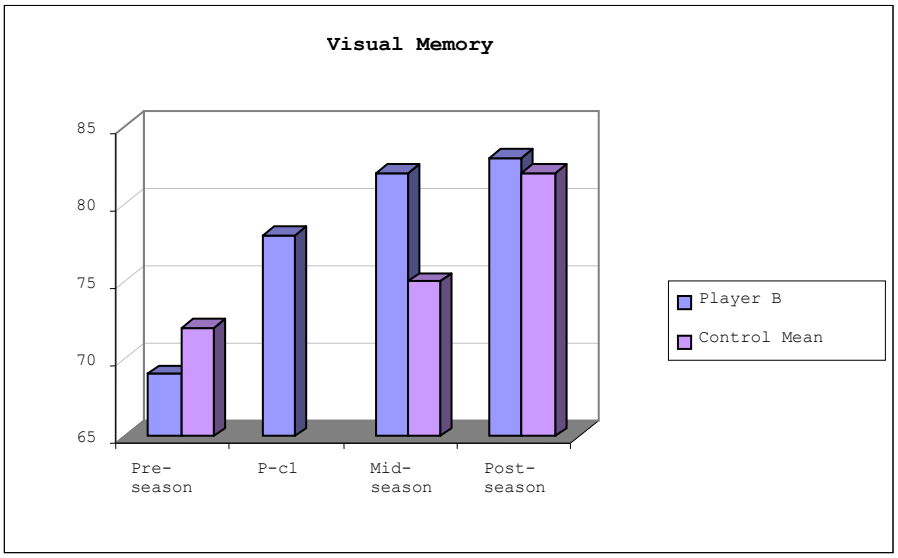


Figure 8.13 ***Player B's*** **ImPACT Visual Motor Speed Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

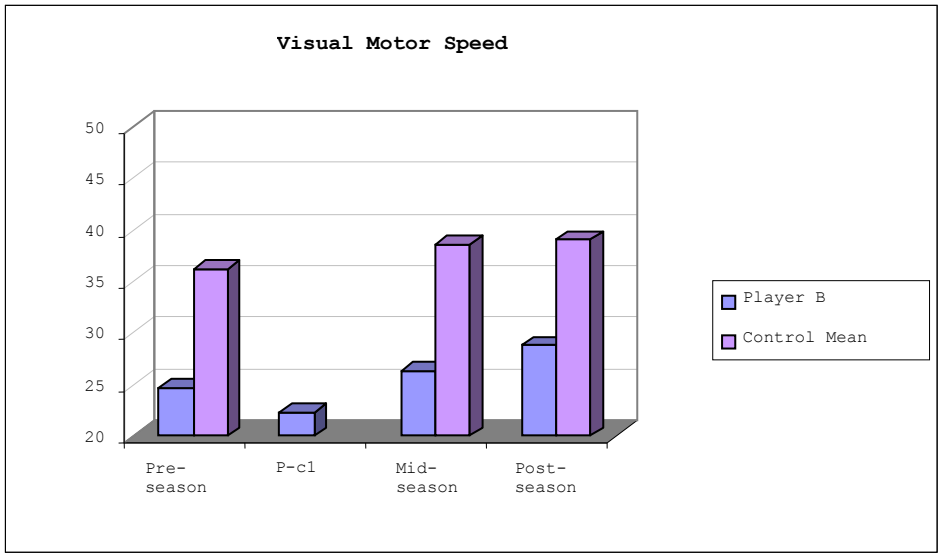


Figure 8.14 ***Player B's*** **ImPACT Reaction Time Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

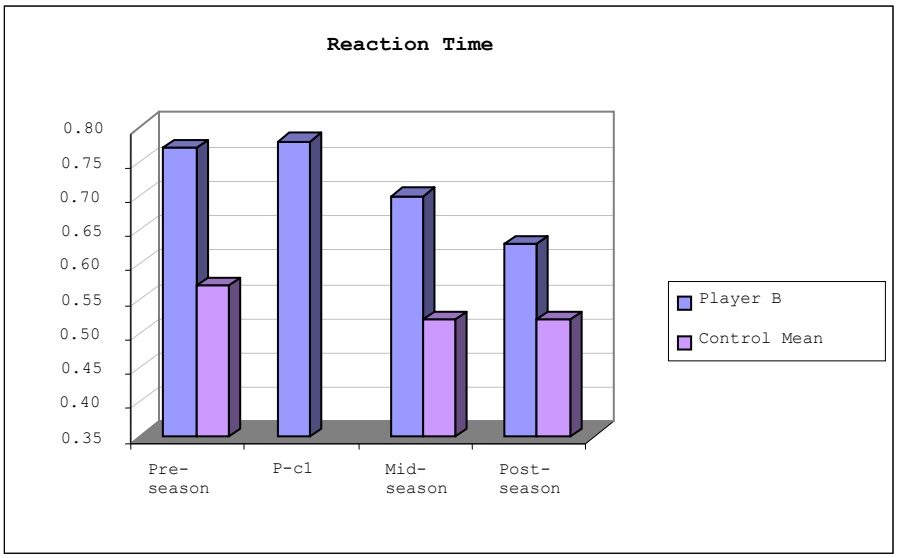


Figure 8.15 ***Player B's*** **Purdue Preferred Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

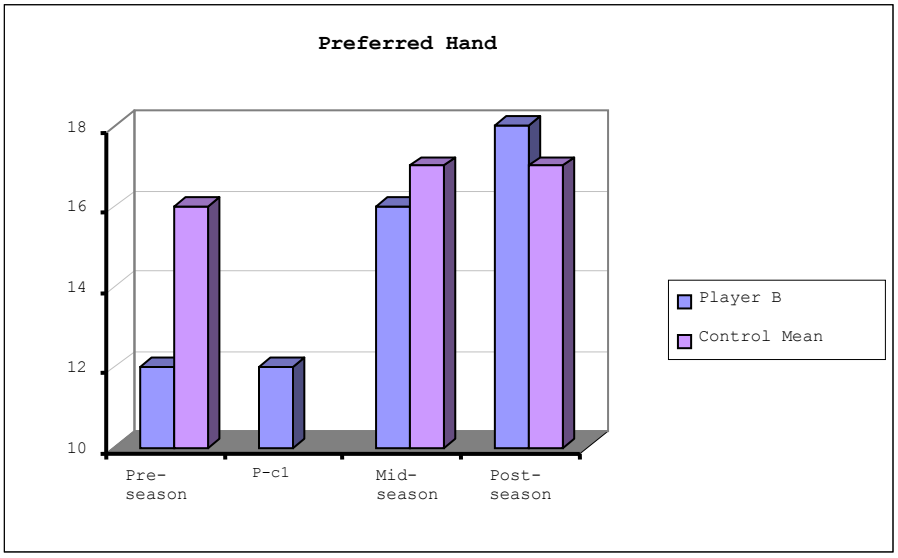


Figure 8.16 ***Player B's*** **Purdue Non-Preferred Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

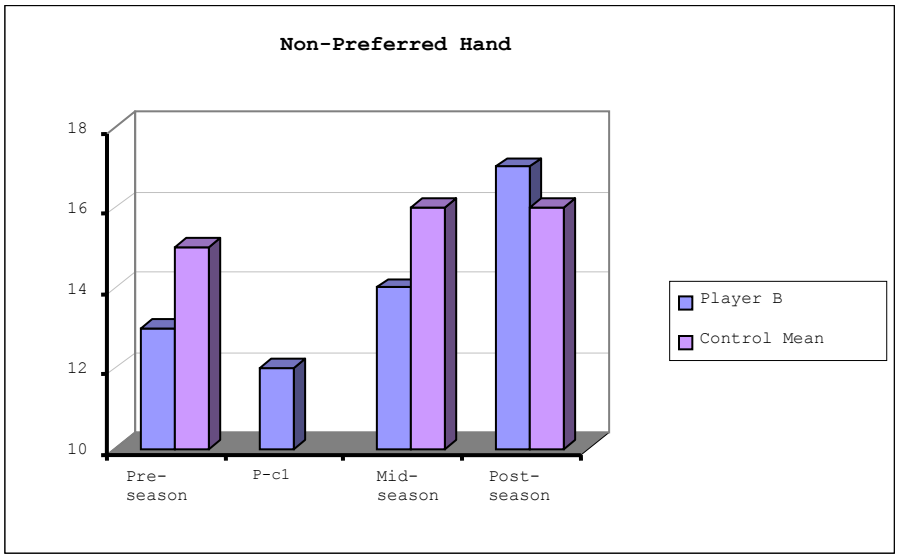


Figure 8.17 ***Player B's*** **Purdue Both Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

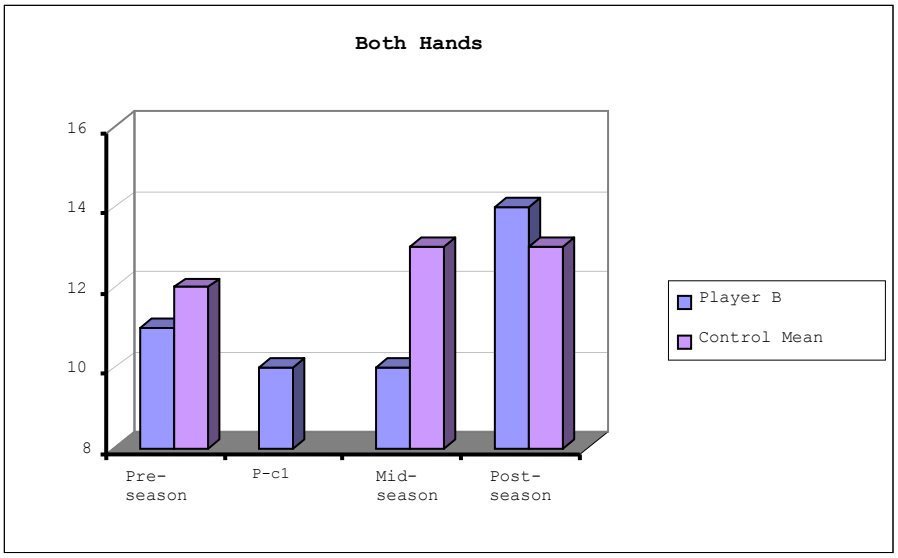
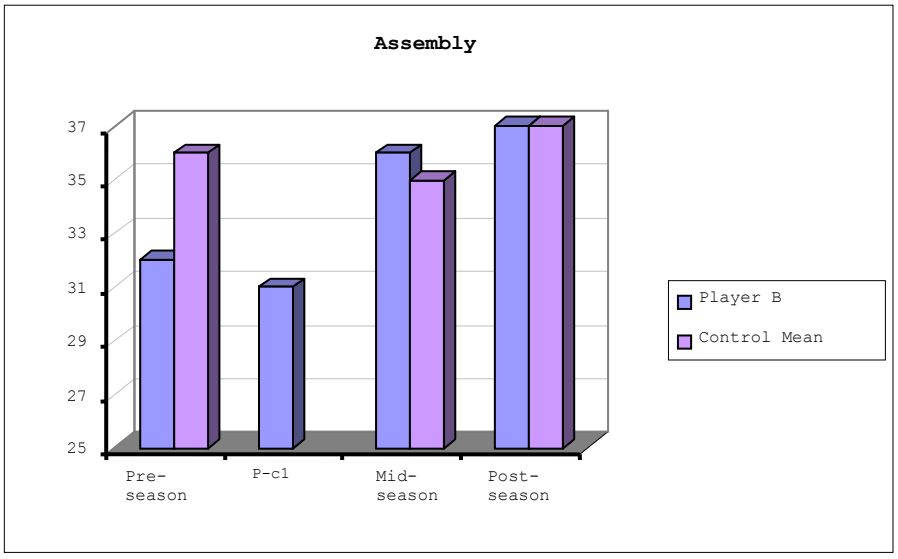


Figure 8.18 ***Player B's*** **Purdue Assembly Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**



8.2.2.2 Player B: Tackling

As tabled in the previous subsection (Table 8.2, page 152), *Player B* was involved in a total of **120** tackles, a number which was substantially higher than the team average of 103.45. A further analysis of *Player B*'s tackling data for the purposes of the present subsection, revealed that he *made* a total of **63** tackles (versus the marginally lower rugby mean of 60.70) of which 68% were made above the waist and 32% were made below the waist (Table 8.10). He *received* a total of **57** tackles (versus the lower rugby mean of 42.75), of which 79% were made above the waist and 21% were made below the waist (Table 8.11). For *Player B*, Grab tackles were the predominant means of making tackles (Figure 8.19), and Head-on tackles were the predominant means of receiving tackles (Figure 8.20).

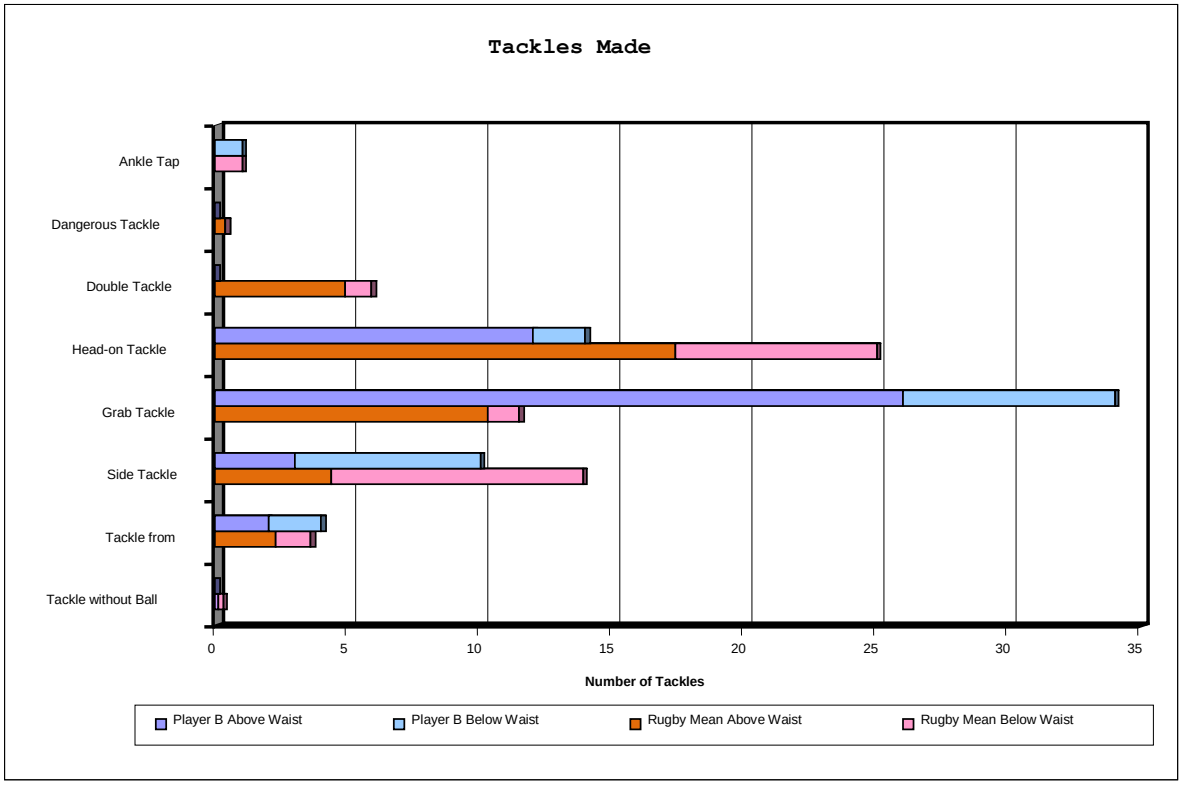
Table 8.10 ***Player B's*** **Number and Type of Tackles Made with Rugby Mean**

	Above Waist		**Below Waist**		**Total**	
	Player B	**Rugby Mean**	**Player B**	**Rugby Mean**	**Player B**	**Rugby Mean**
Ankle Tap	0	0.00	1	1.00	1	1.00
Dangerous Tackle	0	0.38	0	0.00	0	0.38
Double Tackle	0	4.91	0	0.95	0	5.86
Head-on Tackle	12	17.38	2	6.66	14	24.04
Grab Tackle	26	10.33	8	1.24	34	11.57
Side Tackle	3	4.38	7	9.52	10	13.90
Tackle from Behind	2	2.33	2	1.29	4	3.62
Tackle without Ball	0	0.14	0	0.19	0	0.33
Total Tackles	**43**	**39.85**	**20**	**20.85**	**63**	**60.70**
Percentage	**68**	**65**	**32**	**35**	**100**	**100**

Table 8.11 ***Player B's*** **Number and Type of Tackles Received with Rugby Mean**

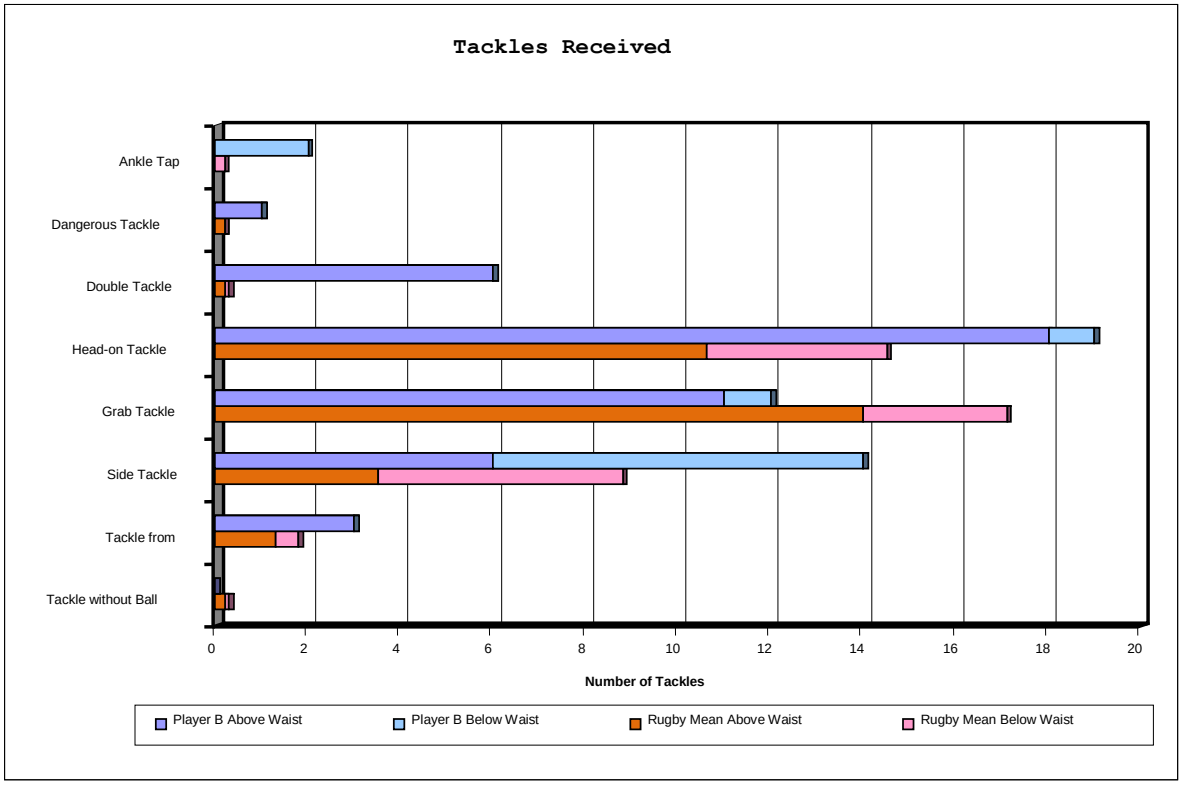
	Above Waist		Below Waist		Total	
	Player B	Rugby Mean	Player B	Rugby Mean	Player B	Rugby Mean
Ankle Tap	0	0.00	2	0.19	2	0.19
Dangerous Tackle	1	0.19	0	0.00	1	0.19
Double Tackle	6	0.19	0	0.09	6	0.28
Head-on Tackle	18	10.61	1	3.90	19	14.51
Grab Tackle	11	13.51	1	3.10	12	16.61
Side Tackle	6	3.52	8	5.31	14	8.82
Tackle from Behind	3	1.29	0	0.52	3	1.78
Tackle without Ball	0	0.24	0	0.14	0	0.37
Total Tackles	**45**	**29.50**	**12**	**13.25**	**57**	**42.75**
Percentage	**79**	**69**	**21**	**31**	**100**	**100**

Figure 8.19 ***Player B's*** **Number and Type of Tackles Made with Rugby Mean**



Note: Rugby Mean (orange and pink) indicated below Player B's performance (dark blue and light blue)

Figure 8.20 ***Player B's*** **Number and Type of Tackles Received with Rugby Mean**



Note: Rugby Mean (orange and pink) indicated below Player B's performance (dark blue and light blue)

8.2.2.3 Player B: Overview

In sum, this was a 31 year old rugby player with estimated average IQ, who indicated no recollection of previous concussions excluding the most recent, suspected and not formally diagnosed concussion. His tackling count over the season overall was consistently higher than the rugby team's averages on Tackles Made, Tackles Received and Total Tackles. At the *pre*-season assessment interval, his scores were lower than expected of an individual with an estimated average IQ, denoting "impaired" performance on ImPACT Reaction Time, "borderline" performance on ImPACT Visual Motor Speed, "low average" performance on Purdue Non-Preferred and "poor" performance on Purdue Preferred and Purdue Assembly. At the *P-c1* assessment interval, compared with the *pre*-season assessment interval, he showed signs of decreased cognitive performance on ImPACT Verbal Memory, ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Non-Preferred, Purdue Both and Purdue Assembly. *Player B* only seems to improve on his *P-c1* scores at the *mid*-season

assessment interval on all measures with the exception of Purdue Both which remained the same. *Player B* seems to improve after the third assessment, whereas the Non-Contact Sports controls improved over *pre*-season quite substantially on the second assessment and sustained it with two additional repeats, with the exception of Purdue Assembly at *mid*-season.

Overall for *Player B*, ImPACT Verbal Memory, ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Non-Preferred, Purdue Both and Purdue Assembly appeared to be the most sensitive and discriminatory indicators of a suspected concussive event. Furthermore, it was evident at the *pre*-season assessment interval that *Player B* was already compromised on ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred and Purdue Assembly, implicating possible residual effects of cumulative concussive and/or subconcussive events, whereas the controls were consistently within the normative ranges for all tests.

8.2.3 Demographic and Clinical History of Player C

GENERAL INFORMATION					
Subject	*Player C*			**Age**	27
Home language	Afrikaans	**Other**	English	**Race:**	White
EDUCATION, OCCUPATION AND ESTIMATE IQ					
Level of Education completed	Grade 12				
Current study, if any	Project Management				
Occupation	Internet Company/Project Manager				
Estimate IQ (Established on the basis of WAIS-III Picture Completion and Matrix Reasoning Scaled Scores using the OPIE-3 Formula)	116 (Above Average)				
MEDICAL AND PSYCHIATRIC HISTORY					
None					
CONCUSSION HISTORY					
Prior Concussions	1				

8.2.3.1 Player C: Neurocognitive Assessment Results

For *Player C,* the neurocognitive assessment results across all assessment intervals (*pre-*, *mid-*, and *post*-season as well as post-concussion) for each measure are tabulated together with the Non-Contact Sports control mean and the US average ranges (Table 8.12), and illustrated further by means of a figure in respect of each separate measure (Figures 8.21 – 8.28). For discussion purposes the results are interpreted in terms of the normative categories for both ImPACT and the Purdue Pegboard, as found earlier in Tables 8.3 and 8.5. The tables and figures for *Player C* appear together at the end of this subsection (page 187-191).

8.2.3.1.1 Memory

ImPACT Verbal Memory (Table 8.12; Figure 8.21)**.** At the *pre*-season assessment interval *Player C*'s Verbal Memory revealed a "borderline" performance relative to the US normative categories. There was a considerable improvement in evidence relative to the *pre*-season score at the *P-c1* assessment interval, denoting an "average" performance relative to the US normative categories. At the *mid-season* assessment interval there was a marginal decrease in evidence from the previous assessment, albeit still denoting an "average" performance relative to the US normative categories. At the *post-season* assessment interval the score showed a substantial improvement on the *pre*-season level, albeit still denoting an "average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player C* started off worse than the controls. The controls remained consistent over the first two assessments and improved at the *post*-season assessment interval, whereas in contrast *Player C* improved in performance immediately post injury; with a marginal worsening in performance at *mid*-season, and surpassing his *pre*-season score at *post*-season.

ImPACT Visual Memory (Table 8.12; Figure 8.22)**.** At the *pre*-season assessment interval *Player C*'s Visual Memory score denoted an "average" performance relative to the US normative categories. At the *P-c1* assessment interval there was a substantial worsening in performance in evidence relative to the *pre*-season score, denoting a "borderline" performance relative to the US normative categories. At the *mid*-season assessment interval

the Visual Memory score revealed good recovery with an improvement on the *pre*-season level, with a further considerable improvement at the *post*-season assessment interval, denoting an "average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player C* was performing better than the controls, except immediately post-injury. The controls showed a gradual improvement from *pre*- to *mid*-season with a sharp improvement at the *post*-season assessment interval, whereas in contrast *Player C* declined in performance immediately post injury, and continued to improve on his performance from the *mid*- to the *post*-season assessment intervals.

8.2.3.1.2 Motor Speed

ImPACT Visual Motor Speed (Table 8.12; Figure 8.23)**.** At the *pre*-season assessment interval *Player C*'s Visual Motor Speed score denoted a "low average" performance relative to the US normative categories. At the *P-c1* assessment interval there was a substantial improvement over the *pre*-season score, denoting an "average" performance relative to the US normative categories. At the *mid*-season assessment interval, however, there was a marginal worsening in performance, albeit still denoting an "average" performance relative to the US normative categories. At the *post*-season assessment interval the score showed improvement and was better than the *pre*-season score, denoting an "average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player C* was consistently performing at a lower level than the controls. The controls showed a sharp improvement from *pre*- to *mid*-season with a more gradual and steady improvement from the *mid*- to *post*-season assessment intervals, whereas in contrast *Player C* improved in performance immediately post injury, marginally decreasing at *mid*-season and only subsequently improving at *post*-season.

ImPACT Reaction Time (Table 8.12; Figure 8.24)**.** At the *pre*-season assessment interval *Player C*'s Reaction Time score denoted an "average" performance relative to the US normative categories. At the *P-c1* assessment interval there was a slight decrease in Reaction Time (i.e. an improvement in performance), denoting a "high average" performance relative to the US normative categories. At the *mid*-and *post*-season assessment intervals the scores

showed no change, denoting a "high average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player C* was consistently performing better than the controls. The controls showed an improved performance due to a decreased Reaction Time score from *pre-* to *mid*-season and stabilised by the *post*-season assessment interval, whereas in contrast *Player C* was initially not performing as well as he could and improved in performance immediately post injury, and stabilised throughout the remaining assessment intervals.

Purdue Preferred (Table 8.12; Figure 8.25)**.** At the *pre*-season assessment interval *Player C*'s Preferred score revealed an "average" performance relative to the Purdue normative categories. At the *P-c1* and *mid*-season assessment intervals the score showed an improved performance to a level that was within the "average" range relative to the Purdue normative categories. At the *post*-season assessment interval there was a marginal decrease in evidence relative to the previous assessment interval, albeit still an "average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player C* was performing at a similar level. The controls showed an improvement at the *mid*-season assessment interval and stabilised by *post*-season, whereas in contrast *Player C* started off marginally lower than the controls, improved immediately post injury, but could not maintain his performance at *post*-season.

Purdue Non-Preferred (Table 8.12; Figure 8.26)**.** At the *pre*-season assessment interval *Player C*'s Non-Preferred score revealed an "average" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was a marginal decrease in evidence, albeit still denoting an "average" performance relative to the Purdue normative categories. At the *mid*-season assessment interval the score returned to the *pre*-season level, and showed no change at the *post*-season assessment interval, denoting an "average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player C* was initially performing at a higher level than the controls. The controls showed an improvement at the *mid*-season assessment interval, whereas in contrast *Player C* possibly was already performing at his ceiling at *pre*-season and did not improve or benefit from practice throughout the season.

Purdue Both (Table 8.12; Figure 8.27)**.** At the *pre*-season assessment interval *Player C*'s Both score revealed an "average" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was a marginal decrease in evidence, denoting a "low average" performance relative to the Purdue normative categories. At the *mid*-season assessment interval the score showed an improvement on the *pre*-season level, and showed no change at the *post*-season assessment interval, denoting an "average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player C* was performing at an equivalent level at all the assessment intervals.

Purdue Assembly (Table 8.12; Figure 8.28)**.** At the *pre*-season assessment interval *Player C*'s Assembly score denoted a "high average" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was a worsening in evidence compared to the *pre*-season score, although still denoting an "average" performance relative to the Purdue normative categories. At the *mid*-season assessment interval the score showed improvement but was not back to the *pre*-season level, albeit still denoting an "average" performance relative to the Purdue normative categories. At the *post*-season assessment interval there was improvement over all the previous assessment intervals to a level denoting a "high average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player C* was performing better than the controls. The controls showed a slight fluctuation in performance at the *mid*-season assessment interval, implicating that the controls possibly started off closer to their ceiling, whereas in contrast *Player C* started off higher than the controls, decreased immediately post injury, struggled to regain his *pre*-season score at *mid*-season, and ultimately surpassed his *pre*-season score at *post*-season.

Table 8.12 ***Player C's*** **ImPACT and Purdue Pegboard Repeat Assessment Scores vs the Non-Contact Sports Control Mean Score and the US Average Range**

	Pre-season 2005/02/10	P-c1 2005/06/14	Mid-season 2005/06/21	Post-season 2005/10/06	US Average[1]
MEMORY					
ImPACT Verbal Memory					
Player C	**75**	88	85	90	83 – 94
Control Mean Score	84	-	85	87	
ImPACT Visual Memory					
Player C	80	**57**	82	92	69 – 94
Control Mean Score	74	-	76	82	
MOTOR SPEED					
ImPACT VMS[2]					
Player C	**30.6**	38.1	36.4	37.1	32.5 - 42.0
Control Mean Score	37.5	-	39.5	39.0	
ImPACT Reaction Time					
Player C	0.52	0.49	0.49	0.49	0.60 - 0.51
Control Mean Score	0.55	-	0.50	0.52	
Purdue Preferred					
Player C	15.00	16.00	17.00	16.00	16.22 (1.81)
Control Mean Score	16.05	-	17.00	17.09	
Purdue Non-Preferred					
Player C	16.00	15.00	16.00	16.00	15.41 (2.08)
Control Mean Score	15.09	-	16.05	16.23	
Purdue Both					
Player C	12.00	**11.00**	13.00	13.00	12.94 (1.29)
Control Mean Score	12.82	-	13.07	13.36	
Purdue Assembly					
Player C	42.00	40.00	41.00	43.00	39.13 (3.58)
Control Mean Score	36.41	-	35.55	36.45	

[1]Note: ImPACT delineated by range; Purdue Pegboard in mean score and Standard Deviation
Bold print represents scores that fall below the lower limit of the average ranges of both ImPACT and the Purdue Pegboard
[2]Note: Visual Motor Speed (VMS)

Figure 8.21 ***Player C's*** **ImPACT Verbal Memory Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

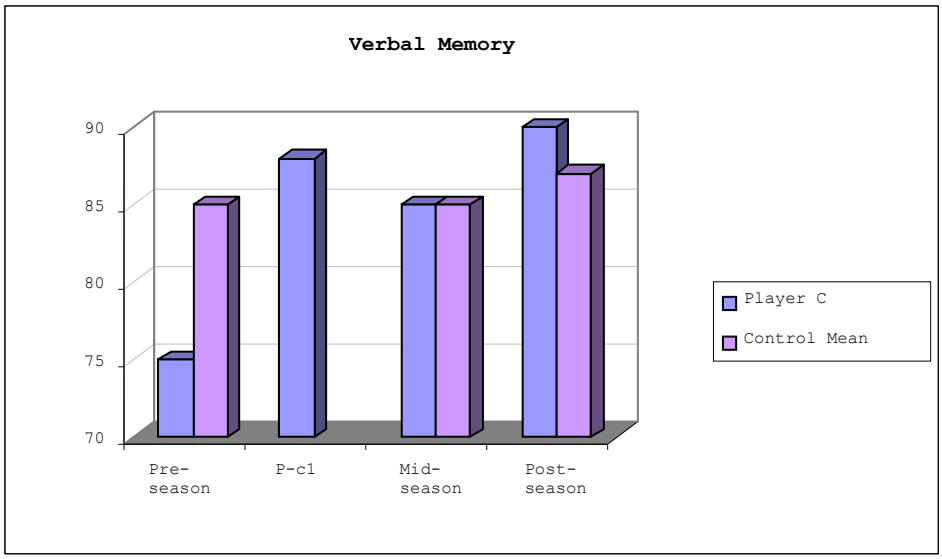


Figure 8.22 ***Player C's*** **ImPACT Visual Memory Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

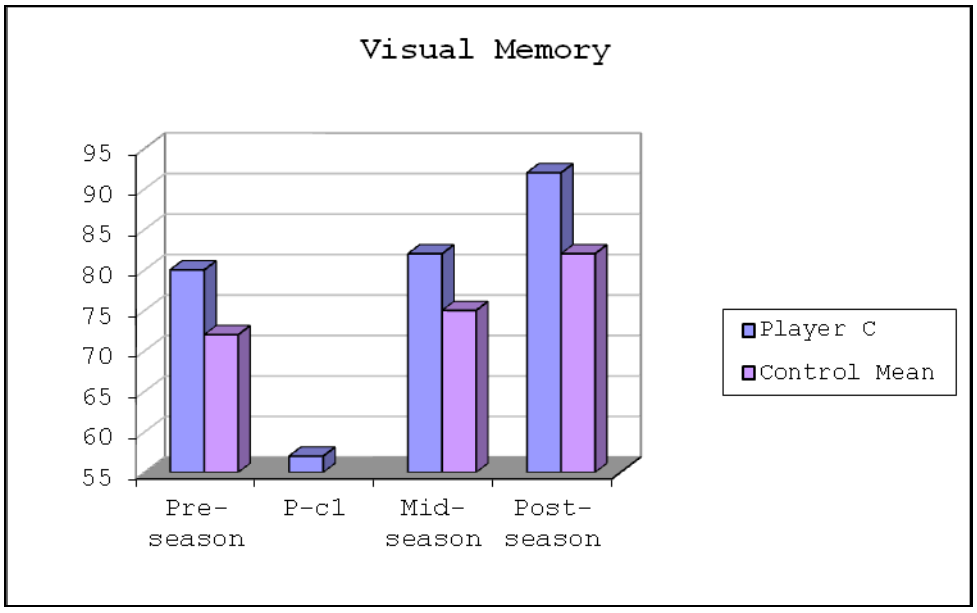


Figure 8.23 ***Player C's*** **ImPACT Visual Motor Speed Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

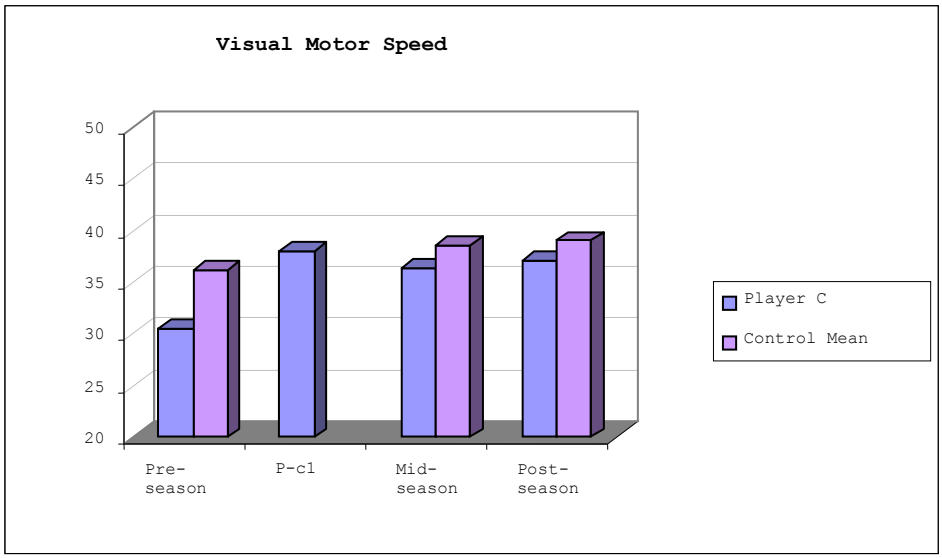


Figure 8.24 ***Player C's*** **ImPACT Reaction Time Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

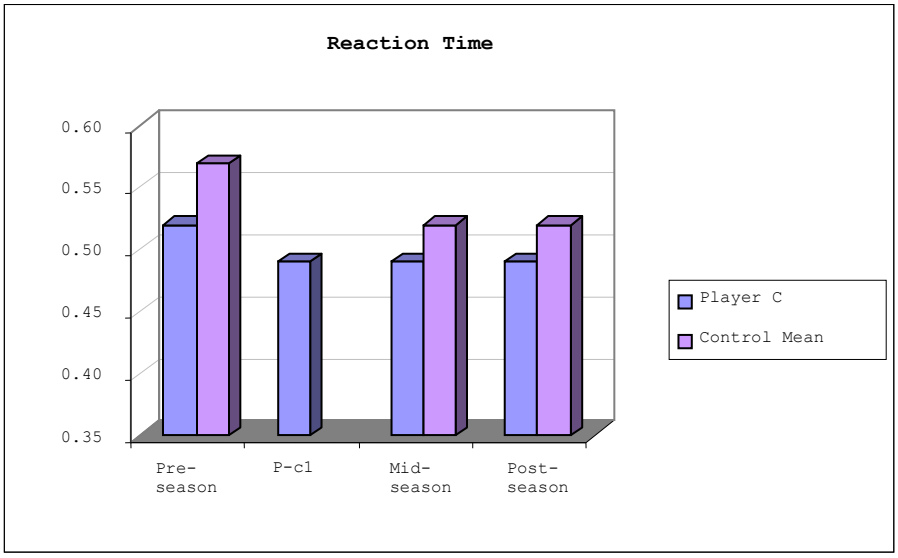


Figure 8.25 ***Player C's*** **Purdue Preferred Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

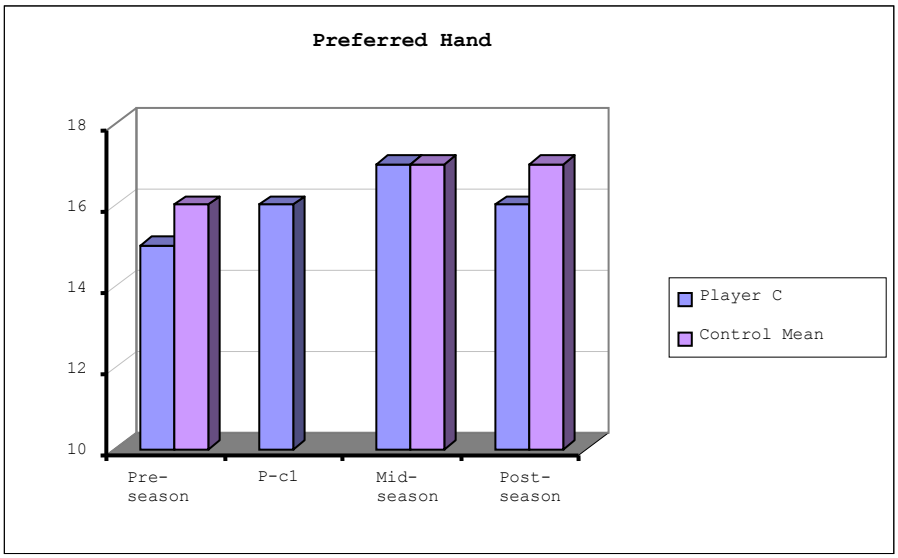


Figure 8.26 ***Player C's*** **Purdue Non-Preferred Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

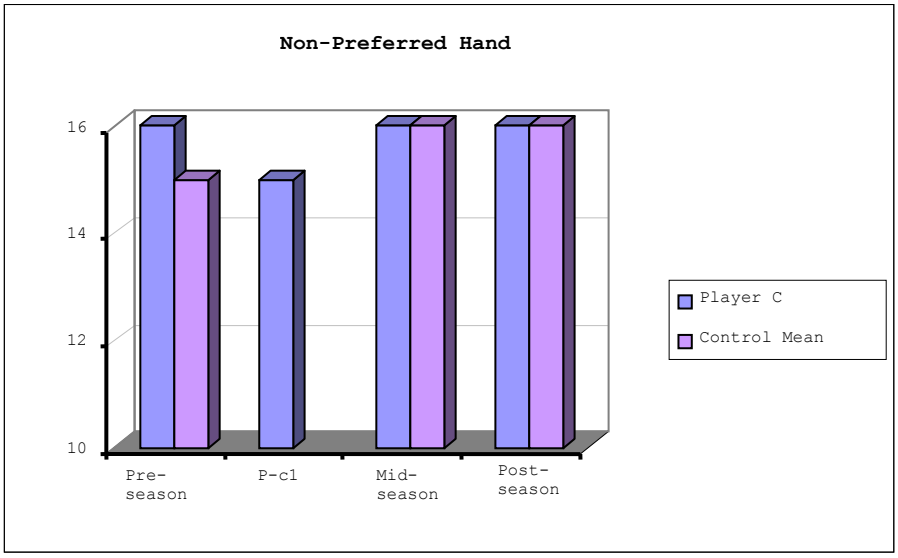


Figure 8.27 ***Player C's*** **Purdue Both Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

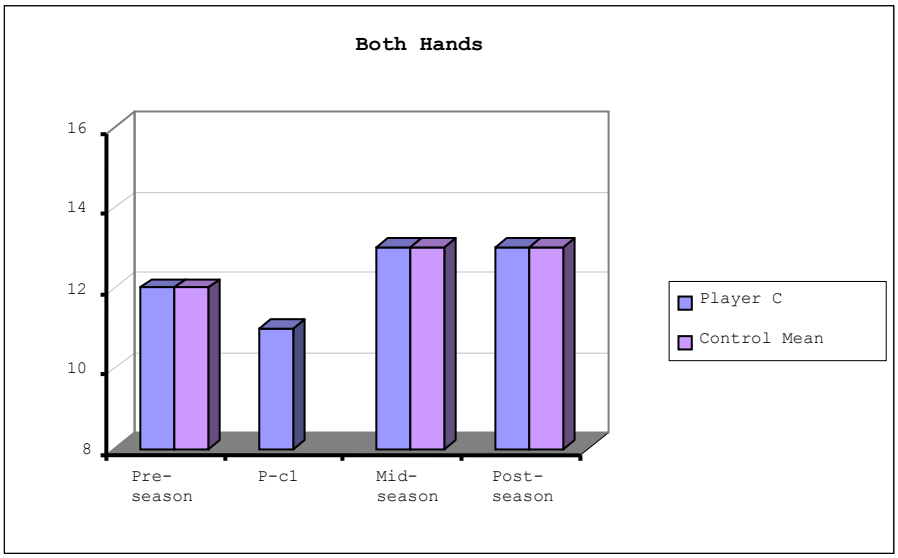
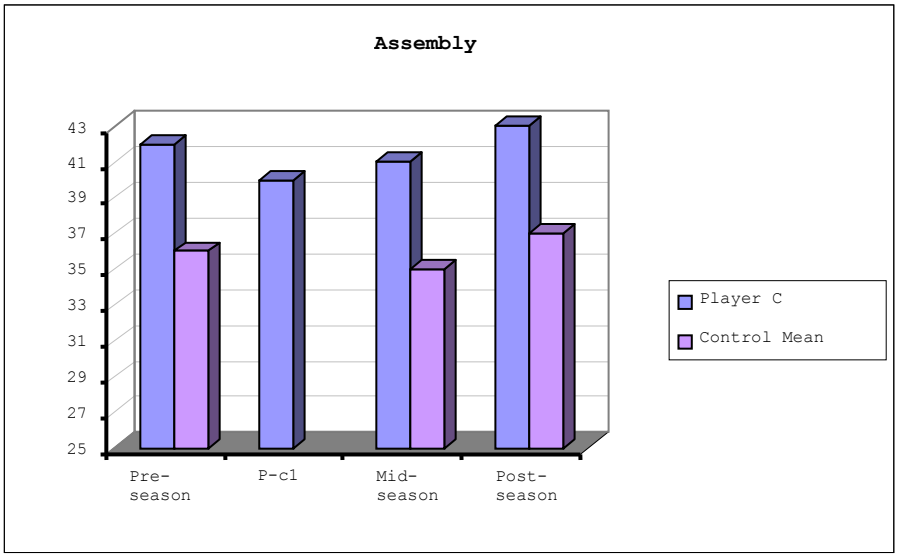


Figure 8.28 ***Player C's*** **Purdue Assembly Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**



8.2.3.2 Player C: Tackling

As tabled in the previous subsection (Table 8.2, page 152), *Player C* was involved in a total of 79 tackles, a number which was lower than the team average of 103.45. A further analysis of *Player C*'s tackling data revealed that he *made* a total of 26 tackles (versus the

substantially higher rugby mean of 60.70) of which 69% were made above the waist and 31% were made below the waist (Table 8.13). He *received* a total of 53 tackles (versus the lower rugby mean of 42.75), of which 72% were made above the waist and 28% were made below the waist (Table 8.14). For *Player C,* Grab tackles were the predominant means of making tackles (Figure 8.29), and Grab tackles and Side tackles were the predominant means of receiving tackles (Figure 8.30).

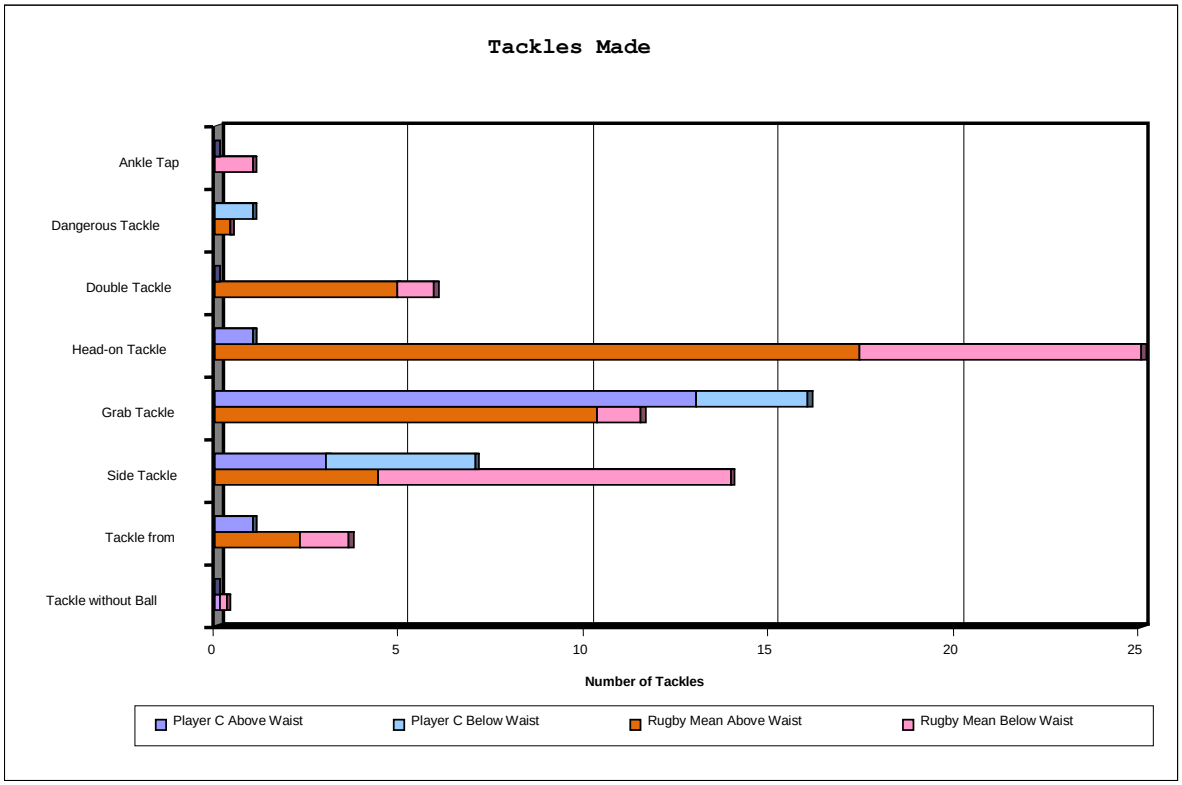
Table 8.13 *Player C's* Number and Type of Tackles Made with Rugby Mean

	Above Waist		**Below Waist**		**Total**	
	Player C	**Rugby Mean**	**Player C**	**Rugby Mean**	**Player C**	**Rugby Mean**
Ankle Tap	0	0.00	0	1.00	0	1.00
Dangerous Tackle	0	0.38	1	0.00	1	0.38
Double Tackle	0	4.91	0	0.95	0	5.86
Head-on Tackle	1	17.38	0	6.66	1	24.04
Grab Tackle	13	10.33	3	1.24	16	11.57
Side Tackle	3	4.38	4	9.52	7	13.90
Tackle from Behind	1	2.33	0	1.29	1	3.62
Tackle without Ball	0	0.14	0	0.19	0	0.33
Total Tackles	**18**	**39.85**	**8**	**20.85**	**26**	**60.70**
Percentage	**69**	**65**	**31**	**35**	**100**	**100**

Table 8.14 *Player C's* Number and Type of Tackles Received with Rugby Mean

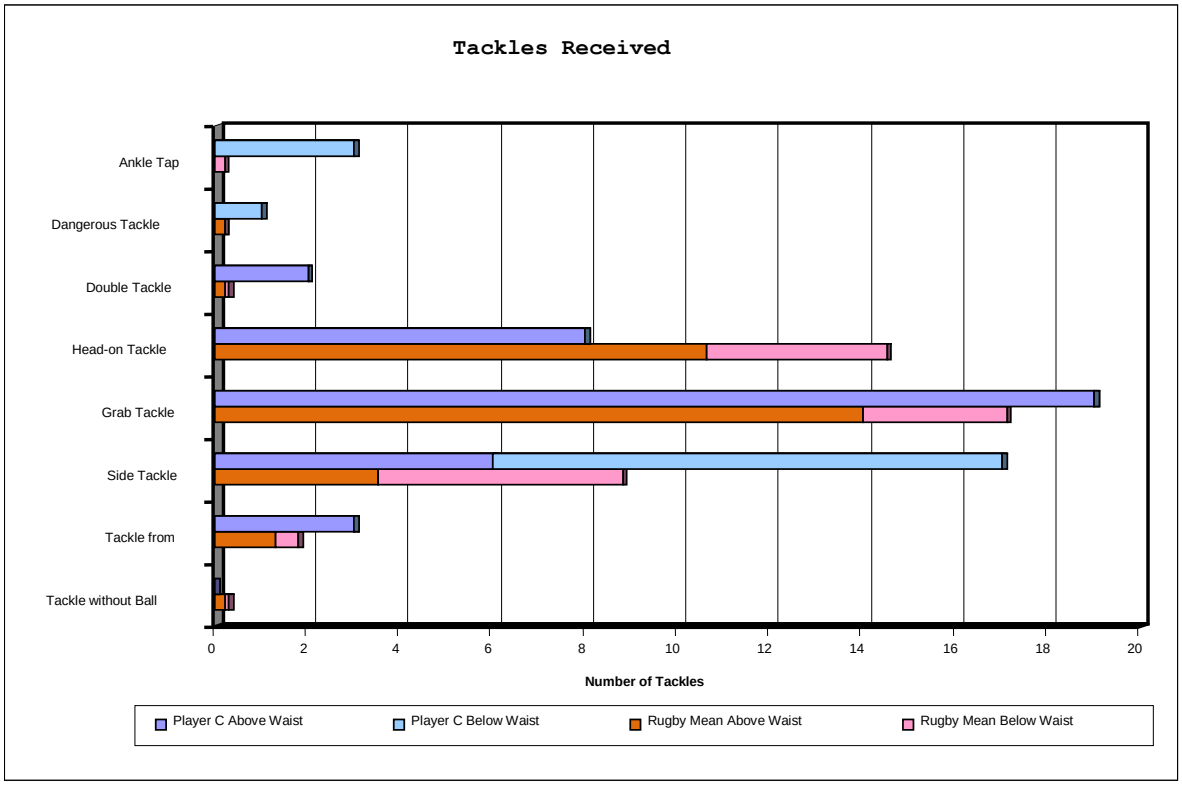
	Above Waist		**Below Waist**		**Total**	
	Player C	**Rugby Mean**	**Player C**	**Rugby Mean**	**Player C**	**Rugby Mean**
Ankle Tap	0	0.00	3	0.19	3	0.19
Dangerous Tackle	0	0.19	1	0.00	1	0.19
Double Tackle	2	0.19	0	0.09	2	0.28
Head-on Tackle	8	10.61	0	3.90	8	14.51
Grab Tackle	19	13.51	0	3.10	19	16.61
Side Tackle	6	3.52	11	5.31	17	8.82
Tackle from Behind	3	1.29	0	0.52	3	1.78
Tackle without Ball	0	0.24	0	0.14	0	0.37
Total Tackles	**38**	**29.50**	**15**	**13.25**	**53**	**42.75**
Percentage	**72**	**69**	**28**	**31**	**100**	**100**

Figure 8.29 ***Player C's*** **Number and Type of Tackles Made with Rugby Mean**



Note: Rugby Mean (orange and pink) indicated below Player C's performance (dark blue and light blue)

Figure 8.30 ***Player C's*** **Number and Type of Tackles Received with Rugby Mean**



Note: Rugby Mean (orange and pink) indicated below Player C's performance (dark blue and light blue)

8.2.3.3 Player C: Overview

In sum, this was a 27 year old rugby player with estimated above average IQ, who reported a history of one prior concussion excluding the most recent, suspected and not formally diagnosed concussion. His tackling count over the season overall was less than the rugby team's averages on Tackles Made and Total Tackles, although his Tackles Received count was marginally higher than that of the rugby team's average. At the *pre*-season assessment interval, his scores were lower than expected of an individual with an estimated above average IQ, denoting "borderline" performance on ImPACT Verbal Memory and "low average" performance on ImPACT Visual Motor Speed. At the *P-c1* assessment interval, compared with the *pre*-season assessment interval, he showed signs of decreased cognitive performance on ImPACT Visual Memory, with subtle signs of decreased cognitive performance on Purdue Non-Preferred, Purdue Both and Purdue Assembly. At the *mid*-season assessment interval, compared with the *P-c1* assessment interval, he showed signs of

decreased cognitive performance on ImPACT Verbal Memory and ImPACT Visual Motor Speed, with subtle signs of decreased cognitive performance on Purdue Preferred at the *post*-season assessment interval. Taking the repeat assessments into consideration *Player C* seemed to get back to or improve on his *pre*-season levels at the *post*-season assessment interval in the modalities of Memory and Motor Speed, with the exception of Purdue Preferred. The Non-Contact Sports controls improved over *pre*-season levels quite substantially on the second assessment and sustained it with two additional repeat assessments, with the exception of Purdue Assembly at the *mid*-season assessment interval.

Overall for *Player C*, ImPACT Visual Memory, Purdue Non-Preferred, Purdue Both and Purdue Assembly appeared to be the most sensitive and discriminatory indicators of a suspected concussive event. Furthermore, it was evident at the *pre*-season assessment interval that *Player C* was already compromised on ImPACT Visual Motor Speed, implicating possible residual effects of cumulative concussive and/or subconcussive events, whereas the controls were consistently within the normative ranges for all tests.

8.2.4 Demographic and Clinical History of Player D

GENERAL INFORMATION					
Subject	*Player D*			**Age**	28
Home language	English	**Other**	Afrikaans	**Race:**	White
EDUCATION, OCCUPATION AND ESTIMATE IQ					
Level of Education completed	Grade 12				
Current study, if any	None				
Occupation	Own Business				
Estimate IQ (Established on the basis of WAIS-III Picture Completion and Matrix Reasoning Scaled Scores using the OPIE-3 Formula)	101 (Average)				
MEDICAL AND PSYCHIATRIC HISTORY					
None					
CONCUSSION HISTORY					
Prior Concussions	± 10				

8.2.4.1 Player D: Neurocognitive Assessment Results

For *Player D,* the neurocognitive assessment results across all assessment intervals (*pre-*, *mid-*, and *post*-season as well as post-concussion) for each measure are tabulated together with the Non-Contact Sports control mean and the US average ranges (Table 8.15), and illustrated further by means of a figure in respect of each separate measure (Figures 8.31 – 8.38). For discussion purposes the results are interpreted in terms of the normative categories for both ImPACT and the Purdue Pegboard, as found earlier in Tables 8.3 and 8.5. The tables and figures for *Player D* appear together at the end of this subsection (page 201-206).

8.2.4.1.1 Memory

ImPACT Verbal Memory (Table 8.15; Figure 8.31)**.** At the *pre*-season assessment interval *Player D*'s Verbal Memory score denoted an "average" performance relative to the US normative categories. There was a substantial improvement in performance relative to the *pre*-season score at the *P-c1* assessment interval at the ceiling of the test, denoting a "very superior" performance relative to the US normative categories. At the *mid*-season assessment interval the score revealed a considerable worsening in performance relative to the two previous assessments, denoting an "average" performance relative to the US normative categories. At the *post*-season assessment interval there was an improvement in evidence compared to the *mid*-season level but did not reach the *pre*-season or *P-c1* level, in the direction that denoted an "average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player D* was performing at a higher level than the controls. The controls remained consistent over the first two assessments and improved at the *post*-season assessment interval, whereas in contrast *Player D* was initially performing better than the controls, reached his ceiling post-injury, but could not regain his *pre*-season performance by the *post*-season assessment interval.

ImPACT Visual Memory (Table 8.15; Figure 8.32)**.** At the *pre*-season assessment interval *Player D*'s Visual Memory score denoted an "average" performance relative to the US normative categories. At the *P-c1* assessment interval there was a considerable improvement in evidence compared to the *pre*-season score albeit still denoting an "average" performance

relative to the US normative categories. At the *mid-season* assessment interval, however, there was a worsening in performance, although still indicating an "average" performance relative to the US normative categories. At the *post-season* assessment interval the score showed a substantial improvement on the previous fluctuating scores, denoting an "average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player D* was performing at a higher level than the controls. The controls showed a gradual improvement from *pre-* to *mid*-season with a sharp improvement at the *post*-season assessment interval, whereas in contrast *Player D* started off higher than the controls, improved considerably immediately post injury, declined at *mid*-season and ultimately improved substantially by the *post*-season assessment interval, implicating that through his fluctuating performance *Player D* possibly did not benefit from practice.

8.2.4.1.2 Motor Speed

ImPACT Visual Motor Speed (Table 8.15; Figure 8.33). At the *pre*-season assessment interval *Player D*'s Visual Motor Speed score denoted a "low average" performance relative to the US normative categories. At the *P-c1* assessment interval there was an indication of improved performance over the *pre*-season level, denoting an "average" performance relative to the US normative categories. At the *mid*-season assessment interval there was a further improvement over the *pre*-season and *P-c1* scores, albeit still denoting an "average" performance relative to the US normative categories. At the *post*-season assessment interval, however, there was a worsening in performance, although better than the *pre*-season level and denoting an "average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player D* was initially performing at a lower level than the controls. The controls showed a sharp improvement from *pre-* to *mid*-season with a more gradual and steady improvement from the *mid-* to *post*-season assessment intervals, whereas in contrast *Player D* was initially not performing as well as he could, improved immediately post-injury, although ultimately after multiple assessments he did not reveal the ability to benefit from practice with *post*-season scores lower than post-injury and *mid*-season assessment levels.

ImPACT Reaction Time (Table 8.15; Figure 8.34)**.** At the *pre*-season assessment interval *Player D*'s Reaction Time score denoted a "low average" performance relative to the US normative categories. At the *P-c1* assessment interval there was a decrease in Reaction Time (i.e. an improvement in performance), and at the *mid*-season assessment interval there was a further improvement in performance due to a decreased Reaction Time on the *pre*-season and *P-c1* scores, denoting an "average" performance relative to the US normative categories. At the *post*-season assessment interval the score showed a marginal worsening compared to the *mid*-season assessment but still denoting an "average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player D* was initially performing at a lower level than the controls. The controls showed an improved performance due to a decreased Reaction Time score from *pre*- to *mid*-season and stabilised by the *post*-season assessment interval, whereas in contrast *Player D* was initially not performing as well as he could, improved immediately post-injury and stabilised from the *mid*-to the *post*-season assessment intervals.

Purdue Preferred (Table 8.15; Figure 8.35)**.** At the *pre*-season assessment interval *Player D*'s Preferred score revealed an "average" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was an improved performance to a level that was within the "high average" range relative to the Purdue normative categories. At the *mid*-season assessment interval the score showed a marginal worsening compared to the previous assessment, denoting an "average" performance relative to the Purdue normative categories. At the *post*-season assessment interval there was an improvement on the *mid*-season assessment and was similar to the *P-c1* level, denoting a "high average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player D* was initially performing at a marginal lower level than the controls. The controls showed an improvement at the *mid*-season assessment interval, whereas in contrast *Player D* was initially not performing as well as the controls, improved immediately post-injury, and although ultimately after multiple assessments he did not reveal the ability to maintain his performance and/or to benefit from practice as fluctuations in performance was in evidence throughout the season.

Purdue Non-Preferred (Table 8.15; Figure 8.36)**.** At the *pre*-season assessment interval *Player D*'s Non-Preferred score revealed performance in the lower limits of the "average" range relative to the Purdue normative categories. At the *P-c1* assessment interval there was an improved performance to a level that was within the "high average" range relative to the Purdue normative categories. At the *mid*-season assessment interval the score showed a marginal worsening compared to the previous assessment denoting an "average" performance relative to the Purdue normative categories. At the *post*-season assessment interval there was an improved performance on the *mid*-season and *Pc-1* scores, denoting a "high average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player D* was initially performing at a lower level than the controls. The controls showed an improvement at the *mid*-season assessment interval, whereas in contrast *Player D* was initially not performing as well as he could, improved substantially immediately post-injury and although ultimately after multiple assessments he did not reveal the ability to benefit from practice and/or to maintain his performance.

Purdue Both (Table 8.15; Figure 8.37)**.** At the *pre*-season assessment interval *Player D*'s Both score denoted an "average" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was a marginal improvement in performance and at the *mid*-season assessment interval the scores showed no change in the direction that denoted "average" performance relative to the Purdue normative categories. At the *post*-season assessment interval there was a further improvement on all the previous assessments, with a performance in the upper limits of the "average" range relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player D* was initially performing at a similar level than the controls. The controls showed an improvement at the *mid*-season assessment interval and stabilised by *post*-season, whereas in contrast *Player D* was initially performing quite similar to the controls, benefited from practice and improved by the *post*-season assessment interval.

Purdue Assembly (Table 8.15; Figure 8.38)**.** At the *pre*-season assessment interval *Player D*'s Assembly score revealed a "poor" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was a worsening in evidence, albeit still

denoting a "poor" performance relative to the Purdue normative categories. At the *mid*-season assessment interval there was a substantial improvement compared to the two previous assessments denoting an "average" performance relative to the Purdue normative categories. At the *post*-season assessment interval there was a further improvement over all the previous assessment intervals to a level in the upper limits of the "average" range relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player D* was initially performing at a lower level than the controls. The controls showed a slight fluctuation in performance on the *mid*-season assessment interval and improved again by *post*-season, implicating that the controls possible started off closer to their ceiling, whereas in contrast *Player D* initially needed practice to surpass all previous scores by the *post*-season assessment interval.

Table 8.15 ***Player D's*** **ImPACT and Purdue Pegboard Repeat Assessment Scores vs the Non-Contact Sports Control Mean Score and the US Average Range**

	Pre-season 2005/02/07	P-c1 2005/04/05	Mid-season 2005/06/05	Post-season 2005/10/10	US Average[1]
MEMORY					
ImPACT Verbal Memory					
Player D	93	100	85	89	83 – 94
Control Mean Score	84	-	85	87	
ImPACT Visual Memory					
Player D	80	93	86	94	69 – 94
Control Mean Score	74	-	76	82	
MOTOR SPEED					
ImPACT VMS[2]					
Player D	**31.9**	35.8	39.3	34.3	32.5 - 42.0
Control Mean Score	37.5	-	39.5	39.0	
ImPACT Reaction Time					
Player D	**0.65**	0.58	0.50	0.51	0.60 - 0.51
Control Mean Score	0.55	-	0.50	0.52	
Purdue Preferred					
Player D	15.00	18.00	17.00	18.00	16.22 (1.81)
Control Mean Score	16.05	-	17.00	17.09	
Purdue Non-Preferred					
Player D	13.00	17.00	16.00	18.00	15.41 (2.08)
Control Mean Score	15.09	-	16.05	16.23	
Purdue Both					
Player D	12.00	13.00	13.00	14.00	12.94 (1.29)
Control Mean Score	12.82	-	13.07	13.36	
Purdue Assembly					
Player D	**32.00**	**30.00**	38.00	40.00	39.13 (3.58)
Control Mean Score	36.41	-	35.55	36.45	

[1]Note: ImPACT delineated by range; Purdue Pegboard in mean score and Standard Deviation
Bold print represent scores that fall below the lower limit of the average ranges of both ImPACT and the Purdue Pegboard
[2]Note: Visual Motor Speed (VMS)

Figure 8.31 ***Player D's*** **ImPACT Verbal Memory Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

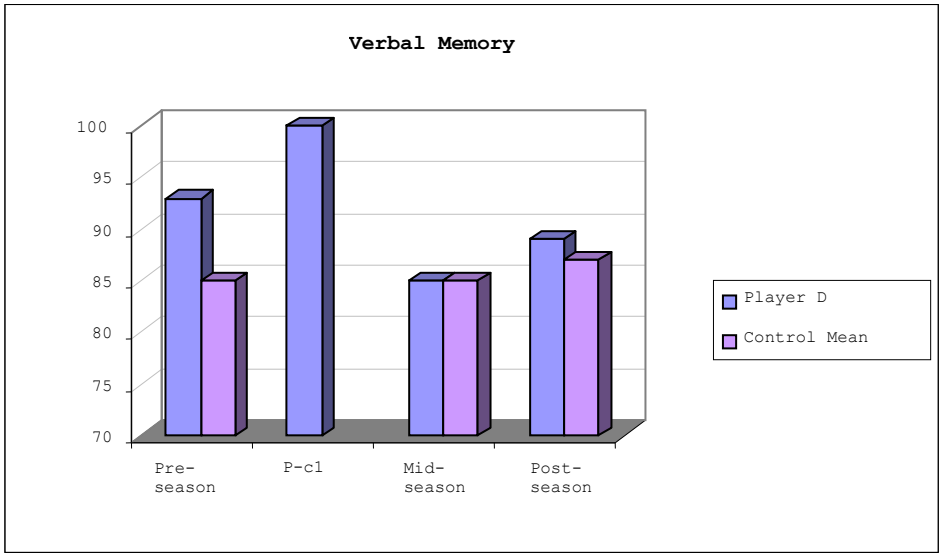


Figure 8.32 ***Player D's*** **ImPACT Visual Memory Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

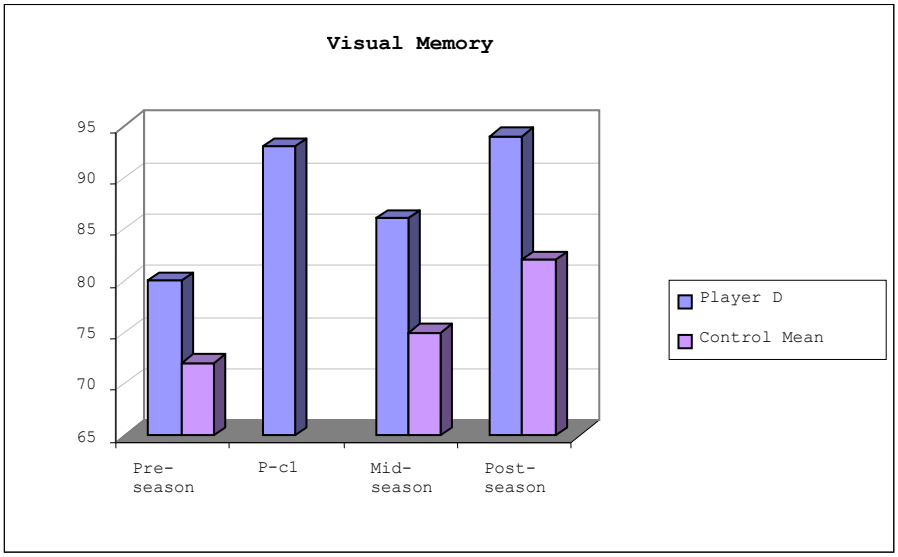


Figure 8.33 ***Player D's*** **ImPACT Visual Motor Speed Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

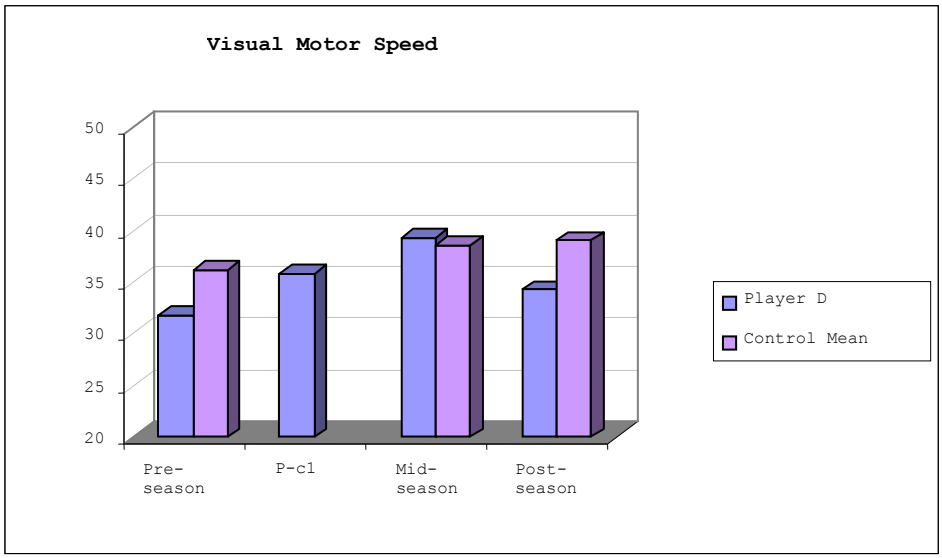


Figure 8.34 ***Player D's*** **ImPACT Reaction Time Composite Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

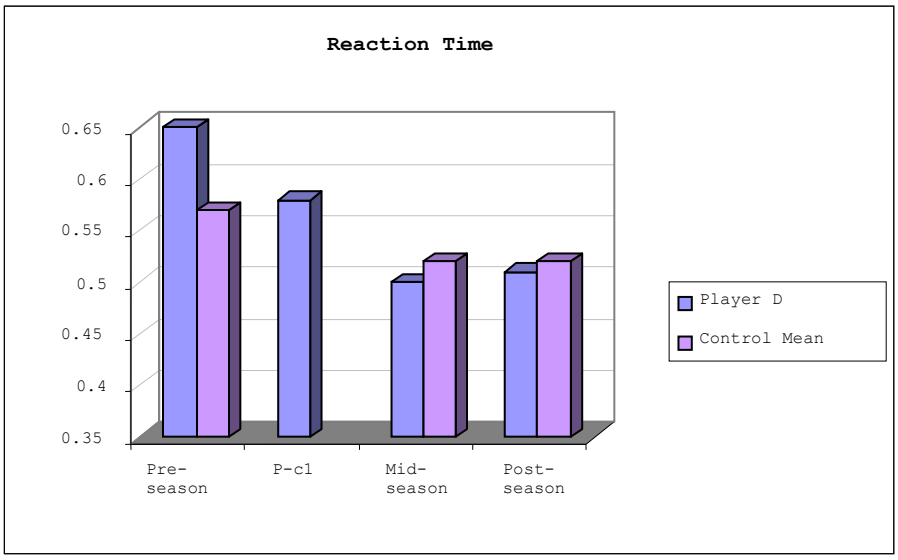


Figure 8.35 ***Player D's*** **Purdue Preferred Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

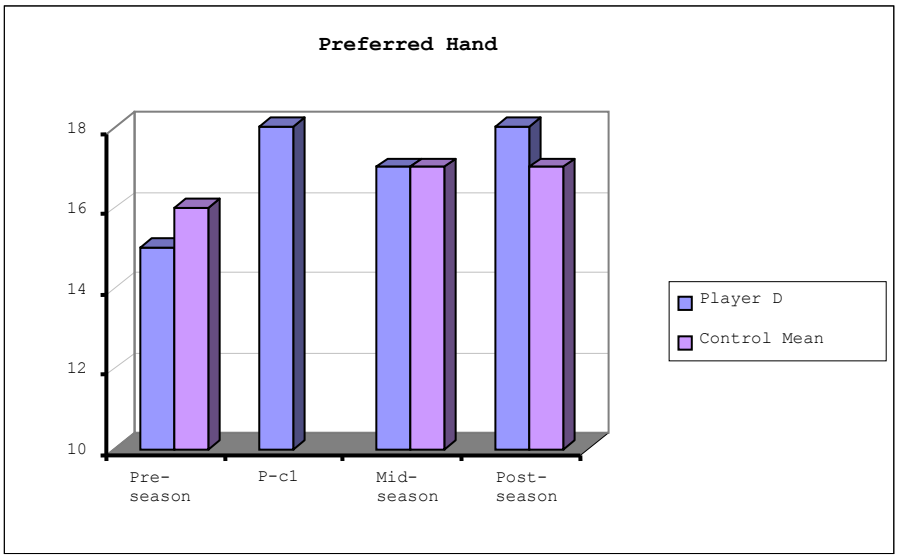


Figure 8.36 ***Player D's*** **Purdue Non-Preferred Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

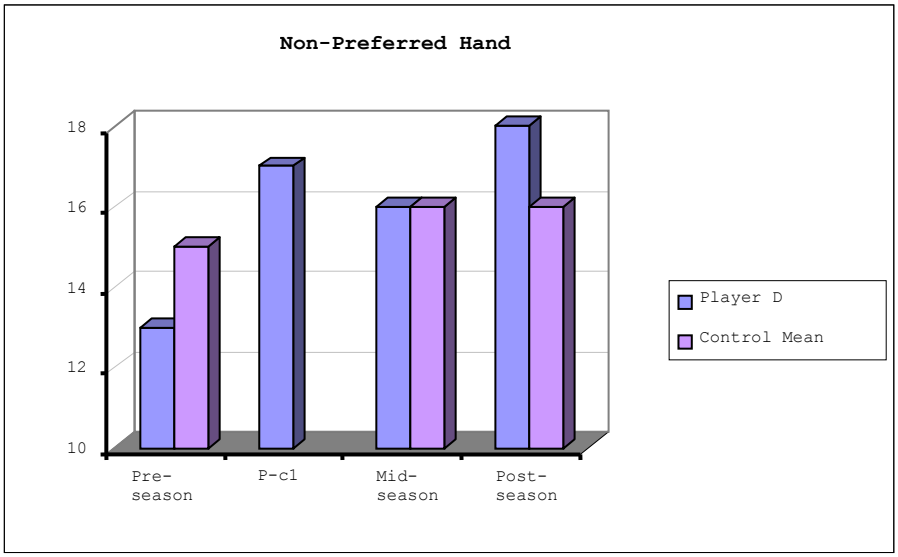


Figure 8.37 ***Player D's*** **Purdue Both Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**

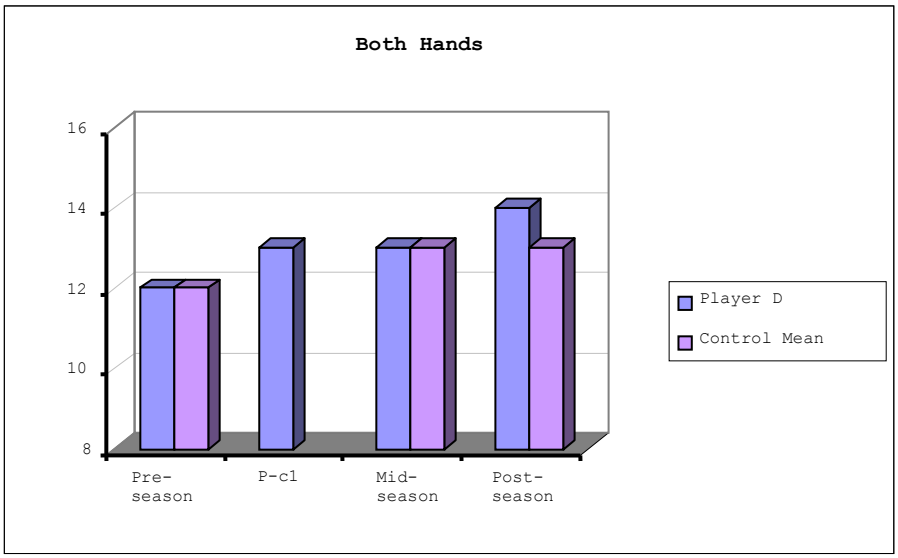
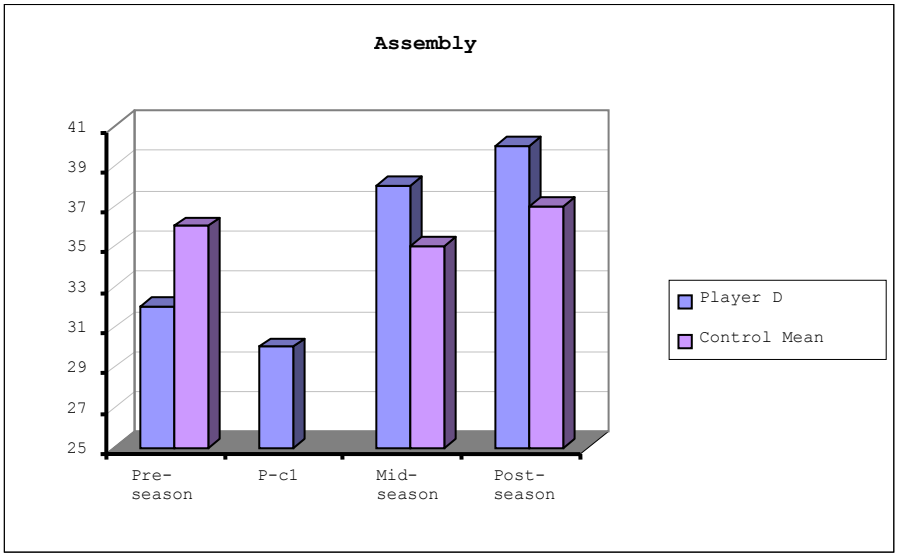


Figure 8.38 ***Player D's*** **Purdue Assembly Scores at the Pre-season, Post-concussion 1, Mid- and Post-season Assessment Intervals**



8.2.4.2 Player D: Tackling

As tabled in the previous subsection (Table 8.2, page 152), *Player D* was involved in a total of **122** tackles, a number which was substantially higher than the team average of 103.45. A further analysis of his tackling data revealed that he *made* a total of **48** tackles (versus the higher rugby mean of 60.70), of which 81% were made above the waist and 19% were made below the waist (Table 8.16). He *received* a total of **74** tackles (versus the substantially lower rugby mean of 42.75), of which 72% were made above the waist and 28% were made below the waist (Table 8.17). For *Player D,* Head-on tackles were the predominant means of making tackles (Figure 8.39), and Head-on tackles were the predominant means of receiving tackles (Figure 8.40).

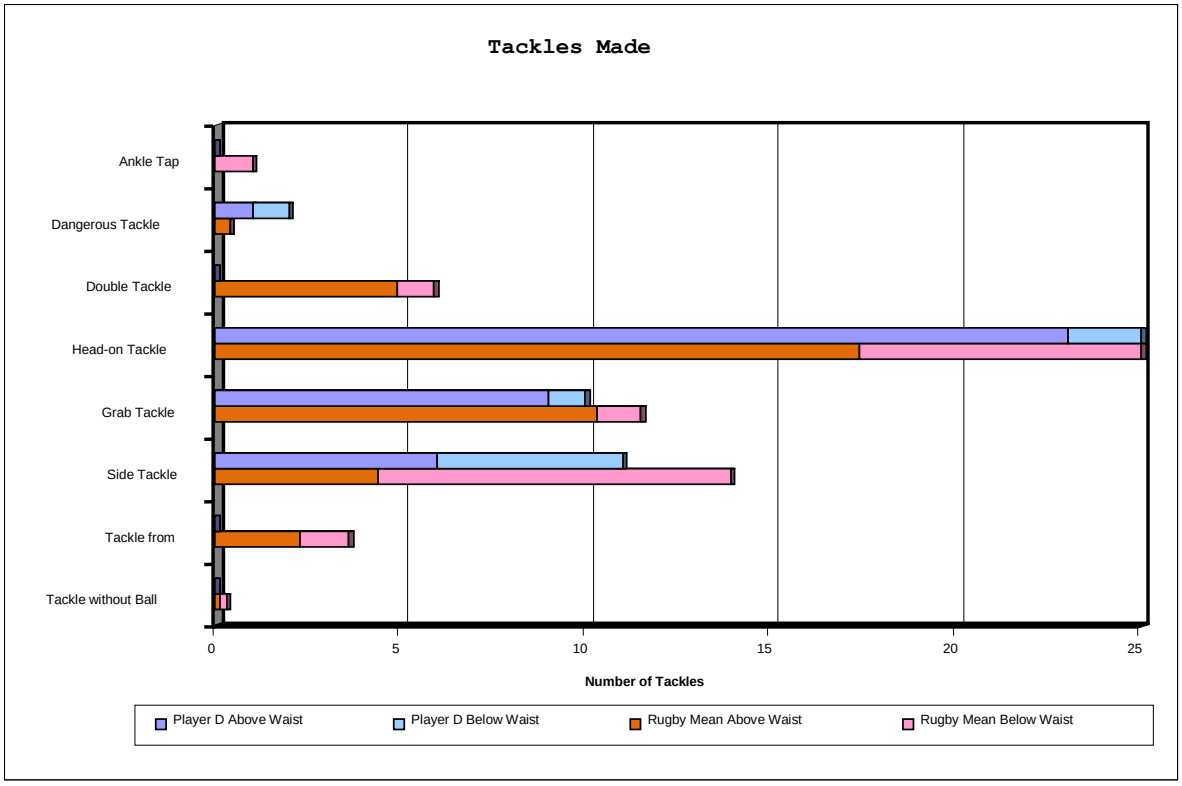
Table 8.16 *Player D's* Number and Type of Tackles Made with Rugby Mean

	Above Waist		**Below Waist**		**Total**	
	Player D	**Rugby Mean**	**Player D**	**Rugby Mean**	**Player D**	**Rugby Mean**
Ankle Tap	0	0.00	0	1.00	0	1.00
Dangerous Tackle	1	0.38	1	0.00	2	0.38
Double Tackle	0	4.91	0	0.95	0	5.86
Head-on Tackle	23	17.38	2	6.66	25	24.04
Grab Tackle	9	10.33	1	1.24	10	11.57
Side Tackle	6	4.38	5	9.52	11	13.90
Tackle from Behind	0	2.33	0	1.29	0	3.62
Tackle without Ball	0	0.14	0	0.19	0	0.33
Total Tackles	**39**	**39.85**	**9**	**20.85**	**48**	**60.70**
Percentage	**81**	**65**	**19**	**35**	**100**	**100**

Table 8.17 ***Player D's*** **Number and Type of Tackles Received with Rugby Mean**

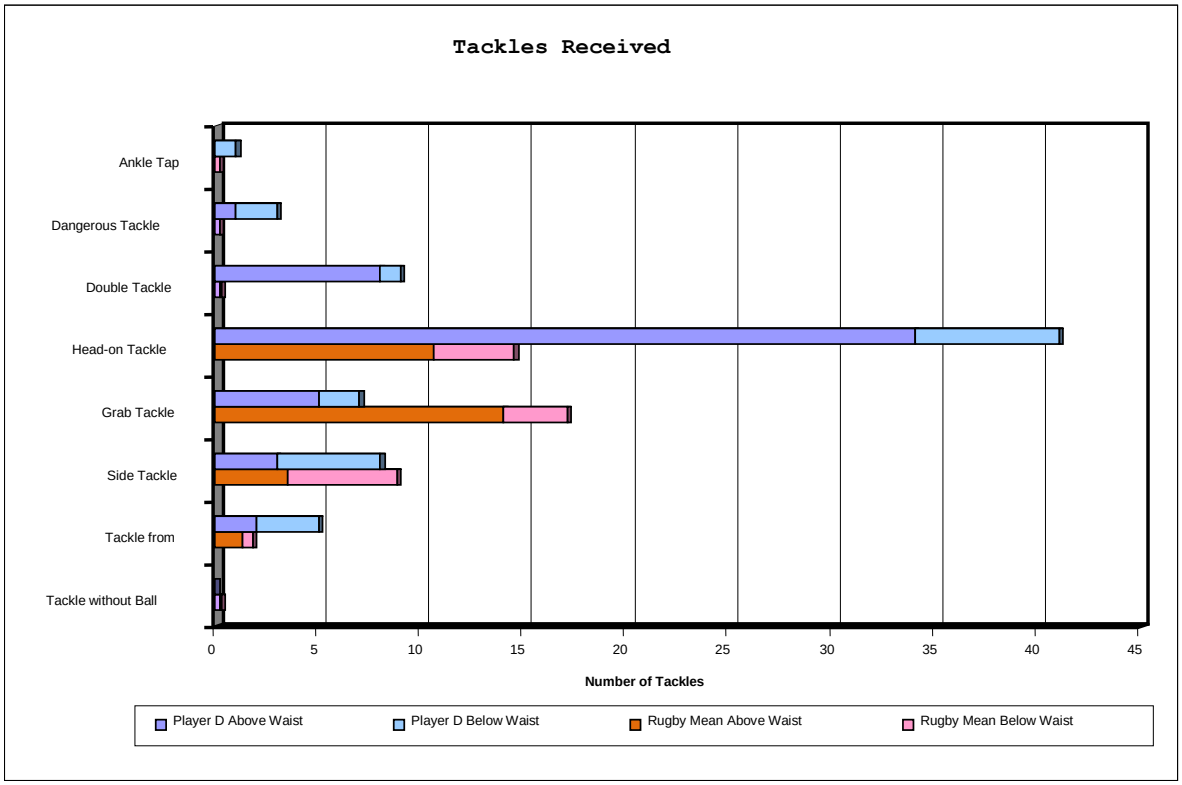
	Above Waist		Below Waist		Total	
	Player D	**Rugby Mean**	**Player D**	**Rugby Mean**	**Player D**	**Rugby Mean**
Ankle Tap	0	0.00	1	0.19	1	0.19
Dangerous Tackle	1	0.19	2	0.00	3	0.19
Double Tackle	8	0.19	1	0.09	9	0.28
Head-on Tackle	34	10.61	7	3.90	41	14.51
Grab Tackle	5	13.51	2	3.10	7	16.61
Side Tackle	3	3.52	5	5.31	8	8.82
Tackle from Behind	2	1.29	3	0.52	5	1.78
Tackle without Ball	0	0.24	0	0.14	0	0.37
Total Tackles	**53**	**29.50**	**21**	**13.25**	**74**	**42.75**
Percentage	**72**	**69**	**28**	**31**	**100**	**100**

Figure 8.39 ***Player D's*** **Number and Type of Tackles Made with Rugby Mean**



Note: Rugby Mean (orange and pink) indicated below Player D's performance (dark blue and light blue)

Figure 8.40 ***Player D's*** **Number and Type of Tackles Received with Rugby Mean**



Note: Rugby Mean (orange and pink) indicated below Player D's performance (dark blue and light blue)

8.2.4.3 Player D: Overview

In sum, this was a 28 year old rugby player with estimated average IQ, who reported a history of ten prior concussions (none formally diagnosed) and excluding the most recent suspected and not formally diagnosed concussion. His tackling count over the season overall was considerably higher than the rugby team's averages on Tackles Received and Total Tackles, although his Tackles Made count was lower than that of the rugby team's average. At the *pre*-season assessment interval, his scores were lower than expected of an individual with an estimated average IQ, denoting "low average" performance on ImPACT Visual Motor Speed and ImPACT Reaction Time and "poor" performance on Purdue Assembly. At the *P-c1* assessment interval, compared with the *pre*-season assessment interval, he showed signs of decreased cognitive performance on Purdue Assembly. At the *mid*-season assessment interval, compared with the *P-c1* assessment interval, he showed signs of decreased cognitive performance on ImPACT Verbal Memory, ImPACT Visual Memory,

Purdue Preferred and Purdue Non-Preferred. At the *post*-season assessment interval, compared with the *mid*-season assessment interval, he showed signs of decreased cognitive performance on ImPACT Visual Motor Speed and ImPACT Reaction Time. Taking the repeat assessments into consideration *Player D* seemed to fluctuate throughout the season and improved his *pre*-season scores at the *post*-season assessment interval, although at *post*-season there were indications of a slight decrease in performance on the *mid*-season scores on ImPACT Visual Motor Speed and ImPACT Reaction Time. The Non-Contact Sports controls improved over *pre*-season quite substantially on the second assessment and sustained it with two additional repeats.

Overall for *Player D*, Purdue Assembly (immediately post-concussive), ImPACT Verbal Memory, ImPACT Visual Memory, Purdue Preferred and Purdue Non-Preferred (*mid*-season), as well as ImPACT Visual Motor Speed and ImPACT Reaction Time (*post*-season), appeared to be the most sensitive and discriminatory indicators of a suspected concussive event. Furthermore, it was evident at the *pre*-season assessment interval that *Player D* was already compromised on ImPACT Visual Motor Speed, ImPACT Reaction Time, and Purdue Assembly, implicating possible residual effects of cumulative concussive and/or subconcussive events, whereas the controls were consistently within the normative ranges for all tests.

8.2.5 Demographic and Clinical History of Player E

GENERAL INFORMATION					
Subject		*Player E*		**Age**	26
Home language	English	**Other**		**Race:**	White
EDUCATION, OCCUPATION AND ESTIMATE IQ					
Level of Education completed		Grade 12, Degree			
Current study, if any		None			
Occupation		Massage Therapist			
Estimate IQ (Established on the basis of WAIS-III Picture Completion and Matrix Reasoning Scaled Scores using the OPIE-3 Formula)		104 (Average)			
MEDICAL AND PSYCHIATRIC HISTORY					
Fractures to the jaw, arm, nose, fingers and sternum					
CONCUSSION HISTORY					
Prior Concussions		1			

8.2.5.1 Player E: Neurocognitive Assessment Results

For *Player E,* the neurocognitive assessment results across all assessment intervals (*pre-*, *mid-*, and *post*-season as well as post-concussion) for each measure are tabulated together with the Non-Contact Sports control mean and the US average ranges (Table 8.18), and illustrated further by means of a figure in respect of each separate measure (Figures 8.41 – 8.48). For discussion purposes the results are interpreted in terms of the normative categories for both ImPACT and the Purdue Pegboard, as found earlier in Tables 8.3 and 8.5. The tables and figures for *Player E* appear together at the end of this subsection (page 215 - 219).

8.2.5.1.1 Memory

ImPACT Verbal Memory (Table 8.18; Figure 8.41)**.** At the *pre*-season assessment interval *Player E*'s Verbal Memory score denoted a "low average" performance relative to the US normative categories. At the *mid*-season assessment interval there was a considerable improvement over the *pre*-season score denoting an "average" performance relative to the US normative categories. There was a substantial improvement compared to the *pre*-season score at the *P-c1* assessment interval and at the ceiling of the test, denoting a "very superior" performance relative to the US normative categories. At the *post*-season assessment interval there was a marginal decrease in evidence relative to the previous assessment, albeit still denoting a "superior" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player E* was initially performing at a lower level than the controls. The controls showed consistent performance over the first two assessments and improved at the *post*-season assessment interval, whereas in contrast *Player E* started off lower than the controls, improved by *mid*-season and improved further post-injury, to ultimately by the *post*-season assessment interval be considerably better than the controls.

ImPACT Visual Memory (Table 8.18; Figure 8.42)**.** At the *pre*-season assessment interval *Player E*'s Visual Memory score denoted a "borderline" performance relative to the US normative categories. At the *mid-season* assessment interval there was a substantial improvement over the *pre*-season score denoting an "average" performance relative to the US normative categories. At the *P-c1* assessment interval there was a marginal worsening in evidence, albeit still with scores denoting an "average" performance relative to the US normative categories. At the *post*-season assessment interval there was a considerable improvement on all the previous assessment scores, denoting a "high average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player E* was initially performing at a lower level than the controls. The controls showed a gradual improvement from *pre*- to *mid*-season with a sharp improvement at the *post*-season assessment interval, whereas in contrast *Player E* was

initially not performing well but benefited from practice and improved substantially at the *post*-season assessment interval with a marginal fluctuation at the *P-c1* assessment interval.

8.2.5.1.2 Motor Speed

ImPACT Visual Motor Speed (Table 8.18; Figure 8.43). At the *pre*-season assessment interval *Player E*'s Visual Motor Speed score revealed a performance in the lower limits of the "average" range relative to the US normative categories. At the *mid*-season assessment interval there was a marginal worsening in performance compared to the *pre*-season level, denoting a "low average" performance relative to the US normative categories. At the *P-c1* assessment interval there was a further worsening in performance compared to the *pre*- and *mid*-season levels, albeit still denoting a "low average" performance relative to the US normative categories. At the *post*-season assessment interval there was an improvement in evidence and better than the *pre*-season level, denoting an "average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player E* was initially performing at a lower level than the controls. The controls showed a sharp improvement from *pre*- to *mid*-season with a more gradual and steady improvement from the *mid*- to *post*-season assessment intervals, whereas in contrast *Player E* was performing lower than the controls and ultimately after multiple assessments he did not reveal the ability to benefit from practice.

ImPACT Reaction Time (Table 8.18; Figure 8.44)**.** At the *pre*-season assessment interval *Player E*'s Reaction Time score denoted a "borderline" performance relative to the US normative categories. At the *mid*-season assessment interval there was a marginal improvement in evidence compared to the *pre*-season assessment, albeit still denoting a "borderline" performance relative to the US normative categories. At the *P-c1* assessment interval there was a further decrease in Reaction Time (i.e. an improvement in performance), however still denoting a "low average" performance relative to the US normative categories. At the *post*-season assessment interval there was again an increase in Reaction Time (i.e. a worsening in performance) denoting a "low average" performance relative to the US normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player E* was consistently performing at a much lower level than the controls.

The controls showed an improved performance due to a decreased Reaction Time score from *pre*- to *mid*-season and stabilised by the *post*-season assessment interval, whereas in contrast *Player E* was continuously performing at a much lower level, did not stabilise or showed indications of benefiting from practice.

ImPACT Preferred (Table 8.18; Figure 8.45)**.** At the *pre*-season assessment interval *Player E*'s Preferred score revealed a "poor" performance relative to the Purdue normative categories. At the *mid*-season assessment interval there was an improvement compared to the previous assessment, denoting an "average" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was a marginal worsening in performance denoting performance in the lower limits of the "average" range relative to the Purdue normative categories. At the *post*-season assessment interval there was an improvement on the previous assessment level denoting an "average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player E* was performing at a lower level than the controls. The controls showed an improvement on the *mid*-season assessment interval, whereas in contrast *Player E* started off low, improved at *mid*-season with a worsening in performance immediately post-injury and ultimately through fluctuating performance regained his *mid*-season score by the *post*-season assessment interval.

ImPACT Non-Preferred (Table 8.18 Figure 8.46)**.** At the *pre*-season assessment interval *Player E*'s Non-Preferred score denoted performance in the lower limits of the "low average" range relative to the Purdue normative categories. At the *mid*-season assessment interval there was an improvement compared to the previous assessment denoting an "average" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was a worsening in performance but similar to the *pre*-season level, denoting performance in the lower limits of the "low average" range relative to the Purdue normative categories. At the *post*-season assessment interval there was yet again an improvement on the *P-c1* assessment score and similar to the *mid*-season level denoting an "average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player E* was performing at a lower level than the controls. The controls showed an improvement at the *mid*-season assessment

interval, whereas in contrast *Player E* initially not performing as well as he could, and after multiple assessments he did not reveal the ability to benefit from practice.

ImPACT Both (Table 8.18; Figure 8.47)**.** At the *pre*-season assessment interval *Player E*'s Both score revealed a "low average" performance relative to the Purdue normative categories. At the *mid*-season assessment interval there was a marginal improvement compared to the previous assessment in the direction that denoted an "average" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was a worsening in performance in the direction that denoted a "low average" performance relative to the Purdue normative categories. At the *post*-season assessment interval there was an improvement on *P-c1* level and similar to *mid*-season, in the direction that denoted an "average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player E* was initially performing at a lower level than the controls. The controls showed an improvement at the *mid*-season assessment interval, whereas in contrast *Player E* started off marginally lower than the controls, and through fluctuating performances he never really benefited from practice.

ImPACT Assembly (Table 8.18; Figure 8.48)**.** At the *pre*-season assessment interval *Player E*'s Assembly score denoted performance in the lower limits of the "low average" range relative to the Purdue normative categories. At the *mid*-season assessment interval there was a substantial worsening in evidence denoting a "poor" performance relative to the Purdue normative categories. At the *P-c1* assessment interval there was a marginal improvement over the *mid*-season assessment score, albeit still denoting a "poor" performance relative to the Purdue normative categories. At the *post*-season assessment interval there was an improvement on all previous assessments in evidence, denoting an "average" performance relative to the Purdue normative categories. In comparison with the mean scores of the Non-Contact Sports Control Group, *Player E* was performing at a lower level than the controls. The controls showed a slight fluctuation in performance on the *mid*-season assessment interval, implicating that the controls possible started off closer to their ceiling, whereas in contrast *Player E* needed practice to eventually surpass his *pre*-season score at *post*-season.

Table 8.18 *Player E's* ImPACT and Purdue Pegboard Repeat Assessment Scores vs the Non-Contact Sports Control Mean Score and the US Average Range

	Pre-season 2005/02/15	Mid-season 2005/07/12	P-c1 2005/08/15	Post-season 2005/10/03	US Average[1]
MEMORY					
ImPACT Verbal Memory					
Player E	**82**	91	100	98	83 – 94
Control Mean Score	84	85	-	87	
ImPACT Visual Memory					
Player E	**52**	85	81	95	69 – 94
Control Mean Score	74	76	-	82	
MOTOR SPEED					
ImPACT VMS[2]					
Player E	32.9	32.3	**31.8**	33.5	32.5 - 42.0
Control Mean Score	37.5	39.5	-	39.0	
ImPACT Reaction Time					
Player E	**0.73**	**0.68**	**0.61**	**0.65**	0.60 - 0.52
Control Mean Score	0.55	0.50	-	0.52	
Purdue Preferred					
Player E	**13.00**	15.00	**14.00**	15.00	16.22 (1.81)
Control Mean Score	16.05	17.00	-	17.09	
Purdue Non-Preferred					
Player E	**12.00**	14.00	**12.00**	15.0	15.41 (2.08)
Control Mean Score	15.09	16.05	-	16.23	
Purdue Both					
Player E	**11.50**	12.00	**11.00**	12.00	12.94 (1.29)
Control Mean Score	12.82	13.07	-	13.36	
Purdue Assembly					
Player E	**33.00**	**26.00**	**30.00**	36.00	39.13 (3.58)
Control Mean Score	36.41	35.55	-	36.45	

[1]Note: ImPACT delineated by range; Purdue Pegboard in mean score and Standard Deviation
Bold print represent scores that fall below the lower limit of the average ranges of both ImPACT and the Purdue Pegboard
[2]Note: Visual Motor Speed (VMS)

Figure 8.41 ***Player E's*** **ImPACT Verbal Memory Composite Scores at the Pre-season, Mid-season, Post-concussion 1 and Post-season Assessment Intervals**

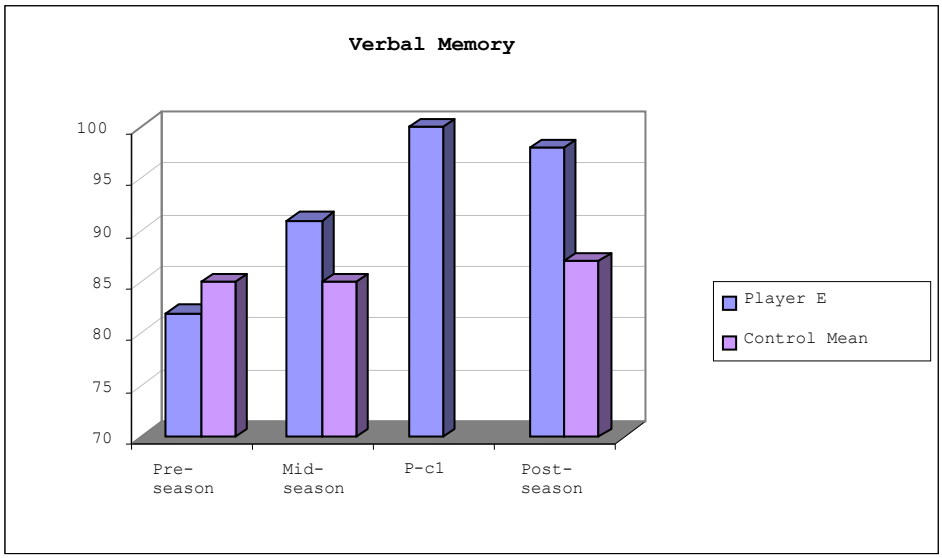


Figure 8.42 ***Player E's*** **ImPACT Visual Memory Composite Scores at the Pre-season, Mid-season, Post-concussion 1 and Post-season Assessment Intervals**

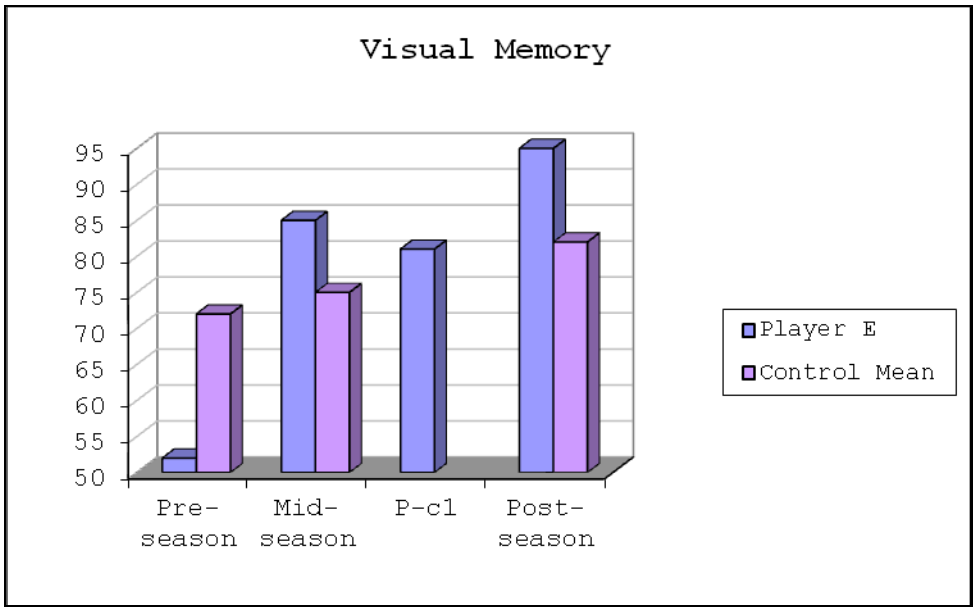


Figure 8.43 ***Player E's*** **ImPACT Visual Motor Speed Composite Scores at the Pre-season, Mid-season, Post-concussion 1 and Post-season Assessment Intervals**

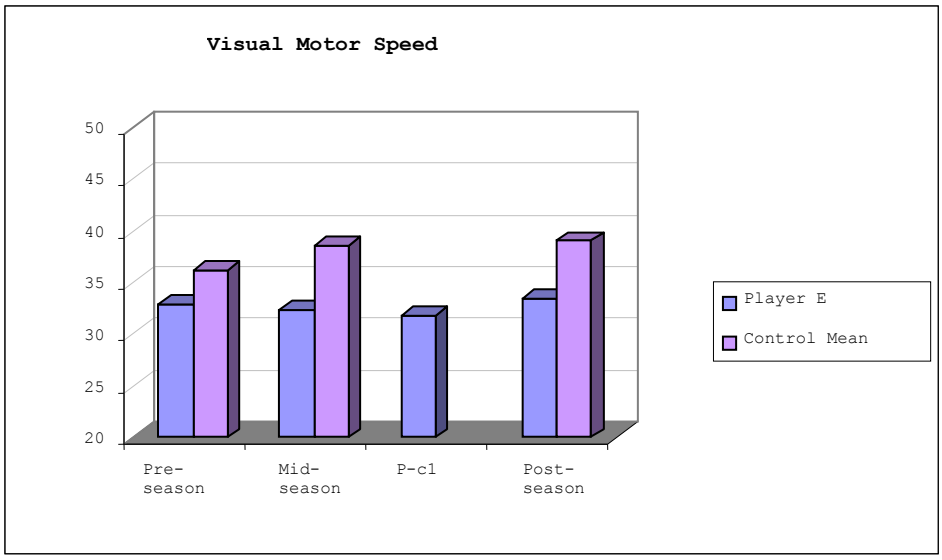


Figure 8.44 ***Player E's*** **ImPACT Reaction Time Composite Scores at the Pre-season, Mid-season, Post-concussion 1 and Post-season Assessment Intervals**

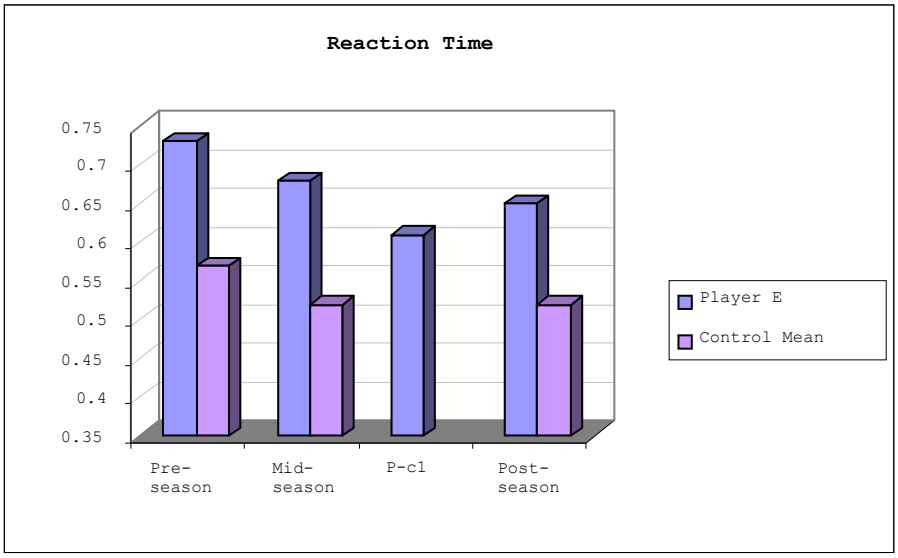


Figure 8.45 ***Player E's*** **Purdue Preferred Scores at the Pre-season, Mid-season, Post-concussion 1 and Post-season Assessment Intervals**

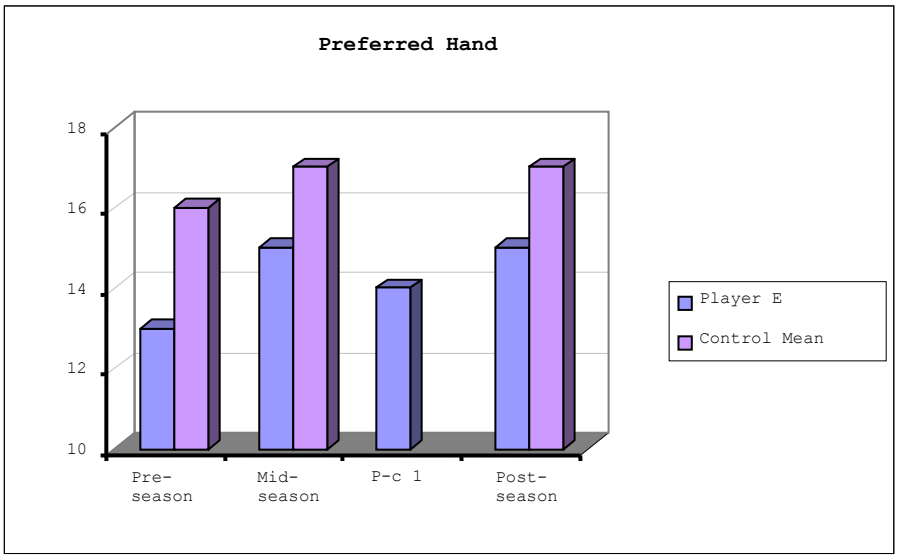


Figure 8.46 ***Player E's*** **Purdue Non-Preferred Scores at the Pre-season, Mid-season, Post-concussion 1 and Post-season Assessment Intervals**

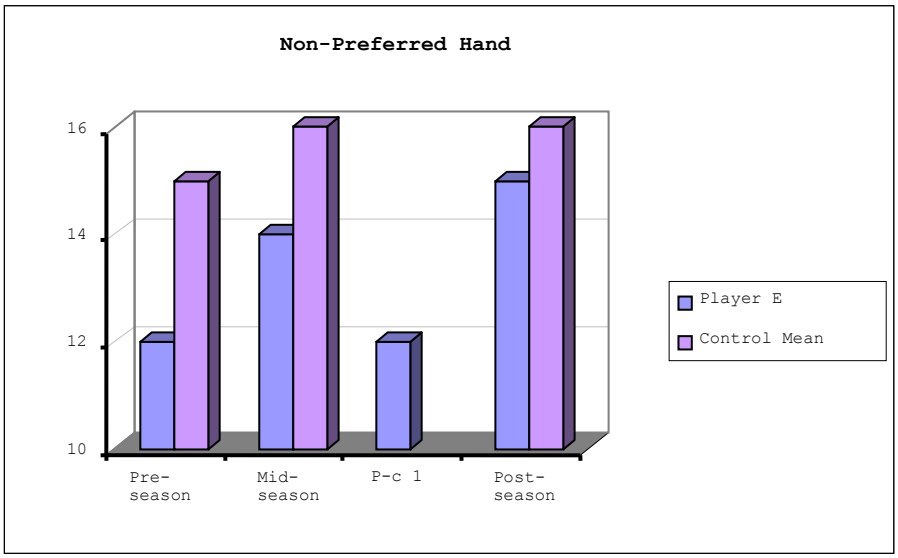


Figure 8.47 ***Player E's*** **Purdue Both Scores at the Pre-season, Mid-season, Post-concussion 1 and Post-season Assessment Intervals**

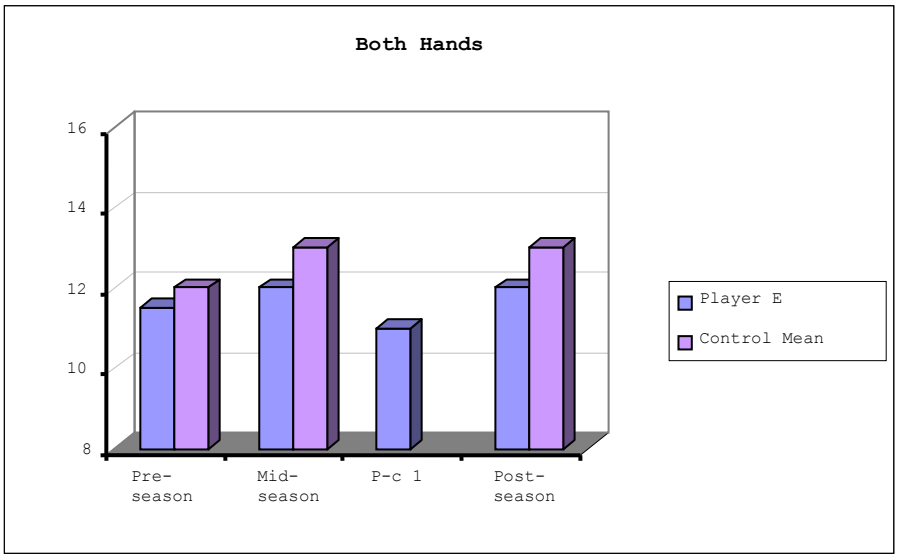
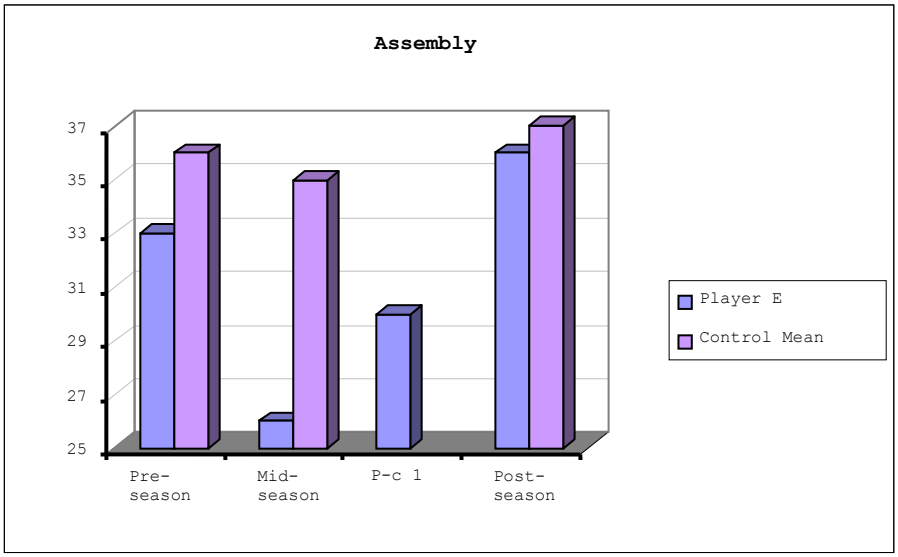


Figure 8.48 ***Player E's*** **Purdue Assembly Scores at the Pre-season, Mid-season, Post-concussion 1 and Post-season Assessment Intervals**



8.2.5.2 Player E: Tackling

As tabled in the previous subsection (Table 8.2, page 152), *Player E* was involved in a total of 114 tackles, a number which was higher than the team average of 103.45. A further analysis of his tackling data revealed that he *made* a total of 43 tackles, (versus the

substantially higher rugby mean of 60.70) of which 84% were made above the waist and 16% were made below the waist (Table 8.19). He *received* a total of 71 tackles (versus the substantially lower rugby mean of 42.75), of which 82% were made above the waist and 18% were made below the waist (Table 8.20). For *Player E*, Head-on tackles and Grab tackles were the predominant means of making tackles (Figure 8.49), and Head-on tackles were the predominant means of receiving tackles (Figure 8.50).

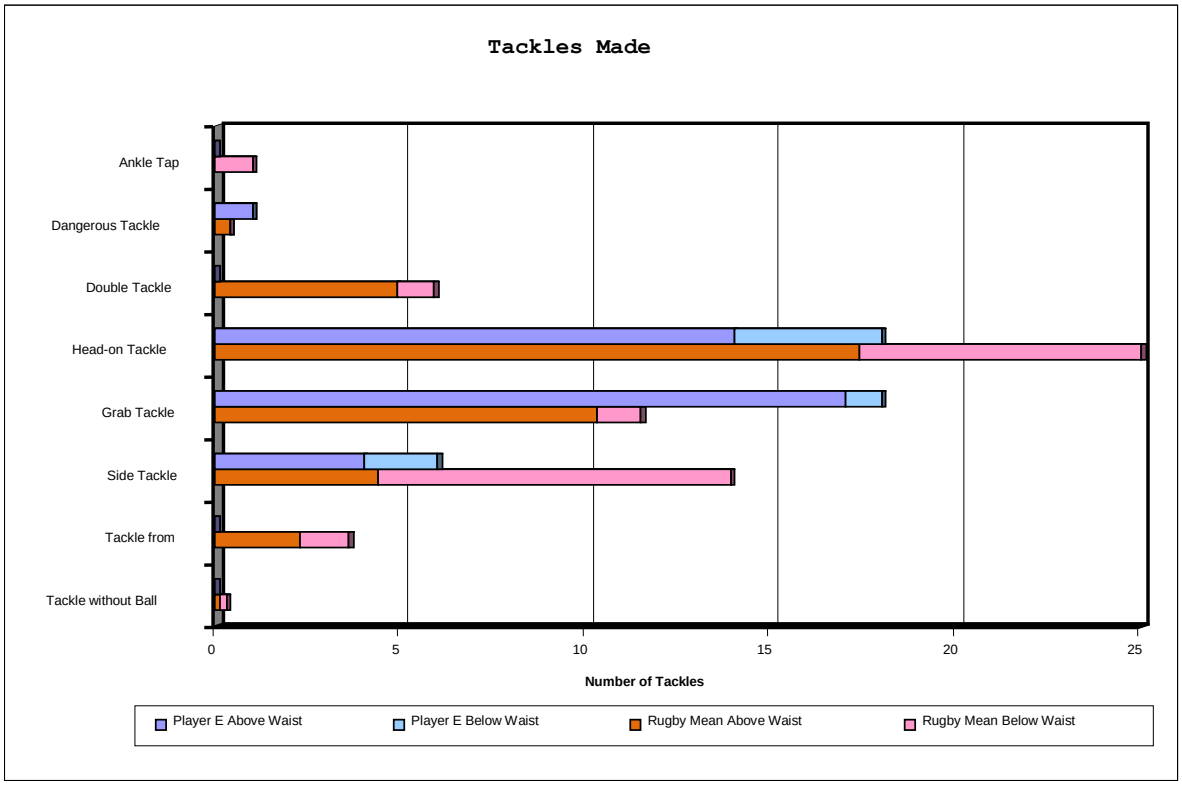
Table 8.19 ***Player E's*** **Number and Type of Tackles Made with Rugby Mean**

	Above Waist		**Below Waist**		**Total**	
	Player E	**Rugby Mean**	**Player E**	**Rugby Mean**	**Player E**	**Rugby Mean**
Ankle Tap	0	0.00	0	1.00	0	1.00
Dangerous Tackle	1	0.38	0	0.00	1	0.38
Double Tackle	0	4.91	0	0.95	0	5.86
Head-on Tackle	14	17.38	4	6.66	18	24.04
Grab Tackle	17	10.33	1	1.24	18	11.57
Side Tackle	4	4.38	2	9.52	6	13.90
Tackle from Behind	0	2.33	0	1.29	0	3.62
Tackle without Ball	0	0.14	0	0.19	0	0.33
Total Tackles	**36**	**39.85**	**7**	**20.85**	**43**	**60.70**
Percentage	**84**	**65**	**16**	**35**	**100**	**100**

Table 8.20 ***Player E's*** **Number and Type of Tackles Received with Rugby Mean**

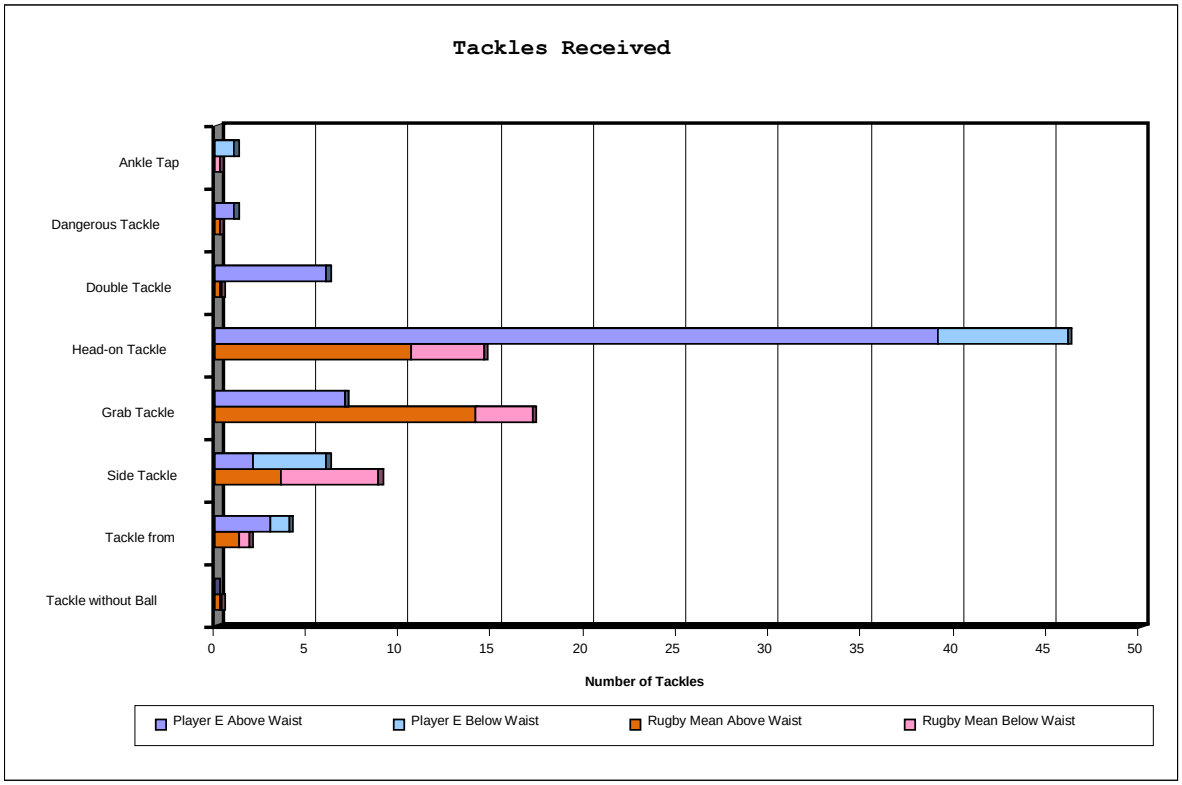
	Above Waist		**Below Waist**		**Total**	
	Player E	**Rugby Mean**	**Player E**	**Rugby Mean**	**Player E**	**Rugby Mean**
Ankle Tap	0	0.00	1	0.19	1	0.19
Dangerous Tackle	1	0.19	0	0.00	1	0.19
Double Tackle	6	0.19	0	0.09	6	0.28
Head-on Tackle	39	10.61	7	3.90	46	14.51
Grab Tackle	7	13.51	0	3.10	7	16.61
Side Tackle	2	3.52	4	5.31	6	8.82
Tackle from Behind	3	1.29	1	0.52	4	1.78
Tackle without Ball	0	0.24	0	0.14	0	0.37
Total Tackles	**58**	**29.50**	**13**	**13.25**	**71**	**42.75**
Percentage	**82**	**69**	**18**	**31**	**100**	**100**

Figure 8.49 ***Player E's*** **Number and Type of Tackles Made with Rugby Mean**



Note: Rugby Mean (orange and pink) indicated below Player E's performance (dark blue and light blue)

Figure 8.50 ***Player E's*** **Number and Type of Tackles Received with Rugby Mean**



Note: Rugby Mean (orange and pink) indicated below Player E's performance (dark blue and light blue)

8.2.5.3 Player E: Overview

In sum, this was a 26 year old rugby player with estimated average IQ, who reported a history of one prior concussion and excluding the most recent suspected and not formally diagnosed concussion. His tackling count over the season overall was considerably higher than the rugby team's averages on Tackles Received and Total Tackles, although his Tackles Made count was lower than that of the rugby team's average. At the *pre*-season assessment interval, his scores were lower than expected of an individual with an estimated average IQ, denoting "borderline" performance on ImPACT Visual Memory, ImPACT Reaction Time; "low average" performance on ImPACT Verbal Memory and Purdue Both; and "poor" performance on Purdue Preferred, Purdue Non-Preferred, and Purdue Assembly. At the *mid*-season assessment interval, compared with the *pre*-season assessment interval, he showed signs of decreased cognitive performance on ImPACT Visual Motor Speed and Purdue Assembly. At the *P-c1* assessment interval, compared with the *mid*-season assessment interval, he showed signs of decreased cognitive performance on ImPACT Visual Memory,

ImPACT Visual Motor Speed, Purdue Preferred, Purdue Non-Preferred, and Purdue Both. At the *post*-season assessment interval, compared with the *P-c1* assessment interval, he showed signs of decreased performance on ImPACT Verbal Memory and ImPACT Reaction Time. Taking the repeat assessments into consideration *Player E* seemed to fluctuate throughout and appeared only to improve at the *post*-season assessment interval, whereas the Non-Contact Sports controls improved over *pre*-season quite substantially on the second assessment and sustained it with two additional repeats.

Overall for *Player E*, ImPACT Verbal Memory (at *mid*-season) and ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Non-Preferred and Purdue Assembly appeared to be the most sensitive and discriminatory indicators of a suspected concussive event. Furthermore, it was evident at the *pre*-season assessment interval that *Player E* was already compromised on ImPACT Verbal Memory, ImPACT Visual Memory, ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both and Purdue Assembly, implicating possible residual effects of cumulative concussive and/or subconcussive events, whereas the controls were consistently within the normative ranges for all tests.

8.3 SYNTHESIS OF PLAYER PROFILES

Club rugby players in South Africa have typically played the sport since their early primary school years, and three of the five identified players have played competitive club rugby for more than ten years, with one player playing competitively for eight years and the youngest player for four years. There were two forward and three backline players. Two of the players had an estimated above average IQ, with the rest with an estimated average IQ. Three of the players were in the age range of 26 – 28, with one player aged 31, and the youngest player being 23.

One of the players reported no prior knowledge of sustaining or previously being treated for a concussion, two of the players indicated knowledge of at least one prior diagnosed concussion (one of them with a loss of consciousness), one of the players recalled four prior diagnosed concussions (two with a loss of consciousness) and one of the players reported the possibility of 10+ undiagnosed concussions since high school.

8.3.1 Neurocognitive Assessment Results

8.3.1.1 Memory

Pre-season. Two of the five identified players showed sensitivity to possible pre-existing concussive and subconcussive events with lowered performance relative to the US normative categories on ImPACT Verbal Memory (*Players C* and *E*) and one of the five identified players demonstrated lowered performance on ImPACT Visual Memory (*Player E*).

Post-concussion follow up. Two of the five identified players showed sensitivity to possible pre-existing concussive and subconcussive events in conjunction with the observed suspected concussive event sustained during the season, with lowered performance relative to the US normative categories on ImPACT Verbal Memory (*Players A* and *B*), and on ImPACT Visual Memory (*Players C* and *E*).

Mid-season. Two of the five identified players showed sensitivity to possible pre-existing concussive and subconcussive events in conjunction with the observed suspected concussive event sustained during the season with lowered performance relative to the US normative categories on ImPACT Verbal Memory (*Players C* and *D*) and on ImPACT Visual Memory (*Players A* and *D*).

Post-season. One of the five identified players showed sensitivity to possible pre-existing concussive and subconcussive events in conjunction with the observed suspected concussive event sustained during the season with lowered performance relative to the US normative categories on ImPACT Verbal Memory (*Player E*).

8.3.1.2 Motor Speed

Pre-season. Three of the five identified players showed sensitivity to possible pre-existing concussive and subconcussive events with lowered performance relative to the US normative

categories on ImPACT Visual Motor Speed (*Players B, C* and *D*), ImPACT Reaction Time and Purdue Assembly (*Players B, D* and *E*). There was evidence of Purdue Preferred, Purdue Non-Preferred and Purdue Both showing sensitivity to possible concussive and subconcussive events with lowered performance relative to the Purdue normative categories for two of the five identified players (*Players B* and *E*).

Post-concussion follow up. Four of the five identified players showed sensitivity to possible pre-existing concussive and subconcussive events in conjunction with the observed suspected concussive event sustained during the season, with lowered performance relative to the Purdue normative categories on Purdue Assembly (*Players B, C, D* and *E*). Three of the five identified players showed lowered performance relative to the Purdue normative categories on Purdue Both and Purdue Assembly (*Players B, C* and *E*). There was evidence of ImPACT Visual Motor Speed (*Players B* and *E*), ImPACT Reaction Time (*Players A* and *B*), and Purdue Preferred (*Players B* and *E*) showing sensitivity to possible concussive and subconcussive events with lowered performance relative to the respective normative categories. However, *Player A* demonstrated lowered performances at his *P-c2* assessment interval on ImPACT Visual Motor Speed, Purdue Both and Purdue Assembly.

Mid-season. Three of the five identified players demonstrated sensitivity to possible pre-existing concussive and subconcussive events in conjunction with the observed suspected concussive event sustained during the season, with lowered performance relative to the US normative categories on ImPACT Visual Motor Speed (*Players B, C* and *E*). Two of the five identified players showed lowered performance relative to the US normative categories on ImPACT Reaction Time (*Players B* and *E*) and Purdue Both (*Players A* and *B*). One of the five identified players showed lowered performance relative to the Purdue normative categories on Purdue Preferred and Purdue Non-Preferred (*Player D*), and on Purdue Assembly (*Player E*).

Post-season. Two of the five identified players demonstrated sensitivity to possible pre-existing concussive and subconcussive events in conjunction with the observed suspected concussive event sustained during the season, with lowered performance relative to the US normative categories on ImPACT Visual Motor Speed (*Players B,* and *D*). One of the five

identified players showed lowered performance relative to the US normative categories on ImPACT Reaction Time (*Players E*). On the Purdue Pegboard, there was no indication of lowered performances relative to the Purdue normative categories on any of the tasks.

8.3.2 Tackling

Three of the five identified players with suspected concussive injury appeared in the upper region of the tackling data. The tackling data indicated that these three identified players (*Players B, D* and *E*) were involved in more tackling situations than the Rugby Group's averages on Tackles Received and Total Tackles. *Player B,* however, was involved in more tackling situations than the Rugby Group's average in all three of the tackling categories, while *Player C* was only higher than the Rugby Group's average on Tackles Received.

Calculating the averages of Tackles Made of the five players, the players made 73.6% of their tackles *above* the waist and 26.4% *below* the waist (compared with the Rugby Group's 65.65% and 34.35% respectively); and calculating the averages of Tackles Received of the five players, the players received 70.6% of their tackles *above* the waist and 29.4% *below* the waist (compared with the Rugby Group's 69% and 31% respectively).

8.3.3 Final Synthesis

Table 8.21 shows the specific individual player's cognitive vulnerability at each of the assessment intervals. *Players B, C, D* and *E* started the *pre*-season assessment interval with lower and already compromised scores, and more specifically with *Player C* on two of the tests, *Player D* on four of the tests, *Player B* on six of the tests, and *Player* E on seven of the tests. It seems legitimate to assume that exposure to frequent head and body collisions impacts negatively on neurocognitive performance.

Looking at the neurocognitive assessment results in descending order the following tests appeared to be sensitive and discriminatory indicators of cognitive vulnerability in players who received a suspected concussive event during the season (Figure 8.51), as assessed at *pre*-season and additional assessment intervals: ImPACT Visual Motor Speed and Purdue Assembly (depressed in eleven instances), ImPACT Reaction Time (depressed in nine

instances), ImPACT Verbal Memory (depressed in eight instances), ImPACT Visual Memory and Purdue Both (depressed in seven instances). The Purdue Pegboard appeared to be the most sensitive test for revealing cognitive vulnerability in fluctuating performances over the assessment intervals (fluctuations in two instances for Preferred, in three instances for Non Preferred and Both, and in four instances for Assembly).

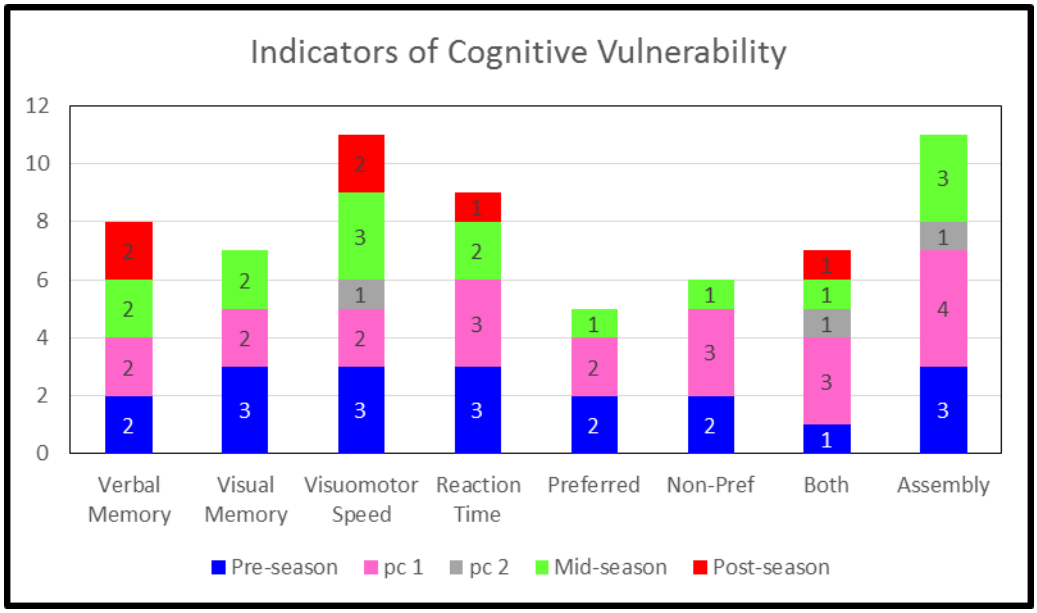
Looking across the seasonal tackling data of the five identified players, with the exception of Player *A*, three of the other players were consistently higher than the Rugby Group averages in Tackles Received and Total Tackles.

Table 8.21 Individual Player Cognitive Vulnerability

	Pre-season	P-c1	P-c2	Mid-season	Post-season
MEMORY					
ImPACT Verbal Memory	C E	A B		C D	D E
ImPACT Visual Memory	B D E	C E		A D	
MOTOR SPEED					
ImPACT VMS	B C D	B E	A	B C E	B D
ImPACT Reaction Time	B D E	A B E		B E	E
Purdue Preferred	B E	B E		D	
Purdue Non-Preferred	B E	B C E		D	
Purdue Both	E	B C E	A	B	A
Purdue Assembly	B D E	B C D E	A	A C E	

Note: Letters of the alphabet refer to each of the five individual players
Visual Motor Speed (VMS)

Figure 8.51 Indicators of Cognitive Vulnerability per test



CHAPTER 9

DISCUSSION

As an introduction to the discussion, the broad aims of this study and the statistical hypotheses are presented. Following this, a discussion of the results are presented in turn for the independent cross-sectional analyses, the dependent prospective analyses, the tackling and correlational analyses, and the individual player analyses. The final section includes an evaluation of the study, followed by the implications of the research outcomes.

9.1 AIMS OF THE STUDY AND STATISTICAL HYPOTHESES

The biomechanical mechanisms of TBI were reviewed in depth in the introductory sections of this thesis (see chapter two). Essentially it was established that a direct or indirect impulsive force to the head, neck or elsewhere on the body may result in a concussive brain injury and may produce alterations at various levels of neurocognitive functioning (Collie et al., 2003; Collins, Lovell & McKeag, 1999; Erlanger et al., 1999). The brain is a relatively multifaceted, and interconnected biological system that is sensitive to mechanical and biochemical injury at multiple levels and in multiple ways as it continues to move even after the skull decelerates rapidly following impact (Withnall et al., 2005). Excessive mechanical forces of linear and rotational head accelerations/decelerations trigger a multi-layered neurometabolic reaction that contribute to overall cerebral vulnerability, traumatic axonal injury and persistent neurocognitive deficits (Giza & Hovda, 2001; Hovda et al., 1999).

There is growing evidence to support the cumulative deleterious neurocognitive effects of repetitive concussive and subconcussive events in contact sports. Whilst some studies report no significant neurocognitive effects between a history of concussion and long-term, persistent neurocognitive effects (Hinton-Bayre & Geffen, 2004; Iverson et al., 2006; Macciocchi et al., 2001), others do report an association between concussion history and long-term neurocognitive effects (Collins, Grindel et al., 1999; Gardner, Shores & Batchelor, 2010; Guskiewicz et al., 2003, 2005; Iverson et al., 2002; Killiam, Cautin & Santucci, 2005;

Moser et al., 2005). Recently, there have been an increased interest in Rugby Union ('rugby' for the purposes of this thesis) into the acute, chronic and cumulative deleterious neurocognitive effects of repeated concussive and subconcussive events (Farace, Ferree, Hollier, Barth & Shaffrey, 2003; Gardner, Shores & Batchelor, 2010; Pettersen & Skelton, 2000; Shuttleworth-Edwards, Border, Reid & Radloff, 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Radloff, Whitefield-Alexander, Smith & Horsman, 2013; Shuttleworth-Edwards, Smith & Radloff, 2008; Shuttleworth-Jordan, Puchert & Balarin, 1993; Thornton, Cox, Whitfield & Fouladi, 2008).

The most prominent neurocognitive deficit following MTBI is the loss of processing speed capacity and includes compromised reaction time, slowed decision-making, impaired motor speed, impaired concentration, impaired memory (Frencham, Fox & Maybery, 2005; Gronwall, 1989, 1987), and typically differentiates MTBI athletes from controls. There is evidence of a positive correlation between lower cognitive performance, increased chronic neurological deficits and possession of the APOE ϵ4 genotype in older players and/or players with a number of years of cumulative exposure to contact sports (Jordan et al., 1997; Kutner, Erlanger, Tsai, Jordan & Relkin 2000; Lishman, 1997). Research based on autopsy data has identified chronic traumatic encephalopathy (CTE) as a pathologically distinct neurodegenerative condition affecting a wide range of individuals, including football players, who have experienced multiple concussions (McKee, Cantu, Nowinski et al., 2009; Omalu, Hamilton, Kamboh, DeKosky & Bailes, 2010). More recently this is confirmed in the literature in a study that suggests an increased risk of neurodegenerative causes of death among retired National Football League players including Alzheimer's Disease, Parkinson disease and amyotrophic lateral sclerosis (ALS) (Lehman, Hein, Baron & Gersic, 2012). An autopsy study by Omalu, Bailes, Hamilton, et al. (2011) reported on pathologic findings of CTE in college-age and professional football players with relatively short playing careers. Small, Kepe et al. (2013) found brain tau deposits in living retired players to be consistent with tau deposition patterns observed in other autopsy studies of CTE.

This research study sought to investigate both the acute and chronic deleterious neurocognitive effects of cumulative and repetitive concussive and subconcussive events of club level rugby players (time frames of within three months and longer than three months,

respectively), that are often below the threshold of obvious symptom presentation. For the amateur adult club level rugby player the potential risks associated with cumulative and repetitive concussive and subconcussive events and neurocognitive effects appear to be largely overlooked since MTBI research has mainly focussed on professional athletes and amateur collegiate or university athletes (Farace et al., 2003; Gardner, Shores & Batchelor, 2010; Hinton-Bayre & Geffen, 2004; Jordan et al., 1997; Kutner et al., 2000; Macciocchi et al., 1996; Matser et al., 1999; McCrory et al., 2000; Pettersen & Skelton, 2000; Shuttleworth-Edwards et al., 2013,2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith & Radloff, 2008; Shuttleworth-Jordan, Puchert & Balarin, 1993; Thornton, Cox, Whitfield & Fouladi, 2008).

To the author's knowledge there are no sport-related MTBI studies (and therefore no rugby studies) that have (i) investigated the cumulative neurocognitive effects of frequent head and body collisions using both computerized and traditional neurocognitive assessment measures in combination with video notational analyses in order to identify MTBI and to monitor different aspects of tackling, (ii) combined a *mid*-season assessment interval with a *pre*- and *post*-season assessment interval, and (iii) included a supplementary series of case study analyses with the traditional comparative group based analyses of rugby players versus non-contact sports controls.

More specifically, therefore, in terms of the above broad methodological parameters, it was decided that the current study would investigate the acute and chronic neurocognitive effects of repetitive effects of concussive and subconcussive events of amateur adult club level rugby players as indicated at *pre*-season, as a history of MTBIs have been associated with lowered *pre*-season baseline performance on visual motor processing speed (Collins, Grindel et al., 1999). Further, to determine whether there was evidence of a combination of persistent acute or sub-acute neurocognitive effects because of frequent and continuous exposure to head and body collisions, *mid*- and *post*-season assessment intervals were included. Due to the possibility of reaction times normalizing during long follow-up, as suggested in earlier studies of Van Zomeren & Deelman (1978), a *mid*-season assessment was included in order to determine what happens between the two more commonly applied assessment intervals. This enabled before, during and after season appraisals of club rugby players to assess the

extent of cumulative neurocognitive deficits in association with the level of participation in the sport.

The present study investigated club level rugby players in comparison with demographically equivalent non-contact sports controls over one sport season (approximately seven months for both groups). The reasoning behind choosing adult club level rugby players as the participant group was that they have been exposed to the effects of multiple concussive and subconcussive events over years of playing the contact game, and possibly do not have all the medical protective factors in place for the identification and follow up of concussive brain injury that may apply at a professional or university/collegiate level. Moreover, the adult level of play is generally more intense than at youth levels, which further supports the fact that as a whole this amateur adult club level group may be particularly vulnerable to residual and cumulative concussive effects.

For the purposes of this study, and over a period of one rugby season, amateur adult club level rugby players were targeted and included in a rugby group, and a mixture of amateur cricket and cycling athletes were targeted and included in a non-contact sports control group. Data were collected from an initial sample of club level rugby players and non-contact sportsmen at the *pre*-season assessment interval (n = 33 and n = 32, respectively). Following a reduction in the sample available for analysis at the *mid*-season assessment interval (due to change of clubs, drafts into provincial teams or work related demands) the sample of club level rugby players versus non-contact sportsmen were both reduced to n = 24. Following the *mid*-season assessment interval the sample was further reduced for similar reasons (due to change of clubs, drafts into provincial teams or work related demands), resulting in a final rugby sample of n = 20 (designated the Rugby Group) and a non-contact sports control sample of n = 22 (designated the Non-Contact Sports Control Group).

These two comparative groups (Rugby and Non-Contact Sports Controls) were then subjected to a series of tests to investigate both the acute and chronic deleterious effects of cumulative and repetitive concussive and subconcussive events of club level rugby players and utilized a widely used and renowned neurocognitive computerized measure, ImPACT (ImPACT, 2004), and a neurocognitive measure of hand-motor speed, the Purdue Pegboard

(Purdue Pegboard, 2002; Lezak et al., 2004). The neurocognitive functions derived from these tests were grouped into two broad domains of functioning, viz., (i) Memory, including ImPACT Verbal Memory and ImPACT Visual Memory, and (ii) Motor Speed, including ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both and Purdue Assembly. The neurocognitive measures (ImPACT and the Purdue Pegboard) were then subjected to independent cross-sectional and dependent prospective analyses and it was considered appropriate in the statistical analyses of the results to make a Bonferroni's adjustment to the *p*-value by a factor of two functional modalities, thereby guarding against Type I error (indicating significance when there is no real difference), but not overly correcting towards stringency via an adjustment for the entire number of subtests administered, thereby protecting against the risk of Type II error (failure to identify true differences). In a study of MTBI where effects may be subtle, albeit not necessarily without relevant clinical implications, it is considered appropriate to apply cautionary procedures against missing the presence of impairment where it is present (Brandt, 2007; Demakis, 2006; Frencham, Fox & Maybery, 2005).

Throughout the season the study incorporated tackling analyses of the rugby playing group (*n* = 20) derived from video notational measures to establish the frequency of tackling. These data were descriptively analyzed and subjected to a series of tentative and speculative exploratory Spearman's correlational analyses, in the knowledge that due to the small sample size that these were exploratory.

From a methodological perspective possible sampling limitations were addressed with the inclusion of a matched control group (the Non-Contact Sports Control Group), with the same age and the closest possible approximation to years of education and estimated IQ as the rugby playing group (the Rugby Group), in order to gauge variation in performance across the *pre-*, *mid-* and *post*-season assessment intervals. There were no significant differences between the means for the Rugby and the Non-Contact Sports Control groups for the variables of age, years of education, and estimated IQ, suggesting that the Rugby and the Non-Contact Sports Control groups were equivalent for age, years of education, and estimated IQ. The age range was 21 to 32 years, the years of education for the sample ranged from 12 to 16 years and the estimated IQ score for the sample ranged from 90 to 118 for both

groups. For the rugby versus the control group respectively, the average age was 26.4 and 25.8 respectively; the average years of education was 13.55 and 13.148 respectively; and the average estimated IQ was 103.95 and 107.18 respectively (p = > .05 in all instances, see table 6.1, page 99). From these descriptive data it is evident that this was a young adult population and was made up of individuals of at least average intelligence.

In contrast to the statistical comparisons revealing that the comparative groups were equivalent for age, level of education and estimated IQ, the concussive history differed significantly between the Rugby Group and the Non-Contact Sport Control Group. The Rugby Group obtained significantly more concussions than the Non-Contact Sports Control Group, thereby, strongly confirming the *non-equivalence* of these two comparative groups on this variable. For the Rugby Group versus the Non-Contact Sports Control Group, the reported concussions was 1.85 and 0.13 respectively (p = 0.005, see Table 6.1, page 99). Therefore, it can be proposed that any deleterious neurocognitive effects in evidence for the Rugby Group on the neurocognitive measures (ImPACT and Purdue Pegboard), as demonstrated on the independent cross-sectional and dependent prospective statistical analyses, could not readily be accounted for on the basis of intergroup differences in age, education and IQ. Rather such effects could be more readily attributed to the repetitive long-term exposure of the club level rugby players to cumulative and sub-concussive effects in association with years of participation in contact sport.

Further in support of the above supposition was the outcome of the video analyses of tackling incidences over the rugby season, which was based on a *within* group investigation pertaining to the Rugby Group only. Clearly it was not possible to do a similar comparative analysis for the Non-contact Sports Control Group in that players from that group would not be engaged in sports that formally involve tackling procedures. Therefore, the issue of equivalence of the Rugby Group and Non-Contact Sports Control Group for age, education and IQ was not of statistical relevance to this aspect of the study. Conceptually, however, the clear demonstration of the Rugby Group having a significantly higher incidence of concussions than a group of non-contact sportsmen warrants further investigation of the total rugby group's concussion data in relation to obtained tackling data, due to a proposed association between these two factors, and that the incidence of tackling within the Rugby Group implies

the presence of multiple possible head jarring incidents in association with tackling that would not have been a characteristic of the control group.

Perusal of the tackling data reveal that on average rugby players in the Rugby Group were involved in a total of 103.45 tackles over one rugby season and on average they made 60.7 tackles and received 42.75 tackles. It would be feasible, taking the average club level rugby player and multiplying the average of tackles over one rugby season by a definitely underestimated average of ten years of exposure to the game, and this translates into the somewhat alarming figure of more than a thousand tackles per individual, excluding any contact practice sessions, each time placing the individual at risk for a head jarring incident with associated risk of cumulative, deleterious neuropathological consequences.

In addition to incorporating tackle averages, the study further extrapolated that the head-on type of tackle is the type of tackle predominately received by rugby players and this reiterates previous research indicating that tackling and being tackled head-on are the most common mechanisms of injury (Garraway, Lee, Macleod, Telfer, Deary & Murray, 1999; Kemp, Hudson, Brooks & Fuller, 2008; Wilson, Quarrie, Milburn & Chalmers, 1999). Furthermore the study revealed that tackling and being tackled above the waist line contributes to higher numbers than those made and received below the waist line. Previous studies also indicated that most injuries are due to high or above the waistline tackles rarely with loss of consciousness, and/or amnesia. (Hinton-Bayre, Geffen & Friis, 2004; Quarrie & Hopkins, 2008).

In short, these tackling data derived on the basis of the within Rugby Group video analyses of all games across the rugby season, provide compelling further evidence in addition to the concussion data, that the Rugby Group differs from the Non-Contact Sports Control Group in terms of possible injury to the brain with associated neurocognitive effects. Therefore it is proposed that any deleterious neurocognitive effects established on the basis of this research are more readily attributed to the repetitive long-term exposure of the club level rugby players to cumulative and sub-concussive effects in association with years of participation in contact sport than other potentially influential variables such as age, education and IQ that are considered to have been well controlled in this study.

Finally, the study combined the abovementioned cross-sectional and prospective paradigm with individual case-based results to investigate the cumulative neurocognitive effects of repetitive concussive and subconcussive events in club level rugby during the course of one rugby season. Supplementary to this aspect of the study, tackling analyses were incorporated into the Rugby Group and case-based analyses in order to take into account the impact of tackling on neurocognitive performance and the individualized effect thereof on said neurocognitive performance. This is a novel aspect to research methodology in the study of neuropsychological effects in rugby, being the first time this has ever been formally done in a neurocognitive study of this type to the author's knowledge. Specifically, the analyses utilised descriptive statistical analyses, and a computerized notational system to assist in the external identification of the frequency of tackles and its relationship to neurocognitive outcome. Specifically, in the case-based analyses, comparisons were made with the individuals' own *pre*-season baseline and the average scores of the Non-Contact Sports Control Group derived from the group based aspect of the study. This was done with a view to understanding the neurocognitive assessment outcome of sports-related MTBI and the relative sensitivity of various neurocognitive assessment measures to this type of injury and the possible influence of tackling incidence on individual players. This combined set of investigative parameters, group and case-based, were considered to more effectively contribute to the understanding of sports-related MTBI, and provided for a much more powerful approach to the study of neurocognitive effects across a rugby season than only one of these investigative parameters alone.

Broadly having relevance to all these complimentary aspects of this investigation, they were all aimed at identifying the acute and residual neurocognitive outcome of sports-related MTBI. It was possible to adopt an overarching interpretive framework in terms of The Brain Reserve Capacity Theory as explicated by Satz (1993), and further elaborated on by Stern (2006, 2003) for the present study. This theory proposes the concept that individuals uniquely possess the capability to withstand and compensate for mild, traumatically induced neuronal loss. When an individual's cognitive reserve is depleted beyond a certain threshold, such as due to concussive and subconcussive events, certain neurocognitive deficits emerge (Jordan, 1997; Randolph, 2001; Satz, 1993; Stern, 2006, 2003; Weight, 1998).

In light of these suppositions in respect of the cognitive reserve conceptual framework, a number of statistical and empirical hypotheses were formulated for the four different aspects of the study as follows:

(i) In respect of the *independent cross-sectional analyses*, utilising a series of independent t-test analyses, it was hypothesized that there would be significant differences between the mean scores of the Rugby Group relative to the Non-Contact Sports Control Group on the neurocognitive measures at each of the assessment intervals (*pre-*, *mid-*, and *post*-season), in the direction of the Rugby Group performing worse than the Non-Contact Sports Control Group, due to postulated exposure to the cumulative effects of head and body collisions during years of rugby participation, including concussive and sub-concussive events during participation in one rugby season.

(ii) In respect of the *dependent prospective analyses*, utilising a series of dependent t-test analyses, it was hypothesized that there would be *either* significant differences in the mean scores on the *neurocognitive measures* for the Rugby Group at the *pre-* versus *mid-* versus *post*-season assessment intervals in the direction of worsening performance for the Rugby Group in contrast to no deterioration in scores for the Non-Contact Sports Control Group, due to the deleterious neuropsychological effects of unreported concussive and sub-concussive events sustained by the rugby players during participation in one rugby season, on top of the long-term effects of concussive and subconcussive events sustained over years of playing rugby, *or* that for the same reason there would be no significant differences in the mean scores on the neurocognitive measures for the Rugby Group at *pre-* versus *mid-* versus *post*-season assessment intervals, in contrast to significant improvement for the Non-Contact Sports Control Group in the mean scores on the neurocognitive measures.

(iii) In respect of the *correlational analyses*, utilising a series of exploratory Spearman's correlational analyses it was hypothesized that due to unreported concussive and

subconcussive events that *more* tackles would be associated with *poorer* neurocognitive performance for the Rugby Group and for the identified individual players due to a long history of participation in contact sport, including those sustained in one rugby season. Further, utilising a series of exploratory Spearman's correlational analyses it was hypothesized that *more* concussions would be associated with *poorer* neurocognitive performance across the three assessment intervals (*pre-*, *mid-* and *post-*season). It was also hypothesized that on the basis of a series of exploratory Spearman's correlational analyses that *more* concussions would be associated with *higher* number of tackles, in all three tackling categories (i) Tackles Made, (ii) Tackles Received, and (iii) Total Tackles.

(iv) Finally, in respect of the *individual player analyses*, utilising descriptive comparisons of each individual player's neurocognitive data from the *pre-*, *mid-* and *post-* season assessments in relation to normative data, their *post-*concussional follow-up data, as well as their tackling data derived from the computerized notational system, it was hypothesized that the outcome would descriptively be comparable with adverse neurocognitive effects identified on the group analyses of the present study and/or that have been reported in the literature for adult athletes who have sustained concussive and subconcussive events on top of a long history of participation in contact sports.

9.2 INTERPRETATION OF RESULTS

This section discusses the results for the (i) Independent Cross-sectional Analyses, (ii) Dependent Prospective Analyses; (iii) Correlational Analyses, and (iv) Individual Player Analyses. As indicated above, the neurocognitive functions targeted in this research were grouped into two broad domains of functioning namely Memory (including ImPACT Verbal Memory and ImPACT Visual Memory), and Motor Speed (including ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both and Purdue Assembly).

9.2.1 Independent Cross-sectional Analyses

The Memory functional modality, made up of ImPACT Verbal Memory and ImPACT Visual Memory, in the independent cross-sectional analyses at the three assessment intervals (*pre-*, *mid-* and *post*-season) revealed no significant results. Overall this functional modality did not discriminate between the Rugby Group and the Non-Contact Sports Control Group. However, in contrast the Motor Speed functional modality, made up of ImPACT Visual Motor Speed, ImPACT Reaction Time, Purdue Preferred, Purdue Non-Preferred, Purdue Both and Purdue Assembly, at the three assessment intervals (*pre-*, *mid-* and *post*-season) revealed most of the tasks being significant in the direction of poorer performance for the Rugby Group, compared with the Non-Contact Sports Control Group. More specifically, the ImPACT Visual Motor Speed and ImPACT Reaction Time were consistently significantly worse for the Rugby group at the three assessment intervals (*pre-*, *mid-* and *post*-season). Overall ImPACT appeared to be more discriminating than the Purdue Pegboard. The findings on the Purdue Pegboard were still in the direction of poorer performance for the Rugby Group, compared with the Non-Contact Sports Control Group, but lacked significance at the *post*-season assessment interval.

The finding that the Rugby Group performed poorer than the Non-Contact Sports Control Group particularly in the Motor Speed modality (incorporating tests of processing speed and hand-motor speed) is consistent with the findings of MTBI studies undertaken with boxing, soccer, American football, Rugby League and Rugby (Barth et al., 1989; Collie et al., 2006; Collins, Field et al., 2003; Cremona-Meteyard & Geffen, 1994; Downs & Abwender, 2002; Gardner, Shores & Batchelor, 2010; Hinton-Bayre et al., 1997; Iverson et al., 2004; Jordan et al., 1997; Maddocks & Saling, 1996; Moriarity et al., 2004; Rawdin et al., 2003; Shuttleworth-Edwards et al., 2013, 2004; Stewart et al., 1994; Warden et al., 2001; Witol & Webbe, 1994; Wilberger, 1993). In terms of the outcome of this study the ImPACT Visual Motor Speed and ImPACT Reaction Time tasks have been consistently sensitive to neurocognitive effects of concussive and subconcussive events across one rugby season even with the inclusion of the additional *mid*-season assessment interval. In respect of ImPACT Reaction Time, this finding supports other MTBI research in that reaction time represents

one of the cognitive domains that has consistently shown to be sensitive to the effects of MTBI (Levin et al., 1987; O'Connor & Burns, 2003).

Finally as indicated above, the ImPACT tests were more consistently discriminating between the two comparative groups at all three assessment intervals than the Purdue Pegboard, however this does not suggest that the Purdue Pegboard does not lack sensitivity per se, as it did differentiate between the *pre*- and *mid*-season assessment intervals, and is consistent with a previous study where it was used and did show up sensitivity (Shuttleworth-Jordan et al., 1993). The Purdue Pegboard appears to lose sensitivity on repeated trials by virtue of not having randomized versions like on the ImPACT test, such that outcome is confounded by the influence of practice effects to be discussed in more detail under the dependent prospective analyses.

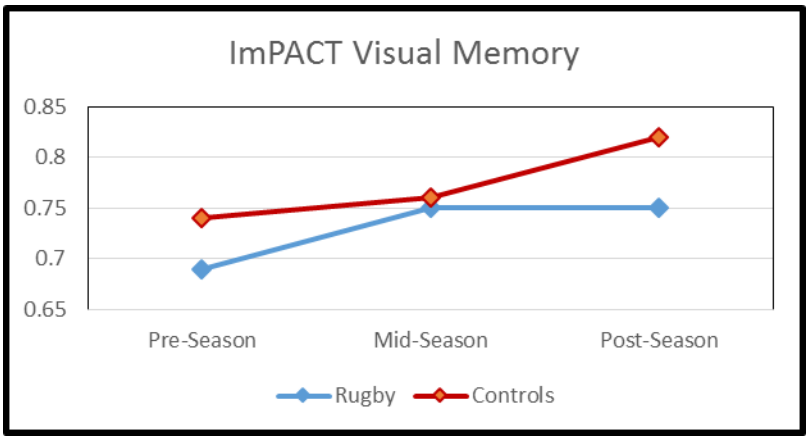
9.2.2 Dependent Prospective Analyses

Firstly, in terms of the Memory functional modality, the dependent t-test comparisons of all measures between the Rugby Group *pre*- versus *mid*- versus *post*-season and the Non-Contact Sports Control Group *pre*- versus *mid*- versus *post*-season revealed that the results for the Rugby Group replicated the results for the Non-Contact Sports Control Group, in respect of a strong trend in the direction of both these groups performing better at *post*-season. The ImPACT Visual Memory task was the only test where the Rugby Group failed to improve with practice by the end of the season in contrast to the Non-Contact Sports Control Group which did improve at the *post*-season assessment interval (Figure 9.1). A likely explanation for this dissociation is that it is the most challenging task in terms of a learning effect over time in terms of its non-geometric arbitrary designs, and thereby serving to discriminate between the two groups.

This finding can be considered clinically meaningful, as a decrement in learning and mental agility is implicated and may suggest a deterioration in neurocognitive processes (Duff, 2012; Duff et al., 2007). The finding accords with a growing body of studies on sports concussion that have revealed similar effects, with lack of learning ability in the contact sports players than controls. Maddocks & Saling (1996) demonstrated significant improvements for

controls when compared to concussed American football players. Similarly, amongst rugby players and non-contact sports controls a significant practice effect was demonstrated for the control group on amongst others, the Purdue Pegboard that was not as strongly in evidence for the rugby group (Shuttleworth-Jordan et al., 1993). In addition, another study on university rugby players revealed a relative absence of practice effects for school and university rugby players versus non-contact sports controls on ImPACT Visual Motor speed after a long test-retest interval of around seven to eight months (Shuttleworth-Edwards et al., 2013).

Figure 9.1 Rugby (Pre- vs Mid- vs Post-season) versus Non-Contact Sports Controls (Pre- vs Mid- vs Post-season) for ImPACT Visual Memory



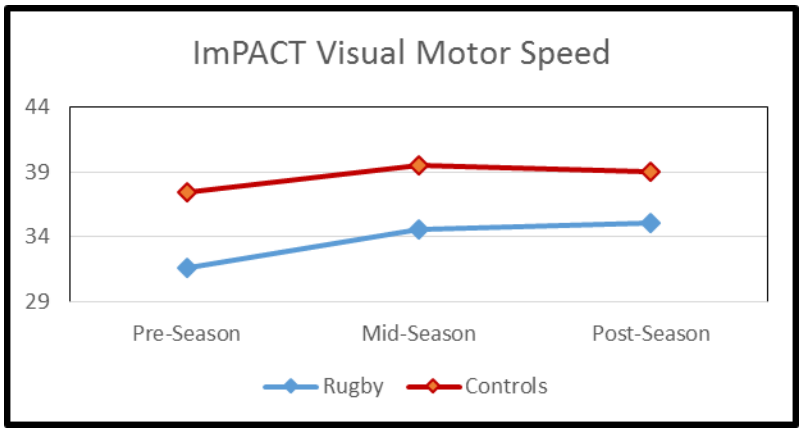
Secondly, in terms of the Motor Speed functional modality, including both the ImPACT and Purdue measures, the dependent t-test comparisons for the Rugby Group and Non-Contact Sports Control Group at *pre*- versus *mid*- versus *post*-season, revealed some broadly similar results for the Rugby Group and the Non-Contact Sports Control Group, in that there was a consistent strong trend in the direction of both the Rugby Group and Non-Contact Sports Control Group performing better at the *post*-season interval compared with the *pre*-season level. However, on closer, more intricate analyses and taking performance across all three test intervals into account, there were some indications of differential effects between the two groups as follows.

On the ImPACT Visual Motor Speed, both groups improved at *mid*-season, but this improvement ceased to occur for the Non-Contact Sports Control Group at *post*-season.

Rather for the Non-Contact Sports Control Group there was a slight dip in performance at *post*-season, whereas in contrast the Rugby Group continued to show extremely marginal improvement (Figure 9.2). As indicated earlier on the basis of the cross-sectional analyses it was evident that the scores of the Non-Contact Sports Control Group were much higher than the Rugby Group. Descriptively at *pre*-season it can be observed that in comparison to the Rugby Group which starts significantly lower (according to the ImPACT normative categories in the Low Average range 28.4-32.4), the score of the Non-Contact Sports Control Group is much higher and closer to the ceiling performance level of that test (according to the ImPACT normative categories in the Average range 32.5-42.0). The Non-Contact Sports Control Group goes on even more closely to approximate the ceiling level of performance at the *mid*-season interval. In contrast, none of the Rugby Group scores approximating the ceiling level of performance such as occurred for the Non-Contact Sports Control Group.

Therefore, it appears that in contrast to the Non-Contact Sports Control Group who started off at *pre*-season performing at a high level approximating the ceiling level, there was much more room for the Rugby Group to continue with improvement, in that at *pre*-season and *mid*-season they were still performing well below the ceiling performance level of that test. These subtle observations of differential performance regarding learning capacity for the Rugby Group over the three assessment intervals, compared with the Non-Contact Sports Control Group, in light of their already depressed scores at the *pre*-season interval, can be seen to have provided a potentially critical additional diagnostic marker of vulnerability in the Rugby Group. In that there was no significant interaction effect, this is nevertheless a subtle indication of differential performance between the two groups that would not have been demonstrated without a third *mid*-season assessment interval that was a unique contribution of the present study.

Figure 9.2 Rugby (Pre- vs Mid- vs Post-season) versus Non-Contact Sports Controls (Pre- vs Mid- vs Post-season) for ImPACT Visual Motor Speed

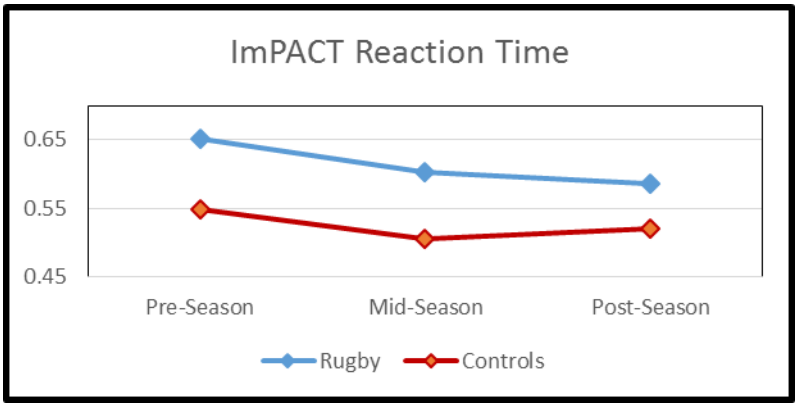


Importantly, the above observations in respect of the pattern of performance on the ImPACT Visual Motor Speed test are compellingly strengthened by the presence of significant interaction effects in evidence for ImPACT Reaction Time (Figure 9.3) and Purdue Both (Figure 9.4), where in both instances the Rugby Group does start significantly poorer at *pre*-season (as indicated on the cross-sectional analyses that implies a measure of neurocognitive vulnerability) than the Non-Contact Sports Control Group, but improves more by *post*-season than the Non-Contact Sports Control Group. (With regard to Figures 9.3 and 9.4, it is important to be aware that these are indeed broadly commensurate findings, not to be confused by the apparent opposite direction of the gradients by virtue of the fact that a *lower* Reaction Time score indicates better performance whereas a *higher* Purdue score indicates better performance). Again this outcome can probably be explained by the fact that the Non-Contact Sports Control Group performed closer to their ceiling and with the inclusion of the third assessment interval there is not much room for improvement.

Important to be noted here, is that had only *pre*- and *post*-season testing been completed such as has been done in most of the prior studies on sports concussion, rather than the three assessment intervals that were completed in the present study, these significant interaction effects serving to highlight signs of additional neurocognitive vulnerability in the Rugby Group compared with the Non-Contact Sports Control Group would have been missed. It is relevant that the Non-Contact Sports Control Group were already performing so close to the ceiling level on these three motor tests (ImPACT Visual Motor Speed, ImPACT Reaction

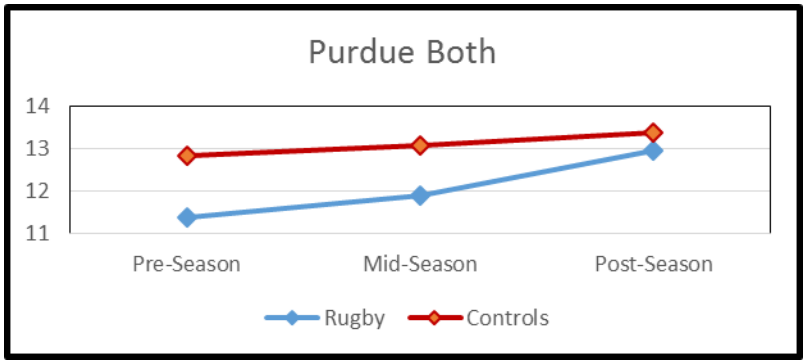
Time and Purdue Both), that they improve on a single retest and then plateau out, whereas the Rugby Group perform so poorly at first that they do not show the same plateau effect on repeat testing as soon as do the Non-Contact Sports Control Group.

Figure 9.3 Rugby (Pre- vs Mid- vs Post-season) versus Non-Contact Sports Controls (Pre- vs Mid- vs Post-season) for ImPACT Reaction Time Composite Score



Note: A lower Reaction Time score indicates better performance

Figure 9.4 Rugby (Pre- vs Mid- vs Post-season) versus Non-Contact Sports Controls (Pre- vs Mid- vs Post-season) for Purdue Both Hands



Note: A higher Purdue score indicates better performance

The overall observed pattern of greater neurocognitive vulnerability for the Rugby Group relative to the Non-Contact Sports Control Group based on their differential comparative performances in learning on ImPACT Visual Motor Speed, ImPACT Reaction Time composite scores and the Purdue Both, that takes place across the three assessment intervals, adds to an accumulating body of research that demonstrates the diagnostic utility of differential effects in neuropathological compromised groups versus normal controls (Duff et al., 2007), as well as studies on sports concussion that have revealed similar effects (Barth et

al., 1989; Shuttleworth-Edwards et al., 2013; Shuttleworth-Jordan et al., 1993). It is understandable in terms of the strongly differentiating feature between the two groups of exposure to repeated concussive and subconcussive events exclusively in the Rugby Group over a long rugby-playing career, including the more recent season, although it is not possible on the basis of the present research to know how much contribution applies to past or more recent concussive events.

9.2.3 Correlational Analyses

A series of exploratory Spearman's correlations were run on *concussions* reported on the biographical questionnaire for the Total Group and the Rugby Group alone in relation to the neurocognitive data at the *pre-*, *mid-* and *post*-season assessment intervals. In terms of these correlations there were significant outcome for Purdue Preferred at *mid*-season, and Purdue Non-Preferred was significant at *mid-* and *post*-season in the hypothesized direction of *more* concussions being associated with *poorer* neurocognitive performance. In addition there were consistent trends in the hypothesized direction for all the other tests except ImPACT Visual Memory and Purdue Assembly where there were inconsistent trends across the three test intervals. However when the same exploratory correlational analysis were run for the Rugby Group alone (i.e., a much reduced sample number compared with the Total Group), there were no significant results and only one consistent trend which was for ImPACT Verbal Memory in the hypothesized direction.

Another series of exploratory Spearman's correlations were run on the Rugby Group concussion data and tackling data (Tackles Made, Tackles Received and Total Tackles). While not significant, these correlations were consistently in the hypothesized direction of *more* concussions being associated with a *higher* number of tackles. Further, in respect of the tackling data, a series of exploratory Spearman's correlational analyses were run for the Rugby Group in the three tackling categories (Tackles Made, Tackles Received and Total Tackles) in relation to the neurocognitive measures (ImPACT and Purdue Pegboard) at each of the three assessment intervals (*pre-*, *mid-* and *post*-season). Overall for all three tackling categories there were no significant results. In terms of consistent trends Purdue Pegboard, and more specifically Purdue Non-Preferred and Purdue Both, were in the hypothesized

direction of *more* concussions being associated with *poorer* neurocognitive performance, albeit lacking significance. There were two consistent trends in the opposite of the hypothesized direction of more tackles being associated with better neurocognitive performance, viz. ImPACT Visual Motor Speed and ImPACT Reaction Time.

Taking all these correlational analyses into consideration, while highly tentative due to the lack of substantive significant outcome, there is a reasonably compelling indication of outcome in the hypothesized direction of *more* concussions and *higher* tackling being associated with *poorer* neurocognitive outcome, particularly on the Purdue Pegboard non-Assembly tasks, and notably there was a highly consistent indication of an association between a *higher* number of concussions and *more* tackles. These correlations were run on a purely exploratory basis, in the knowledge that small sample numbers on such correlational analyses are at risk of Type II error, i.e., failing to demonstrate significance where it exists. For the same reason, it was decided to report on Spearman's rather than the more widely used Pearson's correlation coefficients because Spearman's is a nonparametric rank statistic that measures the strength of a monotone association between two variables, and is used when the researcher knows nothing about the parameters of the variable of interest in the population (hence the name *nonparametric*).

While highly tentative, these indications may be considered to be of important heuristic relevance in the sports concussion literature, and worthy of further investigation. Studies on tackling in rugby to date, have been focused on the biomechanics of concussive injury, player position, injury type and injury site and the causative link with a higher risk of concussive injury (Gabbett et al., 2011; Gabbett, Jenkins & Abernethy, 2011; Gissone et al., 1997; Guskiewicz & Mihalik, 2011; King et al., 2011; Sharp et al., 2001). To the author's knowledge there are no other studies to date that have monitored the incidence of tackling in rugby in itself, or attempted to link such tackling data with the incidence of concussion and/ or neurocognitive effects. The only studies of a somewhat comparable nature are looking at the measured quantity-response relationship on cognitive functioning were soccer studies incorporated the frequency of headers and the number of soccer-related concussions and found lowered neurocognitive performance on focused attention and visual motor processing tasks (Matser et al., 2001; Webbe & Ochs, 2003).

9.2.4 Individual Player Analyses

In respect of the five individual player analyses these will be discussed in terms of the following aspects: (i) the individual players' concussion histories; (ii) the individual players' tackling data; (iii) the individual players' education and IQ levels; and (iv) the individual players' neurocognitive assessment data.

In terms of the *concussion histories* of the five individual players it was not possible to see any substantive differences in neurocognitive outcome between those individuals who reported a prior history of two or more diagnosed concussions and those who reported no prior history of diagnosed concussions. There were trends however, predominantly in the direction of the individuals with two or more concussive events tending to perform *worse* at the *pre*-season neurocognitive assessment interval. A history of MTBI has been associated with lowered *pre*-season baseline performance on visual motor processing speed among American football players (Collins, Grindel et al., 1999). Players with a history of more than one concussion were associated with long-term deficits in visual motor processing speed, reaction time and executive functioning with a trend towards significant lower memory scores (Collins, Grindel, Lovell, Dede, Moser, Phalin, Nogle et al., 1999; Iverson et al., 2002a; Maddocks & Saling, 1996). Rugby players reporting three or more prior concussions performed more *poorly* on *pre*-season neurocognitive assessments and confirms research done by Iverson, Echemendia, LaMarre, Brooks & Gaetz (2012). Reviews of Shuttleworth-Edwards & Whitefield, (2007) also indicated the presence of a learning disability combined with a history of two or more concussions lead to poorer performance on tests of executive functioning and mental processing speed.

In terms of individual players' *tackling data*, these were compared with the Rugby Group means for each tackling category (Tackles Made, Tackles Received and Total Tackles). Looking across the seasonal tackling data of the five individual players, three of the five players were consistently higher than the Rugby Group averages in Tackles Received and Total Tackles, and one player was higher than the Rugby Group averages in all three tackling categories. This comparison was done with the rationale that players tending to be involved

in a higher number of tackles due to their style and position of play, might also be those players who would be likely to sustain subconcussive and concussive events during another rugby season and perform more poorly on neurocognitive measures.

It was evident that *Player B*'s performance at especially the *pre*-season assessment interval, with a consistently higher than the rugby team's average total tackling count over the season, denoted "impaired" performance on ImPACT Reaction Time, "borderline" performance on ImPACT Visuomotor Speed, "low average" performance on Purdue Non-Preferred and "poor" performance on Purdue Preferred and Purdue Assembly. Another player, *Player E*'s performance, also with a considerably higher than the rugby team's averages on Tackles Received and Total Tackles, denoted "borderline" performance on ImPACT Visual Memory, ImPACT Reaction Time; "low average" performance on ImPACT Verbal Memory and Purdue Both; and "poor" performance on Purdue Preferred, Purdue Non-Preferred, and Purdue Assembly. This observation from the small cohort of individual analyses is consistent with the supposition that rugby players involved in more tackles may be incurring a higher number of concussive brain injury incidents with associated deleterious neurocognitive sequelae. Therefore, high tackling statistics might reflect a long term propensity for much tackling, head jarring and associated concussive and subconcussive injury, thereby making these players more vulnerable to neurocognitive decline. On the basis of the individual case analyses described here, it is being proposed that tackling *may* be a critical differentiating factor, by virtue of being associated with increased risk of neuropathological vulnerability in the form of cumulative concussive injury. While this is a highly tentative observation derived from the combined tackling and neurocognitive data for a limited number of case study analyses only, it can once again, as with the correlational analyses be considered to have important heuristic value worthy of further research.

In terms of the *education levels* of the five individual players, there were differences between the players. For education, none of the players had less than 12 years of education, but only two had some tertiary education, one with a diploma and one with a degree. However, there did not seem to be any clear links between those with less years of education and worse performance on the neurocognitive tests. This might be because the range was too small in

that there was no person with a lower level of education than the school leaving Grade 12 level.

In terms of *the estimated IQ levels*, two of the five players were of above average estimated intelligence, and three of the five players were of average estimated IQ. In contrast to the outcome of the possible influence of level of education, it was evident on assessment results that one of the players with estimated *above* average intelligence, had scores denoting a remarkably superior performance on most of the tests at the *pre*-season assessment interval. Even though he showed a relative lowering at the post-concussion follow up on most of the scores he mainly remained in the above average range. Across all the test occasions this player retained his relatively superior performance compared with the other players, and in terms of cognitive reserve theory it would appear that his relatively high initial level of intelligence was protecting him from excessive effects of cumulative brain injury at this stage of his sporting career. It has been repeatedly demonstrated in the literature that a higher level of premorbid intellectual functioning may preserve functional capacity and may compensate for cognitive inefficiency regardless of injury severity, may decrease vulnerability to cognitive deficits and may lead to improved post-injury functioning and recovery (Adams, Parsons, Culbertson & Nixon, 1996; Coffey, Saxton, Ratcliff, Bryan & Lucke, 1999; Kesler, Adams, Blasey & Bigler, 2003; Lezak et al., 2004; Mortimer, 1997; Mortimer & Graves, 1993; Reitan & Wolfson, 1999). Another possible protective factor for this particular player of note, was the fact that he was observed to have lower tackling averages than the overall rugby group (a total of 84 tackles compared with the Rugby Group average of 103.45).

In contrast to the generally superior performance on testing of this player with estimated above average IQ, the other player with estimated above average IQ did not have that kind of generally superior test profile across the test series. Rather, he performed on a relatively average level, much like the other players who only had average estimated IQs. The difference in outcome between these two players, both starting with above average initial IQ, might be explicable in terms of cognitive reserve theory. In terms of the concepts of cognitive and brain reserve theories (Stern, 2006, 2003, and Satz, 1993, respectively), individuals who evidence higher than average IQ scores can be assumed to have higher cognitive reserve because of superior cognitive networks, and are more likely to process

tasks more effectively before demonstrating functional deficits (Stern, 2006, 2003), but not in instances where other vulnerability factors might be at play (Satz, 1993). Vulnerability factors of relevance that potentially differentiated these two individuals are firstly their age (23 and 27 years respectively), in that the second mentioned player was exposed to at least four more years of exposure to tackling maneuvers (at least eight and twelve years respectively). Secondly, despite the fact that both these players received tackles more than the Rugby Group's average (52 and 53 respectively), the second player was exposed to significantly more tackles above the waist (i.e., 72% of the tackles) and this reiterates findings that most injuries are due to tackles above the waistline (Hinton-Bayre, Geffen & Friis, 2004; Quarrie & Hopkins, 2008). Tentatively it might be posed that these differential vulnerability factors between the two players with initial above average IQ, may have cumulatively contributed to the first player being able better to compensate when sustaining a concussive insult than the second player. However, other additional or alternative unidentified vulnerability factors may also have come into play, such as the specific nature and locus of the injury, etc., and other subtle differentiating characteristics between the two players.

In terms of the *neurocognitive assessment results* of the five individual players the following was observed. Four of the five players' own *pre*-season baseline scores revealed lowered performance relative to the US normative categories on most of the neurocognitive measures (*Players B, C, D* and *E* started the *pre*-season assessment interval with lower and already compromised scores, and more specifically with *Player C* on two of the tests, *Player D* on four of the tests, *Player B* on six of the tests, and *Player* E on seven of the tests), thereby implicating the presence of pre-existing neurocognitive vulnerability due to concussive and subconcussive events already at the beginning of the season due to many years of prior participation in the sport. These four players were involved in rugby playing careers ranging from 13 to 18 years. This statistic translates into a possible alarming range of 1106 to 2160 number of tackles over their rugby playing careers based on the specific tackling data for each one of these players as calculated from the video analyses from the present study (*excluding* any contact practice sessions during the season), multiplied by the number of years of participation in the game. Conceptually these observations, based on the descriptive tackling data in conjunction with number of years participating in rugby, provide quite a

compelling measure of corroboration that persistent neurocognitive vulnerability may be expected by the time a rugby player reaches an adult club level of play in association with repetitive high velocity tackling maneuvers.

An examination of the overall neurocognitive assessment results for the individual case-based analyses, revealed that the most sensitive and discriminatory indicators of lowered performance in players were tests of motor speed and specifically the ImPACT Visual Motor Speed, ImPACT Reaction Time and the Purdue Assembly tests (see Table 8.21 and Figure 8.51, pages 226 and 227). This indication from the individual analyses is broadly commensurate with the neurocognitive results found on the independent and dependent comparative group analyses in the current study, where tests of motor speed and specifically ImPACT Visual Motor Speed and ImPACT Reaction Time being consistently significantly depressed for the Rugby Group relative to the Non-Contact Sports controls across all the assessment intervals (*pre-*, *mid-*, and *post*-season). Generally the finding ties up with literature indicating that MTBI typically leads to impairments in processing speed and reaction time (Barth et al., 1989; Eckner, Kutcher, Broglio & Richardson, 2013; Lezak et al., 2004; Lovell, Collins, Iverson, Johnston & Bradley, 2000).

Of further note on the individual analyses, is a phenomenon noted most prominently for the Purdue Pegboard of fluctuating performances over the assessment intervals (fluctuations in two instances for Preferred, in three instances for Non Preferred and Both, and in four instances for Assembly), implicating cognitive vulnerability for these rugby playing individuals in that hand-motor modality. It is of note that the player singled out earlier when discussing the effects of estimated IQ on the individual players' neurocognitive performances, whose scores all tended to be relatively superior across the test intervals, did, however, reveal marked fluctuating performances on the Purdue Pegboard test (fluctuations on Purdue Both and Purdue Assembly). The implication is that he found this test more challenging than the ImPACT subtests such that some latent cognitive vulnerability was revealed that would have been missed without the inclusion of the Purdue in the test battery. Finally, the Purdue Pegboard results for the correlational analyses stood out as consistently implicating that a *higher* number of concussions was associated with *lowered* performance on those tasks. Measures of hand-motor reaction speed, like the Purdue Pegboard, have been

shown to provide accurate indexes of cognitive changes following brain injury, has been reported to be sensitive to the effects of cognitive impairment and is therefore a good measure of diffuse brain injury following concussion (Lezak et al., 2004; Strauss et al., 2006). Research on MTBI and hand-motor functioning in the sports context appears to be minimal, and the only research done on the effect of MTBI on hand-motor speed was done on soccer players utilizing the Finger Tapping Test (Baroff, 1998). Only one rugby study investigating the acute and chronic neurocognitive effects of rugby-related MTBI incorporated a measure of hand-motor speed, the Purdue Pegboard test (Shuttleworth-Jordan et al., 1993). These indications of sensitivity of the Purdue Pegboard to neurocognitive vulnerability in the present study endorse the inclusion of speeded hand motor tasks, and in particular the Purdue Pegboard in studies of this type with a view to substantiating the extent of brain injury effects.

9.2.5 Overall Implications

Taking the neurocognitive results of all the various modes of analysis used in this study, including independent cross-sectional and dependent group analyses, together with the results on the correlational studies and individual case analyses, incorporating concussion and tackling data in the correlational and individual case investigations, there has been a compelling degree of cross-validation of outcome that jointly serves to endorse the presence of neurocognitive vulnerability in the Rugby Group when compared with the Non-Contact Sports Control Group, especially in the motor area, although there are subtle indications of effects in the same direction in the memory modality. The results are highly commensurate with a gathering body of research implicating similar long-term cognitive deterioration amongst players of rugby and other contact sports (Barth et al., 1989; Bernstein, 2002; Dawodu, 2009; Eckner, Kutcher, Broglio & Richardson, 2013; Field et al., 2003; Frencham, Fox & Maybery, 2005; Gaetz & Bernstein, 2001; Gardner, Shores & Batchelor, 2010; Grindel, Lovell & Collins, 2001; Guskiewicz et al., 2005; Hinton-Bayre & Geffen, 2002; Iverson, Gaetz, Lovell & Collins, 2004; Killiam, Cautin & Santucci, 2005; Lezak et al., 2004; Lovell & Collins, 1998; Lovell, Collins, Iverson, Johnston & Bradley, 2000; Matser, Kessels, Jordan, Lezak & Troost, 1998; Matser, Kessels, Lezak, Jordan & Troost, 1999; McCrea et al., 2012; McCrea, Prichep, Powell, Chabot & Barr, 2010; Moser, Schatz & Jordan, 2005;

Pettersen & Skelton, 2000; Rabadi & Jordan, 2001; Reitan & Wolfson, 1999; Shuttleworth-Edwards, Border, Reid & Radloff, 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith & Radloff, 2008; Shuttleworth-Edwards & Whitefield, 2007; Vanderploeg, Curtiss & Belanger, 2005; Webbe & Ochs, 2003; Witol & Webbe, 2003).

Finally, this seemingly robust empirical observation of enhanced neurocognitive vulnerability in the Rugby Group replicated over the multimodal investigation of the present study, can be understood in terms of the concept of cognitive reserve (Satz, 1993; Stern, 2003; 2006). In terms of the combined implications of these theorists, it is understood that the exposure of the participants of the rugby players in the present study, to repeated concussive and subconcussive events sustained over a long rugby-playing career, including the more recent season, would have succumbed to reductions in brain reserve capacity when compared to those individuals from a Non-Contact Sports Control group of equivalent age, education and estimated level of IQ, in turn causing the rugby players to perform more poorly than equivalent Non-Contact Sports controls on cognitive tasks known to be sensitive to the diffuse effects of mild traumatic brain injury.

From the individual case analyses in particular, it was possible to demonstrate subtle indications of how the protective factor of higher IQ on cognitive reserve might retain neurocognitive scores at a relatively high level despite a recent concussive event, as per the predictions arising out of the cognitive reserve theory of Stern (2006; 2003). It was also possible to demonstrate how this protective effect of a relatively high IQ on neurocognitive performance might be diminished by the neuropathological vulnerability factor of repetitive subconcussive brain injury in association with a particularly high frequency of tackling over many years of participation in rugby, as proposed by the brain reserve theory of Satz (1993).

9.3 CRITICAL EVALUATION OF THE STUDY

The present study set out to investigate the neurocognitive outcome of participation in adult club level rugby, within a milieu of cumulating evidence for deleterious effects in association with participation in a contact sport such as rugby. It is considered that this study, although

not without its limitations, has nevertheless provided a valuable contribution to this body of literature. More specifically the strengths and weaknesses of the study are as follows.

9.3.1 Strengths of the Study

Multiple forms of analysis, including independent cross-sectional and dependent group analyses, together with the correlational and individual case analyses, incorporating concussion and tackling data in the correlational and individual case investigations, allowed for the unique cross-validation of the outcome, and a more robust and compelling inference in support of the hypotheses that the Rugby Group will perform worse than the Non-Contact Sports Control Group, due to postulated exposure to the cumulative effects of tackling during years of rugby participation, including concussive and sub-concussive events during participation in one rugby season.

In terms of the group analyses, it is considered that there was good control for the influential variables of age, education and estimate IQ, often not controlled for in studies of this type. Together with concussion data that discriminated between the two groups, this provided for fairly compelling confirmation, within the limitations of cross-sectional research that can never categorically rule out confounding pre-existing differences between groups, that any deleterious neurocognitive effects in evidence for the Rugby Group could not readily be accounted for on the basis of intergroup differences in age, education and IQ. Rather such effects could be more readily attributed to the repetitive long-term exposure of the club level rugby players to cumulative and sub-concussive effects in association with years of participation in contact sport.

A particularly strong aspect of the study was the novel incorporation of video analyses to enable the researcher to define the type and number of tackles made and received during one rugby season. Incorporating tackling data in a neurocognitive study was never done before, and as no players during the season were formally diagnosed with a concussion, this valuable aspect would have gone unnoticed even for a suspected concussive incident if not for the retrospective video analyses of the games. Specifically, these tackling data derived on the basis of the within Rugby Group video analyses of all games across the rugby season,

provided further evidence in addition to the concussion data, that the Rugby Group differs from the Non-Contact Sports Control Group in terms of possible injury to the brain with associated neurocognitive effects, and revealing important heuristic potential for future studies.

In two aspects of the study, the correlational and individual analyses, the overall investigation was enriched by the incorporation of tackling data over one season for all participants in the rugby playing group. Although highly tentative, there were indications that were in the hypothesized direction of higher tackling incidence being associated either with more concussions or poorer neurocognitive performance. This makes conceptual sense due to the intricacy of the causative link between concussive and subconcussive events and compromised neurocognitive function. This is a novel aspect to research methodology in the study of neurocognitive effects in rugby, being the first time this has ever been formally done in a neurocognitive study of this type to the author's knowledge.

In addition to tackling it was an important and relatively novel feature to combine a series of case analyses with a group analysis, and particularly the inclusion of five individuals who were not formally diagnosed with concussion, although perceived on the video analysis to have possibly sustained a head jarring event. In this regard it is of relevance to make reference to the MTBI research of Wilberger et al. (1991). These researchers highlighted how important it is to note how deleterious effects averaged out in a group analysis and may be isolated in individual instances in the form of subtle individualized effects or trends, such as occurred in their research, and such as can be seen to have been demonstrated in the present study. This type of fine individual analysis can protect against the dangers of Type II error, i.e., the chance of missing the presence of clinically meaningful deleterious effects of mild traumatic brain injury when applying group research (Demakis, 2006; Frencham et al., 2005; Reitan & Wolfson, 1999; Ruff, 2005; Woods et al., 2006).

ImPACT was identified for the purpose of the present study, due to it being widely used for evaluating sports concussion, and it was designed to simultaneously evaluate multiple cognitive domains, and have shown to be sensitive to the effects of concussion (Collins & Hawn, 2002; Collins, Iverson, et al., 2003; Lovell & Collins, 2002; Lovell et al., 2004;

Schatz et al., 2006). It was a relative novel idea to include the Purdue Pegboard that proved to be a sensitive and discriminatory indicator of a suspected concussive event. The lack of good discriminatory measures would have put the study at risk of Type II error, i.e., failing to demonstrate significance where it exists, whereas this test choice engendered compelling indications in support of the hypotheses that there would be significant differences between the Rugby Group and the Non-Contact Sports Control Group, and that a higher number of concussions were being associated with lowered neurocognitive performance.

9.3.2 Limitations of the Study

Limitations of this study included the relatively small sample numbers (i.e., Rugby Group $n = 20$, Non-Contact Sports Control Group $n = 22$) and the small number of concussion cases followed up (i.e. $n = 5$). Statistical analyses with $n < 50$ tend to lack sufficient statistical power (a function of sample size, effect size and p-level) for detecting small, medium or possible large effects (Trusty, Thompson & Petrocelli, 2004). When the same small number of subjects are used for all the assessment intervals the standard error is smaller and consequently smaller differences in means are likely to be detected (Peers, 1996). Risk of Type I error, i.e., indicating significance when there is no real difference, is particularly a problem for correlational analyses which should not be conducted on overly small samples as estimates of the correlation are likely to obtain a spuriously-large correlation coefficient in this way.

The limitations of the small sample numbers were to some extent compensated for by virtue of having a well-controlled study for influential variables in the group comparisons, i.e., age, education, and estimated IQ, as well as a wide-ranging set of simultaneous investigations that served as cross-validation for a series of findings that in isolation would have been extremely tentative, but together became more convincing in terms of the robustness of the outcome in the hypothesized direction of deleterious neurocognitive effects in association with participation in club rugby.

Another limitation was that tackling data were only calculated for the actual games and not for any of the contact practice sessions which were likely to result in an underestimation of

exposure to multiple possible head jarring incidents. Again this may have resulted in Type II error in the correlational analyses due to the underestimation of tackling situations and the subsequent subconcussive and concussive effects. However, this does mean that implications in the hypothesized direction such as they were are not likely to be an exaggeration of effects (i.e. Type 1 error).

Finally the test battery, while comprehensive from the neurocognitive point of view, including both the well-renowned ImPACT test in addition to an adjunctive hand-motor test the Purdue Pegboard that revealed sensitivity, there was no analysis of symptom sequelae that would have been an enriching aspect to this study. However, in that the study already incorporated multiple angles of investigation, it was considered to be beyond the scope of what was possible for the present study, and may have contributed to a lack of focus. A limitation of the Purdue Pegboard was the lack of multiple versions to eliminate practice effects. However this proved to be a discriminating feature in using differential practice effects between the groups as a diagnostic feature.

9.4 CLINICAL IMPLICATIONS AND IMPLICATIONS FOR FUTURE RESEARCH

From a clinical perspective, the most striking feature of the five case analyses conducted for the present study is that there is the persuasive indication that the very minor concussive brain injury, that might not normally be identified on the field as a diagnosable concussion (such as occurred in these five instances), does result in brain dysfunction with measurable neurocognitive sequelae. In accordance with a seminal earlier research study that confirmed deleterious neurocognitive effects of the mildest ‘ding’ injury (Lovell, Collins, Iverson, Johnston, & Bradley, 2004), the outcome provides strong support for high attentiveness to the identification of even the mildest concussive head or body jarring event, and the associated need to remove such a player from the field for further neurocognitive follow up and careful medical management in the interests of being ensured of safe return to play. Taken together, the outcome from the Lovell et al. (2004), and present study, that demonstrate the clinical relevance of the mildest spectrum of observable concussive injury, is commensurate with a growing consensus of opinion that calls for *vigilant* identification,

assessment and management of concussive sequelae and recovery among individuals (American Academy of Neurology, 2013; Echemendia et al., 2001; Guskiewicz et al., 2004; McCrory et al., 2013; Moser et al., 2007). In terms of isolating such very mild concussive injuries, the tackling video analyses conducted in the present study appear to be of novel relevance not only in a research context, but certainly in a clinical context. In clinical contexts, the technique provides a pointer in terms of future possible routes to take to ensure more accurate diagnosis of even the mildest, yet clinically relevant concussive injury. While video analysis of games is a common procedure at professional levels of sports participation, this is usually employed with a view to scrutinizing strategies to enhance competitive performance, and/or to replay established injury incidents. The recommendation arising out of the present study is that such video recordings might be used, in addition, with a view to isolating possible concussive incidents that were not obvious enough to halt play, yet may be in need of being followed up for further assessment and management. Unfortunately such a mechanism would be expensive and time consuming, and while optimal might be difficult to implement more widely at present than just at professional levels of play.

The results of the present study give rise to a number of implications for future research, and questions in need of further research, that in turn may serve to inform management of individual outcome. There are persuasive implications arising from the current study that include *tackling* data in addition to the traditional concussion data alone, and utilizing the *case-based* study method rather than group analyses alone, allows for a detailed level of observation that is not possible on a more circumscribed methodological approach. In particular, a large series of case-based analyses in conjunction with video observations of players over several seasons could provide insights into the predominant vulnerability and protective factors on neurocognitive outcome in terms of brain reserve theory that were tentatively explored on this initial study.

Critical, also, in terms of future studies, is the pursuit of longitudinal analysis not only of professional players, but also of *club* level players who tend to be less of an obvious target for such research, but may well serve to ratify persistent cognitive decrements into older age in association with participation in a sport such as rugby as has been implicated on this initial study on that particular cohort. Recent autopsy and neurological studies have given rise to

alarm about such long-term effects (De Beaumont et al., 2009; Omalu et al., 2011; Tremblay et al., 2013), and these need also to be further ratified in terms of chronic neurocognitive outcomes. Research of this type would be made possible through the regularization of *pre*-season and post-concussion testing for athletes involved in a cerebrally hazardous sport such as rugby for all adult levels and types of play, not only professional players, with an approved psychometric instrument that has demonstrated sensitivity to concussion. Given evidence for significant underreporting of this injury, educational programs for athletes and coaches are needed to ensure optimal identification and reporting of concussions for such follow-up neurocognitive investigation. Such education should include raising awareness in participants of these sports, that even where there are not regular confirmed diagnoses of a concussion, the mere exposure to frequent and repeated head and body collisions may have deleterious brain-related consequences, and therefore warrants ongoing monitoring made possible with routinely applied annual or bi-annual neurocognitive testing.

Finally, research on the refinement of suitable measures for neurocognitive evaluation should continue. The present research indicates that a computerized tool such as the ImPACT test has been sensitive in the identification of extremely mild concussive events on the basis of its case analyses, and that it has also shown apparent sensitivity to more persistent effects via the group analyses. The research did reveal overall that the visual motor speed and reaction time composites were the most sensitive, whereas verbal and visual memory were less sensitive, and might be considered to be removed from the test, or the form that they are given in should be revised. For instance, prior doctoral research using the ImPACT test demonstrated that an associate paired recall of verbal material was particularly sensitive to concussion effects (Clark, 2010). Other rugby research has revealed the Digit Symbol Coding Recall task (another associate paired recall task) to differentiate rugby from control groups (Shuttleworth-Edwards, Border, Reid, & Radloff, 2004). The inclusion of paired learning in both the verbal and visual modalities might be a way of retaining verbal and visual recall tasks in the test, but in a way that is more discriminatory. While the Purdue Pegboard incorporated in this thesis did reveal its sensitivity, it is not clear how this aspect of functioning might be pursued in future research, but should be noted for consideration by test developers working in the sports concussion field. For instance a type of timed finger tapping test could easily be incorporated as part of a test such as the ImPACT test. In short,

on-going research of this nature is important to identify which aspects of such a test are the most discriminatory, and to develop them along the most streamlined route possible in the interests of producing a time and cost efficient, yet highly sensitive instrument.

9.5 FINAL WORD

This thesis set out to examine the persistent neurocognitive effects for club level rugby players relative to equivalent non-contact sports controls over one rugby season, in a uniquely multifaceted study that included group analyses supplemented by case analyses of the mildest cases of concussion, and video analyses of tackling data. Despite the limitations of the study, particularly in terms of small sample numbers, the overall outcome can be seen to add to a growing body of literature that implicates deleterious neurocognitive effects in participants of a sport such as rugby due to repetitive head jarring incidents that are intrinsic to the game that involves a sobering number of tackling maneuvers even over one season. The outcome itself, and the measures employed for the research provide valuable heuristic indications for future research studies in this area.

REFERENCES

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

Adams, J.H., Doyle, D. & Ford, I. (1989). Diffuse axonal injury in head injury: definition, diagnosis and grading. *Histopathology, 15*, 49-59.

Adams, R.L., Parsons, O.A., Culbertson, J.L. & Nixon, S.J. (1996). *Neuropsychology for clinical practice.* Washington, DC: American Psychological Association.

Ahmed, S., Bierley, R., Sheikh, J.I., & Date, E.S. (2000). Post-traumatic amnesia after closed head injury: a review of the literature and some suggestions for further research. *Brain Injury, 14(9),* 765-780.

Alexander, M.P. (1995). Mild traumatic brain injury: Pathophysiology, natural history and clinical management. *Neurology, 45*, 1253-1260.

Alla, S., Sullivan, S.J., Hale, L., & McCrory, P. (2009). Self-report scales/checklists for the measurement of concussion symptoms: a systematic review. *British Journal of Sports Medicine, 43(Suppl. 1),* i3-i12.

All Blacks. (2012). Pitch Side Concussion Assessment. New Zealand Rugby Union. Retrieved August 20, 2012, from http://files.allblacks.com/nzru_media_releases/2012/08/ITMC_PSCA_Process.pdf

Amen, D.G., Newberg, A., Thatcher, R., Jin, Y., Wu, J., Keator, D., et al. (2011). Impact of playing American professional football on long-term brain function. *Journal of Neuropsychiatry and Clinical Neuroscience, 23*, 98-106.

American Academy of Neurology. (1997). Practice parameter: The management of concussion in sport (summary statement) - Report of the Quality Standards Sub-committee. *Neurology, 48*, 581-585.

American Academy of Neurology. (2013). AAN releases new sports concussion guidelines. *Medscape*, Mar 18, 2013.

Andersen, T.E., Larsen, Ø., Tenge, A., Engebretsen, L., & Bahr, R. (2003). Football incident analysis: a new video-based method to describe injury mechanisms in professional football. *British Journal of Sports Medicine, 37,* 226-232.

Anderson, L., Schnor, P., Schroll, M., & Hein, H. (2000). All-cause mortality associated with physical activity during leisure time, work, sports, and cycling to work. *Archive of Internal Medicine, 160,* 1621-1628.

Anderson, P. (2012a). Head hits during a single season may affect learning, memory. *Medscape, May 17.*

Anderson, P. (2012b). Traumatic brain injury more common than previously reported. *Medscape, Nov 29.*

Anderson, P., & Murata, P. (2009). New VA/DoD guidelines for concussion and mild traumatic brain injury. *MedscapeCME Clinical Briefs.* Retrieved May 18, 2011, from http://www.medscape.com/viewarticle/714450

Anderson, V., Northam, E., Hendy, J., & Wrennall, J. (2001). Developmental Neuropsychology. Sussex, England: Psychology Press.

Arciniegas, D.B., Anderson, C.A., Topkoff, J., & McAllister, T.W. (2005). Mild traumatic brain injury: A neuropsychiatric approach to diagnosis, evaluation and treatment. *Neuropsychiatric Disease and Treatment, 1(4),* 311-327.

Arnason, A., Tenga, A., Engebretsen, L., & Bahr, R. (2004). A prospective video-based analysis of injury situations in elite male football. *American Journal of Sports Medicine, 32,* 1459-1465.

Asikainen, I. (2001). Long-term functional and vocational outcome of patients with traumatic brain injury. *Academic dissertation, Department of Neurology,* University of Helsinki, Finland.

Asikainen, I., Nybo, T., Müller, K., Sarna, S., & Kaste, M. (1999). Speed performance and long-term functional and vocational outcome in a group of young patients with moderate or severe traumatic brain injury. *European Journal of Neurology, 6,* 179-185.

Aubry, M., Cantu, R.C., Dvořák, J., Graf-Baumann, T., Johnston, K.M., Kelly, J. et al. (2002). Summary and agreement statement of the 1st International Symposium on Concussion in Sport, Vienna 2001. *Clinical Journal of Sports Medicine, 12*, 6-11.

Bailes, J.E. & Cantu, R.C. (2001). Head injuries in Athletes. *Neurosurgery, 48(1)*, 26-46.

Bailes, J.E. & Hudson, V. (2001). Classification of sport-related head trauma: A spectrum of mild to severe injury. *Journal of Athletic Training, 36(30),* 236-243.

Baker, K.L., & Hutchinson, K. (2008). Cognitive evoked auditory potentials and neuropsychological measures following concussion in college athletes. Miami University, Speech Pathology and Audiology.

Baker, R.J. & Patel, D.R. (2000). Sports Related Mild Head Injury in Adolescents. *Indian Journal of Paediatrics, 67(5)*, 317-321.

Baltes, M.M., Kühl, K., Gutzmann, H., & Sowarka, D. (1995). Potential of cognitive plasticity as a diagnostic instrument: A cross-validation and extension. *Psychology and Ageing, 10(2),* 167-172.

Barnett, J.H. & Sahakian, B.J. (2008). Mental capital and wellbeing: Making the most of ourselves in the 21st century. *Government Office for Science.* Retrieved August 2, 2011, from http://www.foresight.gov.uk/Mental

Barnett, J.H., Salmond, C.H., Jones, P.B., & Sahakian, B.J. (2006). Cognitive reserve in neuropsychiatry. *Psychological Medicine, 36,* 1053-1064.

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

Barr, W.B. (2001). Methodological issues in Neuropsychological Testing. *Journal of Athletic* Training, 36(3), 297-302.

Barr, W.B., & McCrea, M. (2001). Sensitivity and specificity of standardized neurocognitive testing immediately following sports concussion. *Journal of the International Neuropsychological Society, 7*, 693-702.

Barr, W.B., Prichep, L.S., Chabot, R., Powell, M.R., & McCrea, M. (2011). Measuring brain electrical activity to track recovery from sport-related concussion. *Brain Injury*, 1-9.

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

Barth, J., Broshek D., Erlanger D., Feldman, D., Freeman J., Kaushik T., Kroger H., Kutner K. (2000). Development and validation of a web-based neuropsychological test protocol for sports-related return-to-play decision-making. *Archives of Clinical Neuropsychology, 18,* 293–316.

Barth, J.T., Freeman, J.R., Broskek, D.K., & Varney, R.N. (2001). Acceleration-deceleration sport-related concussion: The gravity of it all. *Journal of Athletic Training, 36(3),* 253-256.

Barth, J.T., Macciocchi, S.N., Giordani, B., Rimel, R., Jane, J.A., & Boll, T.J. (1983). Neuropsychological sequelae of minor head injury. *Neurosurgery, 13(5),* 529-533.

Barth, J.T., Varney, R.N., Ruchinskas, R.A., & Francis, J.P. (1999). Mild head injury: The new frontier in sports medicine. In Varney, R.N. & Roberts, R.J. (Eds.). *The evaluation and treatment of mild traumatic brain injury*. London: Lawrence Erlbaum Associates, Publishers.

Bathgate, A., Best, J.P., Craig, G., & Jamieson, M. (2002). A prospective study of injuries to elite Australian rugby union players. *British Journal of Sports Medicine, 36*, 265-269.

Bartlett, R. (2001). Performance analysis: Can bringing together biomechanics and notational analysis benefit coaches? *3rd International Symposium of Computer Science in Sport.*

Baugh, C., Stamm, J.M., Riley, D.O., Gavett, B.E., Shenton, M.E., Lin, A., Nowinski, C.J., Cantu, R.C., McKee, A.C., & Stern, R.A. (2012). Chronic traumatic encephalopathy: Neurodegeneration following repetitive concussive and subconcussive brain trauma. *Brain Imaging and Behaviour*, doi:10.1007/s11682-012-9164-5

Bazarian, J.J., Blyth, B., & Cimpello, L. (2006). Bench to bedside: Evidence for brain injury after concussion – Looking beyond the Computed Tomography Scan. *Academic Emergency Medicine, 13,* 199-214.

Belanger, H.G., Curtiss, G., Demery, J.A., Lebowitz, B.K., & Vanderploeg, R.D. (2005). Factors moderating neuropsychological outcomes following mild traumatic brain injury: A meta-analysis. *Journal of the International Neuropsychological Society, 11,* 215-227.

Belanger, H.G., Spiegel, E., & Vanderploeg, R.D. (2010). Neuropsychology performance following a history of multiple self-reported concussions: A meta-analysis. *Journal of the International Neuropsychological Society, 16(2),* 262-267.

Belanger, H.G. & Vanderploeg, R.D. (2005). The neuropsychological impact of sports-related concussion: A meta-analysis. Journal *of the International Neuropsychological Society, 11,* 345-357.

Bender, S.D., Barth, J.T., & Irby, J. (2004). Historical Perspectives. In M.R. Lovell, R.J. Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 3-21). The Netherlands: Swets & Zeitliner Publishers.

Benson, R.R., Gattu, R., Sewick, B., Kou, Z., Zakariah, N., Cavanaugh, J.M., & Haacke, E.M. (2012). Detection of hemorrhagic and axonal pathology in mild traumatic brain injury using advanced MRI: Implications for neurorehabilitation. *Neurorehabilitation, 31,* 261-279.

Bernhardt, D.T. (2009). Concussion. *Emedicine.* Retrieved August 21, 2011, from http://*emedicine.medscape.com/article/92095*

Bernstein, D.M. (2002). Information processing difficulty long after self-reported concussion. *Journal of the International Neuropsychological Society, 8,* 323-346.

Bernstein, D.M. (1999). Recovery from mild head injury. *Brain Injury, 13(3),* 151-172.

Bey, T., & Ostick, B. (2009). Second impact syndrome. *Western Journal of Emergency Medicine, 10,* 6-10.

Bigler, E.D. (2001). The lesion(s) in traumatic brain injury: Implications for clinical neuropsychology. Archives of Clinical Neuropsychology, 16, 95-131.

Bigler, E.D. (2003). Neurobiology and neuropathology underlie the neuropsychological deficits associated with traumatic brain injury. *Archives of Clinical Neuropsychology, 18*, 595-621.

Bigler, E.D. (2010). Neuroimaging in Mild Traumatic Brain Injury. *Psychology, Injury and Law, 3*, 36-49.

Bigler, E.D. & Orrison, Jr, W.W. (2004). Neuroimaging in Sport-related brain injury. In Lovell, M., Echemendia, R.J., Barth, J.T., & Collins, M.W. (Eds.), *Traumatic Brain Injury in Sports* (pp71-94). The Netherlands: Swets & Zeitlinger Publishers.

Binder, L.M. (1986). Persisting symptoms after mild head injury: A Review of the Postconcussive Syndrome. *Journal of Clinical and Experimental Neuropsychology, 8(4),* 323-346.

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

Binder, L.M., Rohling, M.L., & Larrabee, G.J. (1997). A Review of Mild Head Trauma. Part 1: Meta-analytic review of neuropsychological studies. *Journal of Clinical and Experimental Neuropsychology, 19(3),* 421-431.

Bleiberg. J., Cernich, A.N., Cameron, K., Sun, W., Peck, K., Ecklund, J., et al. (2004). Duration of cognitive impairment after sports concussion. *Neurosurgery, 54,* 1073-1080.

Bleiberg. J., Garmoe, W.E., Halpern, E. Reeves, D.L., & Nadler, J. (1997). Consistency of within-day and across-day performance after mild brain injury. *Neuropsychiatry, Neuropsychology, Behavioural Neurology, 10*, 247-253.

Bleiberg. J., Kane, R. I., Reeves, D.L., Garmoe, W.E., & Halpern, E. (2000). Factor analysis of computerised and traditional tests used in mild brain injury research. *Clinical Neuropsychology, 14*, 287-294.

Blessed, A.L., Tomllinson, B.E., & Roth, M. (1968). The association between quantitative measures of dementia and of senile change in the cerebral grey matter of elderly subjects. *British Journal of Psychiatry, 114,* 797-811.

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

Boffano, P., Boffano, M., Gallesio, C., Roccia, F., Cignetti, R., & Piana, R. (2011). Rugby players' awareness of concussion. *Journal of Craniofacial Surgery, 22(6),* 2053-2056.

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

Borg, J., Holm, L., Cassidy, J.D., Peloso, P.M., Carroll, L.J., von Holst, H., et al. (2004). Diagnostic procedures in mild traumatic brain injury: results of the WHO collaborating centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine, Suppl. 43,* 61-75.

Borgaro, S.R., Prigatano, G.P., Kwasnica, C., & Rexer, J.L. (2003), Cognitive and affective sequelae in complicated and uncomplicated mild traumatic brain injury. *Brain Injury, 17(3),* 189-198.

Bowen, A.P. (2003). Second impact syndrome: A rare, catastrophic, preventable complication of concussion in young athletes. *Journal of Emergency Nursing, 29(3),* 287-289.

Brandt, J. (2007). 2005 INS presidential address: neuropsychological crimes and misdemeanors. *The Clinical Neuropsychologist, 21,* 553-568.

Brandt, J., & Benedict, R.H. B. (2001). Hopkins Verbal Learning Test – revised: Professional manual. Florida: Psychological Assessment Resources, Inc.Broglio, S.P., Ferrara, M.S., Macciocchi, S.N., Baumgartner, T.A., & Elliot, R. (2007). Test-retest reliability of computerised concussion assessment programs. *Journal of Athletic Training, 42(4),* 509-514.

Broglio, S.P., Macciocchi, S.N., & Ferrara, M.S. (2007). Sensitivity of the concussion assessment battery. *Neurosurgery, 60,* 1050-1057.

Broglio, S.P., Pontifex, M.B., O'Connor, P., & Hillman, C.H. (2009). The persistent effects of concussion on neuroelectric indices of attention. *Journal of Neurotrauma, 26(9),* 1463-1470.

Broglio, S.P., & Puetz, T.W. (2008). The effect of sport concussion on neurocognitive function, self-report symptoms and postural control. *Sports Medicine, 38(1),* 53-67.

Broglio, S.P., Sosnoff, J.J., Shin, S., He, X., Alcaraz, C., & Zimmerman, J. (2009). Head impacts during high school football: A biomechanical assessment. *Journal of Athletic Training, 44(4),* 342-349.

Brolinson, P.G., Manoogian, S., McNeely, D., Goforth, M., Greenwald, R., & Duma, S. (2006). Analysis of linear head accelerations from collegiate football impacts. *Current Sports Medicine Report, 5(1),* 23-28.

Brooks, J., Fos, L.A., Greve, K.W., & Hammond, J.S. (1999). Assessment of executive function in patients with mild traumatic brain injury. *The Journal of Trauma, 46(1),* 159-63.

Broshek, D.K., Kaushik, T., Freeman, J.R., Erlanger, D., Webbe, F., & Barth, J.T. (2005). Sex differences in outcome following sports related concussion. *Journal of Neurosurgery, 102(5),* 856-863.

Bruce, J.M., & Echemendia, R.J. (2009). History of multiple self-reported concussions is not associated with reduced cognitive abilities. *Neurosurgery, 64(1),* 100-106.

Bruno, L.A., Gennarelli, T.A., & Torg, J.S. (1987). Management guidelines for head injuries in athletics. *Clinical Sports Medicine, 6,* 17-29.

Buddenberg, L., & Davis, C. (2000). Test-retest reliability of the Purdue Pegboard Test. *American Journal of Occupational Therapy, 54,* 555-558.

Bullock, J. (1997). Injury and cell function. In: Reilly, P., Bullock, R. (Eds.) *Head Injury.* London: Chapman & Hall.

Cantu, R.C. (1986). Guidelines for return to contact sport after a cerebral concussion. *Physician Sports Medicine, 14,* 75-83.

Cantu, R.C. (1992). Cerebral concussion in sport: management and prevention. *Sports Medicine,14(1),* 64-74.

Cantu, R.C. (1996). Head injuries in sport. *British Journal of Sports Medicine, 30(4),* 289-296.

Cantu, R.C. (1997). Athletic head injuries. *Clinics in Sport Medicine, 16(3),* 531-542.

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

Cantu, R.C. (2001). Posttraumatic retrograde and anterograde amnesia: Pathophysiology and implications in grading and safe return to play. *Journal of Athletic Training, 36,* 244-248.

Cantu, R.C. (2003). Recurrent athletic head injury: risks and when to retire. *Clinics in Sports Medicine, 22,* 593-603.

Cantu, R.C. & Voy, R. (1995). Second impact syndrome: a risk in any contact sport. *Physician Sports Medicine, 23(6),* 27-28, 31-34.

Capruso, D.X., & Levin, H.S. (1992). Cognitive impairment following closed head injury. *Neurology of Trauma, 10,* 879-893.

Carroll, L.J., Cassidy, J.D., Holm, L., Kraus, J., & Coronado, V.G. (2004). Methodological issues and research recommendations for mild traumatic brain injury: the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine, Suppl. 43,* 113-125

Cassels, C. (2013). PET imaging reveals damage from head injury in living athletes. *Medscape,* Feb 07, 2013.

Cassidy, J.D., Carroll, L.J., Peloso, P.M., Borg, J., von Holst, H., Holm, L., et al. (2004). Incidence, risk factors, and prevention of mild traumatic brain injury: Results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine, Suppl., 43,* 28-60.

Catroppa, C., & Anderson, V. (2009). Neurodevelopmental outcomes of pediatric traumatic brain injury. *Future Neurology, 4(6),* 811-821.

Centers for Disease Control and Prevention. (2003). TBI report to Congress on mild traumatic brain injury in the United States: Steps to prevent a serious public health problem. *National Center for Injury Prevention and Control, Atlanta, GA.*

Chen, J.K., Johnston, K.M., Frey, S., Petrides, M., Worsley, K., & Ptito, A. (2004). Functional abnormalities in symptomatic concussed athletes: An *f*MRI study. *Neuroimage, 22(1),* 68-82.

Cernich, A., Reeves, D., Sun, W., & Bleiberg, J. (2007). Automated Neuropsychological Assessment Metrics sports medicine battery. *Archives of Clinical Neuropsychology, 22(1),* 101-114.

Clark, S.B. (2010). Neurocognitive and symptom profiles of concussed and nonconcussed provincial rugby union players over one season. *Unpublished Doctoral dissertation, Rhodes University, South Africa.*

Coffey, C.E., Saxton, J.A., Ratcliff, G., Bryan, R.N., & Lucke, J.F. (1999). Relation of education to brain size in normal aging: implications for the reserve hypothesis. *Neurology, 53(1),* 189-196.

CogState Sport. (2010). *CogState Sport.* Retrieved October 30, 2010 from http://www.cogstate.com/go/sport

Collie, A., Darby, D., & Maruff, P. (2001). Computerized cognitive assessment of athletes with sports related head injury. *British Journal of Sports Medicine, 35*, 297-302.

Collie, A., Makdissi, M., Maruff, P., Bennell, K., & McCrory, P. (2006). Cognition in the days following concussion: comparison of symptomatic versus asymptomatic athletes. *Journal of Neurology, Neurosurgery and Psychiatry, 77(2)*, 241-255.

Collie, A., Maruff, P., Makdissi, M., McStephen, M., Darby, D.G., & McCrory, P. (2003). Statistical procedures for determining the extent of cognitive change following concussion. *British Journal of Sports Medicine, 38(3),* 273-278.

Collie, A., Maruff, P., McStephen, M., Darby, D.G. (2003). Psychometric issues associated with computerised neuropsychological assessment of concussed athletes. *British Journal of Sports Medicine, 37,* 556-559.

Collins, M.W., Echemendia, R.J., & Lovell, M.R. (2004). Collegiate and high school sports. In M.R. Lovell, R.L. Echemendia, J.T. Barth, & M.W. Collins (Eds.). *Traumatic Brain Injury in Sports.* The Netherlands: Swets & Zeitlinger Publishers.

Collins, M.W., Grindell, S.H., Lovell, M.R., Dede, D.E., Moser, D.J., Phalin, B.R., Nogle, S., et al. (1999). Relationship between concussion and neuropsychological performance in college football players. *The Journal of the American Medical Association, 282(10),* 964-970.

Collins, M.W. & Hawn, K.L. (2002). The clinical management of sports concussion. *Current Sports Medicine Reports,1(1),* 12-21.

Collins, M.W., Iverson, G.L., Lovell, M.R., McKeag, D.B., Norwig, J., & Maroon, J. (2003). On-field predictors of neuropsychological and symptoms deficit following sport-related concussion. *Clinical Journal of Sports Medicine, 13(4),* 222-229.

Collins, M.W., Lovell, M.R., Iverson, G.L., Cantu, R.C., Maroon, J.C., & Field, M. (2002). Cumulative effects of concussion in high school athletes. *Neurosurgery, 51,* 1175-1181.

Collins, M.W., Lovell, M.R., & McKeag, D.B. (1999). Current issues in managing sports-related concussion. *Journal of the American Medical Association, 282*, 2283-2285.

Covassin, T., Stearne, D., & Elbin, R. (2008). Concussion history and postconcussion neurocognitive performance and symptoms in collegiate athletes. *Journal of Athletic Training, 43(2),* 119-124.

Covassin, T., Weiss, L., Powell, J. & Womack, C. (2007). Effects of a maximal exercise test on neurocognitive function. *British Journal of Sports Medicine, 41,* 370-374.

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

Crippen, D.W. (2009). Head Trauma. *Critical Care Medicine, 37(6),* 2057-2063.

Cripps, A., & Livingston, S.C. (2013). The value of balance-assessment measurements in identifying and monitoring acute postural instability among concussed athletes. *Sport Rehabilitation, 22(1),* 67-71.

Crotti, N. (2014). Tackling Sports Concussions with a Digital Camera. Retrieved December 4, 2014, from http://www.qmed.com/mpmn/medtechpulse/tackling-sports-concussions-digital-camera?cid=nl.qmed01.20141204

Cullum, C.M., & Thompson, L.L. (1997). Neuropsychological diagnosis and outcome in mild traumatic brain injury. *Applied neuropsychology, 4(1),* 6-15.

Darby, D. & Walsh, K. (2005). Walsh's Neuropsychology: A Clinical approach – Fifth Edition. Edinburgh: Elsevier Limited.

Dartfish. (2004). Retrieved October 16, 2004 from http://www.dartfish.com

Davidson, R.J. (1988). EEG measures of cerebral asymmetry: conceptual and methodological issues. *International Journal of Neuroscience, 39,* 71-89.

Davis, G.A., Iverson, G.L., Guskiewicz, K.M., Ptito, A., & Johnston, K.M. (2009). Contributions of neuroimaging, balance testing, electrophysiology and blood markers to the assessment of sport-related concussion. *British Journal of Sports Medicine, 43,* 36-45.

Dawodu, S.T. (2009). Traumatic Brain Injury (TBI) – Definition, Epidemiology, Pathophysiology. *eMedicine.* Retrieved August, 14, 2011, from http://emedicine.medscape.com/article/326510-overview

De Beaumont, L., Mongeon, D., Tremblay, S., Messier, J., Prince, F., Leclerc, S., Lassonde, M., & Théoret, H. (2011). Persistent motor system abnormalities in formerly concussed athletes. *Journal of Athletic Training, 46(3),* 234-240.

De Beaumont, L., Théoret, H., Mongeon, D., Messier, J., Leclerc, S., Tremblay, S. et al. (2009). Brain function decline in healthy retired athletes who sustained their last sports concussion in early adulthood. *Brain (132),* 695-708.

Delaney, J. (2005). Concussion risk factors and return-to-play variables. *Physician and Sports Medicine, 33(9),* 6.

Demakis, G.J. (2006). Meta-analyses in neuropsychology: An introduction. *The Clinical Neuropsychologist, 20,* 5-9.

De Monte, V.E., Geffen, G.M., & Kwapil, K. (2005). Test-retest reliability of rapid screening of MTBI. *Journal of Clinical and Experimental Neuropsychology, 27,* 624-632.

De Monte, V.E., Geffen, G.M., & Massavelli, B.M. (2006). The effects of post-traumatic amnesia on information processing following MTBI. *Brain Injury, 20(13-14),* 1345-1354.

Department of Veteran Affairs. (2009). *VA/DoD Clinical practice guideline for management of concussion/mild traumatic brain injury.* Retrieved May 18, 2011, from http://www.healthquality.va.gov/MTBI/concussion_MTBI_full_1_0.pdf

Dick, R.W. (2003). *National Collegiate Athletic Association (NCAA) Injury Surveillance System 2002-2003*. Indianapolis, Indiana: National Collegiate Athletic Association.

Dikmen, S.S., & Levin, H.S. (1993). Methodological issues in the study of mild head injury. *Journal of Head trauma and rehabilitation, 8(3),* 30-37.

Dixon, C.E., Hamm, R.J., Taft, W.C., & Hayes, R.L. (1994). Increased anticholinergic sensitivity following closed skull impact and controlled cortical impact traumatic brain injury in the rat. *Journal of Neurotrauma, 11,* 275-287.

Downs, D.S. & Abwender, D. (2002). Neuropsychological impairment in soccer athletes. *The Journal of Sports Medicine and Physical Fitness, 42,* 103-107.

Duff, K. (2012). Evidence-based indicators of neuropsychological change in the individual patient: relevant concepts and methods. *Archives of Clinical Neuropsychology, 27,* 248-261.

Duff, K., Beglinger, L.J., Schultz, S.K., Moser, D.J., McCaffrey, R.J., Haase, R.F. et al. (2007). Practice effects in the prediction of long-term cognitive outcome in three patient samples: a novel prognostic index. *Archives of Clinical Neuropsychology, 22(1),* 15-24.

Duthie, G., Pyne, D., & Hooper, S. (2003). Applied Physiology and game analysis of Rugby Union. *Sports Medicine, 33(13),* 973-991.

Dvořák, J., McCrory, P., Aubry, M., Molloy, M., & Engebretsen, L. (2009). Concussion sans frontieres. *British Journal of Sports Medicine, 43(1),* i1-i2.

Echemendia, R.J. (2004). Cultural aspects of neuropsychological evaluations in sport. In M. Lovell, M., Echemendia, R.J., Barth, J.T., & Collins, M.W. (Eds.), *Traumatic Brain Injury in Sports* (pp. 435-442). The Netherlands: Swets & Zeitlinger Publishers.

Echemendia, R.J. & Cantu, R.C. (2003). Return to play following sports-related mild traumatic brain injury: The role for Neuropsychology. *Applied Neuropsychology, 10(1),* 45-55.

Echemendia, R.J., Iverson, G.L., McCrea, M., Macciocchi, S.N., Giola, G.A., Putukian, M., & Comper, P. (2013). Advances in neuropsychological assessment of sport-related concussion. *British Journal of Sports Medicine, 47,* 294-298.

Echemendia, R.J., Putukian, M., Mackin, R.S., Julian, L., & Shoss, N. (2001). Neuropsychological test performance before and following sports-related mild traumatic brain injury. *Clinical Journal of Sports Medicine, 11,* 23-31.

Eckner, J.T., Kutcher, J.S., Broglio, S.P., & Richardson, J.K. (2013). Effect of sport-related concussion on clinically measured simple reaction time. *British Journal of Sports Medicine,* doi:10.1136/bjsports-2012-091579

Elbin, R.J., Schatz, P., & Covassin, T.C. (2011). One-year test-retest reliability of the online version of ImPACT in high school athletes. *American Journal of Sports Medicine, 39(11),* 2319-2324.

Ellemberg, D., Henry, L.C., Macciocchi, S.N., Guskiewicz, K.M., & Broglio, S.P. (2009). Advances in sport concussion assessment: From behavioural to brain imaging measures. *Journal of Neurotrauma, 26,* 2365-2382.

Elson, L.M. & Ward, C.C. (1994). Mechanisms and pathophysiology of mild head injury. *Seminars in Neurology, 14,* 8-18.

Erlanger, D.M., Feldman, D., Kutner, K., Kaushik, T., Kroger, H., Festa, J., et al. (2003). Development and validation of a web-based neuropsychological test protocol for sports-related return-to-play decision-making. *Archives of Clinical Neuropsychology, 18,* 293-316.

Erlanger, D.M., Kutner, K.C., Barth, J.T., & Barnes, R. (1999). Neuropsychology of Sports-related head injury: Dementia Pugilistica to Post Concussive Syndrome. *The Clinical Neuropsychologist, 13(2),* 193-209.

Erlanger, D.M., Saliba, E., Barth, J.T., Almquist, J., Webright, W., & Freeman, J. (2001). Monitoring resolution of post-concussion symptoms in athletes: Preliminary results of a web-based neuropsychological test protocol. *Journal of Athletic Training, 36(3),* 280-287.

Evans, R.W. (2004). Post-traumatic headaches. *Neurologic Clinics, 22,* 237-249.

Farace, E., Ferree, R.M., Hollier, J.A., Barth, J.T., & Shaffrey, M.E. (2003). Trails A: Neurocognitive effect of previous concussions in a woman's rugby sample [Abstract]. In K. Podell (Chair), *Sports-related concussion: Focus on high school athletes.* Symposium conducted at The 31st Annual International Neuropsychological Society Meeting, Honolulu, Hawaii. *Journal of the International Neuropsychological Society, 9(2),* 207.

Fazio, V.C., Lovell, M.R., Pardini, J.E., & Collins, M.W. (2007). The relation between post concussion symptoms and neurocognitive performance in concussed athletes. *Neuro Rehabilitation, 22,* 207-216.

Fédération Internationale de Football Association [FIFA]. (2007). FIFA Big Count 2006: 270 million people active in football. Retrieved May 18, 2011, from http://www.fifa.com/aboutfifa/media/newsid=529882.html

Fick, D.S. (1995). Management of concussion in collision sports. *Postgraduate Medicine, 97(2),* 53-60.

Field, M., Collins, M.W., Lovell, M.R., & Maroon, J. (2003). Does age play a role in recovery from sports-related concussion? A Comparison of high school and collegiate athletes. *Journal of Pediatrics, 142(5),* 546-553.

Finch, C.F., McCrory, P., Ewing, M.T., & Sullivan, J. (2013). Concussion guidelines need to move from only expert content to also include implementation and dissemination strategies. *British Journal of Sports Medicine, 47,* 12-14.

Fisher, J. & Vaca, F. (2004). Sport-related concussion in the emergency department. *Top Emergency Medicine, 26,* 260-266.

Frencham, K.A.R., Fox, A.M., & Maybery, M.T. (2005). Neuropsychological studies of mild traumatic brain injury: A meta-analytic review of research since 1995. *Journal of Clinical and Experimental Neuropsychology, 27,* 334-351.

Frenguelli, A., Ruscito, P., Bicciolo, G., Rizzo, S., & Masserelli, M. (1991). Head and neck trauma in sporting activities. *Journal of Cranio-maxillofacial Surgery, 19,* 178-181.

Friedman, G., Froom, P., Sazbon, L., Grinblatt, I., Shochina, M., Tsenter, J., et al. (1999). Apolipoprotein E-epsilon4 genotype predicts a poor outcome in survivors of traumatic brain injury. *Neurology, 52,* 244-248.

Fuller, C.W., Ashton, T., Brooks, J.H., Cancea, R.J., Hall, J., & Kemp, S.P. (2010). Injury risks associated with tackling in rugby union. *British Journal of Sports Medicine, 44(3), 159*-167.

Fuller, C.W., Brooks, J.H., Cancea, R.J., Hall, J., & Kemp, S.P. (2007). Contact events in rugby union and their propensity to cause injury. *British Journal of Sports Medicine, 41:* 862-867.

Gabbett, T. (2003). Incidence of injury in semi-professional rugby league players. *British Journal of Sports Medicine, 37,* 36-44.

Gabbett, T. (2000). Incidence, site and nature of injuries in amateur rugby league over three consecutive seasons. *British Journal of Sports Medicine, 34,* 98-103.

Gabbett, T.J., Jenkins, D.G., & Abernethy, B. (2011). Physical collisions and injury in professional rugby league match-play. *Journal of Science, Medicine and Sport, 14(3),* 210-215.

Gaetz, M. (2004). The neurophysiology of brain injury. *Clinical Neurophysiology, 115,* 4-18.

Gaetz, M. & Bernstein, D.M. (2001). The current status of electrophysiologic procedures for the assessment of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 16(4),* 386-405.

Gaetz, M., Goodman, D., & Weinberg, H. (2000). Electrophysiological evidence for the cumulative effects of concussion. *Brain Injury, 14,* 1077-1088.

Gale, S.D., Johnson, S.C., Bigler, E.D., & Blatter, D.D. (1995). Nonspecific white matter degeneration following traumatic brain injury. *Journal of the International Neuropsychological Society, 1,* 17-28.

Gallus, J., & Mathiowetz, V. (2002). Brief Report—Test–retest reliability of the Purdue Pegboard for persons with multiple sclerosis. *American Journal of Occupational Therapy, 57,* 108–111.

Gardner, A., Shores, E.A., & Batchelor, J. (2010). Reduced processing speed in rugby union players reporting three or more previous concussions. *Archives of Clinical Neuropsychology, 25,* 174-181.

Garraway, M. & MacLeod, D. (1995). Epidemiology of rugby football injuries. *Lancet, 345*, 229-233.

Garraway, W.M., Lee, A.J., Macleod, D.A.D., Telfer, J.W., Deary, I.J., & Murray, G.D. (1999). Factors influencing tackle injuries in rugby union football. *British journal of Sports Medicine, 33,* 37-41.

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

Gennarelli, T.A., & Graham, D.I. (1998). Neuropathology of head injuries. *Seminars of Clinical Neuropsychiatry, 3,* 160-175.

Gennarelli, T.A., Thibault, L.E., Tomei, G., Wiser, R., Graham, D., & Adams, J. (1987). Directional dependence of axonal brain injury due to centroidal and non-centroidal

acceleration. Paper presented at the 31st Stapp Car Crash Conference, Society of Automotive Engineers.

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

Gilchrist, M.D. (2004). Experimental device for simulating traumatic brain injury resulting from linear accelerations. *Strain, 40,* 180-192.

Gissane, C., Jennings, G.C., & Standing, P. (1993). Incidence of injury in rugby league football. *Physiotherapy, 79,* 305-310.

Gissane, C., Jennings, G.C., & White, J. (1998). Injury in summer rugby league football: The experience of one club. *British Journal of Sports Medicine, 32,* 149-152.

Giza, C.C. & Hovda, D.A. (2004). The pathophysiology of traumatic brain injury. In Lovell, M., Echemendia, R.J., Barth, J.T., & Collins, M.W. (Eds.), *Traumatic Brain Injury in Sports* (pp. 45-69). The Netherlands: Swets & Zeitlinger Publishers.

Giza, C.C. & Hovda, D.A. (2001). The neurometabolic cascade of concussion. *Journal of Athletic Training, 36,* 228-235.

Gonzalez, P.G., & Walker, M.T. (2011). Imaging modalities in mild traumatic brain injury and sports concussion. *American Academy of Physical Medicine and Rehabilitation, 3,* S413-S424.

Grindel, S.H., Lovell, M.R., & Collins, M.W. (2001). The assessment of sport-related concussion: the evidence behind neuropsychological testing and management. *Clinical Journal of Sports Medicine, 11,* 134-143.

Gronwall, D. (1987). Advances in the assessment of attention and information processing after head injury. In H.S. Levin, J. Grafman, & H.M. Eisenberg (Eds.), *Neurobehavioural recovery from head injury*. (pp.355-371). New York: Oxford University Press.

Gronwall, D. (1989). Cumulative and persisting effects of concussion on attention and cognition. In H.M. Eisenberg, & A.L. Benton (Eds.), *Mild head injury* (pp.153-162). New York: Oxford University Press.

Gronwall, D. & Wrightson, P. (1974). Delayed recovery of intellectual functioning after minor head injury. *Lancet,2,* 605-609.

Gronwall, D. & Wrightson, P. (1975). Cumulative effects of concussion. *Lancet, 2*, 995-997.

Gronwall, D. & Wrightson, P. (1980). Duration of post-traumatic amnesia after mild head injury. *Journal of Clinical Neuropsychology, 2,* 51-60.

Guskiewicz, K.M. (2003). Assessment of postural stability following sport-related concussion. *Current Sport Medicine Report, 2(1),* 24-30.

Guskiewicz, K.M., Bruce, S.L., Cantu, R.C., Ferrara, M.S., Kelly, J.P., McCrea, M., et al. (2004). National Athletic Trainers' Association Position Statement: Management of Sport-Related Concussion. *Journal of Athletic Training, 39(3),* 280-297.

Guskiewicz, K.M., Marshall, S.W., Bailes, J., McCrea, M., Cantu, R.C., Randolph, C., et al. (2005). Association between recurrent concussion and late cognitive impairment in retired professional football players. *Neurosurgery, 57(4),* 719-26.

Guskiewicz, K.M., Marshall, S.W., Bailes, J., McCrea, M., Harding, J.R., Matthews, A., Mihalik, J.R. & Cantu, R.C. (2007). Recurrent concussion and risk of depression in retired professional football players. *Medical Science of Sports & Exercise, 39(6),* 903-909.

Guskiewicz, K.M., Marshall, S.W., Broglio, S.P., Cantu, R.C., & Kirkendall, D.T. (2002). No evidence of impaired neurocognitive performance in collegiate soccer players. *American Journal of Sports Medicine, 30(2),* 157-62.

Guskiewicz, K.M., McCrea, M., Marshall, S.W., Cantu, R.C., Randolph, C., Barr, W., et al. (2003). Cumulative effects associated with recurrent concussion in collegiate football players. *Journal of the American Medical Association, 290(19),* 2549-2555.

Guskiewicz, K.M., & Mihalik, J.R. (2011). Biomechanics of sport concussion: Quest for the elusive injury threshold. *Exercise and Sports Science Review, 39(11),* 4-11.

Guskiewicz, K.M., Mihalik, J.R., McCrory, P., McCrea, M., Johnston, K., Makdissi, M., Dvořák, J., Davis, G., & Meeuwisse, W. (2013). Evidence-based approach to revising the SCAT2: introducing the SCAT3. *British Journal of Sports Medicine, 47,* 289-293.

Guskiewicz, K.M., Mihalik, J.R., Shankar, V., Marshall, S.W., Crowell, D.H., Oliaro, S., et al. (2007). Measurement of head impacts in collegiate football players: Relationship between head impact biomechanics and acute clinical outcome after concussion. *Neurosurgery, 61(6),* 1244-1252.

Guskiewicz, K.M., Ross, S.E., & Marshall, S.W. (2001). Postural stability and neuropsychological deficits after concussion in collegiate athletes. *Journal of Athletic Training, 36(3),* 263-273.

Haier, R.J., Siegel, A.V., Nuechterlein, K.H., Hazlett, E., Wu, J.C., Pack, J., et al. (1988). Cortical glucose metabolic rate correlates of abstract reasoning and attention studies with positron emission tomography. *Intelligence, 12,* 199-217.

Halstead, M.E., & Walter, K.D. (2010). Sport-related concussion in children and adolescents. *Pediatrics, 126(3),* 597-615.

Halterman, C.I., Langan, J., Drew, A., Rodriguez, E., Osternig, L.R., Chou, L. et al. (2005). Tracking the recovery of visuo-spatial attention deficits in mild traumatic brain injury. *Brain, 129,* 747-753.

Hamill, J., & Knutzen, K.M. (1995). Biomechanical basis of human movement. Williams & Wilkins: A Waverley Company

Hartmann, P., Ramseier, A., Gudat, F., Mihatsch, M.J., & Polasek, W. (1994). Normal weight of the brain in relation to age, sex, body height and weight. *Pathology, 15(3),* 165-170.

Hawkins, R.D., & Fuller, C.W. (1998). An examination of the frequency and severity of injuries and incidents at three levels of professional football. *British Journal of Sports Medicine, 32,* 326-332.

Headminder. (2003). Retrieved October 30, 2010 from http://www.headminder.com

Helwick, C. (2013). Postconcussion self-report does not reflect recovery. *Medscape,* Oct 15.

Hinton-Bayre, A.D. & Geffen, G. (2002). Severity of sports-related concussion and neuropsychological test performance. *Neurology, 59,* 1068-1070.

Hinton-Bayre, A.D. & Geffen, G. (2004). Australian rules football and rugby league. In M.R. Lovell, R.L. Echemendia, J.T. Barth, & M.W. Collins (Eds.). *Traumatic Brain Injury in Sports.* The Netherlands: Swets & Zeitlinger Publishers.

Hinton-Bayre, A.D., Geffen, G., & Friis, P. (2004). Presentation and mechanisms of concussion in professional Rugby League football. *Journal of Science Medicine and Sport, 7(3),* 400-404.

Hinton-Bayre, A.D., Geffen, G., Geffen, L.B., McFarland, K.A., & Friis, P. (1999). Concussion in contact sports: reliable change indices of impairment and recovery. *Journal of Clinical Experimental Neuropsychology, 21(1),* 70-86.

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

Hodgson, V.R., Thomas, L.M., & Khalil, T.B. (1983). The role of impact location in reversible cerebral concussion. Paper presented at the 27th Stapp Car Crash Conference, Society of Automotive Engineers.

Hofman, P.A., Stapert, S.Z., van Kroonenburgh, M.J., Jolles, J., de Kruijk, J., & Wilmink, J.T. (2001). MR imaging, single-photon emission CT, and neurocognitive performance after mild traumatic brain injury. *American Journal of Neuroradiology, 22,* 441-449.

Holli, K.K., Harrison, L., Dastidar, P., Waljas, M., Ohman, J., Soimakallio, S., & Eskola, H. (2009). Texture analysis of corpus callosum in mild traumatic brain injury patients. *BMC Medical Imaging, 10(1),* 1471-2342.

Hoskins, W., Pollard, H., Hough, K., & Tully, C. (2006). Injury in rugby league. *Journal of Science and Medicine in Sport, 9,* 46-56.

Hovda, D.A., Prins, M., Becker, D.P., Lee, S., Bergsneider, M., & Martin, N.A. (1999). Neurobiology of concussion. In Bailes, J.E., Lovell, M.R., & Maroon, J.C. (Eds.), *Sports-Related Concussion* (pp. 327-332). St. Louis: Quality Medical Publishing Inc.

Howell, D.C. (1989). *Fundamental statistics for the behavioural sciences (2nd Ed.).* Boston: PWS-Kent Publishing Company.

Hsu, J.C. (1996). *Multiple Comparisons Theory and Methods*. London: Chapman & Hall.

Hughes, M.D., & Franks, I.M. (2004). Notational analysis of Sport 2nd Edition – a Perspective on improving coaching. London: E & F.N. Spon, (March).

ImPACT. (2005). Retrieved October 16, 2004 from http://www.impacttest.com

ImPACT. (2004). *Clinical Interpretation Manual for ImPACT 3.0.* Pittsburgh, USA: ImPACT.

Ingebrigtsen, T., Romner, B., & Kock-Jensen, C. (2000). Scandinavian guidelines for initial management of Minimal, Mild and Moderate head injuries. *The Journal of Trauma: Injury, Infection, and Critical Care, April,* 760-765.

Iverson, G.L. (2007). Predicting slow recovery from sport-related concussion: The new simple-complex distinction. *Clinical Journal of Sport Medicine, 17(1),* 31-37.

Iverson, G.L. (2005). Outcome from mild traumatic brain injury. *Current Opinion in Psychiatry, 18(3),* 301-317.

Iverson, G.L., Brooks, B.L., Lovell, M.R., Collins, M.W. (2006). No cumulative effects for one or two previous concussions. *British Journal of Sports Medicine, 40,* 72-75.

Iverson, G.L., Echemendia, R.J., LaMarre, A.K., Brooks, B.L., & Gaetz, M. (2012). Possible lingering effects of multiple past concussions. www.hindawi.com/journals/rerp/2012/316575

Iverson, G.L., Gaetz, M., Lovell, M.R., & Collins, M.W. (2004a). Cumulative effects of concussion in amateur athletes. *Brain Injury, 18(5),* 433-443.

Iverson, G.L., Gaetz, M., Lovell, M.R., & Collins, M.W. (2005). Validity of ImPACT for measuring processing speed following sports related concussion. *Journal of Clinical and Experimental Neuropsychology, 27,* 683-689.

Iverson, G.L., Lovell, M.R., & Collins, M.W. (2002a). Validity of ImPACT for measuring the effects of sports-related concussion. *Archives of Clinical Neuropsychology, 17(8),* 769.

Iverson, G.L., Lovell, M.R., & Collins, M.W. (2002b). Immediate Postconcussion Assessment and Cognitive Testing (ImPACT) Normative data version 2.0. Pittsburgh, USA: ImPACT Applications Inc.

Iverson, G.L., Lovell, M.R., Collins, M.W. (2003). Interpreting change on ImPACT following sport concussion. *The Clinical Neuropsychologist, 17(4),* 460-467.

Iverson, G.L., Lovell, M.R., Collins, M.W. (2005). Validity of ImPACT for measuring processing speed following sports-related concussion. *Journal of Clinical and Experimental Neuropsychology, 27,* 683-689.

Iverson, G., Lovell, M., Collins, M., & Norwig, J. (2002). Tracking recovery from concussion using ImPACT: Applying reliable change methodology. *Archives of Clinical Neuropsychology, 17,* 770.

Iverson, G.L., Lovell, M.R., & Smith, S.S. (2000). Does brief loss of consciousness affect cognitive functioning after mild head injury? *Archives of Clinical Neuropsychology, 15,* 643-648.

Jacobs, A., Put, E., Ingels, M., & Bossuyt, A. (1994). Prospective evaluation of technetium-99m-HMPAO SPECT in mild and moderate traumatic brain injury. *Journal of Nuclear Medicine, 35(6),* 942-947.

Jacobs, A., Put, E., Ingels, M., & Bossuyt, A. (1996). One year follow-up of Technetium-99m-HMPAO SPECT in mild head injury. *Journal of Nuclear Medicine, 37,* 1605-1609.

Jagaroo, V. (2009). *Neuroinformatics for neuropsychologists.* New York: Springer Science Medicine.

Jagoda, A..S., Bazarian, J.J., Bruns, J.J., Cantrill, S.V., Gean, A.D., Howard, P.K., et al. (2008). Clinical policy: Neuroimaging and decision-making in adult mild traumatic brain injury in the acute setting. *Annals of Emergency Medicine, 52(6),* 714-748.

Jakoet, I., & Noakes, T.D. (1998). A high rate of injury during the 1995 Rugby World Cup. *South African Medical Journal, 88(1),* 45-47.

Jay, G.W., Goka, R.S., & Arakaki, A.H. (1996). Minor traumatic brain injury: review of clinical data and appropriate evaluation and treatment. *Journal of Insurance Medicine, 27(4),* 262-282.

Jeffrey, S. (2013). AAN releases new sports concussion guidelines. *Medscape, March 18.*

Jeon, I., Kim, O., Kim, M., Kim, S., Chang, C., & Bai, D. (2008). The effect of premorbid demographic factors on the recovery of neurocognitive function in traumatic brain injury patients. *Journal of Korean Neurosurgical Society, 44,* 295-302.

Johnson, R.A. & Wichern, D.W. (2002). *Applied Multivariate Statistical Analysis,* 5th ed. (pp. 305-312). Upper Saddle River, New York: Prentice-Hall

Johnston, K.M., McCrory, P., Mohtadi, N.G., & Meeuwisse, W. (2001). Evidence-Based Review of Sport-Related Concussion: Clinical Science. *Clinical Journal of Sport Medicine, 11,* 150-159.

Jones, D.K., & Leemans, A. (2011). Magnetic Resonance Imaging. In M. Modo & J.W.M. Bulte (Eds.) *Diffusion Tensor Imaging.* (pp. 127-144). New York: Humana Press.

Jordan, B.D. (2004). Genetic aspects of traumatic brain injury. In M.R. Lovell, R.L. Echemendia, J.T. Barth, & M.W. Collins (Eds.). *Traumatic Brain Injury in Sports.* (pp. 358-374). The Netherlands: Swets & Zeitlinger Publishers.

Jordan, B.D. (1997). The shuttle effect: The development of a model for the prediction of variability in cognitive test performance across the adult life span. *Doctoral dissertation, Rhodes University, South Africa.*

Junge, A., Cheung, K., Edwards, T., & Dvořák, J. (2004). Injuries in youth amateur soccer and rugby players – comparison of incidence and characteristics. *British Journal of Sports Medicine, 38,* 168-172.

Kaplan, H.I., Sadock, B.J., & Grebb, J.A. (1994). Synopsis of psychiatry. Baltimore: Williams & Wilkins.

Kelly, J.P., Nichols, J.S., Filley, C.M., Lillehei, K.O, Rubinstein, D., & Kleinschmidt-DeMasters, B.K. (1991). Concussion in sports: guidelines for the prevention of catastrophic outcome. *Journal of the American Medical Association, 226*, 2867-2869.

Kelly, J.P. & Rosenberg, J.H. (1997). Diagnosis and management of concussion in sports. *Neurology, 48*, 575-580.

Kelly, J.P. & Rosenberg, J.H. (1998). The development of guidelines for the management of concussion in sports. *Journal of head Trauma Rehabilitation, 13(2),* 53-65.

Kemp, S.P., Hudson, Z., Brooks, J.H., & Fuller, C.W. (2008). The epidemiology of head injuries in English professional rugby union. *Clinical Journal of Sports Medicine, 18(3),* 227-234.

Kerr, H.A., Curtis, C., Micheli, L.J., Kocher, M.S., Zurakowski, D., Kemp, S.P., et al. (2008). Collegiate rugby union injury patterns in New England: A prospective cohort study. *British Journal of Sports Medicine, 42,* 595-603.

Kerr, R. (1982). Psychomotor learning. NY: CBS College Publishing.

Kesler, S.R., Adams, H.F., & Bigler, E.D. (2000). SPECT, MR and quantitative MR imaging: Correlates with neuropsychological and psychological outcome in traumatic brain injury. *Brain Injury, 14*, 851-857.

Kesler, S.R., Adams, H.F., Blasey, C.M., & Bigler, E.D. (2003). Premorbid intellectual functioning, education and brain size in traumatic brain injury: an investigation of the cognitive reserve hypothesis. *Applied Neuropsychology, 10,* 153-162.

Killam, C., Cautin, R.L., & Santucci, A.C. (2005). Assessing the enduring residual neuropsychological effects of head trauma in college athletes who participate in contact sports. *Archives of Clinical Neuropsychology, 20(5),* 599-611.

King, D., Brughelli, M., Hume, P., & Gissane, C. (2013). Concussions in amateur rugby union identified with the use of a rapid visual screening tool. *Journal of the Neurological Sciences,* http://dx.doi.org/10.1016/jj.jns.2013.01.012

King, D., Hume, P., & Clark, T. (2011). The effect of player positional groups on the nature of tackles that result in tackle-related injuries in professional rugby league matches. *Journal of Sports Medicine and Physical Fitness, 51(3),* 435-443.

Kirkendall, D.T., Jordan, S.E., & Garrett, W.E. (2001). Heading and head injuries in soccer. *Sports Medicine, 31,* 369-386.

Kirkwood, M.W., Randolph, C., & Yeates, K.O. (2009). Returning pediatric athletes to play after concussion: The evidence (or lack thereof) behind baseline neuropsychological testing. *Acta Paediatrica, 98(9),* 1409-1411.

Klein, M., Houx, P.J. & Jolles, J. (1996). Long-term persisting cognitive sequelae of traumatic brain injury and the effect of age. *Journal of Nervous and mental Disease, 184(8),* 459-467.

Koh, J.O., Cassidy, J.D., & Watkinson, E.J. (2003). Incidence of concussion in contact sports: a systematic review of the evidence. *Brain Injury, 17(10),* 901-917.

Kohler, R.M. (2004). Concussion in sport: Practical management guidelines for medical practitioners. *Continuing Medical Education, 22,* 122-125.

Konczak, J., & Timmann, D. (2007). The effect of damage to the cerebellum on sensorimotor and cognitive function in children and adolescents. *Neuroscience and Biobehavioural Reviews, 254(5),* 655-663.

Kontos, A.P., & Collins, M.W. (2013). Sport-related Concussion Research: Advances in Prognosis, Safer Return To Play, and Clinical Treatments. University of Pittsburgh School of Medicine Department of Orthopaedic Surgery Pittsburgh, Pennsylvania

Kontos, A.P., Elbin, R.J., Covassin, T., & Larson, E. (2010). Exploring differences in computerized neurocognitive concussion testing between African American and White Athletes. *Archives of Clinical Neuropsychology, 25(8),* 734-744.

Kozora, E. & Gerber, D. (2004). Special considerations and implications of neuropsychological testing in professional athletes. In M. Lovell, R.J. Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic Brain Injury in Sports* (pp. 358-374). The Netherlands: Swets & Zeitlinger Publishers.

Krull, K.R., Scott, J.G., & Sherer, M. (1995). Estimation of premorbid intelligence from combined performance and demographic variables. *The Clinical Neuropsychologist, 9,* 83-88.

Kurca, E., Sivak, S., & Kucera, P. (2006). Impaired cognitive functions in mild traumatic brain injury patients with normal and pathologic magnetic resonance imaging. *Neuroradiology, 48(9),* 661-669.

Kushner, D.S. (2001). Concussion in sports: Minimizing the risk for complications. *American Family Physician, 64(6),* 1007-1014.

Kutner, K.C., Erlanger, D.M., Tsai, J., Jordan, B., Relkin, N.R. (2000). Lower cognitive performance of older football players possessing Apolipoprotein E epsilon4. *Neurosurgery, 47(3),* 651-657.

Lakhan, S.E., & Kirchgessner, A. (2012). Chronic traumatic encephalopathy: the dangers of getting 'dinged'. *Springerplus 1.* Doi:10.1186/2193-1801-1-2.

Langlois, J.A., Rutland-Brown, W., & Wald, M.M. (2006). The epidemiology and impact of traumatic brain injury: a brief overview. *Journal of Head Trauma Rehabilitation, 21(5),* 375-378.

Leary, T., & White, J.A. (2000). Acute injury incidence in professional county club cricket players (1985-1995). *British Journal of Sports Medicine, 34,* 145-147.

Leclerc, S., Lassonde, M., Delaney, J.S., Lacroix, V., & Johnston, K.M. (2001). Recommendations for grading concussions in athletes. *Sports Medicine, 31(8),* 629-636.

Lehman, E.J., Hein, M.J., Baron, S.L., & Gersic, C.M. (2012). Neurodegenerative causes of death among retired National Football League players. *Neurology,*

Leibovici, D., Ritchie, K., Ledesert, B., & Touchon, J. (1996). Does education level determine the course of cognitive decline? *Age Ageing, 25(5),* 392-397.

Lendon, C.L., Harris, J.M., Pritchard, A.L., Nicoll, J.A., Teasdale, G.M., & Murray, G. (2003). Genetic variation of the APOE promoter and outcome after head injury, *Neurology, 61(5),* 683-685.

Levin, H.S., Amparo, E.G., Eisenberg, H.M., Williams, D.H., High, W.M., McArdle, C.B., & Weiner, D. (1987). Magnetic resonance imaging and computerized tomography in relation to neurobehavioural sequelae at mild and moderate head injury. *Journal of Neurosurgery, 66,* 706-713.

Levin, H.S. & Benton, A.L. (1986). Neuropsychological assessment. In Baker, A.B. & Joynt, R.J. (eds.), *Clinical Neurology,* Volume 1. New York: Harper & Row.

Levin, H.S., Benton, A.L., & Grossman, R.G. (1982). *Neurobehavioural consequences of closed head injury* (2nd ed.). New York: Oxford University Press.

Levin, H.S., Eisenberg, H.M. & Benton, A.L. (Eds.). (1989). *Mild Head Injury*. New York: Oxford University Press.

Levin, H.S., Mattis, S., Ruff, R.M., Eisenberg, H.M., Marshall, L.F., Tabaddor, K., et al. (1987). Neurobehavioural outcome following minor head injury: A three-center study. *Journal of Neurosurgery, 66,* 234-243.

Levin, H., & Smith, D. (2013). Traumatic brain injury: networks and neuropathology. *The Lancet Neurology, 12(1),* 15-16.

Lezak, M.D., Howieson, D.B., & Loring, D.W. (2004). *Neuropsychological Assessment.* (4th ed.). New York: Oxford University Press.

Lipton, M.L., Gulko, E., Zimmerman, M.E., Friedman, B.W., Kim, M., Gellella, E., et al. (2009). Diffusion-tensor imaging implicates prefrontal axonal injury in executive function impairment following very mild brain injury. *Radiology, 252,* 816-824.

Lishman, W.A. (1997). *Organic Psychiatry* (3rd Ed.). Oxford: Blackwell.

Lishman, W.A. (1988). Physiogenesis and psychogenesis in the 'post-concussional syndrome'. *British Journal of Psychiatry, 153,* 460-469.

Longo, U.G., Huijsmans, P.E., Maffulli, N., Denaro, V., De Beer, J.F. (2011). Video analysis of the mechanisms of shoulder dislocation in four elite rugby players. *Journal of Orthopaedic Science, 16(4),* 389-397.

Lovell, M.R. (2002). The relevance of neuropsychological testing for sports-related head injuries. *Current Sports Medicine Reports, 1,* 7-11.

Lovell, M.R. (2004). *ImPACT Version 3.0 Clinical User's Manual.* Pittsburgh, USA: ImPACT Inc.

Lovell, M. R. (2006). Letters to the editor. *Journal of Athletic Training, 41 (2),* 137-138.

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

Lovell, M.R. & Collins, M.W. (2002). New developments in the evaluation of sports-related concussion. *Current Sports Medicine Reports, 1,* 287-292.

Lovell, M.R., Collins, M.W., & Bradley, J. (2004). Return to play following sports-related concussion. *Clinical Sports Medicine, 23,* 421-441.

Lovell, M.R., Collins, M.W., & Fu, F.H. (2003). New technology and sports-related concussion. *Orthopedic Technology Review, 5(1),* 35.

Lovell, M.R., Collins, M.W., Iverson, G.L., Field, M., Maroon, J.C., Cantu, R., et al. (2003). Recovery form mild concussion in high school athletes. *Journal of Neurosurgery, 98,* 296-301.

Lovell, M.R., Collins, M.W., Iverson, G.L., Johnston, K.M., & Bradley, J.P. (2004a). Grade 1 or "ding" concussions in high school athletes. *Journal of Neurosurgery, 98(2),* 296-301.

Lovell, M.R., Collins, M.W., Pardini, J.E., Parodi, a., Yates, A. (2005). Management of cerebral concussion in military personnel: Lessons learned from sports medicine. *Operative Techniques in Sports Medicine, 13,* 212-221.

Lovell, M.R., Echemendia, R.J., Barth, J.T., & Collins, M.W. (2004). *Traumatic brain injury in sports.* The Netherlands : Swets & Zeitlinger

Lovell, M.R., Iverson, G., Collins, M., McKeag, D. & Maroon, J. (1999). Does loss of consciousness predict neuropsychological decrements after concussion? *Clinical Journal of Sports Medicine, 9,* 193-198.

Lovell M.R., Pardini, J.E., Welling, J., Collins, M.W., Bakal, J., Lazar, N., Roush, R., Eddy, W.F., & Becker, J.T. (2007). Functional brain abnormalities are related to clinical recovery and time to return-to-play in athletes. *Neurosurgery, 61(2),* 352-360.

Macciocchi, S.N. & Barth, J.T. (2004). Methodological concerns in traumatic brain injury. In M. Lovell, R.J. Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic Brain Injury in Sports* (pp. 281-298). The Netherlands: Swets & Zeitlinger Publishers.

Macciocchi, S.N., Barth, J.T., Alves, W., Littlefield, L., Jane, A., & Cantu, R.C. (2001). Multiple concussions and neuropsychological functioning in Collegiate Football Players. *Journal of Athletic Training, 36(3),* 303-306.

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

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

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

MacLeod, D.A.D. (1993). Risks and injuries in rugby football. In McLatchie, G.R., & Lennox, C.M.E. (Eds.), *Trauma and sport injuries (pp. 371-381).* London: Butterworth-Heinemann Ltd.

Maddocks, D.L. Dicker, G.D., & Saling, M.M. (1995). The assessment of orientation following concussion in athletes. *Clinical Journal of Sports Medicine, 5*, 32-35.

Maddocks, D.L. & Saling, M.M. (1996). Neuropsychological deficits following concussion. *Brain Injury, 10*, 99-103.

Mahoney, D. (2009). Mild brain injury can have long-term effects. *Family Practice News, Apr,* 39.

Maiden, D., & Dyson, M. (1997). An examination of the validity of the Manual Dexterity Test. *Australian Occupational Therapy Journal, 44,* 177-185.

Makdissi, M., Collie, A., Maruff, P., Darby, D.G., Bush, A., McCrory, P. & Bennell, K. (2001). Computerized cognitive assessment of concussed Australian Rules Footballers. *British Journal of Sports Medicine, 35(5),* 354-360.

Makdissi, M., Darby, D., Maruff, P., Ugoni, A., Brukner, P., & McCrory, P.R. (2010). Natural history of concussion in sport. *The American Journal of Sports Medicine, 38,* 464-471.

Malone, S. (2012). Brain injuries tied to contact sports share patterns. *Medscape, Dec 3.*

Marchi, N., Bazarian, J.J., Puvenna, V., Janigro, M., Ghosh, C., Zhong, J., Zhu, T., Blackman, E., Stewart, D., Ellis, J., Butler, R., & Janigro, D. (2013). Consequences of repeated blood-brain barrier disruption in football players. *PLoS ONE 8(3):* e56805. Doi:10.1371/journal.pone.0056805

Maroon, J.C., Field, M., Lovell, M., Collins, M., & Post, J. (2002). The evaluation of athletes with cerebral concussion. *Clinical Neurosurgery, 49*, 319-332.

Maroon, J.C., Lovell, M.R., Norwig, J., Podell, K., Powell, J.W., & Hartl, R. (2000). Cerebral concussion in athletes: Evaluation and neuropsychological testing. *Neurosurgery, 47(3),* 659-669.

Maroon, J.C., Steele, P.B., Berlin, R. (1980). Football head and neck injuries: an update. *Clinical Neurosurgery, 27*, 414-429.

Martin, G.N. (1998). Human Neuropsychology. London: Prentice Hall.

Mathias, J.L., Beall, J.A., & Bigler, E.D. (2004). Neuropsychological and information processing deficits following mild traumatic brain injury. *Journal of the International Neuropsychological Society, 10*, 286-297.

Mathias, J.L., & Wheaton, P. (2007). Changes in attention and information-processing speed following severe traumatic brain injury: A meta-analytic review. *Neuropsychology, 212*, 212-223.

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

Matser, J.T., Kessels, A.G., Lezak, M., Jordan, B.D., & Troost, J. (1999). Neuropsychological impairment in amateur soccer players. *Journal of the American Medical Association, 282(10)*, 971-973.

Matser, J.T., Kessels, A.G., Lezak, M., & Troost, J. (2001). A dose-response relation of headers and concussions with cognitive impairment in professional soccer players. *Journal of Clinical Experimental Neuropsychology, 23*, 770-774.

Matser, J.T., Kessels, A.G., Lovell, M.R. (2004). Soccer. In Lovell, M.R., Echemendia, R.J., Barth, J.T., & Collins, M.W. (Eds.), *Traumatic Brain Injury in Sports* (pp. 193-208). The Netherlands: Swets & Zeitlinger Publishers.

Mayers, L.B., & Redick, T.S. (2012). Clinical utility of ImPACT assessment for postconcussion return-to-play counseling: psychometric issues. *Journal of Clinical and Experimental Neuropsychology, 34(3),* 235-242.

McAllister, T.W. & Arciniegas, D. (2002). Evaluation and treatment of postconcussive symptoms. *Neurorehabilitation, 17*, 265-283.

McAllister, T.W., Flashman, L.A., Maerlender, A., et al. (2013). Cognitive effects of one season of head impacts in a cohort of collegiate contact sport athletes. *Neurology, 78,* 1777-1784.

McAllister, T.W., Saykin, A.J., Flashman, L.A., Sparling, M.B., Johnson, S.C., Guerin, S.J., et al. (1999). Brain activation during working memory one month after mild traumatic brain injury: A functional MRI study. *Neurology, 53,* 1300-1308.

McCaffrey, R. J., Mihalik, J.P., Crowell, D.H., Shields, E.W., & Guskiewicz, K.M. (2007). Measurement of head impacts in collegiate football players: Clinical measures of concussion after high- and low-magnitude impacts. *Neurosurgery, 61(6),* 1236-1243.

McCaffrey, R. J., Ortega, A., Orsillo, S. M., Nelles, W. B., & Haase, R. F. (1992). Practice effects in repeated neuropsychological assessments. *The Clinical Neuropsychologist, 6,* 32-42.

McClincy, M.P., Lovell, M.R., Pardini, J., Collins, M.W., & Spore, M.K. (2006). Recovery from sports concussion in high school and collegiate athletes. *Brain Injury, 20,* 33-39.

McCrea, M.A. (2008). Mild Traumatic Brain Injury and Postconcussion Syndrome. New York: Oxford University Press.

McCrea, M. (2001). Standardized mental status assessment of sports concussion. *Clinical Journal of Sports Medicine, 11,* 176-181.

McCrea, M., Barr, W.B., Guskiewicz, K., Randolph, C., Marshall, S.W., Cantu, R., Onate, J.A., & Kelly, J.P. (2005). Standard regression-based methods for measuring recovery after sport-related concussion. *Journal of the International Neuropsychological Society, 11,* 58-69.

McCrea, M., Guskiewicz, K.M., Marshall, S.W., Barr, W., Randolph, C., Cantu, R.C., et al. (2003). Acute Effects and Recovery Times Following Concussion in Collegiate Football Players. *Journal of the American Medical Association, 290(19),* 2556-6563.

McCrea, M., Guskiewicz, K.M., Randolph, C., Barr, W., Hammeke, T., Marshall, S.W., Powell, M.R., Ahn, K.W., Wang, Y., & Kelly, J.P. (2012). Incidence, clinical course and predictors of prolonged recovery time following sport-related concussion in high school and college athletes. *Journal of the International Neuropsychological Society, 18,* 1-12.

McCrea, M., Hammeke, T., Olsen, G., Leo, P., & Guskiewicz. K.M. (2005). Unreported concussion in high school football players: Implications for prevention. *Clinical Journal of Sports Medicine, 14(1),* 13-17.

McCrea, M., Iverson, G.L., Echemendia, R.J., Makdissi, M., & Raftery, M. (2013). Day of injury assessment of sport-related concussion. *British Journal of Sports Medicine, 47,* 272-284.

McCrea, M., Kelly, J.P., Kluge, J., Ackley, B., & Randolph, C. (1997). Standardized assessment of concussion in football players. *Neurology, 48,* 586-588.

McCrea, M., Kelly, J.P., Randolph, C., Cisler, R., & Berger, L. (2002). Immediate neurocognitive effects of concussion. *Neurosurgery, 50(5),* 1032-1041.

McCrea, M., Kelly, J.P., Randolph, C., Kluge, J., Bartolie, E., Finn, G., & Baxter, B. (1998). Standardized assessment of concussion (SAC). On site mental status evaluation of the athlete. *Journal of Head Trauma & Rehabilitation, 13,* 27-35.

McCrea, M., Prichep, L., Powell, M.R., Chabot, R., & Barr, W.B. (2010). Acute effects and recovery after sport-related concussion: A neurocognitive and quantitative brain electrical activity study. *Journal of Head Trauma Rehabilitation, 25(4),* 283-292.

McCrory, P.R. (1999). You can run but you can't hide: the role of concussion severity scales in sport. *British Journal of Sports Medicine, 33(5),* 297-298.

McCrory, P.R. (2001). Does second impact syndrome exist? *Clinical Journal of Sports Medicine, 11,* 144-149.

McCrory, P.R. (2002a). Treatment of recurrent concussion. *Current Sports Medicine Reports, 1,* 28-32.

McCrory, P.R. (2002b). What advice should we give to athletes postconcussion? *British Journal of Sports Medicine, 36,* 316-318.

McCrory, P.R. (2003). Brain injury and heading in soccer. *BMJ, 327,* 351-352.

McCrory, P.R. (2009). Summary and agreement statement of the 2nd International Conference on concussion in sport, Prague 2004. *British Journal of Sports Medicine, 39,* 196-204.

McCrory, P.R. & Berkovic, S.F. (1998a). Second impact syndrome. *Neurology, 50,* 677-683.

McCrory, P.R. & Berkovic, S.F. (1998b). Concussion Convulsions. *Sports Medicine, 25(2),* 131-136.

McCrory, P.R., Collie, A., Anderson, V., & Davis, G. (2004). Can we manage sports related concussion in children the same way as in adults? *British Journal of Sports Medicine, 38,* 516-519.

McCrory, P.R., & Johnston, K.M. (2002). Assessing prognostic significance. *The Physician and Sportsmedicine, 30(8),* 43-47.

McCrory, P.R., Johnston, K., Meeuwisse, W., Aubry, M., Cantu, R., Dvořák, J., et al. (2005). Summary and agreement statement of the 2nd International Conference on Concussion in Sport, Prague 2004. *Clinical Journal of Sports Medicine, 15(2),* 48-55.

McCrory, P., Meeuwisse, W., Aubry, M., Cantu, R., Dvorak, J., Echemedia, R. J. et al. (2013). Consensus statement on concussion in sport-the 4th international conference on concussion in sport held in Zurich, November 2012. *Clinical Journal of Sport Medicine, 23,* 89-117.

McCrory, P.R., Meeuwisse, W., Johnston, K., Dvořák, J., Aubry, M., Mollay, M.., Cantu, R. & Cantu, R. (2009). Consensus statement on concussion in sport: the 3rd International Conference on concussion in sport held in Zurich, Nov 2008. *Journal of Athletic Training, 44(4),* 434-448.

McIntosh, A.S., & McCrory, P., (2005). Preventing head and neck injury. *British Journal of Sports Medicine, 39,* 314-318.

McIntosh, A.S., McCrory, P., & Comerford, J. (2000). The dynamics of concussive head impact in rugby and Australian rules football. *Medicine & Science in Sports and Exercise, February,* 1980-1984.

McIntosh, A.S., McCrory, P., Finch, C.F., & Wolfe, R. (2010). Head, face and neck injury in youth rugby: incidence and risk factors. *British Journal of Sports Medicine, 44,* 188-193.

McIntosh, A.S., Savage, T.N., McCrory, P., Fréchède, B.O., & Wolfe, R. (2010). Tackle characteristics and injury in a cross section of rugby union football. *Medical Science of Sports Exercise, 42(5),* 977-984.

McKee, A.C., Cantu, R.C., Nowinski, C.J., Hedley-Whyte, E.T., Gavett, B.E., Budson, A.E., et al. (2009). Chronic traumatic encephalopathy in athletes: Progressive tauopathy after repetitive head injury. *Journal of Neuropathology and Experimental Neurology, 68(7),* 709-735.

McKee, A.C., Stein, T.D., Nowinski, C.J., Stern, R.A., Daneshvar, D.H., Alvarez, V.E., Lee, H., et al. (2013). The spectrum of disease in chronic traumatic encephalopathy. *Brain, 136,* 43-64.

McKenzie, A., Hodge, K. & Sleivert, G. (2000). Smart training for rugby: A complete guide for rugby players and coaches. Auckland, NZ: Reed.

McLean, D.A. (1992). Analysis of the physical demands of international rugby union. *Journal of Sports Science, 10,* 285-296.

McManus, A. (2006). Management of brain injury in non-elite field hockey and Australian football – a qualitative study. *Health Promotion Journal of Australia, 17(1),* 67-69.

McNarry, AF & Bateman, DN (2004). Simple bedside assessment of level of consciousness: comparison of two simple assessment scales with the Glasgow Coma scale. *Anaesthesia, 59 (1),*1365-2044.

Mendez, C.V., Hurley, R.A., Lassonde, M., Zhang, L., & Taber, K.H. (2005). Mild traumatic brain injury: Neuroimaging of sports-related concussion. *Journal of Neuropsychiatry and Clinical Neurosciences, 17(3),* 297-303.

Meehan III, W.P., d'Hemecourt, P., Collins, C.L., Taylor, A.M., & Comstock, D. (2012). Computerized neurocognitive testing for the management of sport-related concussions. *Pediatrics, 129,* 38-44.

Micheli, P.T. & Riseborough, E.M. (1974). The incidence of injuries in rugby football. *Journal of Sports Medicine, 2,* 93-97.

Mihalik, J.P., McCaffrey, M.A., Rivera, E.M., Pardini, J.E., Guskiewicz, K.M., Collins, M.W., & Lovell, M.R. (2007). Effectiveness of mouthguards in reducing neurocognitive deficits following sports-related cerebral concussion. *Dental Traumatology, 23: 14-20.*

Miller, J.R., Adamson, G.J., Pink, M.M., & Sweet, J.C. (2007). Comparison of preseason, midseason, and postseason neurocognitive scores in uninjured collegiate football players. *American Journal of Sports Medicine, 35(8),* 1284-1288.

Mittl, R., Grossman, R., Hiehle, J., Hurst, R., Kauder, D, Gennarelli, T., et al. (1994). Prevalence of MR evidence of diffuse axonal injury in patients with mild head injury and normal CT findings. *American Journal of Neuroradiology, 15,* 1583-1589.

Mortimer, J.A. (1997). Brain reserve and the clinical expression of Alzheimer's disease. *Geriatrics, 52 (Suppl. 2),* 50-53.

Mortimer, J.A., French, L.R., Hutton, J.T., & Schuman, L.M. (1985). Head injury as risk Factor for Alzheimer's disease. *Neurology, 35,* 264-267.

Mortimer, J.A., & Graves, A.B. (1993). Education and other socioeconomic determinants of dementia and Alzheimer's disease. *Neurology, 43(Suppl4),*39-44.

Mortimer, J.A., Van Duijn, C.M., Fratglioni, L., Graves, A.B., Heyman, A., Jorm, A.F., Kokmen, E., Kondo, K., Rocca, W.A., Shalat, S.L., Soininen, H. & Hofman, A. (1991). Head trauma as a risk factor for Alzheimer's disease: A collaborative re-analysis of case-control studies. *International Journal of Epidemiology, 20(2)(Suppl2),* 28-35.

Moser, R.S., Schatz, P., & Jordan, B.D. (2005). Prolonged effects of concussion in high school athletes. *Neurosurgery, 57,* 300-306.

Moser, R. S., Iverson, G. L., Echemendia, R. J., Lovell, M. R., Schatz, P., Webbe, F. M. et al. (2007). NAN position paper. Neuropsychological evaluation in the diagnosis and management of sports concussion. *Archives of Clinical Neuropsychology, 22,* 909-916.

Mrazik, M., Ferrara, M.S., Perterson, C.L., Elliott, R.E., Courson, R.W., Clanton, M.D., *et al*. (2000). Injury severity and neuropsychological and balance outcomes of four college athletes. *Brain Injury, 14(10),* 921-931.

Mushkudiani, N.A., Engel, D.C., Steyerberg, E.W., Butcher, I., Lu, J., Marmarou, A., et al. (2007). Prognostic value of demographic characteristics in traumatic brain injury: results from the IMPACT study. *Journal of Neurotrauma, 24,* 259-269.

Myers, P.T. (1980). Injuries presenting from rugby union football. *The Medical Journal of Australia, 2,* 17-20.

National Center for Injury Prevention and Control. (2007). Report to congress on mild traumatic brain injury in the United States: Steps to prevent a serious public health problem. Atlanta, GA: Centers for Disease Control and Prevention.

Nelson, W.E., Jane, J.A., & Gieck, J.H. (1984). Minor head injury in sports: a new system of classification and management. *The Physician and Sportsmedicine, 12(3),* 103-107.

Nesselroade, J.R., & Salthouse, T.A. (2004). Methodological and theoretical implications of intra-individual variability in perceptual-motor performance. *Journal of Gerontology Series B: Psychological Science Society, 59,* 49-55.

Newcombe, F., Rabbitt, P., & Briggs, M. (1994). Minor head injury: pathophysiology or iatrogenic sequelae? *Journal of Neurology, Neurosurgery, and Psychiatry, 57*, 709-716.

Nicholl, J., & LaFrance, W.C. (2009). Neuropsychiatric sequelae of traumatic brain injury. *Seminars in Neurology, 29(3),* 247-255.

Nicol, A., Pollock, A., Kirkwood, G., Parekh, N., & Robson, J. (2010). Rugby union injuries in Scottish schools. *Trauma, 12(4),* 221-238.

Noakes, T & Du Plessis, M. (1996). Rugby without risk – A practical guide to the prevention and treatment of rugby injuries. Pretoria: J.L. Van Schaik Publishers.

Nortje, J. & Menon, D.K. (2004). Traumatic brain injury: physiology, mechanisms, and outcome. *Current Opinion in Neurology, 17*, 711-718.

Omalu, B., Bailes, J., Hamilton, R.L., et al. (2011). Emerging histomorphologic phenotypes of chronic traumatic encephalopathy in American athletes. *Neurosurgery, 69,* 173-183.

Omalu, B.I., Hamilton, R.L., Kamboh, M.I., DeKosky, S.T., Bailes, J. (2010). Chronic traumatic encephalopathy (CTE) in a National Football League player: case report and emerging medicolegal practice questions. *Journal of Forensic Nursing,* 6, 40 – 46.

Ommaya, A.K. (1996). Head injury mechanisms and the concept of preventive management: A review and critical synthesis. *Journal of Neurotrauma, 12,* 527-546.

Ommaya, A.K. & Gennarelli, T.A. (1974). Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. *Brain, 97,* 633-654.

O'Neil, B., Naunheim, R.S., Prichep, L.S., & Chabot, R.J. (2011). Can quantitative brain electrical activity aid in the triage of mild traumatic brain injured patients. *Annals of Emergency Medicine, 58,* S228.

Orchard, J., James, T., Alcott, E., Carter, S., & Farhart, P. (2002). Injuries in Australian cricket at first class level 1995/1996 to 2000/2001. *British Journal of Sports medicine, 36,* 270-274.

Ostrosky-Solis, F. (2004). Can literacy change brain anatomy? *International Journal of psychology, 39(1),* 1-4.

Oxford Concise Colour Medical Dictionary. (1996). Oxford: Oxford University Press.

Parkinson, D. (2000). Concussion confusion. *Critical Reviews in Neurosurgery, 9,* 335-339.

Parsons, T.D., Notebaert, A.J., Shields, E.W., & Guskiewicz, K.M. (2009). Application of reliable change indices to computerized neuropsychological measures of concussion. *International Journal of Neuroscience, 119,* 492–507.

Peers, I. (1996). Statistical Analysis for Education and Psychology Researchers. London: The Falmer Press.

Pellman, E.J., Lovell, M.R., Viano, D.C., Casson, I.R., & Tucker, A.M. (2004). Concussion in Professional Football: Neuropsychological Testing – Part 6. *Neurosurgery, 55(6),* 1290-1305.

Pellman, E.J., Lovell, M.R., Viano, D.C., & Casson, I.R. (2006). Concussion in Professional Football: Recovery of NFL and high school athletes assessed by computerized neuropsychological testing–Part 12. *Neurosurgery, 58(2),* 1-10.

Pellman, E.J., Powell, J.W., Viano, D.C., Casson, I.R., Tucker, A.M., Feuer, H., Lovell, M.R., Waeckerle, J.F., & Robertson, D.W. (2004). Concussion in professional football: Epidemiological features of game injuries ad review of the literature Part 3. *Neurosurgery, 54,* 81-96.

Pellman, E.J., Viano, D.C., Tucker, A.M., Casson, I.R., & Waeckerle, J.F. (2003). Concussion in professional football: Reconstruction of game impacts and injuries. *Neurosurgery, 53(4),* 799-812.

Perneger, T.V. (1998). What's wrong with Bonferroni adjustments. *British Medical Journal, 316,* 1236-1238.

Petchprapai, N., & Winkelman, C. (2008). Mild traumatic brain injury: Determinants and subsequent quality of life: A review of the literature. *The Journal of Neuroscience nursing, 39,* 260-272.

Pettersen, J.A. & Skelton, R.W. (2000). Glucose enhances long-term declarative memory in mild head-injured varsity rugby players. *Psychobiology, 28,* 81-89.

Podell, K. (2004). Computerized assessment of sports-related brain injury. In M.R. Lovell, M.R., Echemendia, R.L., Barth, J.T., & Collins, M.W. (Eds.), *Traumatic Brain Injury in Sports.* The Netherlands: Swets & Zeitlinger Publishers.

Poirier, M.P. (2003). Concussions: Assessment, management and recommendations for return to activity. *Clinical Pediatric Emergency Medicine, 4,* 179-185.

Poirier, M.P., & Wadsworth, M.R. (2000). Sports-related concussions. *Pediatric Emergency Care, 16(4),* 278-283.

Ponsford, J., Wilmott, C., Rothwell, A., Cameron, P., Kelly, A., Nelms, R., et al. (2000). Factors influencing outcome following mild traumatic brain injury in adults. *Journal of the International Neuropsychological Society, 6,* 568-579.

Pontifex, M.B., O'Connor, P.M., Broglio, S.P., & Hillman, C.H. (2009). The association between mild traumatic brain injury history and cognitive control. *Neuropsychologia, 47,* 3210-3216.

Powell, J.W. (2004). Diagnosis, management and prevention. In M.R. Lovell, R.J. Echemendia, J.T. Barth, & M.W. Collins (Eds.). *Traumatic brain injury in sports* (pp. 23-33). The Netherlands: Swets & Zeitliner Publishers.

Preece, M., & Geffen, G.M. (2007). The contribution of pre-existing depression to the acute cognitive sequelae of mild traumatic brain injury. *Brain Injury, 21(9),* 951-961.

Pretz, L.C. (2007). *Assessment of risk factors in sports-related concussion: Incidence rate and recovery patterns.* Retrieved August 2, 2011, from http://rave.ohiolink.edu/etdc/view?acc_num.miami1177086308

Prichep, L.S., McCrea, M., Barr, W., Powell, M., & Chabot, R.J. (2012). Time course of clinical and electrophysiological recovery after sport-related concussion. *Journal of Head Trauma Rehabilitation,* May 14: PMID 22588360.

Prigatano, G.P. (1986). Neuropsychological rehabilitation after brain injury. London: The John Hopkins University Press.

Provenzano, F.A., Jordan, B., Tikofsky, R.S., Saxena, C., van Heerlum, R.L., & Iehise, M. (2010). F-18 FDG PET imaging of chronic traumatic brain injury in boxers: a statistical parametric analysis. *Nuclear Medicine Communication, 31(11),* 952-957.

Publication Manual of the American Psychological Association, 6th ed. Washington, DC: American Psychological Association, 2010.

Pugh, K.A., & Lipsitz, L.A. (2002). The microvascular frontal-subcortical syndrome of aging. *Neurobiology of Aging, 23,* 421-431.

Purdue Pegboard. (2002). *Instructions and Normative data for Model 32020.* Lafayette Instrument Company: Lafayette, USA.

Putukian, M., & Echemendia, R.J. (1996). Managing successive minor head injuries: which tests guide return to play? *The Physician and Sportsmedicine, 24(11),* 25-38.

Putukian, M., Echemendia, R.J., & Machin, S. (2000). The acute neuropsychological effects of heading in soccer: A pilot study. *Clinical Journal of Sports Medicine, 10,* 104-109.

Putukian, M., Raftery, M., Guskiewicz, K., Herring, S., Aubry, M., Cantu, R.C., & Molloy, M. (2013). Onfield assessment of concussion in the adult athlete. *British Journal of Sports Medicine, 47,* 285-288.

Quarrie, K.L., & Hopkins, W.G. (2008). Tackle injuries in professional Rugby Union. *American Journal of Sports Medicine, 36(9),* 1705-1716.

Rabadi, M.H. & Jordan, B.D. (2001). The cumulative effect of repetitive concussion in sports. *Clinical Journal of Sport Medicine, 11,* 194-198.

Raghupathi, R. (2004). Cell death mechanisms following traumatic brain injury. *Brain Pathology, 14,* 215-222.

Rahnama, N., Reilly, T., & Lees, A. (2002). Injury risk associated with playing actions during competitive soccer. *British Journal of Sports Medicine, 36,* 354-359.

Randolph, C. (2001). Implementation of neuropsychological testing models for the high school, collegiate and professional sport setting. *Journal of Athletic Training, 36(3),* 288-296.

Randolph, C. (2011). Baseline neuropsychological testing in managing sport-related concussion: Does it modify risk? *Current sports Medicine Reports 10(1),* 21-26.

Randolph, C., McCrea, M., & Barr, W.B. (2005). Is neuropsychological testing useful in the management of sports-related concussion? *Journal of Athletic Training, 40(3),* 139-154.

Rangel-Castilla, L., Gasco, J., Hanbali, F., & Salinas, P. (2008). Closed head trauma. http://emedicine.medscape.com/article/251834

Raskin, S.A., Mateer, C.A., & Tweeten, R. (1998). Neuropsychological assessment of Individuals with mild traumatic brain injury. *The Clinical Neuropsychologist, 12(1),* 21-30.

Rassovsky, Y., Satz, P., Alfano, M.S., Light, R.K., Zaucha, K., McArthur, D.L., & Hovda, D. (2006). Functional outcome in TBI I: Neuropsychological, emotional and behavioural mediators. *Journal of Clinical and Experimental Neuropsychology, 28,* 567-580.

Ravdin, L.D., Barr, W.B., Jordan, B., Lathan, W.E., & Relkin, N.R. (2003). Assessment of cognitive recovery following sports related head trauma in boxers. *Clinical Journal of Sports Medicine, 13,* 21-27.

Rees, P.M. (2003). Contemporary issues in Mild Traumatic Brain injury. *Archives of Physical Medicine and Rehabilitation, 84,* 1885-1894.

Reeves, D., Winter, K.P., Bleiberg, J. & Kane, R.L. (2007). ANAM® Genogram: Historical perspectives, description, and current endeavors. *Archives of Clinical Neuropsychology, 22(1),*15-37.

Reitan, R.M. & Wolfson, D. (1985). *The Halstead-Reitan neuropsychological test battery*. Tucson, AZ: Neuropsychology Press.

Reitan, R.M. & Wolfson, D. (1997). The influence of age and education on Neuropsychological performances of persons with mild head injuries. *Applied Neuropsychology, 4(1),* 16-33.

Reitan, R.M. & Wolfson, D. (1999). The two faces of Mild Head Injury. *Archives of Clinical Neuropsychology, 14(2),* 191-202.

Reitan, R.M. & Wolfson, D. (2000). The neuropsychological similarities of mild and more severe head injury. *Archives of Clinical Neuropsychology, 15(5),* 433-442.

Rimel, R.W., Giordani, B., Barth, J.T., Boll, T.J., & Jane, J.A. (1981). Disability caused by minor head injury. *Neurosurgery, 9(3),* 221-228.

Roberts, M. (2011). Footballers: Too many headers 'can damage the brain'. *BBC News, 29 November 2011.*

Rochester, J., Deutsch, B., Nicholas, A., & Lowery, R. (2006). *Prevalence of concussion among NMU football players: A ten year analysis.* Paper presented at the ATR 490 Seminar in Athletic Training. North Michigan University, December 2006.

Ropper, A.H., & Gorson, K.C. (2007). Concussion. *New England Journal of Medicine, 356,* 166-172.

Ruchinskas, R.A., Francis, J.P., & Barth, J.T. (1997). Mild Head Injury in Sports. *Applied Neuropsychology, 4(1),* 43-49.

Rucklidge, J.J., & Tannock, R. (2002). Neuropsychological profiles of adolescents with ADHD: Effects of reading difficulties and gender. *Journal of Child Psychology and Psychiatry, 43,* 988-1003.

Ruff, R.M. (2005). Two decades of advances in understanding of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 20(3),* 5-18.

Ruff, R.M., Evans, R., & Marshall, L.F. (1986). Impaired verbal and figural fluency after head injury. *Archives of Clinical Neuropsychology, 1,* 87-101.

Rutherford, A., Stephens, R., & Potter, D. (2003). The neuropsychology of heading and head trauma in association football (soccer): A Review. *Neuropsychology Review, 13(3),* 153-179.

Rutherford, A., Stephens, R., Potter, D., & Fernie, G. (2005). Neuropsychological impairment as a consequence of football (soccer) play and football heading: Preliminary analyses and report on university footballers. *Journal of Clinical and Experimental Neuropsychology, 27,* 299-319.

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

Satz, P. (2001). Mild head injury in children and adolescents. *Current Directions in Psychological Science, 10(3),* 106-109.

Satz, P.S., Alfano, M.S., Light, R.F., Morgenstern, H.F., Zaucha, K.F., Asarnow, R.F., et al. (1999). Persistent Post-Concussive Syndrome: A proposed methodology and literature review to determine the effects, if any, of mild head and other bodily injury. *Journal of Clinical and Experimental Neuropsychology, 21(5),* 620-628.

Satz, P.S., Cole, M.A., Hardy, D.J., & Rassovsky, Y. (2010). Brain and cognitive reserve: Mediator(s) and construct validity, a critique. *Journal of Clinical and Experimental Neuropsychology, 2,* 1-10.

Saulle, M., & Greenwald, B.D. (2012). Chronic traumatic encephalopathy: a review. *Rehabilitation Research Practice,* 816069.

Saunders, R.L. & Harbaugh, R.E. (1984). The second impact in catastrophic contact-sports head trauma. *Journal of the American Medical Association, 252,* 538-539.

Schatz, P. & Browndyke, (2002). Applications of computer-based neuropsychological assessment. *Journal of Head Trauma Rehabilitation, 17(5),* 395-410.

Schatz, P., Pardini, J., Lovell, M., Collins, M. & Podell, K. (2006). Sensitivity and Specificity of the ImPACT Test Battery for concussion in athletes. *Archives of Clinical Neuropsychology, 21,* 91-99.

Schatz, P. & Sandel, N. (2013). Sensitivity and Specificity of the online version of ImPACT in high school and collegiate athletes. *American Journal of Sports Medicine, 41,* 321-326.

Schatz, P. & Zilmer, E. (2003). Computer-based assessment of sports-related concussions. *Applied Neuropsychology, 10(1),* 42-47.

Scher, A.T. (1987). Rugby Injuries of the spine and spinal cord. *Clinics in Sports Medicine, 6(1),* 87-99.

Schneiders, A.G., Takemura, M., & Wassinger, C.A. (2009). A prospective epidemiological study of injuries to New Zealand premier club rugby union players. *Physical Therapy in Sport, 10(3),* 85-90.

Schoenberg, M.R., Scott, J.G., Duff, K., & Adams, R.L. (2002). Estimation of WAIS-III intelligence from Combined Performance and Demographic Variables: Development of the OPIE-3. *The Clinical Neuropsychologist, 16(4),* 426-438.

Schretlen, D.J. & Shapiro, A.M. (2003). A quantitative review of the effects of traumatic brain injury on cognitive functioning. *International Review of Psychiatry, 15(4),* 341-349.

Schwartz, A. (2009). NFL acknowledges long-term concussion effects. *New York Times, December 20, 2009.*

Schwartz, A. (2010). NFL asserts greater risks of head injury. *New York Times, July 26, 2010.*

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

Shallice, T. (1988). From Neuropsychology to Mental Structure. Cambridge University Press: Cambridge.

Shaw, N.A. (2002). The neurophysiology of concussion. *Progress in Neurobiology, 67(4),* 281-344.

Sherrill-Parrison, S., Donders, J., & Thompson, E. (2000). Influence of demographic variables on neuropsychological test performance after traumatic brain injury. *Clinical Neuropsychology, 14,* 496-503.

Shores, E.A., Lammél, A., Hullick, C., Sheedy, J., Flynn, M., Levick, W. & Batchelor, J. (2008). The diagnostic accuracy of the revised Westmead PTA Scale as an adjunct to the Glasgow Coma Scale in the early identification of cognitive impairment in patients with mild traumatic brain injury. *Journal of Neurology, Neurosurgery and Psychiatry, 79,* 1100-1106.

Shuttleworth-Edwards, A.B. (2008). Central or peripheral? A positional stance in reaction to the Prague statement on the role of neuropsychological assessment in sports concussion management. *Archives of Clinical Neuropsychology, 23,* 479-485.

Shuttleworth-Edwards, A.B., Border, M., Reid, I., & Radloff, S. (2004). South African Rugby Union. In M.R. Lovell, R.L. Echemendia, J.T. Barth, & M.W. Collins (Eds.). *Traumatic Brain Injury in Sports: An international neuropsychological perspective* (pp. 149-168). The Netherlands: Swets & Zeitlinger Publishers.

Shuttleworth-Edwards, A.B., Kemp, R., Rust, A., Muirhead, J., Hartman, N., & Radloff, S. (2004). Cross-cultural effects on IQ test performance: A review and preliminary normative indications on WAIS-III test performance. *Journal of Clinical and Experimental Neuropsychology, 26,* 903-920.

Shuttleworth-Edwards, A.B., Noakes, T.D., Whitefield, V., Roberts, J.C.O., Clark, S., Essack, F., Zoccola, D., Boulind, M., Case, S., & Mitchell, J. (2008). Incidence of concussive injury reported for follow-up management in South African Rugby Union. *Clinical Journal of Sport Medicine, 18(15),* 403-409.

Shuttleworth-Edwards, A.B., & Radloff, S. (2008). Compromised visuomotor processing speed in players of Rugby Union from school through to the adult national level. *Archives of Clinical Neuropsychology, 23(5),* 511-520.

Shuttleworth-Edwards, A.B., Radloff, S.E., Whitefield-Alexander, V.J., Smith, I., & Horsman, M. (2013, 4 August). Practice effects reveal visuomotor vulnerability on school and university rugby players. *Archives of Clinical Neuropsychology.* E-pub ahead of print.

Shuttleworth-Edwards, A.B., Smith, I., & Radloff, S. (2008). Neurocognitive vulnerability amongst university rugby players versus non-contact sport controls. *Journal of Clinical and Experimental Neuropsychology, 30(8),* 870-884.

Shuttleworth-Edwards, A.B., & Whitefield, V.J. (2007). Ethically we can no longer sit on the fence – a neuropsychological perspective on the cerebrally hazardous contact sports. *South African Journal of Sports Medicine, 19(2),* 32-38.

Shuttleworth-Edwards, A.B., Whitefield-Alexander, V.J. (2013). The ImPACT neurocognitive screening test: A survey of South African research, current and projected ethically condoned applications. In K. Cockcroft & S. Laher (Eds.). *Psychological Assessment in South Africa: Research and Applications.* (pp. 443-460). Johannesburg: Wits Press.

Shuttleworth-Jordan, A.B. (1997). Age and education effects on brain damaged subjects: "Negative" findings revisited. *The Clinical Neuropsychologist, 11,* 205-209.

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

Shuttleworth-Jordan, A.B., Puchert, J., & Balarin, E. (1993). Negative consequences of mild head injury in rugby: a matter worthy of concern. In R. Plunkett, & S. Anderson, (Eds.).

Proceedings of the 5th National Neuropsychology Conference (pp. 38-68). Durban: South African Clinical Neuropsychological Association (SACNA), University of Natal.

Signoretti, S., Vagnozzi, R., Tavazzi, B., & Lazzarino, G. (2010). Biochemical and neurochemical sequelae following mild traumatic brain injury: Summary of experimental data and clinical implications. *Neurosurgical Focus, 29(5),* e1.

Silver, J.M., McAllister, T.W., & Yodofsky, S.C. (2005). Textbook of Traumatic Brain Injury. Arlington VA: American Psychiatric Publishing.

Sim, A., Terryberry-Spohr, L., & Wilson, K. R. (2008). Prolonged recovery of memory functioning after mild traumatic brain injury in adolescent athletes. *Journal of Neurosurgery, 108(3),* 511-516.

Small, G.W., Kepe, V., Siddarth, P., Ercoli, L.M., Merrill, D.A., Donoghue, N., Bookheimer, S.Y., Martinez, J., Omalu, B., & Bailes, J. (2013). PET Scanning of Brain Tau in Retired National Football League Players: Preliminary Findings. *The American Journal of Geriatric Psychiatry, 21*(2), 138-144.

Smith, D.H., & Meaney, D.F. (2003). Diffuse axonal injury in head trauma. *Journal of Head Trauma Rehabilitation, 18(4),* 307-316.

Smits, M., Dippel, D.W., Houston, G.C., Wielopolski, P.A., Koudstaal, P.J., Hunink, M.G., et al. (2008). Postconcussion syndrome after minor head injury: Brain activation of working memory and attention. *Human Brain Mapping, 30,* 2789-2803.

Solomon, G., Ott, D., & Lovell, M. (2011). Long-term neurocognitive dysfunction in sports: What is the evidence? *Clinics in Sports Medicine, 30,* 165-177.

Sosnoff, J.J., Broglio, S.P., & Ferrara, M.S. (2008). Cognitive and motor function are associated following mild traumatic brain injury. *Experimental Brain Research, 187,* 563-571.

Sosnoff, J.J., Broglio, S.P., Hillman, C.H., & Ferrara, M.S. (2007). Concussion does not impact intra-individual response time variability. *Neuropsychology, 21(6),* 796-802.

Sosin, D.M., Sniezek, J.E., & Thurman, D.J. (1996). Incidence of mild and moderate brain Injury in the United States, 1991. *Brain Injury, 10,* 47-54.

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

Spreen, O., & Straus, E. (1998). *A compendium of neuropsychological tests* (2nd ed.). New York: Oxford University Press.

Stablum, F. Mogentale, C., Umilta, C. (1996). Executive functioning following mild closed head injury. *Cortex, 32,* 261-278.

Stein, S.C., & Spettell, C. (1995). The head injury severity scale (HISS): a practical classification of closed-head injury. *Brain Injury, 9,* 437-444.

Stephens, R., Rutherford, A., Potter, D., & Fernie, G. (2010). Neuropsychological consequence of soccer play in adolescent UK school team soccer players. *The Journal of Neuropsychiatry and Clinical Neurosciences, 22,* 295-303.

Stephens, R., Rutherford, A., Potter, D., & Fernie, G. (2005). Neuropsychological impairment as a consequence of football (soccer) play and football heading: A preliminary analysis and report on school students (13-16 years). *Child Neuropsychology, 11(6),* 513-526.

Stephenson, S., Gissane, C., & Jennings, D. (1996). Injury in rugby league: A four year prospective study. *British Journal of Sports Medicine, 30,* 331-334.

Stern, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society, 8(3),* 448-460.

Stern, Y. (2003). The concept of cognitive reserve: a catalyst for research. *Journal of Clinical and Experimental Neuropsychology, 25,* 589-593.

Stern, Y. (2006). Cognitive Reserve. *Theory and Applications*. New York: Taylor & Francis.

Stern, Y. (2009). Cognitive Reserve. *Neuropsychologia, 47(10),* 2015-2028.

Stern, Y., Zarahn, E., Hilton, H.J., Flynn, J., DeLaPaz, R., & Rakitin, B. (2003). Exploring the neural basis of cognitive reserve. *Journal of Clinical and Experimental Neuropsychology, 25,* 691-701.

Stewart, W., Gordon, B., Selnes, O., Bandeen-Roche, K., Zeger, S., Tusa, R.J., et al. (1994). Prospective study of central nervous system function in amateur boxers in the United States. *American Journal of Epidemiology, 139,* 573-588.

Stewart, G.W., McQueen-Borden, E., Bell, R.A., Barr, T., & Juengling, J. (2012). Comprehensive assessment and management of athletes with sport concussion. *The International Journal of Sports Physical Therapy, 7(4),* 433-447.

Straumer-Naesheim, T.M., Andersen, T.E., Dvořák, J., & Bahr, R. (2005). Effects of heading exposure and previous concussions on neuropsychological performance among Norwegian elite footballers. *British Journal of Sports Medicine, 39 (Suppl 1),* i70-i77.

Strauss, E., Sherman, M.M.S., & Spreen, O. (2006). A compendium of neuropsychological tests: Administration, Norms, and commentary - Third Edition. Oxford University Press: London.

Sturmi, J.E., Smith, C., & Lombardo, J.A. (1998). Mild brain trauma in sport. *Sports Medicine, 25(6),* 351-358.

Stuss, D.T., & Alexander, M.P. (2000). Executive functions and the frontal lobes: A conceptual view. *Psychological Research, 63,* 289-298.

Stuss, D.T., Stethem, L.L., Hugenholtz, H., et al. (1989). Reaction time after head injury: fatigue, divided and focused attention, and consistency of performance. *Journal of Neurology, Neurosurgery and Psychiatry, 52,* 742-748.

Tapia, M. & Marsh, G.E. (2002). *Interpreting and reporting effect sizes in research investigations.* Paper presented at the Annual Meeting of the Mid-South Educational research association, Chatlanooga, Tennessee.

Tavazzi, B., Vagnozzi, R., Signoretti, S., Amorini, A.M., Belli, A., Cimatti, M., Delfini, R., Di Pietro, V., Finnochiaro, A., & Lazzarino, G. (2007). Temporal window of metabolic brain

vulnerability to concussions: oxidative and nitrosative stresses – Part II. *Neurosurgery, 61,* 390-396.

Teasdale, G. & Jennett, B. (1974). Assessment of coma and impaired consciousness: A practical scale. *Lancet, 2*, 81-84.

Teasdale, G. & Mendelow, D. (1984). Pathophysiology of head injuries. In. Brooks (Ed.), *Closed head injury. Psychological, social and family consequences*. Oxford : Oxford University Press.

Teasdale, G., Nicoll, J.A., Murray, G., & Fiddes, M. (1997). Association of apolipoprotein E polymorphism with outcome after head injury. *Lancet, 350,* 1069-1071.

Teasdale, G., Murray, G., & Nicoll, J.A. (2005). The association between APOE, age and outcome after head injury: a prospective cohort study. *Brain*, (Epub head of print).

Tegeler, C., Kim, J.Y., Collins, G., Steelman, D., Westwood, K., Reynolds, P., Martin, D., Greenberg, J., & Stump, D. (2003). *Transcranial Doppler Ultrasound for concussion in amateur athletes*. 26th Annual Meeting of the American Society of Neuroimaging; March 6-9.

Tegeler, C. (2004). *New developments in neuroscience.* Paper presented at the New Developments in Sports-Related Concussion Conference. Pittsburgh, July 2004.

Tellier, A., Malva, L.C.D., Cwinn, A., Grahovac, S., Morrish, W., & Brennan-Barnes, M. (1999). Mild head injury: a misnomer. *Brain Injury, 13(7),* 463-475.

Templer, D.I., Hartlage, L.C., & Cannon, W.G., (Eds.). (1992). Preventable Brain Damage. *Brain Vulnerability and Brain Health.* New York: Springer Publishing Company.

Terrel, T.R. (2004). Concussion in athletes. *Sports Medicine, Southern Medical Journal, 97(9),* 837-842.

Thompson, J., Sebastianelli, W., & Slobounov, S. (2005). EEG and postural correlates of mild traumatic brain injury in athletes. *Neuroscience Letters, 377,* 158-163.

Thornton, A.E., Cox, D.N., Whitfield, K., & Fouladi, R.T. (2008). Cumulative concussion exposure in rugby players: Neurocognitive and symptomatic outcomes. *Journal of Clinical and Experimental Neuropsychology, 30(4),* 398-409.

Thurman, D.J., Alverson, C., Dunn, K.A., Guerrero, J., & Sniezek, J.E. (1999). Traumatic brain injury in the United States: a public health perspective. *Journal of Head Trauma and Rehabilitation, 14,* 602-615.

Tiffin, J. (1968). Purdue Pegboard examiner's manual. Rosemont, IL: London House.

Tiffin, J., & Asher, E.J. (1948). The Purdue Pegboard: norms and studies of reliability and validity. *Journal of Applied Psychology, 32,* 234-247.

Tommasone, B.A., & McLeod, T.C.V. (2006). Contact sport concussion incidence. *Journal of Athletic Training, 41(4),* 470-472.

Tremblay, S., De Beaumont,L., Henry, L.C., Van Boulanger, Y., Evans, A.C., Bourgouin, P., Poirier, J., Théoret, H., & Lassonde, M. (2012). Sports Concussions and Aging: A Neuroimaging Investigation. *Cerebral Cortex, 23,* 1159–1166.

Tromp, E. & Mulder, T. (1991). Slowness of information processing after traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology, 13(6),* 821-830.

Trusty, J., Thompson, B., & Petrocelli, J.V. (2004). Practical guide for reporting effect size in quantitative research. *Journal of Counselling and Development, 82,* 107-110.

Trzepacz, P.T., & Baker, R. (1993). *The psychiatric mental status examination*. Oxford: Oxford University Press

Tucker, A.M. (1997). Common soccer injuries: diagnosis, treatment and rehabilitation. *Sports Medicine, 23(1),* 21-32.

Turner, R.C., Lucke-Wold, B.P., Robson, M.J., Omalu, B.I., Petraglia, A.L., & Bailes, J.E. (2013). Repetitive traumatic brain injury and development of chronic encephalopathy: A potential role for biomarkers in diagnosis, prognosis and treatment? *Frontiers in Neurology, 3,* 1-11.

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

Tysvaer, A.T, & Einar, A.L. (1991). Soccer injuries to the brain: a neuropsychological study of former soccer players. *The American Journal of Sports Medicine, 19,* 56-60.

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

Tysvaer, A. & Storli, O.V. (1989). Soccer injuries to the brain: a neurologic and electroencephalographic study of active football players. *American Journal of Sports Medicine, 17,* 573-578.

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

Tysvaer, A. & Lochen, E. (1991). Soccer injuries to the brain: A neuropsychologic study of former soccer players. *The American Journal of Sports Medicine, 19,* 56-60.

Uzzell, B.P. (1999). Mild head injury: much ado about something. In Varney, N.R., & Roberts, R.J. (Eds.), *The Evaluation and Treatment of Mild Traumatic Brain Injury*. London: Lawrence Erlbaum Associates, Publishers.

Vagnozzi, R., Tavazzi, B., Signoretti, S., Amorini, A.M., Belli, A., Cimatti, M., Delfini, R., Di Pietro, V., Finocchiaro, A., & Lazzarino, G. (2007). Temporal window of metabolic brain vulnerability to concussions: mitochondrial-related impairment – Part I. *Neurosurgery, 61,* 379-389.

Van Boven, R.W., Harrington, G.S., Hackney, D.B., Ebel, A., Gauger, G., Bremner, J.D., D'Esposito, M.D., Detre, J.A., Haacke, E.M., Jack, C.R., Jagust, W.J., Le Bihan, D., Mathis, C.A., Mueller, S., Mukherjee, P., Schuff, N., Chen, A., & Weiner, M.W. (2009). Advances in neuroimaging of traumatic brain injury and posttraumatic stress disorder. *Journal of Rehabilitation Research & Development, 46(6),* 717-755.

Van Kampen, D.A., Lovell, M.R., Pardini, J.E., Collins, M.W., & Fu, F.H. (2006). The "value added" of neurocognitive testing after sports-related concussion. *American Journal of Sports Medicine, 34(10),* 1630-1635.

Van Zomeren, A.H. & Deelman, B.G. (1978). Long-term recovery of visual reaction time after closed head injury. *Journal of Neurology, Neurosurgery and Psychiatry, 41,* 452-457.

Vanderploeg, R.D., Berlanger, H.G., & Curtiss, G. (2006). Mild traumatic brain injury: Neuropsychological causality modelling. In G. Young, K. Nicholson, & A.W. Kane (Eds.) *Psychological Knowledge in Court.* USA: Springer.

Vanderploeg, R.D., Curtiss, G., & Belanger, H. G. (2005). Long-term neuropsychological outcomes following mild traumatic brain injury. *Journal of the International Neuropsychological Society, 11,* 228-236.

Varney, N.R. & Menefee, L. (1993). Psychosocial and executive deficits following closed head injury: implications for orbital frontal cortex. *Journal of Head Trauma Rehabilitation, 8(1),* 32-44.

Viano, D.C., Casson, I.R., & Pellman, E.J. (2007). Concussion in professional football: biomechanics of the struck player – part 14. *Neurosurgery, 61(2),* 313-327.

Viano, D.C., & Pellman, E.J. (2005). Concussion in professional football: biomechanics of the striking player – Part 8. *Neurosurgery, 56(2),* 266-280.

Von Holst, H., & Cassidy, J.D. (2004). Mandate for the WHO Collaborating Centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine, 43,* 8-10.

Warden, D.L., Bleiberg, J., Cameron, K.L., Ecklund, M.D., Walter, J., Sparling, M.B., et al. (2001). Persistent prolongation of simple reaction time in sports concussion, *Neurology, 57,* 524-526.

Webbe, F.M., & Barth, J.T. (2003). Short-term and long-term outcome of athletic closed head injuries. *Clinical Sports Medicine, 22,* 577-592.

Webbe, F.S. & Ochs, S.R. (2003). Recency and frequency of soccer heading interact to decrease neurocognitive performance. *Applied Neuropsychology, 10(1),* 31-41.

Wechsler, D. (1997). *Manual for the Wechsler Adult Intelligence Scale – Third Edition*. San Antonio: The Psychological Corporation.

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

Weinstein, E., Turner, M., Kuzma, B.B., & Feuer, H. (2013). Second impact syndrome in football: new imaging and insights into a rare and devastating condition. *Journal of Neurosurgical Pediatrics, 11,* 331-334.

Whitefield, V. (2007). "*Glory is temporary, brain injury may be forever". A neuropsychological study on the cumulative effects of sports-related concussive brain injury amongst Grade 12 schoolboy athletes.* Doctoral thesis, Rhodes University, Grahamstown, South Africa.

Wilberger, J.E. (1993). Minor head injuries in American football. *Sports Medicine*, 15(5), 338-343.

Wilberger, J.E., Haag, B., & Maroon, J.C. (1991). Cumulative effects of football related minor head injury. *Presentation of the American Association of Neurological Surgeons Meeting*. New Orleans, Louisiana.

Williams, D.H., Levin, H.S., & Eisenberg, H.M. (1990). Mild head injury classification. *Neurosurgery, 27,* 422-428.

Williams, P. (1984). Epidemiology of rugby injuries: Wales 1982 – 1984. Five Nations Fellowship. University of Wales, College of Medicine. 1 – 42.

Wilson, B.D., Quarrie, K.L., Milburn, P.D., & Chalmers, D.J. (1999). The nature and circumstances of tackle injuries in rugby union. *Journal of Scandinavian Medicine Sport, 2,* 153-162.

Withnall, C., Shewchenko, N., Gittens, R. & Dvořák, J. (2005). Biomechanical investigation of head impacts in football. *British journal of Sports Medicine, 39* (Suppl I)*:* i49-i57.

Witol, A.D. & Webbe, F.M. (2003). Soccer heading frequency predicts neuropsychological deficits. *Archives of Clinical Neuropsychology, 18*, 397-417.

Witol, A.D. & Webbe, F.M. (1994). Neuropsychological deficits associated with football play. *Archives of Clinical Neuropsychology, 9*, 204-205.

Woodard, J., Marker, C., Tabanico, F., Miller, S., Dorsett, E., Cox, L. et al. (2002). A validation study of the Automated Neuropsychological Assessment Metrics (ANAM) in non-concussed high school players. *Journal of the International Neuropsychological Society, 8(2),* 175.

Woods, S.P., Rippeth, J.D., Conover, E., Carey, C.L., Parsns, T.D., & Troster, A.I. (2006). Statistical power of studies examining the cognitive effects of subthalamic nucleus deep brain stimulation in Parkinson's disease. *The Clinical Neuropsychologist, 20,* 27-38.

Wojtys, E.M., Hovda, D., Landry, G., Boland, A., Lovell, M., McCrea, M., & Minkoff, J. (1999). Concussion in sports. *The American Journal of Sports Medicine, 27(5),* 676-687.

Wrightson, P. & Gronwall, D. (1999). *Mild head injury.* Oxford: Oxford University Press.

Yancosek, K.E., & Howell, D. (2009). A narrative review of dexterity assessments. *Journal of Hand Therapy, 22,* 258-270.

Yeudall, L.T., Fromm, D., Reddon, J.R., & Steffanyk, W.O. (1986). Normative data stratified by age and sex for 12 neuropsychological tests. *Journal of Clinical Psychology, 42,* 918-946.

Young, H.D. (1992). University Physics: Extended version with modern Physics (8th Edition). Pittsburgh, Pennsylvania: Addison-Wesley Publishing Company, Inc.

Zhang, L., Yang, K.H., & King, A.I. (2001a). Comparison of brain responses between frontal and lateral impacts by finite element modeling. *Journal of Neurotrauma, 18(1),* 21-30.

Zhang, L., Yang, K.H., & King, A.I. (2001b). Biomechanics of neurotrauma. *Neurological Reserve, 23(2-3),* 144-156.

Zhang, L., Yang, K.H., & King, A.I. (2004). A proposed injury threshold for mild traumatic brain injury. *Journal of Biomechanical Engineering, 126,* 226-236.

APPENDIXES

Appendix A: Consent Form

RHODES UNIVERSITY
DEPARTMENT OF PSYCHOLOGY
CONSENT FORM

I, _____ Club Chairman of Pirates Rugby Club have been informed of the nature of the research which will be conducted by a Rhodes university doctoral student, Diana Zoccola, on the effects of concussion in club rugby.

I understand that:

1. The abovementioned student is conducting the concussion research as a requirement for a PhD degree at Rhodes University in collaboration with the MRC/UCT Research unit for Exercise Science and Sports Medicine, Newlands, South Africa.

2. The research will involve predominantly the first two teams, who will be assessed using internationally validated computer-based neuropsychological screening batteries and a hand-motor speed test, pre-, mid- and post season. The initial pre-season assessments will take approximately one hour; all subsequent assessments will take 30-40 minutes. Follow-up assessments of concussed players will take place within 6 hours (i.e. on the same day) of injury and then again at weekly intervals, until resolution of symptoms. Pre-, mid- and post-season testing as well as concussion follow-ups will take place on the club premises. In addition, players will be requested to fill out a brief demographic questionnaire with medical background and a symptom checklist, with relevance to the research.

3. This study does not interfere with or substitute for good medical practice. It is therefore advised that all players with concussion should be seen as soon as possible by their general practitioner or other medical practitioners and should not return to contact sport for at least 3 weeks from the time of injury and thereafter on the advice of the medical practitioner.

4. Participation in this research is strictly voluntary and players have the right to withdraw from the study at any stage. Players must contact the researcher in order to sign a withdrawal form should they not wish to continue.

5. The information collected on individual players will be strictly confidential and will only be made available to the medical practitioner and the coach on request. This information may form part of the management decision in individual cases. However, the researcher will not be held accountable for medical decisions made by medical practitioners or coaches on the basis of that information.

6. Data arising out of this project will be used for thesis and publication purposes only by the collaborating universities.

I hereby give consent for those players who will be participating in this research project to be assessed by the abovementioned researcher.

Signed at _____ on _____

_____

Chairman Researcher Witness

Appendix B: General Information and consent

GENERAL INFORMATION AND CONSENT

Please take note of the following information, which will be explained by the researcher.
I am conducting research on concussion management as a requirement for a PhD degree at Rhodes University.

1. What I need is some basic information which will be a help to the coach and doctors when managing the after effects of a head injury – should you get concussed whilst playing sport or any other reason during the year. This is the latest way that sports concussion is being managed in other parts of the world (e.g. USA and Australia), and is already in place for the Springboks and All Black rugby teams. To my knowledge Pirates Rugby Club will be the first rugby club in South Africa to have this system in place.

2. I am going to ask some questions, and collect some scores on a number of small tasks. This will include a co-ordination test, as well as a computer-based test. Both assessments will not take longer than one 40-minute period.

3. The information collected pre-season, as well as mid- and post-season, will be totally confidential, and will only be looked at individually should you have a head injury. Following a head injury, people will be retested using some of the tests, and the results will be compared with those obtained from the first assessment. From this comparison it will be possible to gauge the recovery process, and the coach and doctor will be able to make more informed decisions about how serious the concussion is, and when you will be fit to go back to play. The researchers only act in an advisory capacity, and do not take responsibility for the final decision about return to play.

4. The follow-up testing after a concussion will be done at the rugby Club – but you will be given all that information if you get concussed.

5. The information obtained will also be used for research purposes, where the identity of the players involved will not be of any importance, and will not be made known.

6. It is very important to do your best, and to be as accurate and honest as possible when you answer the questions. If the information you give at the time is not accurate, and/or you cannot do your best on the tests for any reason, this might cause the doctor to make a wrong assessment of the seriousness of the head injury, and would not be of benefit to you medically. If at the end of the session you believe you have not been able to do your best for any reason, please inform the researcher. Reasons might be because you have a headache, are worrying about something else, or have felt distracted because of external noise, and so on.

7. Is there anything you don't understand, or are unhappy about? Are there any further questions? If you are happy to go ahead, please sign the following consent paragraph.

I _____ , understand the nature of the research project as specified above. I understand that my participation in the research is strictly voluntary and that I have the right to withdraw from the study at any stage.

SIGNED: _____ DATE: _____

Appendix C: Biographical Questionnaire

GENERAL INFORMATION					
Subject				**Age**	
Home language		**Other**		**Race:**	
EDUCATION, OCCUPATION AND ESTIMATE IQ					
Level of Education completed					
Current study, if any					
Occupation					
Estimate IQ (Established on the basis of WAIS-III Picture Completion and Matrix Reasoning Scaled Scores using the OPIE-3 Formula)					
MEDICAL AND PSYCHIATRIC HISTORY					
CONCUSSION HISTORY					
Prior Concussions					