NEUROCOGNITIVE AND SYMPTOM PROFILES OF CONCUSSED AND NONCONCUSSED PROVINCIAL RUGBY UNION PLAYERS OVER ONE SEASON

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ABSTRACT

Neurocognitive and symptom profiles of concussed and nonconcussed adult provincial rugby union players were investigated over one rugby season, including early season (baseline), intermittent postconcussion, and end of season testing. In a non-equivalent quasi-experimental design, nonconcussed (n = 54) and concussed (n = 17) rugby groups were compared with demographically equivalent noncontact sport controls (n = 37, and n = 17, respectively). Measures included the ImPACT cognitive and symptom composites, and the WMS-III Visual Reproduction and Verbal Paired Associates subtests. The independent and dependent comparative analyses in respect of both nonconcussed and concussed groups, provided cross-validation of poorer acute and/or chronic neuropsychological outcomes for the rugby groups on the ImPACT Reaction Time, Visual Motor Speed, Impulse Control and Symptom composites, and the WMS-III Verbal Paired Associates. The finding of significantly poorer scores on Verbal Paired Associates up to 24 days post concussion for the rugby players versus controls, was longer than the 7 – 10 day recovery period frequently cited in the literature. The overall implication of the study is that even in a group with high cognitive reserve such as these provincial level athletes, there may be prolonged acute recovery, as well as permanent deleterious neuropsychological consequences of cumulative concussive injury in association with a sport such as rugby. Accordingly, the move towards careful *individualised* postconcussion monitoring of neurocognitive functioning is endorsed, including early identification of any significant permanent reductions in cognitive reserve. Sensitivity of the ImPACT test might be enhanced via inclusion of a verbal associate learning task.

DEDICATION

In memory of Pete McD
who died as a result of
rugby-related MTBI

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CHAPTER ONE: INTRODUCTION

It was a cold day in the winter of 1977 when life finally ebbed away from a young man, barely twenty-one years of age. The young man, Pete, died as a result of a traumatic brain injury sustained from playing rugby. Lying lifeless against the white starched bed sheet in the intensive care unit, Pete bore no resemblance to that of a healthy young rugby player. As tears rolled down the face of the young student nurse who had nursed him since his admission to intensive care, she recalled how fit and vibrant Pete had looked when running onto the field at the rugby match she had attended ten days previously. She could barely reconcile that the motionless body with a grossly swollen head and puffed eye slits each measuring more than 15 centimetres, was the same young man. "Why had Pete agreed to play rugby when his doctor had told him not to play sport for six weeks, because he had sustained a concussion the week prior to this fatal second concussion? Why couldn't the neurosurgeon stop the extensive brain haemorrhages? How will his team mates, and the opponent who tackled him, feel when they hear that he has died? His poor family will be devastated! His mother, who having sat beside him at every opportunity, holding his hand, will be especially inconsolable. This is utterly devastating. What an appalling waste of young life given tragically to the game of rugby..." she thought as she slowly lifted the sheet to gently cover his face – a symbolic act of the finality of life. That was more than thirty years ago, when little was known about second impact syndrome, yet Pete's death continues to weigh heavily in her heart. She has watched many rugby matches since, and her passion for the sport continues unabatedly. That same young nurse is now a mature psychologist and author of this thesis. If in any small way she can make a contribution to the scientific findings about rugby-related concussion that might help prevent needless deaths and brain injury happening to other young people, then such is her prime motivation for writing this thesis.

1.1 INTRODUCTION

Rugby Union, played globally in at least 100 countries and watched by millions, is regarded as one of the most exciting sports in modern times (International Rugby Board, 2009a, 2010a, 2010b). In South Africa rugby union is played widely at school and adult level; rugby matches draw large crowds of supporters and televised rugby matches are

watched by thousands of viewers, and - considering the following that rugby has in South Africa - it could almost be considered 'the opium of the people'. Rugby involves a high level of bodily contact and is aptly termed a 'collision' sport (Jeanty & Della Porta, 2009; Maxwell & Visek, 2009; Shuttleworth-Edwards & Radloff, 2008). Consequently rugby results in many injuries of which concussion accounts for 11 to 35.9% of these injuries (e.g., Collins, Micheli, & Yard, 2008; Kohler, 2004; McIntosh, 2005; McIntosh et al., 2009; McIntosh, McCrory, Finch, & Wolfe, 2010; Myers, 1980; Nathan, Goedeke, & Noakes, 1983; Nicol, Pollock, Kirkwood, Parekh, & Robson, 2010; Roux, Goedeke, Visser, Van Zyl, & Noakes, 1987; Shuttleworth-Edwards, Noakes et al., 2008; Shuttleworth-Edwards, Smith, & Radloff, 2008; Sparks, 1981). Despite this high incidence of rugby-related concussion, currently there are eight known published neuropsychological research studies, investigating mostly the chronic neurocognitive deficits and related symptoms resultant from rugby-related concussions, and therefore a need exists for further research. Specifically, there is a need to investigate if adult rugby players, particularly at a professional and provincial level, experience chronic neuropsychological deficits and related symptom complaints as a result of Mild Traumatic Brain Injury (MTBI), in the form of concussive and subconcussive events, sustained over many years of playing the sport. Furthermore, if chronic neurocognitive deficits and related symptom complaints do exist, then the added impact that one season of rugby participation has on these deficits warrants investigation. Moreover, research is required to examine the acute neurocognitive deficits and symptoms arising from rugby-related concussion and the recovery thereof, among adult rugby players.

Pertinent to such research, therefore, are general assessment issues related to neuropsychological assessments that need to be taken into account. This includes the particular neurocognitive functions required to be tested as a result of MTBI, and the selection of appropriate neuropsychological tests to assess these functions and postconcussive symptoms. Originally neuropsychological research and assessment of sport-related MTBI focused on traditional paper and pencil tests (Collins, Echemendia, & Lovell, 2004), however in more recent years computer-based neuropsychological test batteries have developed for sport-related MTBI assessment (Pretz, 2007). Several versions of computer-based neuropsychological programs have been developed in recent years, for example, CogState Sport, Concussion Resolution Index (Headminder), Automated Neuropsychological Assessment Metrics (ANAM), and Immediate Post-

concussion Assessment and Cognitive Testing (ImPACT) (Aubry et al., 2002). Although some controversy exists regarding computerised testing (e.g., Schatz & Browndyke, 2002; Schatz & Zilmer, 2003: Shuttleworth-Edwards & Border, 2002), for the purpose of the present study, ImPACT was identified as being neuropsychologically sophisticated and it assesses a wide range of both neurocognitive functions and postconcussive symptoms. However, traditional neuropsychological tests may incorporate crucial aspects, not included in the ImPACT computerised test, which might reveal sensitivity to MTBI. Therefore, in light of the sensitivity of memory to MTBI in general (e.g., Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005; Frencham, Fox, & Maybery, 2005; Lezak, Howieson, & Loring, 2004) and sport-related MTBI (e.g., Belanger & Vanderploeg, 2005; Collins, Field et al., 2003; Covassin, Stearne, & Elbin, 2008; De Beaumont et al., 2009; Echemendia, Putukian, Mackin, Julian, & Shoss, 2001; Gaetz, Goodman, & Weinberg, 2000; Guskiewicz et al., 2005; Hatfield, Bieliauskas, Begloff, Steinberg, & Kauszler, 2004; Iverson, Gaetz, Lovell, & Collins, 2004b; Iverson, Lange, & Franzen, 2005; Killam, Cautin, & Santucci, 2005; Lovell, Collins, Iverson et al., 2003; Matser, Kessels, Jordan, Lezak, & Troost, 1998; McClincy, Lovell, Pardini, Collins, & Spore, 2006; Mihalik et al., 2005; Parker, Osternig, van Donkelaar, & Chou, 2007; Peterson & Skelton, 2000; Shuttleworth-Edwards, Border, Reid, & Radloff, 2004) it was decided for the purpose of the present study to incorporate the WMS-III Verbal Paired Associates and Visual Recognition subtests in conjunction with the ImPACT test.

A number of rugby-related MTBI studies (Shuttleworth-Edwards, Border et al., 2004, Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008) have evaluated and interpreted findings in terms of the Reserve theory, as an appropriate heuristic model. The basic premise of the Reserve theory, is that each individual is conceptualised as having a cognitive reserve which explains variability among individuals, and that when this cognitive reserve is depleted beyond a certain threshold, such as during the normal aging process or the onset of neurological disease, then in association with central nervous system dysfunction neurocognitive deficits emerge (Jordan, 1997; Randolph, 2001; Satz, 1993; Stern, 2002; Stern et al., 2003). Various researchers have extrapolated this model, to account for the different outcomes and variability found among individuals on neuropsychological testing, following a similar degree of sport-related MTBI severity, (e.g., Collins, Grindel et al., 1999; Heilbronner et al., 2009; Shuttleworth-Edwards, Border et al., 2004, Shuttleworth-Edwards & Radloff,

2008; Shuttleworth-Edwards, Smith et al. 2008). Reserve theory, therefore, was applied as an overall conceptual framework for the interpretation of outcome in respect of the current study.

Against the above-mentioned empirical rationale, and conceptual backdrop of Reserve theory, a cross-sectional and prospective study was designed with the objective of addressing the following key research questions:

(1) Whether or not rugby players at adult provincial level suffer chronic neurocognitive and related symptomatic sequelae, as a result of long-term involvement in playing rugby, due to concussive and subconcussive events sustained;
(2) Whether or not rugby players at adult provincial level suffer compounded chronic neurocognitive and related symptomatic sequelae, as a result of playing rugby over one season, due to concussive and subconcussive events sustained;
(3) How rugby players at adult provincial level recover from MTBI in terms of neurocognitive and related symptomatic sequelae, the variability of which can be understood in terms of the Reserve theory; with practical implications being related to team planning measures when a team player has been concussed;
(4) The extent to which rugby concussive and subconcussive events can be understood in terms of and in support of the Reserve theory.

1.2 STRUCTURE OF THE THESIS

The thesis is bound in two volumes. Volume 1 contains the theoretical framework of the thesis (Chapters one to six). Volume Two contains the research study (Chapters seven to nine), followed by the references and appendixes. For ease of reference, all tables and figures are prefixed with an arabic numeral in order of their first mention in the thesis**.** *Tables are placed at a convenient point in the text following their first mention in Chapters two and three, and tables and figures will be placed at the end of the applicable section in which they are first mentioned in the text in Chapters seven and eight.*

Volume One:
Chapter one provides the background and introduction to this thesis.

Chapter two provides information on traumatic brain injury (TBI) in general in terms of definition, types, mechanisms and biomecchanisms of injury, neurophysiological consequences and rating of severity, in order to put MTBI in context.

Chapter three describes MTBI in terms of definition and related terminology, mechanisms and biomecchanisms of injury, neurophysiological consequences, rating of severity, neuropsychological consequences pertaining to neurocognitve deficits, symptom complaints and their recovery, the cumulative and chronic effects of MTBI, and factors affecting prognosis. The Reserve theory is then discussed.

Chapter four describes the team contact sports with high-risk for MTBI, and examines the epidemiology of TBI, MTBI, and sport-related MTBI with reference to team contact sports in general and to rugby union specifically.

Chapter five focuses on the management and assessment of MTBI. Details about general medical management and related MTBI assessment techniques are provided. Neuropsychological assessment is discussed in terms of neurocognitive assessment and assessment of symptoms. General assessment issues are highlighted, as well as the various neuropsychological functions assessed in MTBI. Both traditional (pencil and paper) as well as computerised neuropsychological tests are elucidated, with specific emphasis on the ImPACT computerised test.

Chapter six outlines neuropsychological research findings pertaining to MTBI in team contact sports in relation to their impact on specific neurocognitive functions and symptom reports. Findings pertaining to MTBI in rugby specifically are described.

Volume Two:

Chapter seven encompasses the methodology for this study.

Chapter eight provides the results, pertaining to (i) the General Rugby group and comparative General Control group, and (ii) the Concussed Rugby group and comparative Matched Control group.

Chapter nine concludes the thesis with a discussion. This is then followed by the references and appendixes.

CHAPTER TWO: TRAUMATIC BRAIN INJURY

The focus of this chapter is on traumatic brain injury (TBI) in general. Initially TBI will be defined and presented in terms of its two types (penetrative and closed), following which the mechanisms and biomechanisms affecting the severity of brain injury will be provided. The neurophysiological consequences of TBI will then be elucidated. Finally, the manner in which TBI severity is categorised as 'mild', 'moderate' and 'severe' will be described, in order to provide the context for the subsequent more specific exposition of mild traumatic brain injury (MTBI), which is the focus of this thesis.

2.1 DEFINITION OF TRAUMATIC BRAIN INJURY (TBI)

The definition of TBI has been inconsistent and variable according to circumstances and the specialised discipline for which it is being defined (Dawodu, 2009; Ruff & Jurica, 1999). Moreover, the era in which various definitions arose, and given that the presentation of TBI is characterised by a number of diverse signs and symptoms, add to further disparity amongst the various definitions. The most recent, relatively succinct and inclusive definition of TBI appears to be that of the Department of Veteran Affairs (2009a, p. 16), whereby traumatic brain injury is defined as:

> "A traumatically induced structural injury and/or physiological disruption of brain function as a result of an external force that is indicated by new onset or worsening of at least one of the following clinical signs, immediately following the event", that includes any period of loss of or decreased level of consciousness; any posttraumatic amnesia in the form of memory loss for events immediately prior to or following the injury; any alteration in mental status at the time of injury e.g., confusion, disorientation or slowed thinking etc.; any neurological deficits e.g., weakness, praxis, paresis/plegia, aphasia, difficulties with balance, vision, etc. that may or may not be transient; or an intracranial lesion. "External forces may include any of the following events: the head being struck by an object, the head striking an object, the brain undergoing an acceleration/deceleration movement without direct external trauma to the head, a foreign body penetrating the brain, forces generated from events such as a blast or explosion, or other forces yet to be defined." (Department of Veteran Affairs, 2009a, p. 16).

The above criteria generally define TBI. Not every individual exposed to an external force sustains a TBI, however an individual who is exposed to an external force and manifests any one of the above signs and symptoms is considered to have experienced a TBI (Department of Veteran Affairs, 2009a). Therefore, TBI is a brain insult caused by an external mechanical force rather than a congenital or degenerative insult to the brain, in association with an altered or diminished state of consciousness, that may lead to temporary or permanent cognitive, physical, and psychosocial functional impairment (Dawodu, 2009). 'Traumatic brain injury' is a more specific term than 'head injury', the term commonly used in the past (Anderson, Northam, Hendy, & Wrennall, 2001; Lezak et al., 2004; Lovell, Echemendia, Barth, & Collins, 2004; Rees, 2003; Ruff, 2005; Silver, McAllister, & Yudofsky, 2005), in that the latter could refer also to non-brain injury, e.g., injury to the jaw or face (Lezak et al., 2004: Von Holst & Cassidy, 2004). Therefore, for this study, the term 'traumatic brain injury' (TBI) is used despite citing literature that refers to the condition as 'head injury'.

Falling within the broad rubric of the above definition of TBI, two broad types of injury can be distinguished, viz., penetrative and closed TBI.

2.2 PENETRATIVE (OPEN) AND CLOSED BRAIN INJURIES

Traumatic brain injuries are either 'penetrative' (open) or 'closed' brain injuries (Nicholl & LaFrance, 2009). A penetrative brain injury involves penetration of the skull, and where the brain or dura mater is partially exposed (Lezak et al., 2004). Penetrative brain injuries are commonly caused by gun shot wounds, penetrative foreign bodies such as tools or everyday objects (Lezak et al., 2004), bomb or landmine explosions, and shrapnel. A 'closed' brain injury has no penetration of the skull, but involves the forward and backward thrust and/or rotation of the brain (e.g., Bell, Neal, Lettieri, & Amonda, 2008; Lezak et al., 2004). Closed brain injuries are commonly caused by an impact to the brain as a result of either an accelerating object impacting on the slower moving head or body, or by a slower moving object that decelerates the moving body and head. Causes of closed brain injuries include falls, vehicle accidents, assaults or being accidentally struck by objects or bodies (National Centre for Injury Prevention and Control, 2007). Closed brain injury is the form of brain injury referred to in this study, and in the text whenever the term 'traumatic brain injury' (TBI) is used, this pertains to 'closed' as opposed to

'penetrative' brain injury. The more detailed mechanisms of closed TBI are described below.

2.3 MECHANISMS AND BIOMECHANISMS OF CLOSED TBI

The brain is protected by three membranes (the inner pia mater, the middle arachnoid layer and the outer dura mater) and floats within the Cerebral Spinal Fluid (CSF) that fills the ventricles and subarachnoid space (Whitefield, 2007). The brain can therefore withstand quite substantial translation and deformation, if the head is subject to significant forces (Crippen, 2009). The head need not be impacted upon directly to subject the brain to significant forces in that 'impulsive loading', the forceful impact to another part of the body, can set the head in motion (Bailes & Cantu, 2001; Poirier, 2003). Basic principles of physics can partly explain how significant forces can result in brain injury when the head or body is rapidly accelerated and/or decelerated. Newton's First Law of Motion states that force equals mass times acceleration, or acceleration equals force divided by mass (Cantu, 1998a, 1998b). Accordingly, once an object is in motion, it maintains a constant velocity until sufficient force in the opposite direction acts on it and stops it. There are a number of ways in which this phenomenon typically occurs, as described below.

When the head stops moving suddenly (e.g., by the impact from a heavy rugby player running into another player), the brain continues to move within the skull at the original velocity (e.g., of the running rugby player) until it strikes the inside of the skull. Translational acceleration can occur, whereby the head moves in a straight line in relation to the brain's centre of gravity. In addition, rotational acceleration can occur, whereby the brain rotates its centre of gravity (Lezak et al., 2004), in that the brainstem is relatively fixed in relation to the skull, and turns as the skull turns, whereas the cerebrum does not (Nicholl & LaFrance, 2009). Angular acceleration is the amalgamation of both translational and rotational acceleration as a result of the movement of the neck and head on impact (Lezak et al., 2004). Rotational acceleration may be implicated in loss of consciousness (Parker, 2001). The strain resulting from rotational acceleration of the brain within the skull and the translational force can cause the delicate blood vessels and neuronal fibres to stretch and shear. The shearing of axons or axon clusters causes diffuse axonal injury (DAI), and shearing of blood vessels causes small haemorrhages, resulting

in multiple microscopic lesions throughout the brain. These lesions usually occur within the cerebral and brain stem white matter, including the cerebellum in severe injuries (Lezak et al., 2004). Apart from microscopic lesions, rotational force can result in larger lesions, for example intracerebral haemorrhages (Lampert & Hardman, 1984).

Coup injuries are those that occur directly below the site of impact, while contracoup injuries occur contralateral to the site of impact (Rangel-Castilla, Gasco, Hanbali, & Salinas, 2008). Therefore contusions are likely to occur at both the coup and contracoup sites (Lezak et al., 2004; Rangel-Castilla et al., 2008). At the coup point of impact, the skull moves inwards while the adjacent areas compensate by bending outwards, which then is followed by rebound effects (Lezak et al., 2004). Contracoup effects are particularly pronounced in the orbital and temporal regions, often involving local neuronal loss and ultimately subcortical demylenation (Lishman, 1999). Deceleration on impact can also result in occipital coup and frontotemporal contracoup lesions (Barth et al., 1989).

In more severe brain injuries, a variety of pathological changes has been found on postmortem examination. Some of these injuries are primarily as a result of direct physical damage to the brain parenchyma, and others are the result of secondary complications such as oedema, anoxia and vascular disturbances (Lishman, 1999). Vascular lesions are also evident, in the form of punctuate haemorrhages, sometimes accompanied by small and large infarcts. Vascular lesions may also result from reduced cerebral blood flow at the time of injury, hypotension, embolism and increased intracranial pressure occluding the arteries or vessel spasm following the injury, which can lead to necrosis of major cerebral arteries and brain tissue (e.g., Lezak et al., 2004; Lishman, 1999). Widespread damage to nerve fibres can be detected, with myelin breakdown, reabsorption and the formation of retraction balls occurring mainly in the central white matter of the hemispheres, the long tracts of the brain stem, and the corpus callosum. Once the acute stage has resolved, varying degrees of gliosis and cerebral atrophy (such as cyst formation, ventricular diverticula or ventricular enlargement) may occur (Lishman, 1999).

All of the mechanisms discussed above may cause biochemical changes related to perfusion and energy demands that are not fully understood. Furthermore, as pointed out

by Bernhardt (2009), it is uncertain whether findings from experimental animal models or human studies involving more severe brain injuries, can be extrapolated to precisely reflect the mechanisms and pathophysiology of the milder spectrum of TBI that is the focus of this study. However, it can be assumed that in all likelihood the mechanisms and pathological changes are similar for mild and severe TBI, including both the initial features (primary brain injury) and complications arising (secondary brain injury) such as will be elucidated below, in that the nature and extent of these factors influence TBI severity and outcomes (Hemmer, 2000).

2.4 NEUROPHYSIOLOGICAL CONSEQUENCES OF TBI

Traumatic brain injuries are often divided into two categories: primary brain injury and secondary brain injury. Primary brain injury is the initial structural (anatomical and physiological) injury to the brain as a direct result of the traumatic impact (Crippen, 2008; Rangel-Castilla et al., 2008). DAI, contusions, haematomas and haemorrhages are examples of primary brain injuries (Rangel-Castilla et al., 2008). Secondary brain injury is the resultant series of physiological changes initiated by the original trauma that add further complications. Elevated intracranial pressure, cerebral oedema, hypoxia, acidosis, systemic hypotension, epilepsy, hyrdocephalus, ischaemia, and herniation of the brain tissue are examples of secondary brain injuries (e.g., Barth et al., 1989; Bayir, Clark, & Kochanek, 2002; Crippen, 2008; Rangel-Castilla et al., 2008). The treatment of brain injury is directed at preventing or minimising secondary brain injury (Crippen, 2008; Rangel-Castilla et al., 2008). Primary and secondary brain injuries are discussed in more detail below.

2.4.1 Primary Brain Injuries

Primary brain injuries include DAI, cerebral contusions, epidural, subdural and intracerebral haematomas, in addition to Duret, subarachnoid and intraventricular haemorrhages. Rangel-Castilla et al. (2008) explain that DAI is not caused by contact injury alone, but also by acceleration injury, particularly rotational injury, whereby mechanical forces act on the long axons, resulting in axonal structural failure. The relatively incompressible brain is unable to tolerate shear or tensile strain, particularly on rapid strain, and is particularly vulnerable to lateral rotation, although more able to

tolerate sagittal movements. The mechanical forces of the head striking a surface are enough to dissect axons into proximal and distal segments. The greater the number of axons implicated in DAI, the greater the neurologic deficits and the stronger the likelihood and persistence of unconsciousness. Approximately 30 to 40% of individuals who die as a result of TBI show evidence of DAI and ischaemia on postmortem examination (Rangel-Castilla et al., 2008).

Cerebral contusions tend to be greater at the coup site when on impact the head is at rest, and at the contracoup site when the head is in motion such as with a fall or a motor vehicle accident (Lishman, 1999). Under conditions of rapid acceleration or deceleration, regardless of whether or not there is contact (e.g., whiplash injury), a single contusion or multiple contusions can occur in the deeper brain structures, particularly the brainstem and corpus callosum (Lezak et al., 2004). Cerebral contusions usually occur in the frontal and temporal lobes, and may be an adjunct to skull fracture. Contusions tend to expand from 24 hours to 10 days after injury, and this delayed enlargement of intraparenchymal contusions is the prevalent cause of deterioration or death in TBI.

Epidural haematomas occur in 5 to 15% of patients with a fatal TBI and 1 to 2% of all cases admitted with TBI suffer with an epidural haematoma (e.g., Liebeskind, 2009; Rangel-Castilla et al., 2008). Epidural haematomas are the result of bleeding from the middle meningeal artery, or from the diploic vessels as a result of an overlying skull fracture (Rangel-Castilla et al., 2008). Skull fractures occur in 85 to 95% of adult cases of epidural haematoma, but less so in paediatric cases due to the plasticity of the immature calvaria (Liebeskind, 2009). Usually these fractures are temporal skull fractures (Bernhardt, 2009), resulting in the pool of blood stripping the dura mater away from the skull (Lezak et al., 2004). Therefore, epidural haematomas mostly occur over the temporoparietal area, but can also occur over the frontal, parietal and posterior areas. Epidural haematomas that occur over the posterior area near the brain stem are of concern in that the individual may remain conscious until shortly before death, or may experience an initial brief period of unconsciousness followed by a lucid period and then have subsequent deterioration over the next 15 to 30 minutes until the brain stem is compromised by pressure from the haematoma (e.g., Bernhardt, 2009; Rangel-Castilla et al., 2008; Lezak et al., 2004). The postconcussion syndrome (characterised by headaches,

vertigo, dizziness, emotional lability, fatigue, restlessness and difficulty concentrating) is believed to be the delayed result of an epidural haematoma (Liebeskind, 2009).

A subdural haematoma is a pool of blood that forms over the cerebral hemispheres in the space between the dura mater and brain arachnoid (Lezak et al., 2004; Lishman, 1999; Rangel-Castilla et al., 2008). Subdural haematomas are the result of the brain parenchyma shifting during violent head motion, resulting in bleeding from vessels on the inner side of the dura mater or the brain's surface (e.g., Lezak et al., 2004; Rangel-Castilla et al., 2008). In the case of a bridging vein rupture, the haematoma forms in the frontoparietal parasagittal region, and with an arterial rupture the haematoma forms in the temporoparietal region (Rangel-Castilla et al., 2008). Approximately 20 to 40% of cases admitted with severe TBI suffer a subdural haematoma (Rangel-Castilla et al., 2008). A subdural haematoma is rare in MTBI, and its classic presentation is an acute and persistent loss of consciousness (Bernhardt, 2009). Intracerebral haematomas may form from bleeding within the brain tissue or from the direct rupture of blood vessels within the brain, and usually occur in the frontal and temporal lobes (Lezak et al., 2004). Blood may also pool in the basal cisterns, which impedes the flow of CSF, and results in hydrocephaly (Lishman, 1999). The entire spectrum of intracranial contusions and haematomas that have been described are known to occur in sport-related brain injuries (Bruno, Gennarelli, & Torg, 1987).

Mechanical forces may impact on cerebral circulation causing numerous forms of micro and macro intracerebral haemorrhages. Duret haemorrhages are small punctuate haemorrhages of the midbrain and pons caused by arteriole stretching at the time of primary injury, but are particularly lethal when they arise in the brainstem (Rangel-Castilla et al., 2008). Subarachnoid haemorrhaging can occur after any degree of TBI and usually presents as a worsening headache with increasingly elevated intracranial pressure after the initial injury (Bernhardt, 2009). In more severe TBI, an intraventricular haemorrhage often accompanies other intracranial haemorrhages. An intraventricular haemorrhage predisposes the individual to intracranial hypertension and posttraumatic hydrocephaly, requiring treatment with an intraventricular catheter or ventriculoperitoneal shunt (Rangel-Castilla et al., 2008).

The various structural brain injuries discussed above can in some instances result in secondary consequences that exacerbate the brain injury. These are described below.

2.4.2 Secondary Consequences

Secondary consequences involve subsequent intracranial insults to the brain, such as elevated intracranial pressure, herniation, cerebral oedema, cerebral ischaemia (including vasospasm, hypoxia and anoxia), epilepsy, hydrocephaly and brain death. They also include secondary systemic insults such as hyperglycaemia, hypercapnia, hypotension, anaemia, hyponatraemia and endocrine disorders (e.g., Crippen, 2009; Lishman, 1999; Rangel-Castilla et al., 2008). A few of these consequences are now discussed.

Crippen (2009) helps elucidate various concepts explaining the reaction of the brain to trauma. Due to the inelasticity of the skull, when the volume within the skull increases, the intracranial pressure increases. The Monro-Kellie doctrine states that due to the inelasticity of the skull, the intracranial volume (V i/c [intracranial volume] = V [brain] + V [cerebrospinal fluid] + V [blood]) is fixed. In an adult, the intracranial volume is typically 1500 millilitres, with approximately 85 to 90% being accounted for by the brain, less than 3% being accounted for by the cerebrospinal fluid, and 10% by the intravascular cerebral blood. When a considerable brain injury results in cerebral oedema, the intracranial volume increases, and because the intracranial volume is ‘fixed’ and the brain has extremely limited compliance, this results in increased intracranial pressure.

A further concept is that of cerebral blood flow. In non-injured, non-hypertensive persons, a constant amount of blood flow to the brain is maintained through autoregulation, whereby the arterioles dilate or constrict within a specific range of blood pressure. When, however, the mean arterial pressure falls below 50 mm Hg or rises above 150 mm Hg, a pressure-passive flow arises in that the arterioles can no longer autoregulate and the cerebral blood flow becomes totally dependent on blood pressure. When the mean arterial pressure falls below 50 mm Hg the brain is at risk of cerebral ischaemia, and when the mean arterial pressure rises above 160 mm Hg, the increased cerebral blood flow results in increased intracranial pressure. A drastically increased and uncontrolled intracranial pressure may cause herniation of the brain (Crippen, 2009).

Herniation of the brain occurs when the brain slides through the openings within the falx or the tentorium and over the free dural edges, with the brain frequently injured by the dural edge. Crippen (2009) describes the process as follows. The total intracranial compartment is divided into three compartments by the falx cerebri and the tentorium cerebelli dural structures. The falx cerebri separates the left and right hemispheres of the brain, dividing the supratentorial compartment into two halves. The tentorium cerebelli separates the supratentorial compartment (cerebral hemispheres) from the infratentorial compartment (the brain stem and cerebellum). There are five types of herniation: (i) transtentorial, when the uncus of the temporal lobe herniates the tentorium causing pressure on the third cranial nerve and the pupil to dilate, (ii) subfalcine, when the cingulate gyrus of the frontal lobe herniates the falx, compromising anterior cerebral blood flow to the frontal lobe medial areas, (iii) central, when each of the cerebral hemispheres herniates the tentorium, resulting in considerable pressure upon the upper brainstem, (iv) cerebellar, when the cerebellum herniates upwards through the tentorium, resulting in considerable compression of the upper brainstem, and (v) tonsillar, when the cerebellar tonsils herniate downwards through the foramen magnum causing considerable compression of the lower brainstem and upper cervical spinal cord. Herniation of the brain across the dural structures can result in irreversible or fatal brain injury (Crippen, 2009).

Cerebral oedema, the engorgement of brain tissue, can also raise intracranial pressure (Lishman, 1999). Vasogenic oedema occurs when water and solubles diffuse into the brain as a result of a breach in the blood-brain barrier, causing the fluid to accumulate in the white matter. It is uncertain whether the more common form, cellular oedema, is caused by the injured brain cells intake of extracellular potassium or outtake of sodium, chloride or sodium hydrocarbate (Rangel-Castilla et al., 2008).

Cerebral ischaemia, the inadequate supply of oxygen to the brain as a result of hypoperfusion or hypoxia, can be caused by uneven cerebral blood flow (e.g., vasospasm or focal dysregulation in an injured area of the brain) or respiratory alkalosis whereby the raised affinity of the haemaglobin for oxygen hinders oxygen release. The traumatised brain is extremely sensitive to a lack of oxygen (Rangel-Castilla et al., 2008) and cerebral anoxia can cause necrosis in the cortical sulci, Ammon’s horn and basal ganglia, and result in the loss of Purkinje cells from the cerebellum (Lishman, 1999).

Endocrine disorders are briefly discussed as they may have an impact on typical symptoms following TBI, such as sleep difficulties. Endocrine disorders following TBI have the potential to contribute to morbidity, in that marked changes of the hypothalamo-pituitary axis in the acute phase of TBI have been documented (Behan, Phillips, Thompson, & Agha, 2008). Eighty percent of patients have evidenced gonadotropin deficiency, 18% growth hormone deficiency, 16% corticotropin deficiency, and 40% vasopressin abnormalites resulting in inappropriate anti-diuresis or diabetes insipidus. In addition, 25% of long-term TBI survivors exeperience deficiency of one or more of the pituitary hormones (Behan et al., 2008). Jeffrey (2010) describes research using polysomnography, that confirms sleep disturbances, reduced sleep efficiency and increased wake after sleep onset in patients with TBI fourteen months after injury, in comparison with uninjured control subjects. Findings revealed reduced evening melatonin production in the TBI group, in addition to increased levels of anxiety and depression that may contribute to these problems. However, when controlling for depression, slow-wave sleep was also higher in patients with TBI compared with controls. This is because the mechanical effects of the brain damage disrupts brain structures that regulate sleep, including the production of melatonin, that then increases slow-wave sleep. In turn, a lack of sleep can impact on postconcussive neurocognitive functions such as attention, concentration and working memory, and can cause irritability (Jeffrey, 2010). The most severe secondary consequence of TBI is brain death, defined as “the irreversible loss of all brain function” (Dixon & Malinoski, 2009, p. 4) and therefore the absence of brainstem functioning, and is diagnosed via comprehensive clinical evaluation (e.g., Dixon & Malinoski, 2009; Gandey, 2010).

Overall, the damage to the cerebral white matter as a result of primary DAI and vascular injuries, and in some instances the ensuing secondary injuries (in the forms of elevated intracranial pressure, herniation, cerebral oedema, and hypoxia), in combination are considered to affect the severity and therefore the outcome of brain injury. The severity of any brain injury is based on the outward signs of the described neurophysiological primary and secondary injuries, however the specific modus operandi used for rating severity are described below.

2.5 RATING TRAUMATIC BRAIN INJURY SEVERITY

The range of traumatic brain injuries are categorised from mild to severe. The mildest form involves a slight knock to the head without notable adverse effects on the brain or mental status functioning. The severest form involves protracted coma or a vegetative condition (Lezak at al., 2004). Due to many factors involved in brain injury, ranging from the force and site of impact and the subsequent neurophysiological damage involved, to the individual's resilience or vulnerability in terms of factors such as age and premorbid functioning, it is difficult to categorise the severity of brain injury precisely. Furthermore, there is often "insufficient evidence to establish a definite threshold for the severity of the closed head injury" (American Psychiatric Association, 1994, p. 704). In an attempt to overcome the difficulty in rating brain injury severity, various methods of assessing severity have been introduced. The Glasgow Coma Scale (GCS), developed by Teasdale and Jennett (1974) and based on neurological responses, appears to be the most commonly used rating scale for assessing severity (Arciniegas, Anderson, Topkoff, & McAllister, 2005; Petchprapai & Winkelman, 2007). Alternatively, time measures of loss of consciousness (LOC) and posttraumatic amnesia (PTA) have been correlated with the severity and outcome of TBI and are used as severity measures (Levin, Williams et al., 1988; Newcombe, Rabbitt, & Briggs, 1994). Other classification systems devised to assist in TBI severity rating are described below.

2.5.1 Glasgow Coma Scale (GCS)

The GCS is a quantitative assessment providing a general guide to the level of injury severity, whereby the improvement and deterioration in decreased consciousness and coma can be assessed during the initial stages of brain damage (e.g., Crippen 2009; Teasdale & Jennett, 1974). The GCS score is calculated according to the patient's responses on three neurological functions: eye opening, motor response and verbal response (see Table 1). The total minimum GCS score is 3 and the maximum score is 15. Scores of 13-15 indicate mild TBI or the absence of TBI, 9-12 indicates moderate TBI, and 3-8 indicates severe TBI (Clinical Psychology Associates, 2008; Lezak et al., 2004; Teasdale & Jennett, 1974).

As the standard for assessing TBI, the GCS permits objective, reproducible evaluation of neurological status and is a relatively easy means of monitoring the individual's

neurological functioning over time (Rangel-Castilla et al., 2008). Furthermore, the GCS is recognised internationally as the most valid and reliable measure of severity, although its reliability depends on the absence of confounding features such as blood alcohol, sedation or concomitant factors (e.g., Fisher & Mathieson, 2001; Rangel-Castilla et al., 2008; Ruff & Grant, 1999). These factors include endotracheal intubation preventing speech, periorbital trauma possibly hindering eye opening, and paralysis possibly preventing the desired motor response (Lezak et al., 2004; Rangel-Castilla et al., 2008). The GCS motor component score is the most predictive component for TBI severity and has the strongest correlation with neurological outcome (Bay & McLean, 2007; Rangel-Castilla et al., 2008).

While moderate and severe TBI are relatively clearly defined and validated on the GCS, the criteria for MTBI (apart from a GCS score of 13-15) is less clear, mostly as a result of the variable presentation of MTBI (Comerford, Geffen, May, Medland, & Geffen, 2002). Therefore, with less predictive validity for GCS scores implying 'mild' severity, the classification of MTBI has been less uniform across medical research and sports settings (e.g., Clinical Psychology Associates, 2008; Segatore & Way, 1992). Hence, modifications to the GCS in the forms of the GCS-Extended (GCSE) and the GCS 15 have been proposed to cover MTBI. The GCSE includes an additional subscale to incorporate PTA, with a scale ranging from 0 to 7, and covering the presence of amnesia for more than 3 months (0) through to no amnesia (7). The GCS 15, in addition to a GCS score of 15, assesses three risk levels (low, intermediate and high risk) according to the presence of symptoms, posttraumatic amnesia and loss of consciousness. Despite these proposed models, and given that the GCS lacks sensitivity to MTBI, the GCS continues to receive support in clinical practice guidelines (Bay & McLean, 2007).

Table 1

Glasgow Coma Scale Response and Scores

Eye opening	Score	Motor response	Score	Verbal response	Score
No response	1	No response	1	No response	1
To painful stimulation (not to face)	2	Extensor posturing to pain	2	Utters / groans incomprehensible sounds	2
To speech stimuli	3	Flexor posturing to pain	3	Utters inappropriate words, not sentences	3
Spontaneous eye opening	4	Withdrawal movements as response to pain	4	Converses but is disoriented or confused; answers	4
		Localising movements to pain	5	Oriented to person, place and date and converses normally	5
		Follows verbal commands to move	6		

(Information sourced from Clinical Psychology Associates, 2008; Centers for Disease Control and Prevention, 2003; Lezak, 1995, based on Teasdale & Jennett, 1974).

2.5.2 Loss of Consciousness (LOC)

Consciousness refers to the awareness of oneself and surroundings, hence loss of consciousness (LOC) is defined as an "unawareness or inability to respond to the environment…but does not include transient confusion or other alterations of mental status (e.g., feeling dazed, disoriented, or confused)" (Petchprapai & Winkelman, 2007, p 3). Various theories have been proposed as to how LOC occurs as a result of TBI. These include the Reticular theory which postulates that the brainstem's reticular formation is temporarily paralysed following a blow; the Centripetal Hypothesis which postulates that mechanically induced force strains and disrupts the brain function; the Pontine Cholinergic System theory which postulates that cholinergic neurons are activated and consequently suppress behavioural responses; and the Convulsive Hypothesis which postulates that changes in general neuronal firing results in LOC (Shaw, 2002; Mendez, Hurley, Lassonde, Zhang, & Taber, 2005). However, it is uncertain whether one particular mechanism or a combination of mechanisms, suitably accounts for LOC. Levels of consciousness range on a continuum from being fully alert, to drowsiness, stupor, and finally coma (Lezak et al., 2004). Crippen (2008, 2009) notes that the clinical assessment of states of consciousness, dependent on visual and palpable findings from the physical examination, may be classified according to one of the categories in Table 2.

Consciousness is controlled and structurally produced by the cerebral hemispheres, pons and the medulla structures. These structures are interconnected by the reticular formation (beginning in the medulla, extending to the midbrain, and forming the reticular activating system) that modulates the perception of events and integrates responses (Crippen, 2008; Crippen, 2009). Accordingly, impairments of consciousness are considered indicative of pathological conditions in the brain, with a correlation between persistent or profound consciousness impairment and white matter lesions of greater depth, although some patients remain unconscious despite no lesion presence (e.g., Lishman, 1987; Rangel-Castilla et al., 2008). In general, however, the depth and duration of coma is considered indicative of TBI severity, particularly if the duration is of hours or days (Cantu, 2001). In contrast, the duration of unconsciousness lasting many hours can result in a complete and uneventful recovery (Lishman, 1999). Furthermore, in the case of MTBI, sport-related studies have found no correlation between the presence or absence of LOC and neuropsychological outcome (Iverson, Lovell, & Smith, 2000; Lovell, Iverson, Collins, McKeag, & Maroon, 1999).

Table 2

Levels of Consciousness

CATEGORY	DESCRIPTION OF STATE
Cloudy consciousness	Mild information processing speed defects caused from macro tearing and the disruption of intercellular connectivity and connectivity between brain regions, and mechanical and/or biochemical vascular compromise resulting in areas of impaired or nonfunctional tissue in the parenchyma. Cloudy consciousness may occur after mild-to-moderate brain trauma and can persist for several months. Long-term memory normally remains intact, but memory for recent events may be diminished.
Lethargy	There appears to be decreased alertness and ability to perform tasks that are usually accomplished effortlessly. Individuals may rouse briefly in response to stimuli, but then settle into inactivity when alone. Awareness of the immediate environment is retained.
Obtundation	There appears to be a decrease in alertness and awareness. Individuals rouse briefly in response to stimuli and can follow simple commands. Inactivity when stimulation ceases. Unawareness of the immediate environment.
Stupor	Unable to communicate clearly but responds to painful stimuli, e.g., withdraws from painful stimuli. Individual settles back into inactivity when stimuli cease.
Coma	No response to even intense stimuli.
Brain death	Irreversible cessation of entire brain function.

(Information sourced from Crippen, 2008, 2009)

2.5.3 Posttraumatic Amnesia (PTA)

Posttraumatic Amnesia (PTA) in relation to TBI refers to the memory loss following brain injury. Although no universally acceptable definition for PTA exists (Ahmed, Bierley, Sheikh, & Date, 2000), PTA is often considered to comprise two types of amnesia: retrograde amnesia and anterograde amnesia (Cantu, 2001). Retrograde amnesia is the memory loss for or inability to remember recent events or information experienced pretrauma (Cantu, 2001; Levin, Benton, & Grossman, 1982; Lynch & Yarnell, 1973). Therefore, the measurement of retrograde amnesia duration is from the actual time of the closest incident prior to the injury that can be recalled, up until the time of the injury. Retrograde amnesia is usually of short duration, lasting from a few seconds to a minute, except in severe TBI cases when the duration may be days or weeks (Lishman, 1999).

On the other hand, anterograde amnesia is the memory loss for or inability to form new memories in order to remember events or new information experienced posttrauma, and is often considered the hallmark of PTA or as being synonymous with PTA (e.g., Ahmed et al., 2000; Cantu, 2001; Levin et al., 1982). Therefore, the measurement of anterograde amnesia duration commences at the time of injury and ends at the time that continuous memory returns (Lishman, 1987, 1999; Petchprapai & Winkelman, 2007; Swann & Teasdale, 1999). Determining exactly when continuous memory returns is difficult in that it is not when the individual first registers an experience, but when the registration and memory for such is constant, and the individual can give a clear and successive account of what is happening in his or her immediate environment (e.g., Gronwall & Wrightson, 1980; Lezak, 1995; Lishman, 1999). Termination of PTA is usually abrupt, except in severe injuries when there are often difficulties with long-term memory, and in some instances patchy, islands of memory appear before restoration of continuous memory (Lishman, 1999).

The PTA condition includes impaired orientation and therefore proposals have been made that PTA be termed ‘posttraumatic confusional state’ (Cantu, 2001; Stuss et al., 1999). The usual sequence of orientation recovery is for person, followed by place, and then time (Levin, 1989). Due to some degree of temporal disorientation, PTA duration can only be determined with some certainty retrospectively (Lishman et al., 1999). However, when assessing PTA duration, although questions aimed at assessing short-term memory may

be answered proficiently, neurocognitive recovery is often inadequately restored for memory traces to be laid down as a permanent record (Lishman et al., 1999). Therefore, the disoriented individual finds it difficult to acquire, learn and retrieve new information (Baddeley, Harris, Sunderland, Watts, & Wilson, 1987).

When an individual suffers coma, then traditionally PTA duration covers coma duration and the period of anterograde amnesia (Lezak et al., 2004). However, including coma duration is misleading, the reason being that patients with a coma of short duration and yet with prolonged amnesia, are considered to have an equivalent PTA duration to patients who experience prolonged coma (Ahmed et al., 2000; Levin et al., 1982). Hence, Levin et al. (1982) proposed that the measure of PTA duration should commence following coma. Generally, PTA duration is approximately four times the duration of coma, and although PTA duration is linked and broadly proportional to coma duration, the two each measure discrete yet overlapping neurologic events (e.g., Brooks, Symington, Beattie, & Campsie, 1989; Levin et al., 1982; Lezak et al., 2004; Richardson, 1990).

PTA duration is generally considered more reliable than the duration of unconsciousness in assessing TBI severity (Cantu, 2001; Lezak, 1995). Although there is no agreement that PTA is a superior or more sensitive predictor of prognosis than depth and duration of unconsciousness, PTA has been found to be more accurate than the length of coma in predicting cognitive outcome two years post injury (e.g., Cantu, 2001; Lezak et al., 2004). Additionally, PTA duration has been found to be the strongest predictor of neurocognitive recovery at six months and also between two to five years post injury (Levin, Papanicolaou, & Eisenberg, 1984). However, while some authors consider that PTA duration is useful for prognosis in only more severe cases of TBI or in certain extreme cases of MTBI (e.g., Levin et al., 1982; Lezak et al., 2004), other authors consider that PTA is the most dependable prognostic marker in cases of MTBI generally (e.g., Cantu, 2001; Stuss et al., 1999). Given that PTA duration is considered relevant to assessing the severity and outcome of TBI, a useful table based on work by Bigler (1990), that incorporates PTA duration as a direct measure of TBI severity and accommodates MTBI, is provided (Table 3).

PTA duration correlates well with the GCS ratings (Levin et al., 1982; Lezak et al., 2004). However, PTA duration has been found to be superior to the admission GCS in predicting prognosis, according to outcomes measured on the Glasgow Outcome Scale twelve months post injury (Katz & Alexander, 1994; van der Naalt, van Zomeren, Sluiter, & Minderhoud, 1999).

Table 3

Estimates of Brain Injury Severity Based on Duration of Posttraumatic Amnesia (PTA)

Severity Estimate	*PTA Duration*
Very mild	Less than 5 minutes
Mild	5-60 minutes
Moderate	1-24 hours
Severe	1-7 days
Very Severe	1-4 weeks
Extremely Severe	Longer than 4 weeks

(Information sourced from Lezak et al., 2004, p.160, based on Bigler, 1990)

2.5.4 Classifications of Traumatic Brain Injury

In order to address the medical fraternity's requirement that codes be supplied for various illnesses and injuries, the International Classification of Diseases (ICD-10) (2006) provides codes pertaining to TBI severity, but no clear diagnostic measures for severity classification. Regarding the clinical criteria for diagnosing TBI, the Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV) published by the American Psychiatric Association (1994), includes neither a classification system for TBI in general nor for MTBI specifically. However, the section 'Criteria Sets and Axes Provided for Further Study' in the DSM-IV, includes the Postconcussional Disorder. Review of the literature reveals that the Department of Veteran Affairs (2009a; 2009b) VA/DoD Clinical Practice Guideline for Management of Concussion/Mild Traumatic Brain Injury, provides a classification of TBI severity, which appears to be the most recently published evidence-based severity guideline. For the VA/DoD classification

purposes, severity of TBI is based on structural imaging findings, time delineated LOC, alterations in consciousness/mental status and PTA, and the best GCS score obtained within 24 hours of TBI. While the severity level of TBI is of prognostic value, it may not be predictive of the patient's ultimate functional outcome (Department of Veteran Affairs, 2009b; Rassovsky et al., 2006). The VA/DoD classification is provided in Table 4.

Overall, GCS ratings and the duration of both LOC and PTA are the main measures employed in classifying TBI as mild, moderate or severe. Such measures may in turn provide some indication of prognosis, albeit of limited application in predicting the final outcome of TBI.

Table 4

VA/DoD Classification of TBI Severity

Criteria	**Mild**	**Moderate**	**Severe**
Structural imaging	Normal	Normal or abnormal	Normal or abnormal
Loss of Consciousness (LOC)	0–30 min	> 30 min and < 24 hours	> 24 hrs
Alteration of consciousness/mental state (AOC) *	a moment up to 24 hrs	> 24 hours. Severity based on other criteria	
Posttraumatic amnesia (PTA)	0–1 day	> 1 and < 7 days	> 7 days
Glasgow Coma Scale (best available score in first 24 hours)	13-15	9-12	< 9

* Alteration of mental status must be immediately related to the trauma to the head. Typical symptoms would be: looking and feeling dazed and uncertain of what is happening, confusion, difficulty thinking clearly or responding appropriately to mental status questions, and being unable to describe events immediately before or after the trauma event.

(Sourced from Department of Veteran Affairs, 2009b, p. 8)

2.6 OVERVIEW

This chapter provided a brief expose of TBI in general. TBIs are postulated to occur along a continuum, with MTBI at the mild end of the TBI severity continuum (Guskiewicz et al., 2004; Mahoney, 2009; McCrea, 2008; Reitan & Wolfson, 2000; Satz, 2001) and therefore, the pathophysiology along the severity continuum is deemed similar.

Specifically, sport-related concussions, which will be elaborated upon in the following chapters, may not be limited to mild TBI severity. In some instances these concussions involve moderate TBI severity (Chamelian & Feinstein, 2004*;* King, Crawford, Wenden, Caldwell, & Wade, 1999; McCauley, Boake, Levin, Contant, & Song, 2001; Parkinson, 2000; Petchprapai & Winkelman, 2007; Shaw, 2002; Terrell, 2004). In other instances these concussions involve severe TBI, that may be fatal should a second MTBI event be incurred while recovery from an initial MTBI event is incomplete (Bailes & Cantu, 2001; Bender, Barth, & Irby, 2004; Bowen, 2003; Heilbronner & Ravdin, 2004). Moreover, consensus on the precise delineation of mild TBI from more severe forms of TBI is questionable, and therefore MTBI cannot be understood as a totally discrete entity from TBI in general (Reitan & Wolfson, 2000). Nevertheless, there are specific issues that pertain particularly to MTBI, and these warrant detailed exposition, as will be pursued in the following chapter.

CHAPTER THREE: MILD TRAUMATIC BRAIN INJURY

The focus of this chapter is on mild traumatic brain injury (MTBI), the least severe form of TBI on the TBI severity continuum. Initially, MTBI will be defined, and the terms 'concussion' and 'subconcussion' in relation to MTBI presented. The mechanisms and biomechanisms and subsequent neurophysiological consequences of MTBI will then be reviewed. The most immediate signs of MTBI will then be discussed, including loss of consciousness (LOC), and posttraumatic amnesia (PTA) specifically as they relate to MTBI, and following this the rating of MTBI severity will be briefly elucidated. Hereafter a description of the (i) neurocognitive and (ii) symptomatic neuropsychological consequences of MTBI are described. The resolution of MTBI and factors affecting outcome are presented. The chapter concludes with a description of the Reserve theory, encompassing the Cognitive Reserve and Brain Reserve theories. This forms the theoretical foundation for this study, being proposed as a central mechanism underlying interindividual variability in respect of MTBI outcome.

3.1 DEFINITION OF MILD TRAUMATIC BRAIN INJURY (MTBI)

Historically, definitions of mild traumatic brain injury (MTBI) have been vague and ambiguous (Leclerc, Lassonde, Delaney, Lacroix, & Johnston, 2001; Ruff & Jurica, 1999). The inconsistent presentation of MTBI, in the form of varied signs and symptoms, seemingly accounts for difficulty of defining MTBI concisely and accurately (Leclerc et al., 2001; Martineau, Kingma, Bank, & Valovich-McLeod, 2007). Therefore, no single definition of MTBI is unanimously or universally accepted in the literature (Cantu, 1992, 1996, 1997b; Guskiewicz, Weaver, Padua, & Garrett, 2000; Heilbronner et al., 2009; Kontos, Collins, & Russo, 2004; Leclerc et al., 2001; Lovell, Collins, & Bradley, 2004; Poirier & Wadsworth, 2000; Pretz, 2007; Powell, 2004; Rutherford, Stephens, & Potter, 2003; Satz, 2001).

A number of the more prominent definitions of MTBI are summarised in Table 5. From the definitions in the table, it is evident that various authors and bodies have used different criteria in their MTBI definitions (e.g., GCS scores, LOC, PTA, and the presence of abnormal neurological signs and symptoms). Only a few definitions include GCS scores of 13-15 (e.g., ACRM, 1993; Carroll, Cassidy, Holm, Kraus, & Coronado,

2004; Rimel, Giordani, Barth, Boll, & Jane, 1981). The possible reason for the exclusion of GCS scores is that the GCS criteria need to be administered within a few hours of injury and not retrospectively. This is because a GCS score can improve considerably in the time between injury and medical evaluation and therefore may not necessarily indicate brain injury if assessment is delayed (Rees, 2003; Ruff, 2005; Ruff & Jurica, 1999).

In contrast, all definitions in the table include some disturbance in consciousness. Kelly et al. (1991) appear to have provided the first definition stating that LOC need not be a necessary prerequisite for MTBI. However, in the definitions where LOC is specifically stipulated, all indicate LOC need not be present, apart from the definition proposed by Rimel et al. (1981) that requires the presence of LOC for 20 minutes or less. None of the tabled MTBI definitions requires LOC in excess of 30 minutes duration.

Apart from Russell and Smith (1961), PTA is not generally mentioned in the tabled MTBI definitions prior to 1993 (e.g., Rimel et al., 1981; Kelly et al., 1991). The American Congress of Rehabilitation Medicine (ACRM, 1993) provided the definition of MTBI specifying PTA and neurological symptoms as the key diagnostic criteria for MTBI (Ruff, 2005). After 1993, most definitions refer specifically to amnesia or memory dysfunction, with the exception of the three Concussion in Sport Group (CISG) definitions (e.g., Aubry et al., 2002; McCrory, Johnston et al., 2005; McCrory et al., 2009). The Concussion in Sport Group (CISG) considers that the type, extent and duration of MTBI symptoms are more important than PTA, in that estimates PTA duration are unreliable and variable (McCrory, Johnston et al., 2005).

The ACRM (1993) definition is the most widely accepted MTBI definition in the USA (e.g., Department of Veteran Affairs, 2009a, 2009b; Willer & Leddy, 2006). The ACRM (1993) defined MTBI as a traumatically induced physiological disruption of brain functioning, as a result of the head being struck by or striking an object, or indirect trauma to the head incorporating an acceleration/deceleration movement to the brain (i.e., whiplash). This results in one or more of the following: LOC for a period of 30 minutes or less; a GCS score of 13-15; retrograde amnesia or PTA with the latter not exceeding a 24 hour period; an alteration of mental state, e.g., feeling dazed, disorientated or

confused; and the presence of one or more focal neurological signs that may or may not be transient, e.g., double vision, loss of taste, smell or balance. The definition includes the proviso that imaging, encephalography or neurological evaluations may be normal.

The most recent evidenced-based consensus document on MTBI, is the VA/DoD Clinical Practice Guideline: management of concussion/mild traumatic brain injury (Department of Veteran Affairs, 2009a, 2009b). The Department of Veteran Affairs (2009a, 2009b), used the ACRM (1993) definition of MTBI it its consensus document. The Department (2009a, 2009b), recommends that a diagnosis of MTBI be made when there is injury to the head in the form of blunt trauma or acceleration/deceleration forces, resulting in at least one of the following:

1. Any observed or self-reported period of (i) transient disorientation, confusion or impairment in consciousness, (ii) memory dysfunction for the period immediately before or following the time of injury, (iii) LOC of less than 30 minutes duration; and

2. Observed signs of impaired neurological or neuropsychological function soon after injury that support the diagnosis, e.g., headache, fatigue, irritability, dizziness, or diminished concentration, although such cannot be used for diagnostic purposes without loss of or alterations in consciousness (Department of Veteran Affairs, 2009b, p. 9).

The ACRM (1993) is clear in its differentiation of mild TBI from moderate TBI (Flanagan, 1999). It defines moderate TBI as any non-penetrative traumatic force to the brain resulting in the alteration of consciousness in the form of unconsciousness persisting beyond thirty minutes, or a posttraumatic amnesia persisting beyond 24 hours. Thus, MTBI is defined as any non-penetrative traumatic force to the brain resulting in the alteration of consciousness (that may or may not include loss of consciousness for a period of less than thirty minutes) or a posttraumatic amnesia persisting for less than 24 hours (ACRM, 1993). For the purposes of this study, the ACRM (1993) definition is used. However, MTBI includes signs and symptoms that show as neuropsychological and functional impairment, as a result of underlying structural impairment. Therefore, structural impairment (i.e., neurophysiological impairment) is included in this study's definition, in that it appears to be a necessary condition for functional impairment to occur (e.g., Cantu, 1996; 2001; Mathias, Beall, & Bigler, 2004; Rutgers et al., 2007; Tellier et al., 1999).

Table 5
A Selection of MTBI/Concussion Definitions

Source	GCS SCORE	LOC (minutes)	PTA (hours)	Other factors
Russell & Smith, 1961	-		< 1	Transient disturbance in consciousness
Committee on Head Injury, 1966	-			Clinical syndrome involving immediate and transient posttraumatic impairments in neural function due to brain stem involvement, e.g., alteration of consciousness, disturbances in vision and balance.
Rimel, Giordani, Barth, Boll, & Jane, 1981	13-15	LOC ≤ 20	-	< 48 hours hospitalisation and LOC for 20 minutes or less.
Kelly et al., 1991	-	None or brief LOC	-	Trauma induced alteration in mental status that may or may not involve loss of consciousness.
ACRM, 1993 American Congress of Rehabilitation Medicine, 1993	13-15	None or LOC ≤ 30	≤ 24	Traumatically induced physiological disruption of brain functioning, also involving an alteration of mental state (e.g., dazed, disoriented or confused) and one or more focal neurological signs (may or may not be transient).
AAN, 1997 American Academy of Neurology (AAN), 1997	-	None or brief LOC	May be Present	Trauma induced alteration in mental status that may or may not involve loss of consciousness. Confusion and amnesia, the hallmarks of concussion, may follow immediately or several minutes after a blow to the head.
AOSSM, 1997 American Orthopaedic Society for Sports Medicine Wojtys, Hovda, Landry, Boland, Lovell, McCrea et al., 1999	-	None or brief LOC	May be present	Alteration in cerebral function by a direct or indirect (rotation) to the head. One or more signs and symptoms, e.g., LOC, amnesia, cognitive and memory dysfunction, difficulty concentrating, light-headedness, vertigo, tinnitus, blurred vision, photophobia, headache, nausea, vomiting, or balance disturbance. Delayed signs and symptoms, e.g., sleep irregularities, fatigue, lethargy, personality changes, depression, or an inability to perform usual daily activities.
NCIP, 2003 National Center for Injury Prevention and Control, 2003	-	None or LOC < 30	May be present	Injury to the head (blunt trauma or acceleration/deceleration forces). One or more of the following: transient disorientation/confusion/impaired consciousness; memory dysfunction; LOC less than 30 minutes; seizures; symptoms, e.g., headache, fatigue, irritability, dizziness, impaired concentration, in support of diagnosis but in conjunction with LOC or altered consciousness; (infants/young children- vomiting, lethargy, irritability).

Table continues on page 29

Table continues from page 28

Table 5
A Selection of MTBI/Concussion Definitions

Source	GCS SCORE	LOC (minutes)	PTA (hours)	Other factors
Concussion in Sport Group (CISG), Vienna, 2001 (Aubry et al., 2002)	_	None or brief LOC	_	A biomechanical force to the head or body, induces pathophysiological changes that act on the brain resulting in (i) neurological impairment (acute onset, short duration, resolving spontaneously), (ii) acute neuropathological changes (functional disruption rather than structural damage), (iii) clinical syndromes (graded, usually sequential set, sometimes including LOC, and (iv) no structural abnormalities on neuroimaging.
CISG, Prague, 2004 (McCrory, Johnston, Meeuwisse, Aubry, Cantu, Dvorak et al., 2005)	_	None or brief LOC	_	Same as CISG definition above (Aubry et al., 2002), with the added proviso that symptom may be persistent or prolonged. Adding simple concussion - no complications, resolves in 7-10 days, and complex concussion - complications, not resolved in 10 days, and specific sequelae such as LOC or convulsions.
CISG, Zurich, 2008 (McCrory, Meeuwisse, Johnston, Dvorak, Aubry, Molloy et al., 2009)	_	None or brief LOC	_	Same as CISG definition above (McCrory, Johnston et al., 2005), but excludes the simple and complex concussion classification. Alludes to concussion and MTBI being separate entities, but no clarification of differences between the two terms is provided.
WHO Collaborating Centre Task Force on MTBI (Carroll, Cassidy, Holm, Krauss, & Coronado, 2004)	13-15 ≥30 minutes post injury	None or LOC ≤30	None or < 24	Acute injury as a result of mechanical energy to the head from external physical forces (not due to another injury or effects of medication, drugs or alcohol) with one or more of the following: (i) LOC as described, or (ii) PTA as described, or (iii) confusion or disorientation, or (iv) transient neurological abnormalities or focal signs (e.g., intracranial lesions not requiring surgery and seizures).
VA/DoD (2009) (Department of Veteran Affairs, 2009a, 2009b)	-	Transient confusion, disorientation, or impaired consciousness or LOC < 30	Memory dysfunction immediately pre- or post-injury	Injury to the head from either a blunt trauma, acceleration or deceleration forces, or a blast resulting in one or more of the following: a) transient confusion, disorientation or impaired consciousness; memory dysfunction for period immediately pre- or post-injury; LOC for less than 30 minutes, and b) signs soon after injury of neurological or neuropsychological dysfunction supporting MTBI diagnosis (but not used for diagnosis in absence of impaired consciousness or LOC), e.g., headache, dizziness, irritability, poor concentration and fatigue. Severity is judged by the characteristics at the time of injury, as opposed to assessment of symptom severity at varying points postinjury.

3.2 OTHER TERMS RELATING TO MTBI

The term ‘mild traumatic brain injury’ has been used interchangeably with the terms ‘minor brain injury’, ‘minor head injury’, ‘mild head injury’, ‘cerebral concussion’ and ‘concussion’ (Barth, Varney, Ruchinskas, & Francis, 1999; Bender et al., 2004; Levin, Eisenberg, & Benton, 1989; Maroon et al., 2000; Von Holst & Cassidy, 2004). The term concussion is frequently used to describe MTBI (Flanagan, 1999). However, concussion may differ conceptually from MTBI, and is therefore discussed below. Additionally, the term subconcussion is often used in literature pertaining to MTBI and is defined in section 3.2.2.

3.2.1 The Term Concussion

The word concussion is derived from Latin, *concussus* or *concutera*, meaning, “to shake violently” (Cantu, 2001; Maroon et al., 2000, p. 662; Philips, 2003). Concussion is not a recent phenomenon in that the signs and symptoms of this condition appear in the early writings of Hippocrates (460-370 BC), Galen (129-216 AD), and Pare (16th century) who used the term ‘*commotio cerebri*’ to describe this condition (Bender, Barth, & Irby, 2004; Maroon et al., 2000; McCrory & Berkovic, 2001).

Several unsuccessful attempts have been made to differentiate MTBI and concussion. For example, it is proposed that an intracranial haemorrhage or lesion on standard neuroimaging is excluded in concussion, but not in MTBI, suggesting that concussion is a milder form of MTBI (e.g., Anderson et al., 2006; Legome & Alt, 2009; Levine, 2010). In contrast, it has been argued that concussion is not limited or unique to MTBI and can transpire in moderate TBI (Chamelian & Feinstein, 2004*;* King et al., 1999; McCauley et al., 2001; Parkinson, 2000; Petchprapai & Winkelman, 2007; Shaw, 2002; Terrell, 2004). A further difference posited is that concussion is a ‘mechanism’ of injury, whereas MTBI is the result of injury (Nelson, Jane, & Gieck, 1984). Furthermore, although concussion can arise as the result of various traumas, some authors consider concussion a specific type of MTBI unique to that sustained through sport (Levine, 2010; Lezak et al., 2004; Petchprapai & Winkelman, 2007).

Given that concussion is often regarded as a type of MTBI sustained through sport, the term concussion frequently appears in sport literature, in reference to mild injuries in contact sport and not routinely involving LOC (Lovell et al., 1999). Contact sport-related MTBI typically involves multiple MTBI events over the years of sport participation, and is likely to incur more pronounced effects than a single non sport-related MTBI. Therefore, sport-related and non-sport-related MTBI could be viewed as separate entities (e.g., Binder, Rohling, & Larrabee, 1997; Carroll, Cassidy, Holm et al., 2004). In 2008, the CISG panel considered separating concussion from MTBI in their definition, proposing that concussion and MTBI are two separate constructs (McCrory et al., 2009). However, this was not implemented.

Although it appears that MTBI is the popular and commonly accepted scientific term in recent literature, the terms MTBI and concussion are frequently used interchangeably within documents (e.g., Anderson et al., 2001; de Kruijk, Leffers, Menheere et al., 2002; Department of Veteran Affairs, 2009a, Department of Veteran Affairs, 2009b; Flanagan, 1999; Guskiewicz et al., 2004; Lezak et al., 2004). Therefore, these terms are used interchangeably in this study, regardless of the term used in the literature, on the grounds that these two terms refer to the same condition.

3.2.2 The Term Subconcussion

Reference is often made to subconcussion or 'microtrauma' (e.g., Erlanger, Kutner, Barth, & Barnes, 1999; Green & Jordan, 1996; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008). 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, 2007a, p. 33). Although subconcussive effects have only been demonstrated in experimental studies on animals, there is reasonable evidence to extrapolate the existence of such events to humans (e.g., Killam et al., 2005; Kutner, Erlanger, Tsai, Jordan, & Relkin, 2000; Rutherford, Stephens, Potter, & Fernie, 2005; Shuttleworth-Edwards, Smith et al., 2008). These proposed incidents of microtraumatic brain injury, that are neither outwardly detected nor severe enough to warrant being termed a concussion *per se*, are postulated to have cumulative effects as occurs with diagnosable concussive events, with the implication of resulting in chronic neuropsychological

sequelae (Erlanger et al., 1999; Killam et al., 2005; Rutherford et al., 2003; Shuttleworth-Edwards, Smith et al., 2008).

3.3 MECHANISMS AND BIOMECHANISMS OF MTBI

The mechanisms and biomechanisms for TBI (see Chapter 2) are similar for MTBI (e.g., Bernhardt, 2009; Gaetz, 2004). These processes may be of a reduced intensity compared to the more severe forms of TBI, or one individual may exhibit less vulnerability than another to mechanisms with a force of equivalent intensity. However, the force at the time of injury does not necessarily determine the severity of injury, because sometimes a high level of force does not result in injury and sometimes a low level of force does result in injury (Cantu, 1996). Nevertheless, in order for a MTBI to occur, there needs to be a trauma preceding the brain injury, in the form of external physical impact to the body or head, or a blunt force causing rotation, acceleration or deceleration to the head (Alexander, 1995; ACRM, 1993; American Psychiatric Association [APA], 1994; Centers for Disease Control and Prevention (CDC), 2004; Department of Veteran Affairs, 2009a, 2009b; Petchprapai & Winkelman, 2008).

Until the early 1990s, MTBI was considered a temporary disruption of consciousness with no chronic sequelae, and if these occurred they were considered reversible. However, studies have since found that MTBI is likely to result in both structural and functional changes to the brain, with the potential to induce persistent chronic neuropsychological sequelae (Gaetz & Bernstein, 2001). Accordingly, the neurophysiological and neuropsychological consequences of MTBI will be described.

3.4 NEUROPHYSIOLOGICAL CONSEQUENCES OF MTBI

The neurophysiological changes induced by MTBI include structural changes and biochemical changes in the brain tissue at a micro level. As indicated earlier, structural injuries were not previously suspected in MTBI, however it is now known that they do occur (e.g., Bigler, 2003; Cantu, 2001; Gaetz, 2004; Rutherford et al., 2003). These structural changes, in the form of DAI, haemorrhages and contusions, will be discussed first. This will be followed by a description of the biochemical changes (neurochemical

and metabolic) involved in MTBI. Finally, secondary neurophysiological consequences, as a result of a repeat MTBI event, will be elucidated.

3.4.1 Structural Changes

In the event of MTBI, brain areas below the coup and contracoup sites may be injured as a result of translational forces, resulting in contusions and the shearing of the cortical surface (Lezak et al., 2004; Rutherford et al., 2003). Diffuse or multifocal brain injury is likely to occur as a result of rotational forces (Barth et al., 1999). These diffuse microscopic lesions form when the white fibrous axonal tracts (that connect the cortex to the midbrain and brain stem) and their radiating axons shear as a result of impact forces, retract and produce balls of axoplasm termed axonal bulbs (e.g., Lezak et al., 2004; Rutherford et al., 2003). Axonal bulbs can also occur when axonal neurofilaments undergo compaction from five minutes until six hours post injury, with a subsequent secondary axotomy occurring some weeks later (Giza & Hovda, 2004). Neural and vascular damage to the parasagittal white matter of the cerebrum and the junctions of the cerebral cortex's grey-white matter, as a result of the impact forces encountered in MTBI, commonly occur in the anterior and inferior frontotemporal areas of the brain. Damage may also affect the reticular system and upper brainstem resulting in possible loss of consciousness, or the corpus callosum and diencephalic structures adjacent to the third ventricle, which might account for long-term memory impairment that sometimes results from MTBI (Kirkendall, Jordan, & Garrett, 2001; Rutherford et al., 2003; Silver, McAllister, & Arciniegas, 2009).

3.4.2 Biochemical Changes

Trauma to the brain also causes neurochemical changes at the micro level, exposing neurons to metabolic stress and vulnerability (Hovda et al., 1999; Rutherford et al., 2003). These neurochemical changes at a cellular level involve a hypermetabolic phase characterised by an interrelated efflux of potassium, an energy crisis (in the form of glycolysis, decreased oxidative metabolism and increased lactate production), an influx of calcium, an efflux of magnesium, and alterations in neurotransmission, and is then followed by a hypometabolic phase. Giza and Hovda (2004) explain the neurochemical changes in MTBI, based on studies of experimental TBI models, as follows. During the

hypermetabolic phase, in the first few minutes after a concussive impact, extracellular potassium levels increase dramatically and loss of consciousness may result. The increase in extracellular potassium is caused by disruption to the membrane of the neuron cells when the axons are stretched and open channels for the efflux of the voltage-dependent potassium. This depolarisation of the neurons stimulates the neurotransmitter glutamate that exacerbates the potassium efflux further. In an attempt to restore the membrane potential (i.e., the equilibrium of sodium and potassium), increased quantities of adenosine triphosphate (ATP) are consumed which initiates cellular glycolysis. Therefore, precisely when there is an increased demand for ATP, the ATP production is reduced, which triggers an energy crisis in the brain (Giza & Hovda, 2004).

Concurrent with decreased levels of ATP in the brain, decreased oxidative metabolism occurs. Reduced oxidase activity has been found in the ipsilateral cerebral cortex day one post injury, recovered day two post injury, reinstated day three, becoming most pronounced day five, and fully restored day ten post injury. However, decreased activity day ten post injury has been found in some regions, including the ipsilateral hippocampus. While this period of hyperglycosis and decreased oxidative metabolism ensues, there is a resultant and concomitant increase in lactate production from the glia. Increased lactate production has been associated with neuronal dysfunction in that it induces acidosis, membrane damage, cerebral oedema and blood brain permeability (Giza & Hovda, 2004). Lactate may, however, be a possible substitute fuel source during the energy crisis (Tsacopoulos & Magistretti, 1996).

Simultaneous to hyperglycosis and decreased oxidative metabolism, there is an influx of calcium to the neuron cells. However, the elevated intracellular calcium level results in dysfunction of the oxidative metabolism, increases the cellular demand for ATP, and activates proteases causing cell damage or death and axonal dysfunction by breaking down the microtubules and neurofilaments. Calcium has been shown to accumulate in the striatum ipsilateral to the injury, cerebral cortex and dorsal hippocampus, with elevated levels enduring for at least 48 hours and levels normalising by day four post-injury. In addition, following brain injury, intracellular magnesium levels reduce and remain low for up to four days. Magnesium is required to maintain the cellular membrane potential and to initiate protein synthesis, and therefore its depletion results in a further influx of calcium (Giza & Hovda, 2004).

Further to these events, there is a concomitant alteration in neurotransmission lasting for days up to months, which is a further possible mechanism accounting for long-term deficits following MTBI. The down regulation of excitatory neurotransmitter receptor functions, particularly of the glutamatergic, cholinergic and adrenergic transmission, has been associated with neurocognitive deficits in attention, memory and learning. Inhibitory neurotransmitter receptor functions, particularly loss of gamma amino butyric acid (GABA) functions, are suggestive of a relationship between disinhibition and seizure development, as a result of MTBI. Concurrent to these ionic and metabolic events, the cerebral blood flow decreases up to fifty percent and may remain at lowered levels for many days post injury, limiting the brain's ability to respond to the increased energy demands. Hypermetabolism and decreased cerebral blood flow result in intracellular acidosis (Giza & Hovda, 2004). Thus, although MTBI may not initially result in extensive neuronal damage, the surviving neurons are vulnerable to minor changes in cerebral blood flow or anoxia that may incur extensive neuronal loss (Cantu, 2001). Following the hypermetabolic phase, a hypometabolic phase occurs, lasting up to ten days (Bowen, 2003; Giza & Hovda, 2004). Whether this hypometabolic phase is protective or increases vulnerability to further injury is unknown, but the disparity between energy demands and energy delivery is considered to account for impairments in consciousness, cognition and memory following MTBI and to cause predisposition to secondary brain injuries (Giza & Hovda, 2004).

3.4.3 Secondary Neurophysiological Consequences

Secondary brain injuries include vulnerability to a second MTBI event, particularly during the acute phase of recovery when the metabolic and ionic derangements can result in cell death. The brain may also be vulnerable weeks after injury, when the neurotransmitter systems are not yet fully restored, and the risk for aberrant connections could result in permanent dysfunction, which may or may not involve cell loss (Giza & Hovda, 2004).

The most serious secondary brain injury in MTBI is Second Impact Syndrome (SIS), although controversy surrounds the definition and existence of SIS (Bernhardt, 2009; Fischer & Vaca, 2004; McCrory, 2001; McCrory & Berkovic, 1998; Mendez et al., 2005). Second impact syndrome is a condition in which an individual sustains a second,

albeit mild, concussion when not fully recovered from a first concussion, and which can cause profound disability, coma or sudden death (Bailes & Cantu, 2001; Baker & Hutchinson, 2008; Bender et al., 2004; Bowen, 2003; Fick, 1995; Heilbronner, & Ravdin, 2004; Kontos et al., 2004; Martineau et al., 2007). When SIS occurs, within two to five minutes following the second impact, it is believed that herniation of the brain and brain stem failure, are implicated (Bailes & Cantu, 2001). Hence the conscious individual appears stunned but within seconds to minutes, collapses into a coma and respiratory failure ensues (Bailes & Cantu, 2001).

Although SIS can occur within days or weeks of the primary MTBI event, it more frequently occurs during the ten-day phase following an initial MTBI, when the brain cells are not completely destroyed but remain in a vulnerable state, and when there may be minor changes in cerebral blood flow or raised ICP, oedema, and apnoea (Bailes & Cantu, 2001; Bender et al., 2004; Bernhardt, 2009; Echemendia & Cantu, 2004; Kelly et al., 1991; Kontos et al., 2004; Martineau et al., 2007; Wojtys et al., 1999). Therefore, closely spaced MTBI events are a more serious risk for SIS, than MTBI events spaced further apart (Bailes & Cantu, 2001; Echemendia & Cantu, 2004).

SIS is rare and its incidence is unknown (Bailes & Cantu, 2001; Cantu, 1995; Cantu & Voy, 1995; Giza & Hovda, 2004; Martineau et al., 2007; McCrory & Berkovic 1998; McCrory, Berkovic, & Cordner, 2000), although a few probable cases have been reported (e.g., Bailes & Cantu, 2001; Martineau et al., 2007; Matser, Kessels, & Lovell, 2004; Mendez et al., 2005). The morbidity rate for SIS is given as 100% and the mortality rate 50% (Martineau et al., 2007). SIS is reported to affect persons less than 21 years of age (Cantu, 1992; Hatfield et al., 2004). However, other authors report that SIS is more prevalent amongst males aged 16 -24 years (McCrory & Berkovic, 1998).

3.4.4 Signs of MTBI

Signs, the outward physical manifestation of MTBI, that may ensue immediately following injury include: delayed cognitive responses in following directions or answering questions, the display of inappropriate emotions (e.g., crying or laughing), inappropriate behaviour playing sport (e.g., running away from the ball or passing the ball to the wrong player), loss of consciousness (LOC) or alteration in consciousness,

confusion, posttraumatic amnesia (PTA), pupillary asymmetry, seizures, slurred speech, vacant stare, vomiting, weakness or numbness in legs or arms (e.g., Department of Veteran Affairs, 2009a; Lovell, Collins, & Bradley, 2004; Powell, 2004). LOC and PTA (already detailed in Chapter 2) are now briefly discussed in relation to MTBI specifically.

Loss of Consciousness (LOC)

Although LOC may occur following MTBI, LOC is not a necessary prerequisite in defining or diagnosing MTBI (e.g., American Academy of Neurology (AAN), 1997; ACRM, 1993; Aubry et al., 2002; Carroll, Cassidy, Holm et al., 2004; Kelly et al., 1991; Kibby & Long, 1996; McCrory, Johnston et al., 2005; McCrory et al., 2009; Ruff, 2005; Ruff & Jurica, 1999; Wojtys et al., 1999). Furthermore, LOC is seldom evident following MTBI and occurs in less than 25% of sport-related MTBIs (e.g., Collins, Grindel et al., 1999; Delaney, Lacroix, Leclerc, & Johnston, 2002; Guskiewicz et al., 2000; Leclerc et al., 2001; Lovell, Bradley, Collins, & Burke, 2003; Macciocchiet al., 1996; McCrea, Barr et al., 2005; Moser et al., 2007). The presence of LOC is not a useful predictor of MTBI severity or outcomes, with no studies found in support of the converse (e.g., Collins, Grindel et al., 1999; Erlanger, Kaushik et al., 2003; Guskiewicz et al., 2004; Lovell et al., 1999; Maroon, Field, Lovell, Collins, & Post, 2002; McMillan, 1997). However, the CISG consider that LOC of more than one-minute duration may alter MTBI management, citing Jennett and Bond (1975) that LOC is predictive of outcome in severe TBI (e.g., McCrory et al., 2009). In contrast, other MTBI studies have found no evidence that LOC results in either specific acute or persistent cognitive deficits, nor in poorer outcomes. Instead, some authors suggest that amnesia, reported memory difficulties, and symptom complaints, are more predictive of outcome than LOC (e.g., Collins, Iverson et al., 2003; Erlanger. Kaushik et al., 2003; Iverson, Lovell, & Smith, 2000; Leininger, Gramling, Farrell, Kreutzer, & Peck, 1990; Lovell et al., 1999; Maddocks & Saling, 1996).

Posttraumatic Amnesia (PTA)

Amnesia or on-field confusion are both generally viewed as important indicators of MTBI (Manko, 2003). The severity of concussion has commonly been evaluated according to the duration of PTA, mostly typified by anterograde amnesia, when the concussed individual is unable to remember events occurring after the injury in a continuous fashion, as a result of memory traces not being reliably consolidated and committed to memory (Ahmed et al., 2000). It is, however, unusual for retrograde amnesia to be reported in the

absence of PTA following MTBI (Paniak, MacDonald, Toller-Lobe, Durand, & Nagy, 1998). Where PTA was deemed a necessary diagnostic prerequisite for MTBI, the provisions for PTA duration were extremely variable and ranged from less than an hour to less than 48 hours (e.g., ACRM, 1993; Carroll, Cassidy, Holm et al., 2004; Kibby & Long, 1998; Miller, 1986; Russell & Smith, 1961). However, estimates of PTA length are subjective and, therefore, unreliable in assessing MTBI severity or outcome (Dikmen & Levin, 1993). Therefore PTA or its duration is excluded from a number of MTBI definitions as a diagnostic prerequisite, and instead the possible presence of amnesia or memory dysfunction is alluded to, with no specified duration, (e.g., AAN, 1997; Aubry et al., 2002; Committee on Head Injury, 1966; Department of Veteran Affairs, 2009a; Dikmen & Levin, 1993; Kelly et al., 1991; McCrory, Johnston et al., 2005; McCrory et al., 2009, Rimel et al., 1981; Wojtys et al., 1999).

The presence of PTA, however, may be implicated in MTBI neurocognitive outcomes. De Monte, Geffen and Massavelli (2006) found that patients who had experienced PTA following MTBI evidenced impaired neurocognitive deficits on information processing, on the Digit Symbol Substitution Test, and impaired verbal memory, on the Speed of Comprehension test, compared with those who had not experienced PTA. Additionally, Hinton-Bayre and Geffen (2002) found PTA was a reliable indicator of MTBI impairment two days postinjury, but the duration of PTA and duration of MTBI were not associated. Collins, Iverson et al. (2003) found that decreased memory scores on neurocognitive testing or an increase in symptom reports two days post MTBI, were significantly associated with a four time likelihood of having experienced PTA and at least five minutes of mental status changes at the time of MTBI.

3.5 RATING MILD TRAUMATIC BRAIN INJURY SEVERITY

Several authors and bodies involved with MTBI research and management, between 1963 and 2004, attempted to classify the severity of MTBI in the form of grading systems or guidelines (Cantu, 2001; Leclerc et al., 2001; Maroon et al., 2002; Peloso, Carroll et al., 2004). The aims of these grading systems were to clearly delineate MTBI in terms of severity, assist medical management in MTBI diagnosis, help assess prognosis, and in certain instances provide return to play guidelines (Whitefield, 2007). Typically, these grading systems classified MTBIs into three grades; with grade 1 being considered the

mildest and grade 3 the most severe. Criteria used in delineating these grades included GCS scores, LOC, PTA, neuroimaging abnormalities, the presence of symptoms, or a combination thereof. For this report, the more prominent grading systems are summarised in Table 6 (at the end of this section). LOC is a criterion in all the grading systems in the table, apart from three involving neuroimaging (e.g., Williams, Levin, & Eisenberg, 1990; Hsiang, Yeung, Yu, & Poon, 1997). PTA is a criterion in four of the grading systems (e.g., Cantu, 1986, 2001; Colorado Medical Society, 1991; Ruff, 2005).

The three concussion grading systems most commonly used were the Cantu Grading System for Concussion, the Colorado Medical Society Grading System for Concussion, and the American Academy of Neurology (AAN) Practice Parameter Grading System for Concussion (Hinton-Bayre & Geffen, 2002; Lovell, Collins, & Bradley, 2004; Titolo, 2009). However, several studies found limited evidence in support of these grading systems. For example, Hinton-Bayre and Geffen (2002) found little concordance within and between the Cantu, AAN and Colorado grading systems; Collins, Iverson et al. (2003) and Erlanger, Kaushik et al. (2003) found LOC an unreliable criterion in predicting MTBI outcome; and Lovell, Collins, Iverson, Johnston and Bradley's (2004) study invalidated the use of concussion grading scales with return to play guidelines. Peloso, Carroll et al. (2004), reviewed and empirically assessed 41 MTBI guidelines. They found that only three (two related to paediatric and one related to adult populations) were evidence based, and that the recommendations of two of these three guidelines, were based on expert opinion. More specifically, in reference to the 18 sport-related MTBI guidelines, none was evidenced based or provided standard measures for grading MTBI during competition. They also varied widely in both return to play recommendations and the number of concussions an athlete could sustain before this impacted on a career (Peloso, Carroll et al., 2004).

Apart from Cantu's Evidenced Based Grading System (Cantu, 2001), none of the grading systems pertaining to adults was based on empirical evidence, and they represented a combination of anecdotal experience, medical consensus and clinical impressions (e.g., Bailes & Hudson, 2001; Cantu, 1997; Leclerc et al., 2001; Lovell, Collins, & Bradley, 2004; Lovell, Collins, Iverson et al., 2004; Maroon et al., 2002; McCrory, 1999a, 1999b; Peloso, Carroll et al., 2004; Terrell, 2004). Furthermore, none of the grading systems have been validated prospectively (Leclerc et al., 2001; McCrory, 1999a). Although these

grading systems differed in several respects, in their favour is that most emphasised the need for athletes to be asymptomatic before resuming sport (Aubry et al., 2002; Guskiewicz et al., 2000; Kelly & Rosenberg, 1997; Kelly et al., 1991; Leclerc et al., 2001; McCrory, Johnston et al., 2005; Quality Standards Subcommittee of the American Academy of Neurology, 1997; Randolph et al., 2009).

In 2004, after MTBI grading systems lost favour, the CISG introduced a new retrospective form of severity classification by categorising concussions as simple or complex **(**McCrory, Johnston et al., 2005). This new classification, however, was neither empirically based nor validated, and neglected the individualised approach recommended for MTBI management i.e., neuropsychological testing in cases of simple concussion (e.g., Iverson, 2007; Meeuwisse, 2007; Shuttleworth-Edwards, 2008). Thus, in 2008 the CISG abolished the simple/complex concussion delineation (McCrory et al., 2009).

In summary, the overarching limitations of the classification systems are their lack of an empirical basis for concussion recovery and empirical validation (Guskiewicz et al., 2000; Leclerc et al., 2001; McKinley, 2007). No grading system was found superior or has been universally adopted, and consequently these arbitrarily devised systems have been abandoned (Alla, Sullivan, Hale, & McCrory, 2009; Anderson & Murata, 2009; Bernhardt, 2009; Heilbronner et al., 2009; Leclerc et al., 2001; Lovell, Collins, Iverson et al., 2004; Maroon et al., 2002; Maroon et al., 2000; Mendez et al., 2005; Peloso, Carroll et al., 2004; Rutherford et al., 2003; Terrell, 2004). As a consequence, an individualised approach to MTBI management has evolved, in which MTBI severity is not rated. Instead, the evaluation, assessment and management of MTBI, is based on the individual's neurocognitive and symptomatic recovery, and includes return to play of sport implementation in a gradual, step-wise fashion upon recovery (Lovell, Collins, & Bradley, 2004; McCrory et al., 2009; Meehan & Bachur, 2009).

Table 6

A Selection of Various Authors Diagnostic Criteria in MTBI Classification

Source	GCS SCORE	LOC (minutes)	PTA (hours)	Other factors
1963 Congress of Neurological Surgeons (Maroon, Field, Lovell, Collins & Post, 2002)	- - -	None < 5 > 5		Transient neurologic disturbance: Level I: RTP when complete neurological recovery Level II: RTP after 1 week if neurological recovery Level III: Manage as severe TBI including diagnostic testing
Cantu Grading System (Cantu, 1986)	- - -	None < 5 > 5	< 0.5 > 0.5 < 24 > 24	Grade I: RTP 1 week after symptom resolution unless imaging abnormalities Grade II: RTP 2 weeks after symptom resolution unless imaging abnormalities Grade III: No sport for a month, thereafter RTP is asymptomatic for a week.
Levin, Lippold, Goldman, Handel, High, Eisenberg, et al., 1987	-	-	-	Uncomplicated MTBI: No CT or MRI abnormalities Complicated MTBI: CT or MRI abnormalities
Williams, Levin, & Eisenberg, 1990	13-15 13-15			Uncomplicated MTBI: No CT intracranial abnormalities Complicated MTBI: CT intracranial abnormalities
Colorado Medical Society - revised 1991 (Cantu, 2001)	- - -	- - LOC	- PTA	Grade I: Confusion without amnesia Grade II: Confusion with amnesia Grade III
AAN Practice Parameter (Kelly & Rosenberg, 1997)	- - -	- - LOC even if brief		Grade I: Brief confusion, symptoms or mental status abnormalities last <15 m Grade II: Brief confusion, symptoms or mental status abnormalities last >15 m Grade III
Hsiang, Yeung, Yu, & Poon, 1997	15 15 13-14	- - -	- - -	Low risk: No acute radiological abnormalities High risk: Acute radiological abnormalities High risk: No acute radiological abnormalities
Evidence-Based Cantu Grading System for Concussion (Cantu, 2001)	- - -	None < 1 > 1	< 0.5 > 0.5 < 24 > 24	Grade I (mild): Symptoms < 30m Grade II (moderate): Symptoms > 30 m < 24 h Grade III (severe): Symptoms > 1 wk
Borgaro, Prigatano, Kwasnica and Rexer, 2003	- - -	None/ Brief LOC LOC		Uncomplicated: No neuroimaging space occupying lesions, no symptoms or primarily emotional symptoms as opposed to cognitive symptoms Complicated: Neuroimaging space occupying lesions and primarily cognitive symptoms as opposed to emotional symptoms
Ruff, 2005	- - -	None/Transient < 5 5-30	< 1 minute 1 minute – 12 h 12-24 hours	Type I: One or more neurological symptoms Type II: One or more neurological symptoms Type III: One or more neurological symptoms
CISG (Prague 2004) (McCrory, Johnston et al., 2005)	- -	- None/ > 1	- -	Simple: Recovery between day 7-10 postinjury. No complications Complex: Symptomatic or cognitive deficits more than 10 days postinjury or on exertion and/or LOC > 1 m or convulsions.

3.6 NEUROPSYCHOLOGICAL CONSEQUENCES OF MTBI

MTBI can result in a variety of neuropsychological functional impairments, in the form of neurocognitive deficits, in conjunction with various emotional, behavioural and physical symptom complaints, that may impact significantly on physical, mental and social well being (Bigler, 2003; Cantu, 2001; Gaetz, 2004; Gaetz & Bernstein, 2001; Rutherford et al., 2003; Wilner, 2010). Typical cognitive sequelae of MTBI (e.g., impaired attention and working memory, processing speed, reaction time, cognitive flexibility and executive functioning, learning and memory), as well as typical affective and behavioural sequelae of MTBI (e.g., executive dysfunctions involving disinhibition, irritability, argumentativeness, aggression and impaired judgment) correspond to impairments involving frontotemporal lesions (e.g., Cantu, 2001; Shuttleworth-Edwards & Whitefield, 2007a; Skinner, 2008). For ease of reference, despite their interrelatedness, the neurocognitive consequences and symptoms resulting from MTBI, will be discussed separately below.

3.6.1 Neurocognitive Consequences of MTBI

Lezak et al. (2004) conceptualise the neurocognitive functions inferred from behaviour, including neurocognitive test performance, to include receptive functions (i.e., the ability to acquire, classify and integrate information), learning and memory (i.e., the ability to store and retrieve information), thinking (i.e., the ability to organise/reorganise information), and expressive functions (i.e., the ability to communicate or act upon information). MTBI contributes to impairments in these areas of neurocognitive functioning that can impact on academic or work abilities (Pettersen & Skelton, 2000). However, these functions work in an interrelated way, and therefore cannot be considered as discrete. For example attention, as a separate function, underlies these cognitive functions. Moreover, a specific neurocognitive deficit revealed on psychometric testing, may not be limited to one area of functioning (Lezak et al., 2004). The reasons are that the tasks involved in a specific psychometric test, seldom measure a pure process related to a specific function (Lezak et al., 2004). There is great variability between tests that are purported to measure a specific function, and furthermore few tests are sensitive to the subtle effects of MTBI, unless their task demands are particularly cognitively challenging (e.g., Frencham et al., 2005; Lezak et al., 2004).

In support of the difficulties in delineating cognitive functions, Frencham et al. (2005), in their meta-analysis of 17 MTBI studies, found a significant effect for attention and concentration, but only when combining the domains of attention, working memory and speed of processing, as opposed to small effects in the domains of memory and executive functioning. In this thesis, the most commonly documented MTBI neurocognitive impairments will be discussed as separately as is possible (given these difficulties of separating out pure functions), including those of attention and concentration, information processing, visuomotor processing (psychomotor) speed, reaction time, memory, and executive functioning (e.g., Covassin et al., 2008; Echemendia et al., 2001; Erlanger, Kaushik et al., 2003; Erlanger et al., 1999; Frencham et al., 2005; Hatfield et al., 2004; Hinton-Bayre, Geffen, Geffen, McFarland, & Friis, 1999; Iverson, Lovell, & Collins, 2005; Lubit, 2008; Maddocks & Saling, 1991; Reitan & Wolfson, 2000).

Impairments in Attention and Concentration

Attention, involved in sensory reception and perception, is the brain's reflexive or voluntary alertness to incoming stimuli, and is conceptualised as sequential processes involving different brain systems (Lezak et al., 2004). Attention span refers to the amount of information that can be grasped effortlessly at the same time. Selective attention, commonly referred to as concentration, is the ability to focus on particular stimuli, while competing distractions are suppressed from awareness. Sustained attention is the ability to maintain attention over time. Divided attention is the ability to attend to multiple operations within a complex task, or to attend to more than one task simultaneously. Alternating attention is the ability to shift focus between stimuli or tasks (Lezak et al., 2004).

Attention is one of the most sensitive indicators of acute and chronic neuropsychological impairment following MTBI (Frencham et al., 2005). MTBI is known to result in deficits in attention, particularly selective attention, sustained attention and divided attention (e.g., Bernstein, 2002; Collie, Makdissi, Maruff, Bennell, & McCrory, 2006; Collins, Grindel et al., 1999; Echemendia et al., 2001; Flanagan, 1999; Gentilini et al., 1985; Lezak et al., 2004; Macciocchi, Jeffrey, Alves, Rimel, & Jane, 1996; Mathias et al., 2004; Pettersen & Skelton, 2000; Shuttleworth-Edwards & Radloff, 2008; Stuss et al., 1985). Apart from divided attention, deficits in attention have presented in MTBI patients one month postinjury (e.g., Gentilini et al., 1985; Mathias et al., 2004). Binder, Rohling and Larrabee

(1997) in their meta-analysis of 8 MTBI prospective studies comprising 11 samples tested at least three months after injury, found significant small effects for attention and concentration, although causation was not clearly demonstrated.

Reduced Speed of Information Processing and Visuomotor Processing

Information processing speed, the rate at which cognitive activities can be completed, is reliant on the efficiency of the attentional system (Catroppa & Anderson, 2009). On the other hand reduced information processing speed can affect attentional processes. Therefore, it is uncertain whether MTBI affects information processing *per se*, or is the result of reduced attention (e.g., Catroppa & Anderson, 2009; Lezak et al., 2004; Ponsford, Sloan, & Snow, 1995). Slowed information processing speed is a significant effect following TBI generally (e.g., Mathias & Wheaton, 2007; Tromp & Mulder, 1991). Reduced speed of information processing has frequently been found following MTBI (e.g., Cantu, 1996; Covassin et al., 2008; Echemendia et al., 2001; Flanagan, 1999; Gaetz & Bernstein, 2001; Gronwall 1989; Gronwall & Wrightson, 1974, 1975; Hinton-Bayre, Geffen, & McFarland, 1997; Mathias et al., 2004; Pettersen & Skelton, 2000; Ponsford et al., 2000). Furthermore, reduced information processing performance has differentiated MTBI patients from controls in multiple studies (e.g., Comerford et al., 2002; Gronwall, 1989; MacFlynn Montgomery, Fenton, & Rutherford, 1984; Mathias et al., 2004). Information processing speed is considered the underlying construct in decreased cognitive functions following MTBI and therefore of complex tasks involving the integration of various cognitive systems, and is the most sensitive indicator of deficits (Hinton- Bayre et al., 1997). This is borne out by the findings that the dual information processing tasks of finger tapping and word repetition appear to be more diagnostic of MTBI in females; and that impaired information processing has been associated with PTA, which implicates memory (e.g., De Monte et al., 2005; De Monte, Geffen, & Massavelli, 2006).

Reduced information processing speed often shows as slowed reaction time (Lezak et al., 2004; Zheng, Myerson, & Hale, 2000). Studies of information processing following TBI have demonstrated that reaction time slows as task complexity increases, and that information processing is affected by the novelty rather than motor complexity of an experimental drawing task (Tombaugh, Rees, Stromer, Harrison, & Smith, 2007; Tromp & Mulder, 1991). Among MTBI patients, impaired information processing has been

found within 24 hours of injury, and deficits on slower tactile reaction time and particularly on visual reaction times have been revealed one month post MTBI (Comerford et al., 2002; Mathias et al., 2004). Following MTBI, information processing speed typically returns to normal within one to six months, although slight deficits have been evidenced eight years post MTBI (e.g., Bernstein, 2002; Gronwall 1989; MacFlynn et al., 1984; Mathias et al., 2004). In addition, the severity and duration of this functional impairment appears exacerbated following repeat incidents of MTBI (Cantu, 2001; Gronwall & Wrightson, 1974, 1975; Pettersen & Skelton, 2000). Visuomotor processing speed, specifically, results in deficits or a lack of practice effects following MTBI in comparison to controls (e.g., Covassin et al., 2008; Farace, Ferree, Hollier, Barth, & Shaffrey, 2003; Gardner, Shores, & Batchelor, 2010; Mathias et al., 2004; Parker et al., 2007; Shuttleworth-Edwards, Border et al. 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Jordan, Puchert, & Balarin, 1993). Deficits in visuomotor processing speed have consistently differentiated concussed rugby players from controls (e.g., 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).

Reaction Time Deficits

Slowed reaction time, in the absence of a specific motor disability, represents overall mental slowing and is the most typical and meaningful feature of diffuse brain damage and MTBI (e.g., Sosnoff, Broglio, Hillman & Ferrara, 2007). Therefore, MTBI often results in deficits in reaction time (Collie, Makdissi et al., 2006; Collins, Grindel et al., 1999; Covassin et al., 2008; Gentilini, Nichelli, & Schoenhuber, 1989; Macciocchi et al., 1996; MacFlynn et al., 1984; Maddocks & Sailing, 1996; Pettersen & Skelton, 2000). Bleiberg, Kane, Reeves, Garmoe and Halpern (2000) point out that slowed reaction time with differences of 200 ms and less, have been used to differentiate concussed individuals from nonconcussed individuals (Bohnen, Jolles, Twijnstra, Mellink, & Wijnen, 1995; Hugenholtz, Stuss, Stethem, & Richard, 1988; MacFlynn et al., 1984). Furthermore, following MTBI, reaction time slows as the test complexity increases, and slowed reaction time is of a longer duration among symptomatic athletes as opposed to asymptomatic athletes (Collie, Makdissi et al., 2006; Tombaugh et al., 2007). Athletes who reported visual symptoms, as opposed to those who did not, evidenced slowed

reaction time and visual motor speed, and an increase in symptom scores, that only recovered four to seven days following MTBI (Tinker, 2010).

Simple reaction time can recover as early as five or ten days following MTBI (e.g., Bleiberg et al., 2004; Parker et al., 2007). On the other hand, choice reaction time, in terms of correct as opposed to incorrect responses, has remained slowed one month postinjury (Halterman et al., 2005). Visuospatial attention, involving measures of reaction time, appears highly susceptible to the neural effects of MTBI, as demonstrated in a study that found mild impairment for the orienting component of attention and substantial impairment for the executive component of visual attention following MTBI (Halterman et al., 2005). Among patients, slowed reaction time and including slowed choice reaction time, has been shown three months post MTBI (Gentilini et al., 1989; Hugenholtz et al., 1988). Although Cremona-Meteyard and Geffen (1994) found there was not a slowing in simple reaction time following MTBI, response to cued visual targets was reduced within two weeks of injury, and a year later this deficit in visuospatial attention persisted.

Impairments in Memory

Memory in General

Memory, the capacity to retain and use information for adaptive reasons, is a complex function characterised by various forms of memory impairment (Lezak et al., 2004). A brief explanation of a few select forms of memory follows. Sensory information (e.g., visual and verbal format) is transferred to short-term memory, i.e., the immediate memory span, in which capacity is limited and information is displaced by new incoming information (unless information is rehearsed and transferred to long-term memory). Short-term memory includes working memory in the form of temporary storage and organisation of information required for complex cognitive tasks, e.g., comprehension, learning, and reasoning (Baddeley, 1992). On the other hand, long-term memory is a more permanent store of past memories and learned information and has less limited capacity than short-term memory. Long-term memory includes declarative (explicit) memory and nondeclarative (implicit) memory. Declarative memory includes the long-term memories for facts (semantic memory) and autobiographic events (episodic memory), that can be consciously recalled (Lezak et al., 2004; Ullman, 2004). Nondeclarative memory includes item-specific implicit memory and procedural memory (for recall of procedures required to carry out a task) that is non-conscious. Prospective

memory is the recall ability to carry out future actions (Lezak et al., 2004). Therefore, the types of memory, and possible associated impairments, are numerous.

Memory is one of the cognitive functions most vulnerable to impairment as a result of closed brain injury (Capruso & Levin, 1992; Levin, Goldstein, High, & Eisenberg, 1988; Pettersen & Skelton, 2000). The abilities to register, learn and retrieve information require memory skills, and therefore damage to the cortex, as found in MTBI, can result in impaired learning and memory (Catroppa & Anderson, 2009). Deficits in learning are considered a sound predictor of outcome (Shores et al., 2008). No particular assessment demonstrates memory deficits for all individuals and no single neurocognitive test can measure all types of memory deficits following MTBI (Frencham et al., 2005; Lezak et al., 2004). Moreover, memory can be affected by deficits in attention and information processing speed (Lezak et al., 2004). In addition, for purposes of factor analyses, memory could not be divided successfully into either acquisition/learning and delayed recall, or into visual and verbal memory. Therefore, it is best that memory is represented as a single factor in MTBI studies (e.g., Frencham et al., 2005).

Memory impairment, in the form of retrograde and anterograde amnesia, is an important neuropsychological on-field marker for immediate diagnosis and assessment purposes of sport-related MTBI (Barth et al., 1989; Yarnell & Lynch, 1973). Beyond these two forms of amnesia, several studies on MTBI in contact sport have demonstrated memory impairments as a result of concussion affecting short-term memory, working memory, verbal memory and visual memory (e.g., Belanger & Vanderploeg, 2005; Echemendia et al., 2001; Gaetz et al., 2000; Guskiewicz et al., 2005; Hatfield et al., 2004; Iverson, Lange, & Franzen, 2005; Killam et al., 2005; Matser et al., 1998; Parker et al., 2007; Pettersen & Skelton, 2000; Shuttleworth-Edwards, Border et al., 2004).

Importantly, a review of meta-analyses on outcome of MTBI almost invariably point to memory as being affected. Frencham et al. (2005) in a meta-analysis of 17 studies, found the neurocognitive effects of MTBI to be small, with memory in particular being affected in the early stages following injury, and that most neurocognitive impairments resolve after three months. Similarly, Belanger et al. (2005) in a meta-analysis of 39 prospective MTBI studies, found delayed memory and fluency most affected, but overall neurocognitive recovery occurred within three months of injury. Schretlen and Shapiro

(2003), in a meta-analysis of 39 cross-sectional MTBI studies, found that cognitive functions generally recover within the first few weeks and return to pretrauma functional levels within one to three months following MTBI. In sport-related MTBI specifically, Belanger and Vanderploeg (2005) found in the prospective samples of their meta-analysis of 21 sport-related studies, that memory acquisition, delayed memory, and global cognitive functioning, were the functions most affected by MTBI. They also found that overall neurocognitive recovery occurred within seven days of injury, apart from delayed memory that remained problematic 7 days postinjury.

Repeat incidents of MTBI may have a cumulative effect and even a single MTBI incident may have a chronic effect on neurocognitive impairment, including memory (e.g., Flanagan, 1999; Pettersen & Skelton, 2000; Manko, 2003; Martineau et al., 2007). A study, that induced a condition of hypoxic stress, found deficits on both vigilance and memory tasks (both involving digits) in asymptomatic individuals, who had suffered a single MTBI event one to three years previously, compared with controls. This suggests that under stress conditions, chronic memory and vigilance deficits manifest as a result of MTBI, resembling the level of decrement found immediately following MTBI (Ewing, McCarthy, Gronwall, & Wrightson, 1980).

Verbal Memory

Verbal memory is a complicated function that is not limited to but includes attention, consolidation and retrieval (Hatfield et al., 2004). Those suffering TBI, of varying severity, have been found to exhibit impaired performance on long-term memory for verbal information or declarative memory measures (Levin, Benton, & Grossman, 1982; Lezak, 1979; Pettersen & Skelton, 2000). Those suffering MTBI have significantly more difficulty than controls with various measures of verbal learning, verbal fluency and verbal memory, with a tendency to retrieve fewer words, evidencing less accuracy and making more errors with retrieval (e.g., Guilmette & Rasile, 1995; Kurca, Sivak, & Kucera, 2006; Mathias et al., 2004; Raskin & Rearick, 1996). Among MTBI patients, impaired recall of word learning has been revealed within 24 hours of MTBI (Comerford et al., 2002). 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), but made significant gains at one month, and additional gains three months postinjury (Ruff et al., 1989).

Visual Memory

In respect of visual memory, the most common perceptual deficits resulting from TBI involve those of visual perception that include measures of visual attention, scanning, colour perception, recognition, organisation and interference; and visual memory deficits have been found following MTBI (e.g., Lezak et al., 2004). Impairments in visual memory, usually employing tests that also measure visuospatial functioning, are common following MTBI. There is no consensual definition of visuospatial functioning, therefore visuospatial impairment is defined on the basis of tests used, rather than in conceptual terms. However, most tests designed to assess visuospatial functioning measure visual integrity in terms of analysis and synthesis, without looking at the constructs involved in visuospatial processing (Jagaroo, 1999, 2009). Visuoperceptual functioning, notably organisation, can be assessed via copying tasks of complex figures, and visual memory can be assessed via recall and reproduction of the figures. Deficits in visual memory, visuoprocessing, visuospatial and visuoperceptual functioning have been demonstrated following MTBI, particularly the long-term consequences thereof (e.g., McLatchie et al., 1987; Ross, Casson, Siegel, & Cole, 1987; Jordan, Matser, Zimmerman, & Zazula, 1996; Matser et al., 1998; Matser, Kessels, Lezak, Jordan, & Troost, 1999; Matser, Kessels, Lezak, & Troost, 2001).

Executive Dysfunction

Executive functioning, often referred to as cognitive control or executive control, covers a wide range of cognitive and behavioural functions, for example goal directed behaviour, dealing with novel situations, inhibition, initiation and monitoring of actions, cognitive flexibility, monitoring feedback, multitasking, planning, problem-solving, resisting interference, sequencing, sustaining attention, verbal reasoning and working memory (e.g., Chan, Shum, Toulopoulou, & Chen, 2008; Pontifex et al., 2009). Chan et al. (2008) refer to these functions as the 'cold' component of executive functioning, in that they are relatively logical and mechanical cognitive operations, not requiring emotional arousal. The 'hot' component of executive functioning involves emotional arousal and includes beliefs, desires, experiencing rewards/punishment, regulation of one's social behaviour, interpreting complex emotions in social situations, and making decisions involving

personal or emotional interpretation. Impairments to either the 'cold' or 'hot' components of executive functioning impact dramatically on the individual's optimal performance in everyday life and in the development and maintenance of social relationships. Chan et al. (2008) propose that the dorsolateral prefrontal cortex mediates the 'cold' component, and the ventromedial or orbitofrontal cortex mediates the 'hot' component. Therefore the inability to plan, coordinate, and sequence behaviours is a common consequence of TBI, resulting primarily from impairment to the frontal cortex (Knight & Titov, 2009). However, Stuss and Alexander (2000) point out that executive functioning is not a unitary function, but involves distinct processes implicating the frontal lobes, but no specific region can be associated with a specific process. Alvarez and Emory (2006) also argue that the executive system involves multiple cortical and subcortical structures of which the frontal lobes are but one aspect. Impairment in executive functioning is usually observed as changes in an individual's everyday behaviour, however some neuropsychological tests are considered sensitive to impairments in executive functioning and in differentiating individuals with and without MTBI (e.g., Brooks, Fos, Greve, & Hammond, 1999). Unfortunately, some of these neuropsychological tests lack ecological validity, in that an individual with frontal lobe damage may experience difficulties with everyday life, and yet perform equally as well as unimpaired controls on the tests (Chan et al., 2008; Shallice & Burgess, 1991). Belanger et al. (2005) and Belanger and Vanderploeg (2005) in their meta-analyses of acute neuropsychological outcomes following MTBI, found the smallest overall effect sizes for executive functions, in comparison with other functions, such as memory, fluency, global cognitive ability and language.

Summary of Neurocognitive Consequences of MTBI

Overall, these neurocognitive functions that are sensitive to the effects of MTBI, reveal reduced attention skills, processing speed, reaction time and memory skills, that show within days following MTBI, with adverse effects found to persist up until three months in some studies. Executive dysfunction appears to have a small overall effect size in comparison to other affected neurocognitive functions. Memory appears to be the neurocognitive function commonly affected by MTBI, but usually recovering within three months postinjury (e.g., Belanger et al., 2005; Belanger & Vanderploeg et al., 2005).

3.6.2 Symptoms of MTBI

Symptoms, the subjective experiences or sensations that the individual feels or complains of, are experienced by between 29 to 90% of individuals soon after an MTBI event, of which 80 to 100% of individuals will attribute one or more symptoms to the injury (e.g., Arciniegas et al., 2005; Legome & Alt, 2009). However, MTBI symptoms are not always apparent soon after injury and may evolve over time (Echemendia et al., 2001; Lovell, Collins et al., 2004). MTBI does not have any pathognomic signs and symptoms, in that its symptoms are not unique to MTBI, and can occur in healthy persons or in other conditions (Department of Veteran Affairs, 2009a). For example, studies have found no significant differences between MTBI patients and non-MTBI patients in the development, prevalence, persistence and types of symptoms (e.g., Meares et al., 2008; Necajauskaite, Endziniene, & Jureniene, 2005). Accordingly, and with valid reason, the variety of symptoms occurring in association with MTBI do not necessarily form part of all MTBI definitions. Therefore, the symptoms following MTBI should only be attributed to MTBI if they do not form part of a pre-existing condition, unless the intensity of such is exacerbated following the injury (APA, 1994; Department of Veteran Affairs, 2009a). However, it is the onset of symptoms following MTBI, the intensity of symptoms experienced, and the number of co-occurring symptoms compared to the person's normal functioning, that are of importance (Department of Veteran Affairs, 2009a).

Common self-reported symptoms following MTBI are headache, poor memory, reduced attention, slowed thinking, drowsiness, fatigue, irritability and difficulty functioning at work (e.g., Anderson et al., 2006; Arciniegas et al., 2005; Binder, 1986; Dikmen, Machamer, & Temkin, 2001; Gronwall & Wrightson, 1974; Levin et al., 1987). Headache is the most common symptom, and fatigue, drowsiness, feeling slowed down, and cognitive difficulties are amongst the most commonly reported symptoms following sport-related MTBI (e.g., Fazio, Lovell, Pardini, & Collins, 2007). Additionally, apathy and affective disturbances or lability are typical MTBI symptoms (Arciniegas et al., 2005). Symptoms suggestive of cranial nerve damage include nausea, vertigo/dizziness, blurred vision, diplopia, sensitivity to light and noise, hearing loss, tinnitus, and diminished sense of taste or smell (Legome & Alt, 2009). Findings pertinent to the more commonly reported symptoms will be elucidated on below.

Headache

The single most common MTBI symptom is headache, occurring in 70 to 92% of individuals sustaining MTBI (e.g., Collins, Iverson et al., 2003; Department of Veteran Affairs, 2009a; Evans, 2004; Guskiewicz, McCrea, Marshall et al., 2003; Guskiewicz, Ross, & Marshall, 2001; Legome & Alt, 2009; Lovell, Collins, & Bradley, 2004; Lovell et al., 2006, 2007; McCrory, Ariens, & Berkovic, 2000; Packard, 2008). Headaches may not present immediately following injury, but may occur minutes or hours later, normally developing within 7 days of injury and usually dissipating within 2 to 4 weeks of injury (Bernhardt, 2009; Department of Veteran Affairs, 2009a; Lovell, Collins, & Bradley, 2004). Headaches, notably migraines, may result in protracted recovery from MTBI (e.g., Collins, Field et al., 2003; de Kruijk, Leffers, Menheere et al., 2002; Mihalik et al., 2005). The presence of a headache within 24 hours of MTBI is associated with a poorer outcome, and headaches of more than seven days duration have been associated with incomplete neurocognitive recovery (e.g., de Kruijk, Leffers, Menheere et al., 2002; Martineau et al., 2007). Furthermore, concussed athletes with a headache, compared with concussed athletes without a headache, have been found to experience more on field changes in mental status, report more symptoms, and perform worse on both neurocognitive testing (particularly memory and reaction time) and balance testing (e.g., Collins, Field et al., 2003; Register-Mihalik, Mihalik, & Guskiewicz, 2008). Headaches and concentration difficulties both lasting more than three hours, in combination with LOC and retrograde amnesia, may be indicative of a more serious brain injury (Asplund, McKeag, & Olsen, 2004; Martineau et al., 2007).

Headache is also the most persistent MTBI symptom (e.g., Rimel et al., 1981). Patients reporting headache, dizziness or nausea initially following injury, have been found to experience more severe symptom complaints six months post injury, and more than 20% of individuals reported headache as the most common symptom within 12 months of injury (e.g., Arciniegas et al., 2005; de Kruijk, Leffers, Menheere et al., 2002; Delaney, Abuzeyad, Correa, & Foxford, 2005; Flanagan, 1999; Martelli, Grayson, & Zasler, 1999; Ryan & Warden, 2003).

Headache is a postconcussive symptom involving controversy, in that some studies classify an injury as a concussion on the basis of headache presence, with the view that a headache is a transient alteration in mental status (Terrell, 2004). However, headaches are

known to occur in uninjured football players following football matches or practice, and therefore it is uncertain whether a headache fully represents a concussive or subconcussive event (Terrell, 2004). Pertinent to this are findings by McCrory, Heywood and Coffey (2005), in which 80% of elite Australian footballers reported headaches in the absence of MTBI (49% during competitive matches and 60% during training), which did not differ significantly from their community sample. However, 34% of headaches were classified as migraines (significantly higher than their community sample), which, although uncertain, may lend support to the presence of cumulative effects from subconcussive trauma occurring amongst contact sport athletes. However, it also supports the notion that the prevalence of headaches in the absence of MTBI makes it difficult to rely on headache as a diagnostic marker of MTBI.

Dizziness and Impaired Coordination and Balance

Dizziness is an extremely common somatic complaint post MTBI and may confound neuropsychological assessment (e.g., Arciniegas et al., 2005; Chamelian & Feinstein, 2004; Department of Veteran Affairs, 2009b; Flanagan, 1999). Almost 30% of individuals suffer dizziness, altered coordination and impaired balance, following MTBI (Department of Veteran Affairs, 2009b). Studies have found that up to half the patients experiencing postconcussive dizziness, evidence abnormalities on brainstem evoked potentials (e.g., Wilberger, 1993). Consequently, MTBI patients may exhibit a significant sway in Romberg testing or find the finger-nose-finger test difficult, and these signs might alert the clinician to a persistent injury (Bernhardt, 2009). Recently, coordination, balance and postural control are being used medically as a valid and reliable means of assessing neurological motor functioning and for monitoring recovery following MTBI (e.g., Guskiewicz, 2000; McCrory et al., 2009). Postural instability may represent a sensory integration difficulty in which there is failure to use the visual system effectively, and has been found to last for a minimum of three days following MTBI (e.g., Barker & Patel, 2000; Guskiewicz, Riemann, Perrin, & Nashner, 1997; Guskiewicz et al., 2000; McCrea et al., 2003). Deficits in postural stability have been evidenced well in excess of three days following MTBI, and in the absence of deficits on neurocognitive testing. For example, postural instability was significantly evident day 1 among eleven concussed athletes not evidencing deficits on neurocognitive testing and only improved to the level of the control participants day 10 postinjury (Guskiewicz et al., 1997). Other studies have documented long-term deficits in dynamic motor functioning, in the forms of impaired

balance control when walking and in gait tasks, despite neurocognitive recovery and the resolution of reported symptoms (Catena, van Donkelaar, & Chou, 2009; Chou, Kaufman, Walker-Rabatin, Brey, & Basford, 2004; Parker et al., 2006, 2007).

Fatigue

Fatigue is considered to be the third most common symptom following MTBI (Department of Veteran Affairs, 2009b). However, in one MTBI study, fatigue was reported as the most common symptom amongst the MTBI group, and the related symptom - doing things slowly - was rated as the fifth most common symptom and showed the highest difference in endorsement between the MTBI and control groups (Paniak et al., 2002). Fatigue may be the primary effect of CNS dysfunction, or a secondary effect to a sleep disturbance or depression following MTBI, and may confound neuropsychological assessment (Arciniegas et al., 2005; Department of Veteran Affairs, 2009b).

Feeling Mentally Foggy

Feeling mentally foggy or a step removed from one's environment, is a common subjective complaint post MTBI, with findings revealing that 17% athletes in one study who experienced this symptom 5 to 10 days post MTBI compared with those who did not, evidenced significantly more symptoms, reduced memory performance and slowed reaction time and processing speed on the ImPACT composites. This suggests that fogginess may indicate increased risk for poorer outcomes in MTBI (Iverson et al., 2004b).

Sleep Disturbances

Sleep disturbances, usually in the form of difficulty falling or staying asleep, sleeping less or more than normal, frequently occur within seven days following MTBI. They may confound neuropsychological assessment performance (Arciniegas et al., 2005; Department of Veteran Affairs, 2009b; Rao & Rollings, 2002).

Visual and Olfactory Impairments

Visual impairments, in the forms of blurred vision, seeing bright lights, diplopia or sensitivity to light, occur in up to 50% of persons within seven days following MTBI, and usually resolve within a month (e.g., Cantu, 2001; Department of Veteran Affairs,

2009b). However, in one sport-related MTBI study, blurred vision was reported by 70% of the athletes (McCrory, Ariens et al., 2000). The Department of Veteran Affairs (2009b) report that anosmia (olfactory impairment) is relatively uncommon, occurring in up to 25% of persons, following MTBI. However, no other literature suggests that anosmia occurs this frequently in sport-related MTBI. Anosmia is considered a marker of orbital frontal pathology and its related psychosocial deficits (Varney & Meneffee, 1993).

Learning Difficulties

Problems with new learning and academic difficulties are often encountered for up to three months post MTBI - in that attention, concentration, processing speed and memory are negatively affected (e.g., Frencham et al., 2005; Kontos et al., 2004; Lezak et al., 2004; Lovell et al., 1999). Despite reports of being asymptomatic, high school athletes experiencing two or more concussions, have evidenced lower academic grade averages than their peers. This possibly is due to the concussive incidents or is characteristic of those vulnerable to concussion, or a combination of these factors, the consequences of which may be detrimental to passing exams or achieving a scholarship (Shuttleworth-Edwards & Whitefield, 2007a).

Affective Disturbances

Depression is often a functionally impairing chronic symptom following TBI, occurring in 10 to 77% of cases, mostly in the first year postinjury, and with incidence of up to 17% of cases 3 to 5 years postinjury. In addition, an increased risk remains for developing depression in the decades following injury (e.g., Arciniegas et al., 2005; Hibbard, Uysal, Kepler, Bogdany, & Silver, 1998; Silver et al., 2009). Affective disturbances in individuals suffering both complicated and uncomplicated MTBI, compared with controls, have been evidenced (Borago, Prigatano, Kwasnica, & Rexer, 2003). Furthermore, depression and anxiety occur in more than a third of individuals with persistent symptoms post MTBI (Ropper & Gorson, 2007). Factors that may contribute to developing depression following MTBI, include a history of anxiety and mood disorders, psychosocial problems, substance abuse, or injury factors that may include damage to either hemisphere or interruption to the serotonergic functions (e.g., Nicholl & LaFrance, 2009; Silver et al., 2009). Other factors that may contribute to developing depression post MTBI, include a number of postconcussive symptoms perceived as severe, including headache, blurred vision and dizziness, or psychosocial problems, such as a lack of a

social support system (Silver et al., 2009). Depression following MTBI increases cognitive dysfunction, aggression, anger and suicide risk. Therefore its expedient treatment alleviates other postconcussive symptoms (e.g., fatigue, irritability and sleep difficulties) and improves quality of psychosocial functioning (e.g., Silver et al., 2009).

Iverson (2006) conducted a small study on non-MTBI university students suffering with depression, finding significantly slowed performance on ImPACT reaction time and visual motor speed, and a trend towards poorer performance on the verbal memory composite compared with healthy controls. It seems there may be an overlap between the symptoms of depression and MTBI, and in MTBI cases reporting the symptom depression, it is uncertain whether MTBI or depression primarily predispose the individual to impaired neurocognitive functioning. Aggression and mania are relatively rare consequences following MTBI (Arciniegas et al., 2005; Hibbard et al., 1998).

The symptoms described above are the most common MTBI symptoms. However, other symptoms also occur with MTBI and are included in various studies, symptom checklists or rating scales, and are listed below. Typically, the symptoms fall into three broad domains, that overlap between various articles and symptom scales (e.g., Department of Veteran Affairs, 2009a; McCrory, Johnston et al., 2005; Lewandowski, Rieger, Smyth, Perry, & Gathje, 2009; Piland, Motl, Guskiewicz, McCrea, & Ferrara, 2006; Roe, Sveen, Alvsaker, & Bautz-Holter, 2009). MTBI symptoms pertaining to the three domains include:

(i) *Physical or somatic symptoms*: balance problems, dizziness and coordination difficulties, drowsiness, fatigue, headache, hearing complaints (e.g., hyperacusis or tinnitus), nausea, numbness or tingling, sleep disturbances (e.g., difficulty falling asleep, sleeping more or sleeping less), visual problems (e.g., blurred vision and/or photosensitivity) and vomiting

(ii) *Cognitive symptoms*: attention and concentration difficulties, confusion, feeling mentally foggy, feeling slowed down, impaired executive functioning, impaired judgment, learning difficulties, memory difficulties, and slowed processing speed

(iii) *Behavioural, neurobehavioural and emotional symptoms*: agitation, aggression, anxiety, depression, impulsivity, irritability, feeling more emotional, sadness and nervousness.

There is disparity in the literature as to when acute MTBI symptoms resolve, but it has been reported that, in most cases, symptoms usually dissipate within fourteen to thirty five days following MTBI (e.g., Gronwall & Wrightson, 1974, Parker et al., 2007; McCrea et al., 2003). However, in some cases symptoms may resolve immediately or may resolve and then re-occur (e.g., Cantu, 1992; Guskiewicz et al., 1997; Kontos et al., 2004). On one hand, the varied and sometimes subtle symptoms associated with MTBI may not always be easy to detect or are only accentuated with exercise, yet on the other hand symptoms can take months to resolve with long-term mental, physical, occupational or social consequences (e.g., Bay & McLean, 2007; Borg, Holm, Cassidy et al., 2004; McCauley et al., 2001; McManus, 2006). Additionally, the development and duration of symptoms may also be impacted upon by other variables, for example symptoms that occur within 14 days of MTBI have been associated with pain, a prior affective or anxiety disorder, higher IQ, and are 3.33 times more likely in females than males (e.g., Kashluba et al., 2004; Meares et al., 2008). Paniak et al. (2002) found MTBI patients endorsed significantly more symptoms than controls one month postinjury. However, this was not the case in their follow-up study three months postinjury, and only three symptoms - doing things slowly, fatiguing quickly and poor balance - were endorsed as significantly higher by the MTBI group.

In cases of repeat MTBI, the signs and symptoms in are inconsistent. A history of MTBI has been associated with poorer outcomes in terms of persistent symptoms (e.g., Binder et al., 1997; Ponsford et al., 2000). However, in a study of 650 NFL athletes, 160 athletes had repeat MTBI, of which 51 had sustained three or more MTBI incidents, and the signs and symptoms were similar for both single and repeat MTBI athletes. However, somatic symptom complaints tended to be higher for athletes who sustained a repeat MTBI, compared with their initial MTBI (27.5% versus 18.8%), although athletes who sustained three or more MTBI incidents had symptoms similar to players with a single MTBI (Pellman, Viano, Casson, Tucker et al., 2004). Erlanger, Kaushik et al. (2003) and

Iverson (2007) found a history of MTBI was unrelated to either the number or duration of symptoms.

Persistent and prolonged symptoms are considered to form a post concussion syndrome, around which there is considerable debate. While this framwork is not used for this study, the issues do have pertinence to sport-related MTBI symptom profiles.

Post Concussion Syndrome (PCS)

Terms used to describe the condition of persistent or prolonged postconcussive symptoms that arise from a brain injury, including MTBI, are used inconsistently in the literature and include: Post Concussion Syndrome (PCS), Postconcussional Syndrome, Postconcussion Disorder and Persistent Postconcussive Syndrome (e.g., Arciniegas et al., 2005; McCrea et al., 2003; Landre, Poppe, Davis, Schmaus, & Hobbs, 2006; Smith, 2006). Persistent Postconcussive Syndrome (PPS) usually denotes the presence of symptoms persisting beyond six months, although some authors use this term to denote symptoms persisting beyond three months (Legome & Alt, 2009). In respect of PCS, there is no consensus on when prolonged MTBI symptoms can be considered to constitute this syndrome, in that the presence of symptoms may be required to persist for at least four weeks or in excess of three months (e.g., APA, 1994, 2000; Kashluba, Casey, & Paniak, 2006; WHO, 1993).

The World Health Organization's (WHO) International Classification of Diseases Tenth Revision's [ICD-10] (WHO, 1993), criteria for the Postconcussional Syndrome, is a history of brain trauma and loss of consciousness, with at least three symptoms present four weeks postinjury. The Diagnostic and Statistical Manual of Mental Disorders (DSM), versions DSM-IV and DSM-IV-TR (APA, 1994, 2000] include Postconcussional Disorder in the criteria sets and axes for further study. The DSM-IV and DSM-IV-TR (APA, 1994, 2000) criteria for a Postconcussional Disorder is a history of closed head trauma in which three or more symptoms occur shortly after injury and remain present in excess of three months postinjury, and cognitive assessment (APA, 1994) or documented evidence (APA, 2000) need to prove attention or memory deficits in excess of three months. Additionally, the DSM-IV (APA, 1994) states that a significant cerebral concussion needs to be manifested by two of the three following specific criteria: LOC in excess of five minutes, PTA in excess of 12 hours, or seizure onset or notable worsening

of a pre-existing seizure condition within six months of injury. The ICD-10, DSM-IV and DSM-IV-TR criteria are limited in defining PCS for the reason that concussion does not usually result in LOC, PTA in excess of 12 hours, or seizures (e.g., Ruff & Grant, 1999). Furthermore, research has established little concordance between the ICD-10 and DSM-IV criteria, in that among a group of moderate and mild TBI patients, 64% measured on the ICD-10 and 11% measured on the DSM-IV, met a PCS diagnosis three months postinjury (Boake et al., 2005; McCauley et al., 2008).

Differing criteria used to diagnose PCS culminates in the incidence of PCS being uncertain and variable (e.g., Kashluba et al., 2006; Ingebrigsten, Waterloo, Marup-Jensen, Attner, & Romner, 1998). PCS incidence reports range from 50% individuals experiencing PCS one month post MTBI, through 15 to 40% individuals experiencing PCS symptoms three months post MTBI, and 5% individuals experiencing persistent or disabling symptoms for a year or more post MTBI (e.g., APA, 1994; Ingebrigsten et al., 1998; Legome & Alt, 2009; Wrightson & Gronwall, 1998). In contrast, other authors report that the incidence of PCS ranges from 3 to 5% (Department of Veteran Affairs, 2009; Iverson, 2007; McCrea, 2007). The prevalence of PCS is reportedly higher in females than males (King, 2003). Neither the severity of brain injury nor the number of prior concussions is predictive of who will develop PCS, and although commonly occurring as a result of a MVA, it is seen in athletes, particularly those who have suffered multiple concussions in one sports season (e.g., Arciniegas et al., 2005; Bailes & Cantu, 2001; Bernhardt, 2009; Gerberich, Priest, Boen, Staub, & Maxwell, 1983; Macciocchi et al., 1996).

The most common PCS symptoms are recurrent or persistent headaches, memory impairment, dizziness, concentration problems, ataxia, photosensitivity, sensitivity to noise, loss of libido, attention problems, vertigo, anxiety, depression, irritability and fatigue (e.g., Bailes & Cantu, 2001; Bernhardt, 2009; Cullum & Thompson, 1997; Hickey, 2004). However, the number, extent and duration of PCS symptoms are said to be modified by a number of various preinjury, injury and postinjury factors, or a combination thereof (e.g., Bailes & Cantu, 2001; Gouvier, Cubic, Jones, Brantley, & Cutlip, 1992; Ingebrigsten et al., 1998; Legome & Alt, 2009; Silver et al., 2009).

The existence of PCS as a condition has been controversial for more than 140 years, particularly when litigation is involved (e.g., Evans, 2004; Santalucia & Feldmann, 2000). The greatest controversy is that symptoms commonly associated with PCS are neither specific nor unique to concussion or the PCS condition, in that they occur among the general population or in conditions such as depression, chronic fatigue or somatisation (e.g., Chan, 2001; Fryatt, Rose, & Slessor, 2001; Grouvier, Uddo-Crane, & Brown, 1998; Iverson & McCracken, 1997; Lees-Haley & Brown, 1993; McAllister & Arciniegas, 2002; McCrory & Johnston, 2002; Neppe & Godwin, 1999; Smith, 2006; Willer& Leddy, 2006). Therefore, it is argued that PCS cannot be defined as a syndrome, in that there is neither consistent symptom presentation linkage, nor coupling of symptoms during the resolution period as found in traditional syndromes (e.g., Arciniegas et al., 2005; Silver et al., 2009; Smith, 2006). In order to overcome criticism that persistent symptoms found in the PCS condition reflect neither a syndrome nor disorder, the Department of Veteran Affairs (2009a, 2009b) introduced the term Persistent Post-Concussive Symptoms to describe MTBI symptoms that persist beyond one month, despite initial treatment.

Various theoretical explanations for PCS have been put forward and debate exists as to whether PCS symptoms are organic or psychologically based (Legome & Alt, 2009). It has been posited that initial MTBI symptoms are organic, and those persisting beyond one to three months are psychological or non-organic. However, this is debatable, in that imaging studies have revealed organic brain injury a year following MTBI among those with PCS and those with minimal neuropsychological impairment (e.g., Legome & Alt, 2009; Robinson, 2010; Uzzell, 1999; Yang, Tu, Hua, & Huang, 2007). The Physiogenesis theory suggests that MTBI causes neuropathological changes in brain functioning and that post concussion symptoms are associated with neurocognitive impairment or neuroimaging abnormalities. The Psychogenesis theory holds that post concussion symptoms reflect premorbid adjustment difficulties and/or postinjury factors (Barclay, 2009). Another explanation for PCS is the cognitive-behavioural model based on the study by Ferguson, Wiley, Mittenberg, Barone and Schneider (1999). In this model, it is proposed that athletes with a history of MTBI underestimate preinjury symptoms, and athletes (both with and without a history of MTBI) overestimate the symptoms expected post MTBI compared with preinjury symptoms. Therefore, athletes with MTBI will overestimate postconcussive symptom changes in line with their symptom expectations.

The recognition of Post Concussion Syndrome as an entity remains unresolved, and therefore will not be utilised as a conceptual basis for discussing symptoms in the present study. Rather, symptoms will be discussed in terms of the relatively unstructured model typically adopted in research, that employs inventories designed to tap into and measure symptom sequelae, and will be described in detail in Chapter 5.

3.6.3 Resolution of Neuropsychological Consequences of MTBI

Neuropsychological resolution following MTBI, in terms of neuropsychological sequelae (including both neurocognitive and symptom reports) and recovery duration, varies widely interindividually. Furthermore, symptom reports do not always correlate with neurocognitive impairment intraindividually (e.g., McCrory, Johnston et al., 2005; Skinner, 2008). The acute and chronic stages of such MTBI resolution will be discussed, and evidence suggesting that cumulative brain injury from repeat MTBI events may lead to chronic traumatic brain injury, will be presented.

Acute and Chronic Stages of MTBI Resolution

In terms of neurocognitive recovery, the pattern appears the same for both general MTBI and sport-related MTBI (Bleiberg et al., 2004; Bieliauskas, Begloff, Steinberg, & Kauszler, 2004; Hinton-Bayre & Geffen, 2004; Lovell, Collins, Iverson et al., 2003; Shuttleworth-Edwards, Border et al., 2004; Stephens, Rutherford, Potter, & Fernie, 2005). However, in terms of reported symptom resolution, the pattern may differ between general MTBI and sport-related MTBI, depending on motivational factors. General MTBI patients may have no reason or benefit in under-reporting symptoms, while athletes may under-report symptoms in order to expedite return to play (e.g., Aubry et al., 2002; Sturmi, Smith, & Lombardo, 1998; Tucker, 1997).

Literature reporting on the average time in which MTBI neuropsychological resolution generally occurs in most MTBI cases is variable, and relatively few evidence-based studies have been undertaken on the length of time for both cognitive and symptom recovery among athletes (e.g., McCrea et al., 2003, 2010; McCrory, 2002a). Some authors report that the neuropsychological effects of MTBI resolve in two to seven days (e.g., Bernhardt, 2009; Ellemberg, Henry, Macciocchi, Guskiewicz, & Broglio, 2009; Iverson, 2007; McCrea, Barr et al., 2005; McCrea et al., 2003; Pellman, Lovell, Viano,

Casson, & Tucker, 2004; Pellman, Lovell, Viano, & Casson, 2006; Pellman, Viano, Casson, Arfken, & Powell, 2004). Other authors report that the neuropsychological effects of MTBI resolve within ten days (e.g., 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; Hugenholtz & Richard, 1982; Iverson, Gaetz, Lovell, & Collins, 2004a; Macciocchi et al., 1996; McCrory et al., 2009; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008; Manko, 2003). Overall, it is assumed that 80 to 90% of concussions resolve within seven to ten days, although children and adolescents may take longer to recover (e.g., McCrory et al., 2009; Halstead & Walter, 2010).

There are, however, instances in which recovery from MTBI takes longer than ten days (e.g., Gentilini et al., 1985; Kontos et al., 2004; Levin, Mattis et et al., 1987; Silver et al., 2009). However, it appears that most individuals recover within three months, and few cases experience chronic neuropsychological effects persisting beyond three months (e.g., Arciniegas et al., 2005; Barth et al., 1983; Belanger & Vanderploeg, 2005; Bernstein, 2002; Collins, Grindel et al., 1999; Echemendia et al., 2001; Gentilini et al., 1985; Hugenholtz et al., 1988; King 1996; Leininger et al., 1990; Levin, Mattis et al., 1987; Ponsford et al., 2000; Segalowitz, Bernstein, & Lawson, 2001; Shuttleworth-Edwards, 2004; Shuttleworth-Edwards & Whitefield, 2007a; van der Naalt, 2001; Vanderploeg, Curtiss, & Belanger, 2005).

MTBI effects that persist longer than three months are considered chronic and relatively permanent (Barth et al., 1989; Bernstein, 2002; Frencham et al., 2005; Reitan & Wolfson, 1999; Schretlen & Shapiro, 2003; Segalowitz et al., 2001; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007a; Van der Naalt, 2001; Vanderploeg et al., 2005). Therefore, from the literature, it appears that symptoms and neurocognitive impairments are considered ‘acute’ when occurring within three months, and ‘chronic’ when persisting in excess of three months, following MTBI (Barth et al., 1989; Bernstein, 2002; Frencham et al., 2005; Schretlen & Shapiro, 2003; Segalowitz et al., 2001; Shuttleworth-Edwards & Radloff, 2008; Van der Naalt, 2001; Vanderploeg et al., 2005).

Therefore for purposes of this thesis, the terms acute and chronic will pertain to the time frames of within three months and following three months, respectively.

It has been proposed that between 7 and 40% of individuals may suffer persistent MTBI symptoms three to six months following injury, with 1 to 20% experiencing symptoms beyond six months (Arciniegas et al., 2005; Belanger et al., 2005; Ingebrigsten et al., 1998; Leininger et al., 1990; Shuttleworth-Edwards, Smith et al., 2008; Silver et al., 2009). In contrast, de Kruijk, Leffers, Menheere et al. (2002) found from their evidence-based study, that 28% of patients had not fully recovered six months post MTBI, as evidenced by at least one symptom complaint, and of those who had reported three initial symptoms, notably headache, nausea or dizziness, 50% had not fully recovered after six months. Therefore, the actual incidence of recovery duration is not known with certainty, however, a small minority of individuals may experience effects from six months up until three years post MTBI **(**e.g., Barth et al., 1983; Department of Veteran Affairs, 2009a; Kontos et al., 2004; Hugenholtz et al., 1988; Leininger et al., 1990).

Apart from paediatric studies of psychosocial changes post MTBI (e.g., Hayman-Abello, Rourke, & Fuerst, 2003), only one adult study was found, that investigated persistent neurobehavioural changes from preinjury baseline until six and twelve months post MTBI, in comparison with controls (e.g., Hartlage, Durant-Wilson, & Patch, 2001). In the study, behavioural changes, perceived both by the individuals and by spouses/close family members, were measured on a 68-item scale, found sensitive to behaviour changes as a result of brain injury. No individuals had a premorbid history of psychiatric disorders. All MTBI individuals were involved in litigation at some point post MTBI. However, the independent rating by family members was considered as mitigating any effects of pending litigation, and provided evidence of any changes that the individuals lacked insight in perceiving themselves. In general, these independent ratings did not differ from the ratings of individuals. The MTBI individuals were divided into two groups, one rated at approximately six months and the other at approximately twelve months post MTBI, in comparison with the same control group. Chi-square analyses revealed that at six months post MTBI, 39 of the 68 behaviours were significantly endorsed as having changed since baseline by more of the post MTBI individuals than the controls, and at twelve months post MTBI, 36 of these 39 behaviours were significantly endorsed as having changed since baseline by more of the post MTBI individuals than the

controls. At twelve months, there was no change in the symptoms less polite and more quarrelsome. The symptom, more concerned, lowered significantly. The symptoms less cheerful, more distractible, and more afraid, increased significantly. It was postulated that neurogenic rather than psychogenic factors were accountable for these consistently occurrent neurobehavioural consequences, although psychogenic factors might account for the recognition of impaired functioning (Hartlage et al., 2001.

Relationship between Neurocognitive Recovery and Symptom Recovery

Typically concussed athletes evidence elevated symptom scores for at least as long as impairments on neurocognitive testing are evident (Peterson, Ferrara, Mrazik, Piland, & Elliott, 2003; Randolph, Barr, & McCrea, 2006; Randolph, McCrea, & Barr, 2005; Randolph et al., 2009; McCrea et al., 2003). Accordingly, Erlanger, Kaushik et al. (2003) found symptom duration related significantly to impaired neurocognitive test scores. Although neurocognitive recovery and symptom recovery may be simultaneous or on occasion neurocognitive recovery precedes the resolution of symptoms, it is not uncommon for reported symptom resolution to precede neurocognitive recovery (e.g., Broglio, Macciocchi, & Ferrara, 2007a; Covassin et al., 2008; Fazio et al., 2007; Guskiewicz et al., 2001; Hugenholtz et al., 1988; Lovell, Collins, Iverson et al., 2004; Manko, 2003; McCrory & Shrier, 2002; Warden et al., 2001). Lovell (2002) points out that cognitive and symptom recovery may not be highly correlated, in that they may reflect two distinct neurobehavioural processes.

Two studies have found that although athletes reported being asymptomatic four to five days postinjury, neurocognitive and memory deficits persisted up to seven to ten days postinjury (Lovell, Collins, Iverson et al., 2003; Manko, 2003). Fazio et al. (2007) found that symptomatic concussed athletes performed significantly more poorly on neurocognitive testing than asymptomatic concussed athletes or controls. However, the asymptomatic concussed athletes' poorer neurocognitive test scores than the controls, was suggestive of neurocognitive impairment, despite reports of being asymptomatic. In further support of reported symptom resolution preceding neurocognitive recovery, Broglio, Macciocchi and Ferrara (2007a) found that within 72 hours of MTBI, athletes were symptomatic and 81% showed deficits on at least one ImPACT composite score compared with baseline testing. However, when the athletes reported being asymptomatic, 38% continued to show neurocognitive impairments on at least one

ImPACT composite score compared with baseline testing. Competitive sportsmen tend to under-report symptoms, in hopes of returning to play, and therefore the importance of neurocognitive testing cannot be overemphasised in terms of making appropriate return to play decisions, even in the absence of symptoms (e.g., Echemendia et al., 2001; Erlanger, Saliba et al., 2001; Fazio et al., 2007; Pellman, Lovell et al., 2004; Van Kampen, Lovell, Pardini, Collins, & Fu, 2006).

During the chronic stage of MTBI recovery, one study found that patients with persistent symptoms six months postinjury, compared with the asymptomatic patients, evidenced deficits in attention and information processing (Bohnen, Jolles, & Twijnstra, 1992). It could be surmised from these findings, that in cases of chronic symptom duration, it is likely that chronic neurocognitive deficits persist.

Cumulative Effects of MTBI

Some authors posit that a mild once-off MTBI poses minimal risk to symptomatic and neurological functioning with little or no chronic sequelae, in that the effects of MTBI are subtle and transient (e.g., Belanger et al., 2005; Hinton-Bayre & Geffen, 2004; Iverson, 2007; Schretlen & Shapiro, 2003). However, there is converging evidence that cumulative and more permanent neuropsychological impairments arise from repeat MTBI, possibly as a consequence of neuronal attrition, and which may be detrimental to an athlete's well-being (e.g., Bailes & Cantu, 2001; Gronwall, 1989; Iverson et al., 2004a, 2004b; Manko, 2003; Pettersen & Skelton, 2000; Rutherford et al., 2003; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007a; Shuttleworth-Jordan et al., 1993; Wilner, 2010). The potential risk for long-term adverse effects, as a result of multiple incidents of MTBI over months or years, is now well recognised and therefore MTBI sustained through sport can no longer be considered benign (e.g., AAN, 1997; Bailes & Cantu, 2001; Kushner, 2001; McLatchie & Jennett, 1994; Wilner, 2010). Sporting bodies are starting to realise the long-term consequences resulting from MTBI, for example the National Football League recently acknowledged both the risk of sustaining MTBI, and the long-term effects of MTBI (Schwartz, 2009, 2010).

Two possible factors contributing towards cumulative brain injury, are the residual effects of both concussive and subconcussive events as a result of playing contact sport

(Bernstein, 1999; King, 1997; Erlanger et al., 1999; Rutherford et al., 2003). It is proposed that subconcussive episodes (microtraumatic brain injuries) that are below the threshold for symptom presentation, may collectively result in long-term neuropsychological consequences (Rutherford et al., 2003; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Jordan et al., 1993). It has been proposed that subconcussive blows to the head may cause as much, or more damage, than a single concussive event. Furthermore, it is speculated that in the days following a repeat trauma, the severity of focal cerebellar ischaemic lesions that could arise, may be proportional to the number of subconcussive events sustained. However, it is difficult to establish where the residual effects of subconcussion begin and those of concussion end (e.g., Barth et al., 1989; Webbe & Barth, 2003). Despite these conceptual difficulties, overall cumulative concussive and subconcussive injury is considered to slow the recovery of neurological functioning (Bailes & Cantu, 2001; Mendez et al., 2005).

The risk of cumulative injury works in two ways. Firstly, an MTBI event causes vulnerability towards sustaining a further MTBI event, and secondly multiple MTBI events are posited to have additive negative neuropsychological effects. Empirical research findings supporting these two factors are now discussed. Following a seemingly full recovery from an incident of MTBI, residual sequelae are considered to increase vulnerability towards CNS stressors, such as alcohol, fatigue or hypoxia, and importantly towards sustaining a further MTBI (Gronwall & Wrightson, 1975; Shuttleworth-Jordan et al., 1993). Having sustained one concussion is a significant risk factor for sustaining a further concussion (Bender, Barth, & Irby, 2004; Bernhardt, 2009; Cantu, 1998c, 2001; Guskiewicz, McCrea, Marshall et al., 2003; Guskiewicz, Marshall et al., 2007; Guskiewicz, Mihalik et al., 2007; Kontos et al., 2004; Martineau et al., 2007; McCrory & Berkovic, 1998; Moser et al., 2007). Following an initial concussion, the risk of sustaining a further concussion is three to six times greater than the risk for an individual who has never been concussed sustaining a first concussion, and the risk is exacerbated further following a history of three or more concussions (e.g., Bernhardt, 2009; Cantu, 2001; Guskiewicz, McCrea et al., 2003). Accumulatively, therefore, each prior MTBI event increases an athlete's risk of a subsequent MTBI event by 50%, and football players with a history of more than three MTBI events are more likely to sustain a further MTBI in a season than football players with a history of two or less MTBI events (Pretz, 2007).

Not only is a history of MTBI considered to decrease the threshold for sustaining a future MTBI event, it also increases the risk of overcoming a future MTBI incident and prolongs recovery (Bender, Barth, & Irby, 2004; Collins et al., 2002; Kontos et al., 2004; Slobounov, Slobounov, Sebastianelli, Cao, & Newell, 2007). Moreover, should an athlete participate in sport prior to the brain recovering from MTBI, the neurologic effects of a further MTBI, particularly if spaced closely together, increases the likelihood of prolonged recovery, chronic neuropsychological effects, and may even be catastrophic (e.g., Bailes & Cantu, 2001; Lovell, Collins, & Bradley, 2004; Manko, 2003; Putukian & Echemendia, 1996). Numerous authors and studies attest to the prolonged recovery or cumulative, long-term, negative neuropsychological consequences among athletes with a history of repeat MTBI, in comparison with athletes sustaining an initial MTBI (e.g., Covassin et al., 2008; Flanagan, 1999; Gaetz, Goodman, &Weinberg, 2000; Gronwall & Wrightson, 1975; Guskiewicz et al., 2000, 2003; Iverson et al., 2004a; Killam et al., 2005; Lovell, Collins, Iverson et al., 2004; Martineau et al., 2007; Moser & Schatz, 2002; Moser, Schatz, & Jordan, 2005; Rutherford et al., 2003; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth- Edwards & Whitefield, 2007a).

A history of one previous MTBI event has been associated with slower neurocognitive recovery among collegiate football athletes (e.g., Guskiewicz, McCrea, Marshall et al., 2003). A history of two or more MTBI events compared with a single MTBI event, has been associated with significantly poorer performance in information processing speed and executive functioning, more prominent adverse effects in the first two days postinjury, an increased duration of impairment on verbal memory and reaction time measures, slower recovery, increased symptom reports, and significantly suppressed P3 amplitude event-related potentials (e.g., Collins, Grindel et al., 1999; Covassin et al., 2008; De Beaumont, Brisson, Lassonde, & Jolicoeur, 2007; Gronwall and Wrightson, 1975; Iverson et al., 2004a). One study looking at chronic effects found that secondary school athletes with a history of two or more MTBI events evidenced similar cognitive performance to athletes who had sustained MTBI in the past week (Moser et al., 2005).

Athletes with a history of three or more MTBI events have demonstrated neurophysiological evidence of long-term changes, diminished memory performance, slower processing speed, and a greater number of postconcussion symptoms reported at baseline testing (e.g., Gaetz et al., 2000; Gardner et al., 2010; Guskiewicz et al., 2000;

Iverson et al., 2004a). Athletes sustaining a fourth MTBI compared with athletes without a history of MTBI, have evidenced more severe on-field presentation, with six times more PTA experienced, more significant and acute memory deficits, and slower recovery (e.g., Collins et al., 2002; Covassin et al., 2008; Guskiewicz, McCrea, Marshall et al., 2003; Iverson, 2007; Iverson, Brooks, Lovell, & Collins, 2006; Iverson et al., 2004a). A transcranial magnetic stimulation study investigating the effects of multiple sport-related concussions on motor cortex integrity, adds further support to the cumulative effects of MTBI. It found that these athletes exhibited a prolonged cortical silent-period duration, which was further exacerbated when sustaining a subsequent concussion (De Beaumont, Lassonde, Leclerc, & Théoret, 2007).

In contrast, some authors have not evidenced an association between poorer neurocognitive test performance on computerised or traditional neurocognitive tests for athletes with a history of MTBI, compared with athletes without a history of MTBI, or when comparing the effects of two mild MTBIs sustained two weeks apart (e.g., Broglio, Ferrara, Piland, Anderson, & Collie, 2006; Bruce & Echemendia, 2009; Collie, McCrory, & Makadissi, 2006; Iverson, Brooks, Lovell, Collins, 2006; Iverson, 2007; Macciocchi et al., 1996). There have also been contradictory findings in respect of concussion history and symptom reports. On one hand, following MTBI, no differences in symptom reports were revealed between athletes with a history of two or more MTBI events and those without a history of MTBI (Covassin et al., 2008). On the other hand, following MTBI, athletes with a history of two and three MTBI events have reported more symptoms than athletes with no history of MTBI (Collins, Grindel et al., 1999; Gaetz et al., 2000). Although the study by Pretz (2007) found no relationship between the number of days to recovery and the number of previously sustained MTBI events, athletes with a history of three or more MTBI events, appear to experience greater MTBI initial symptom severity. The latter finding adds to the evidence that three or more MTBI events may result in chronic neurophysiological changes, which may cause a decline in neuropsychological test performance.

Persistent mild cognitive deficits suggestive of cumulative brain injury have been evidenced in athletes playing soccer, football and rugby that, particularly among older, comparatively more senior level athletes, is indicative of the additive effects of concussive and subconcussive injuries resultant from participation in contact sport (e.g.,

Baroff, 1998; Cremona-Meteyard & Geffen, 1994; Matser et al., 1998; 1999; Rutherford et al., 2005; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards & Whitefield, 2007a; Sortland & Tysvaer, 1989; Spear, 1995; Tysvaer & Lochen, 1991; Tysvaer, Storli, & Bachen, 1989).

Overall, studies have evidenced chronic sequelae as a consequence of repeat MTBI events. For example, athletes who have sustained multiple MTBIs, are likely to perform more poorly relative to nonconcussed athletes on baseline cognitive testing, and evidence more prominent adverse effects in the first two days postinjury, experience slower recovery, and report more subjective and persistent postconcussive symptoms (e.g., Collins, Echemendia, & Lovell, 2004; Covassin et al., 2008; Gaetz et al., 2000; Iverson et al., 2004a; Guskiewicz, McCrea, Marshall et al., 2003; Martineau et al., 2007; Shuttleworth-Edwards, Border et al., 2004). Accordingly, the evidence of cumulative injury casts doubt on the supposed transient nature of MTBI, and it can be assumed that structural damage below the threshold for detection exists (Mathias et al., 2004; McAllister & Arciniegas, 2002; Shuttleworth-Edwards & Whitefield, 2007a). Therefore, in particular reference to rugby, due to player-to-ground and player-to-player collisions, the numerous concussive and subconcussive injuries that occur are likely to result in cumulative brain injury (Shuttleworth-Edwards & Whitefield, 2007a).

Chronic Traumatic Brain Injury (CTBI)

The terms chronic traumatic brain injury (CTBI) and chronic traumatic brain encephalopathy (CTE) are the terms commonly used to describe the cumulative and long-term neurological consequences of repetitive subconcussive and concussive events, primarily described in boxing studies, but anticipated amongst various contact sports such as soccer, football, ice hockey and rugby (e.g., McKee et al., 2009; Rabadi & Jordan, 2001). CTBI is a form of progressive neurological deterioration with associated Parkinson's features, and is characterised by disturbances in behaviour, personality, memory, speech and gait (e.g., Lees, 1997; McKee et al., 2009). Not only is it the severity of prior MTBI events, but the frequency of these events may be a more important factor, in causing an accumulative effect, from which CTBI develops (Delaney, 2005; Rochester at al., 2006).

As early as 1957, Blonstein and Clark studied CTBI among amateur boxers who had suffered severe concussions or had been knocked out more than once some time previously. They found that 42% suffered EEG and neurological abnormalities suggestive of deleterious chronic cerebral changes (Barth et al., 1989). Further boxing studies on CTBI found that subconcussive events, not necessarily resulting in knockouts, caused residual long-term deficits, that there is a direct relationship between brain damage and length of boxing career, that the greater the number of knockouts the greater the impairments evidenced on EEG readings and CT scans, and that the effects of multiple MTBIs are cumulative among both amateur and professional boxers (e.g., Barth et al., 1989; Casson, Sham, Campbell, Tarlau, & DiDomenico, 1982; Casson et al., 1984; Kaste et al., 1982).

A study on retired football players suggests a link between repeat MTBI and an increased risk for depression, which lends further support to the chronic impact of MTBI on neuropsychological functioning (Guskiewicz, Marshall et al., 2007). A recent study compared healthy retired hockey and football athletes (mean age 60.8 years) who had sustained 1 to 5 sport-related MTBIs 27 to 41 years previously with 21 healthy retired athletes who had not sustained a concussion (mean age 58.9 years). The results showed significantly delayed and attenuated ERP P3a and P3b components, with significantly prolonged cortical silent periods, significant reductions in bradykinesia, and poorer performance on episodic memory and response inhibition on the neuropsychological tests. The study also provided preliminary evidence for cognitive (alterations of episodic memory and frontal lobe functions) and motor aberrations in late adulthood, even after one or two concussions (De Beaumont et al., 2009). More recently, repetitive MTBI experienced in contact sport is associated with a motor neuron disease, namely amyotrophic lateral sclerosis (ALS), for which TDP-43 proteinopathy has been identified as the first pathological marker, in the brains and spinal cords of athletes diagnosed with ALS (McKee et al., 2010). Furthermore, a history of prior head injury is an associated risk factor for Alzheimer's Dementia (e.g., Mortimer et al., 1991; Shuttleworth-Edwards & Whitefield, 2007a).

3.6.4 Factors affecting MTBI Prognosis

Initial symptom presentation following MTBI, particularly symptoms that become progressively more imminent in the months following injury, or appear disproportionate to objective neuropsychological testing or injury history, may be attributable to particular premorbid or postmorbid factors (Arciniegas et al., 2005). Vanderploeg, Belanger and Curtiss (2006) suggest that the focus should not be on which factors influence MTBI outcome, but rather on determining the cause of symptoms in each case. They suggest that symptom patterns could be attributed to one of the following or a combination thereof: normal population variance, brain dysfunction, premorbid or comorbid medical or psychiatric problems, malingering or lack of effort on examination, and other factors such as poor social support, self-belief, coping skills or expectations (Vanderploeg et al., 2006). Therefore, prognosis following MTBI cannot be generalised, in that many factors contribute towards the outcome. Important factors that may influence MTBI prognosis, include but are not limited to, age, education, gender, genetic factors, history of previous MTBI, position of play, pre-existing neurologic and psychiatric conditions, and under-reporting or non-recognition of MTBI. These are now discussed further.

Age

Adults, adolescents and children respond differently to MTBI. Children are considered more susceptible and vulnerable to MTBI and the negative effects of MTBI including cerebral oedema and subdural haematomas, although different responses may relate to cerebral maturation, rather than age *per se* (e.g., Anderson et al., 2001; Giza & Hovda, 2004; Halstead & Walter, 2010). Therefore sport-related MTBI that occurs during specific brain development stages, may have enduring effects on neural pathway development, experience-dependent plasticity, neurotransmission and metabolism - with the resultant cognitive potential being diminished (Giza & Hovda, 2004). In 2008, the CISG panel acknowledged that children respond differently to MTBI physiologically, take longer to recover and encounter specific risks compared with adults, and conservative return to play management was proposed for children (McCrory et al., 2009).

Empirical studies have revealed that secondary school football players take longer to recover than older professional or university athletes, and that there is an association

between a younger age and increased difficulty on visual and verbal memory tasks (Collins, Lovell, Iverson, Ide, & Maroon, 2006; Field et al., 2003; Majerske et al., 2008; Pellman, Lovell, Viano, & Casson, 2006). In contrast, some authors suggest increasing age is a disadvantage in MTBI outcome, in that there is an association between being aged over 30 years and poorer MTBI outcome. Among those aged 15 to 56 years, impaired memory and visuomotor problem solving skills three months post MTBI, were more apparent as age increased and education decreased (e.g., Barth et al., 1983; Leininger et al., 1990; Wrightson & Gronwall, 1981).

Education

The outcomes of MTBI and neuropsychological test performance are influenced by the level and quality of education (Shuttleworth-Jordan, 1996). This will be discussed in terms of the Reserve theory, in section 3.7 below. On the other hand, individuals and their families should be educated on the recognition of MTBI, its typical symptoms and the importance of monitoring these symptoms, and how to recognise chronic MTBI symptoms. Possessing this type of knowledge is associated with a decrease in developing chronic MTBI symptoms, an improvement in symptoms three months postinjury, and better outcomes in the first year following injury (e.g., Arciniegas et al., 2005; Paniak et al., 2000; Silver et al., 2009).

Exercise

Initially, following MTBI, an athlete should be withheld from sport, physical and cognitive exertion, so that the brain's increased energy demands following MTBI are not taxed further, and to avoid intensifying symptoms and prolonging neurocognitive recovery (e.g., Canadian Academy of Sport Medicine Concussion Committee, 2000; Canadian Pediatric Society, 2006; Giza & Hovda, 2001; Halstead & Walter, 2010). High levels of physical activity following MTBI have been associated with poorer neurocognitive performance. However, moderate levels of physical activity below the symptom threshold, have been associated with stronger neurocognitive performance within 33 days of MTBI and improvement in the PCS condition (Leddy et al., 2010; Majerske et al., 2008). Due to these conflicting views, physical activity following MTBI, warrants further empirical investigation.

Gender

Gender is a possible risk factor for MTBI, and may influence injury severity (McCrory et al., 2009). Extensive empirical evidence suggests that female athletes have a higher risk of sustaining MTBI than male athletes (Covassin, Swanik, & Sachs, 2003; Delaney et al., 2002; Pretz, 2007; Grindel, Lovell, & Collins, 2001; Solomon, Johnston, & Lovell 2006). The reasons for females being more at risk than males may be hormonal, physiological or morphological, in that males have greater cortical neuronal densities and females have a greater number of neuronal processes (Broshek et al., 2005). In contrast, females may have a lower risk for sustaining sport-related MTBI than males, in that their form of play may be less intense and their collisions of lower velocity compared with males (e.g., Cassidy et al., 2004; Shuttleworth-Edwards & Whitefield, 2007a).

In regard to neurocognitive testing, studies on gender analyses amongst sport cohorts have shown that males and females differ, with females outperforming males on visuoperceptual functioning tests, although memory functioning tests between the genders provides mixed results (Barr, 2003; Guskiewicz et al., 2002; Shuttleworth-Edwards & Whitefield, 2007a). Gender differences have been found on ImPACT neurocognitive testing. In one study, although no gender differences were found at baseline testing, females performed significantly worse than males on visual memory, two to eight days post MTBI (Covassin, Schatz, & Swanik, 2007). In contrast, another study on ImPACT neurocognitive baseline test performance, found gender differences for athletes with a history of multiple concussions. For athletes with a history of two and three concussions, females performed better on the verbal memory composite than males. Females with a history of three or more concussions performed better on the visual memory composite, than in males with a history of two or three concussions. Overall, females performed significantly better than males on both the visual motor speed and reaction time composites (Covassin et al., 2010). Therefore, it may be concluded, that females show greater deficits than males in visual memory in the acute stage following MTBI. However, in the instance of cumulative MTBI, males develop greater cumulative deficits than females in reaction time, visual motor speed, visual and verbal memory, or it may be that males failed to report their MTBI history as accurately as females.

For symptom reports, females appear to have a later onset of symptom complaints, are more vulnerable for poorer outcomes, experience more chronic symptom complaints, and

are at increased risk for PCS three months post MTBI, in comparison with males (e.g., Dick, 2009; Preiss-Farzanegan, Chapman, Wong, Wu, & Bazarian, 2009; Shuttleworth-Edwards & Whitefield, 2007a). Although it has been posited that females have more intense and persistent MTBI symptoms in that the female brain has greater cortical metabolic demands than the male brain, Pretz (2007) found no significant difference between genders in the number of days it took to recover following either an initial or second MTBI event. However, findings between the genders at baseline testing are contradictory. Pretz (2007) found that males obtained a significantly higher symptom score than females, possibly due to previous unreported injuries with lingering effects, while Covassin et al. (2006) found females endorsed significantly more symptoms than males. In one study, although no gender differences were found at baseline testing on ImPACT, two to eight days following MTBI, males reported a significantly higher incidence of the symptoms vomiting and sadness compared with females (Covassin, Schatz, & Swanik, 2007). Barnes et al. (1998) found both genders reported the symptom headache, although males complained more of blurred vision, tingling, and numbness, and females complained more of dizziness and feeling dazed, post MTBI.

Genetic Factors

Growing evidence suggests a genetic predisposition towards the adverse effects of TBI. Jordan (2004), explains that the rationale for this derives from similar histopathology abnormalities found in both cumulative TBI and Alzheimer's Dementia (AD), particularly that of amyloid deposition, changes in cholinergic activity and in some instances neurofibrillary tangles. Central to this rationale, is the influence of the Apolipoprotein E (APOE) genotype. The APOE is a molecule found in intraneuronal neurofibrillary tangles, and in extracellular amyloid plaques. Therefore, the physiological functions of APOE have been postulated as being directly or indirectly involved with neurofibrillary tangle formation, amyloid deposition, cholinergic activity, transportation and metabolism of cholesterol, antioxidant activity, repair and regeneration of injury to the CNS, regulation of alpha-1-antichymotrypsin levels, and medial temporal lobe atrophy and memory. The three isoforms of APOE are APOE2, APOE3 and APOE4, with the APOE4 allele having been soundly established as a risk factor for developing AD (Jordan, 2004). In one study, the APOE4 allele - in conjunction with a history of TBI - was reported to synergistically increase the risk of developing AD by ten-fold, while another study found either the APOE4 allele or TBI each had a risk factor for developing

AD, but neither would confound the other in exacerbating the risk (e.g., Jordan, 2004; Mayeux et al., 1995; O'Meara et al., 1997). However, each of the APOE genotypes and TBI are risk factors for developing AD, therefore the presence of both the APOE genotype and a history of TBI, may well add to the risk for developing AD (Jordan, 2004).

Carriers of the APOE4 allele may have an increased risk for poorer outcomes following severe TBI or repetitive MTBI, although the role of the APOE4 allele on outcome following MTBI is less certain, and the relationship between MTBI, APOE4 status and the development of dementia remains ambiguous (e.g., Arciniegas et al., 2005; Chamelian, Reis, & Feinstein, 2004; Friedman et al., 1999; Heilbronner et al., 2009; Jellinger, 2004; Jordan et al., 1997; Lichtman, Seliger, Tycko, & Marder, 2000). However, a higher frequency of APOE4 carriage has been found among older athletes and in patients, showing chronic cognitive impairments, often found in MTBI. These include findings of impaired memory and attention, among active and retired boxers who had participated in more than 12 professional bouts, among older active football players demonstrating poorer performance in attention, information processing speed and accuracy, and among TBI patients with poorer outcomes in the cognitive rather than the motor domain and poorer outcomes six months post injury (e.g., Arciniegas et al., 2003; Jordan et al., 1997; Kutner et al., 2000; Seliger, Lichtman, & Polsky, 1997; Teasdale, Nicoll, Murray, & Fiddes, 1997). In other studies, only 3% of patients with the APOE4 allele compared with 31% of patients without the APOE4 allele, had favourable outcomes, and those with the APOE4 allele were five times more likely to endure more than a week of unconsciousness (e.g., Friedman et al., 1999; Jordan, 2004). However, there may be an overrepresentation of the APOE4 allele among persons failing to fully recover from MTBI, rather than presence of the allele influencing outcome, or it could be the presence of the allele together with exposure to MTBI being multiplicative rather than additive in terms of risk for CTBI (Arciniegas et al., 2005; Clausen, McCrory, & Anderson, 2005).

In contrast, other studies have not found significant associations between poor neuropsychological or functional outcomes, and the possession of the APOE4 allele in MTBI cases or among active and retired boxers who had participated in less than 12 professional bouts. However, the small number of bouts fought could suggest that the

reserve threshold had not been reached (e.g., Heilbronner et al., 2009; Jordan et al., 1997). A prospective study on the association between APOE4, age (range 0 to 93 years), and outcome on Glasgow Outcome Scale six months following TBI, found no association between outcome and the APOE4 genotype. The results showed that 36% APOE4 carriers and 33% APOE4 non-carriers evidenced unfavourable outcomes, although a significant interaction between APOE4 and age was revealed. APOE4 carriers aged 0 to 15 years had a reduced prospect of favourable outcome, and thereafter the adverse outcomes of APOE4 carriage reduced steadily with age and by age 55 to 60 years, appeared neutralised (Teasdale, Murray, & Nicoll, 2005).

However, the response to and recovery from MTBI may be influenced by possession or non-possession of a specific genotype. Whether such operates independently or in conjunction with other factors that influence the risk for and outcome of MTBI is uncertain, and warrants further investigation (e.g., Aubry et al., 2001; McCrory et al., 2004, 2009).

History of Previous MTBI

Studies on the cumulative and chronic effects of MTBI, discussed in detail above, provide support that a history of previous MTBI lowers the threshold for sustaining a subsequent MTBI, and that these athletes experience poorer outcomes than those with no history of previous MTBI (Collins, Grindel et al., 1999; Shuttleworth-Edwards, Border et al., 2004). Therefore, athletes with a history of MTBI should be managed conservatively when experiencing a subsequent MTBI, and although Quigley's rule may be advocated - that following three MTBI incidents involving LOC an athlete should discontinue actively participating in sport – this is not scientifically validated (e.g., Barth et al., 1989; McCrory, 2002a; Pretz, 2007; Ruchinskas, Francis, & Barth, 1997).

Position of Play

Studies have indicated that the position of play in team sports is also a risk factor for sustaining MTBI (e.g., Delaney, Lacroix, Leclerc, & Johnston, 2000; Delaney, Lacroix, Gagne, & Antoniou, 2001; Pretz, 2007; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Jordan et al., 1993). Among football studies, players in the offensive position, particularly the quarterbacks, have been found to be at greater risk for sustaining MTBI, than those in the defensive position

(Delaney et al., 2000; Pretz, 2007). However, other studies have found the quarterbacks, running backs, the defensive linesman, and tight ends, at high risk for MTBI (Delaney et al., 2001, 2002; Pretz, 2007). Amongst rugby players, the forwards seem at more risk than the backs for sustaining MTBI (Nathan, Goedeke, & Noakes, 1983; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Jordan et al., 1993). However, Brooks, Fuller, Kemp and Reddin (2005) found that among professional rugby players, that the seasonal incidence of MTBI was higher among backs than in forwards.

Pre-existing Neurologic and Psychiatric Conditions

A pre-existing neurological or psychiatric disorder potentially results in more severe MTBI sequelae, and is therefore attributed as a risk for persisting neuropsychological deficits following MTBI (Collins, Grindel et al., 1999; McCrory et al., 2004; Shuttleworth-Edwards, Border et al., 2004). Therefore, scholastic difficulties as a result of MTBI can be compounded by the presence of a comorbid psychiatric or neurological disorder. For example, it has been found among university football players with a history of two or more concussions, that those suffering a learning disability, as opposed to those who did not, performed more poorly on tests of executive functioning and mental processing speeds (Collins, Grindel et al., 1999; Shuttleworth-Edwards & Whitefield, 2007a). A study on the contribution of pre-existing depression to the acute cognitive sequelae of MTBI, found that participants with depression did not perform significantly more poorly than participants without depression overall. However, the MTBI depressed group performed more poorly than the MTBI non-depressed group on word recognition within 24 hours of MTBI, suggesting an interaction between depression and word recognition (Preece & Geffen, 2007). One study found that MTBI patients compared with non-MTBI patients, performed significantly worse on cognitive measures but did not differ in terms of postconcussive symptom reports. However, cognitive performance was unrelated to emotional distress, and yet postconcussive symptoms reports were consistently related to emotional distress (Landre et al., 2006). These findings suggest that reliance on objective neurocognitive testing in determining recovery from MTBI, may be more predictable than reliance on symptom reports that have an underlying emotional basis.

Under-reporting or Non-recognition of MTBI

It is well documented that athletes under-report MTBI for several reasons, including fear of being withheld from competition, motivation to participate in sport, or an unwillingness to let the team down. In addition, under-reporting is often due to non-recognition that a concussion has occurred, not considering an injury severe enough to warrant medical attention, not realising the significance and importance of symptoms, or being reluctant to describe postconcussive symptoms (e.g., Bailes & Cantu, 2001; Barker & Patel, 2000; Bernhardt, 2009; Covassin, Swanik & Sachs, 2003; Cunningham, 2007; Field et al., 2003; Gerberich et al., 1983; Kaut, DePompei, Kerr, & Congeni, 2003; Kelly et al., 1991; Kontos et al., 2004; LaBotz, Martin, Kimura, Hetzler, & Nichols, 2005; Lovell, Collins, Iverson et al., 2004; Martineau et al., 2007; McCrea, Hammeke, Olsen, Leo, & Guskiewicz, 2005; Pretz, 2007; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards, Noakes et al., 2008; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards & Whitefield, 2007a; Tommasone & Valovich McLeod, 2006). Therefore, an alarming factor affecting prognosis is that often the MTBI event is unrecognised or unreported, which poses great risk for repeat concussion (e.g., Shuttleworth-Edwards, Noakes et al., 2008; Shuttleworth-Edwards & Whitefield, 2007a). In a follow-up study of patients visiting an ED for concussion, 88.6% did not recognise that they had suffered concussion, and 28.2% were engaging in at least one activity that posed a high risk for repeat concussion, and therefore the risk of second impact syndrome (Delaney et al., 2005).

All the above risk factors are considered to affect MTBI prognosis in general. However, inextricably bound to many of the above factors, is the notion of cognitive or brain reserve, that serves both as an additional risk factor affecting MTBI prognosis, and as the theoretical underpinning for this study. It will therefore be discussed below.

3.7 RESERVE THEORY

Initially an explanation of the concept of a Reserve theory will be given, and the differences between Cognitive Reserve and Brain Reserve theories will be provided. Extensions to the reserve theories will then be reviewed, following which evidence of empirical findings in support of the Reserve theory will be briefly elucidated. Finally, the general concept of a combined Reserve theory will be discussed.

3.7.1 The Concept of the Reserve Theories

Based on there being little direct relationship between the extent of brain pathology and the severity of resultant neuropsychological outcomes, the concept of a reserve (i.e., brain or cognitive reserve) arose in order to modulate the relationship between brain pathology and outcomes (Satz, 1993; Stern, 2002; Barnett & Sahakian, 2008). Therefore, reserve accounts for the discrepancy between the degree of brain pathology and its clinical outcome, in that brain pathology of the same magnitude may result in different levels of neurocognitive impairment and rates of recovery between individuals (Stern, 2009). In essence, reserve is a potential mechanism or protective factor that mediates the individual's resistance to neurocognitive impairment that results from brain pathology caused by aging, disease or injury. It provides an explanation for the variability between individual responses to brain pathology (e.g., MTBI) in terms of symptom presentation and neurocognitive functioning (e.g., Barnett & Sahakian, 2008; Shuttleworth-Edwards, Border et al., 2004; Stern, 2002). The reserve concept has been presented in a variety of terms that include, but are not limited to, brain reserve, brain reserve capacity, cognitive reserve, and neuronal or neural reserve (Barnett, Salmond, Jones, & Sahakian, 2006; Katzman, 1993; Mortimer et al., 1991; Satz, 1993; Stern, 2002, 2003, 2009). The various terms relating to the reserve theories appear to be used interchangeably in the literature, however there are differences between these theories pertaining to these terms.

Reserve theories can be broadly classified into passive or active models, with the former referring to the hardware of brain function and the latter to the software of brain function, i.e., the brain processes (e.g., Barnett & Sahakian, 2008; Katzman, 1993; Richards & Deary, 2005; Stern, 2003, 2009). The passive model of brain reserve is based on structural differences, such as the brain size or quantity and density of neurons or synapses (e.g., Barnett & Sahakian, 2008; Stern, 2009). The active model of cognitive reserve is based on cognitive processes where when faced with damage, the brain actively attempts to cope by means of pre-existing neural processes or by recruiting neural compensatory strategies (Stern, 2002; Stern, 2009). Satz, Cole, Hardy and Rassovsky (2010) point out that neural reserve resembles the brain reserve concept, and neural compensation resembles the cognitive reserve concept. Stern (2009) points out that the demarcation between brain reserve and cognitive reserve is not straightforward, in that they can be considered to run in parallel and the two terms are often used interchangeably

in the literature. From the descriptions provided below, it will be seen that both theories overlap one another, to the extent that they are inextricably bound, in that one cannot demarcate at which point structural or functional aspects can account for interindividual differences in outcome from brain pathology, notably MTBI. However, these two terms and their theoretical underpinnings do differ, and therefore cognitive reserve and brain reserve will be discussed initially to delineate the two entities.

Cognitive Reserve Theory

Cognitive Reserve theory postulates that interindividual differences exist in the cognitive processes or neural networks that underlie task performance, and that therefore some people cope better than others with brain pathology or insult. This theory is based on two forms of reserve: (i) neural reserve that refers to interindividual variability of brain networks or cognitive paradigms underlying task performance in the healthy brain (some individuals with greater capacity and more efficient or flexible processes can cope better with brain pathology), and (ii) neural compensation that refers to interindividual variability in compensating for disruptions to standard processing networks as a result of brain pathology, whereby alternate brain networks not usually underlying task performance in the healthy brain, are used to maintain or improve performance (Stern, 2009). Compensation is not limited to using an alternate brain network, but may involve compensatory neurogenesis or regeneration of an underlying network (e.g., Kozorovitskiy & Gould, 2003; Stern 2006). Therefore, two individuals may have the same neural reserve, and yet one individual with efficient neural compensation processes may tolerate a larger brain insult prior to the appearance of its clinical manifestation, than the other individual (Stern, 2009).

The active model does not propose a critical threshold at which functioning is impaired, rather the focus is on the processes that maintain function when the brain is damaged (Stern, 2009). Therefore, when confronting a task in the presence of pathology, the brain could use an existing cognitive paradigm to maintain functioning and operate effectively. However, should this not be possible in light of depleted cognitive reserve, the brain may compensate in order to maximise performance and use an alternate cognitive paradigm to carry out the task (Stern, 2002). Accordingly, individuals suffering brain pathology, but who evidence high IQ scores, levels of education and occupational attainment, independently or synergistically, are assumed to have a high cognitive reserve as a result

of superior brain networks or cognitive strategies, and are more likely to process tasks more effectively before demonstrating functional deficits (Stern, 2002). While Dennis, Spiegler and Hetherington (1999) consider cognitive reserve to equate to intelligence, Barnett and Sahakian (2008) propose that it can be considered as an asset that is both innate (e.g., brain size and hardwiring) and malleable, in that it may be stimulated through leisure, educational and work activities throughout the course of life. The malleable aspect of cognitive reserve during childhood is supported by a study in which childhood educational attainment and cognitive ability had the greatest influence on cognitive reserve in later life. From this it was concluded that reserve is not fixed, and the combined influence of these childhood factors in addition to parental background, serves as a dynamic process promoting physical and cognitive health (Richards & Sacker, 2003).

Brain Reserve Theory

The notion of brain reserve, and the associated Brain Reserve theory, is typically described in terms of being a threshold theory (Satz, 1993; Stern, 2009). The medical concept of a 'threshold' is not new, and refers to a level or cut-off point at which a particular effect occurs. Conceptually, a threshold is a hypothetical protective level, which once surpassed induces a desirable or undesirable reaction in response to this hypothetical level being reached. A medical example is the pain threshold. It is assumed that different individuals have differing pain thresholds or levels of pain tolerance, whereby an individual with a low pain threshold is more susceptible to experiencing pain at a low intensity, and an individual with a high pain threshold is able to withstand a greater intensity of pain. In comparison, in terms of Brain Reserve theory, Satz (1993) postulates that each individual is conceptualised as having a neural reserve, the interindividual threshold differences which explain variability among individuals, and that when this reserve is depleted beyond a certain threshold during the normal aging process or as a result of brain pathology, then central nervous system dysfunction or deficits emerge (Randolph, 2001; Satz, 1993; Stern, 2002, 2009; Stern et al., 2003). These reserve differences are considered quantitative – such as a larger brain or a greater number of neurons or synaptic gaps - and include life experiences that can impact on brain anatomy, for example through neurogenesis or the up-regulation of compounds promoting neural plasticity (Stern, 2009).

Stern (2002, 2009) considers the threshold model of brain reserve to be a passive model, in that there is a fixed threshold or cutoff for every person, below which functional impairment results. Stern also considers it as a quantitative model, whereby in a specific type of brain pathology or in the instance of cumulative brain damage, the sum result has the same effect once the critical level of neural depletion is reached. However, individual differences in how the brain processes cognitive tasks as a result of brain pathology, are not accounted for in the threshold models (Stern, 2009). Although Stern (2002) considers brain reserve a passive model, Satz et al. (2010) argues that this is not the case, in that the reserve adapts based on experience, age or injury, by inducing compensatory and regenerative mechanisms that assist in maintaining or repairing neuronal functioning, for example by neurogenesis or synaptogenesis.

Other Reserve Theories

Other proposals of reserve have arisen, based on the Cognitive and Brain Reserve theories. Based on the Brain Reserve theory, Jordan (1997) developed the Shuttle-effect Model to explain interindividual variability in aging. In this model, the process of aging is conceptualised as a progression of cumulative mild brain insults that progressively lower the reserve, and due to there being interindividual variability in the pre-existing reserve upon which these insults impact, this accounts for the variability in the expression of cognitive aging. This model can be extrapolated to the cumulative effects of sport-related MTBI insults over time. Christensen and colleagues proposed that the Brain Reserve hypothesis incorporates indicators of brain reserve, e.g., brain volume, and indicators of cognitive reserve, such as education and intelligence (Christensen et al., 2007; Satz et al., 2010). Valenzuela and colleagues (Valenzuela, 2008; Valenzuela, Breakspear, & Sachdev, 2007) consider the concept reserve as a single construct of lifetime experience that incorporates brain reserve and observable behaviour relating to complex mental activity. Satz et al. (2010) note that Valenzuela's concept is a behavioural approach based on neurocomputational flexibility, and believe it ignores the basis of neural reserve (e.g., brain volume) and cognitive reserve (e.g., intelligence as a factor) on the grounds that these factors cannot be modified.

Richards and Deary (2005) have also extended these reserve theories further, by conceiving both the structural and functional approaches to reserve, as a lifetime of input, and consequently developed the Life Course Model of Cognitive Reserve. Wallack

(2006) explains that more recently, the Default State theory of reserve is being posited as a neurological version of cognitive reserve. The Default State theory explains the association between life experience and physiological change. Default activity patterns are conceptualised as having a default mode, whereby the default mode is turned off for cognitive tasks requiring attention, but as one ages the inability to turn off the default mode relates to cognitive decline and an inability to perform tasks requiring undivided attention. Research for this model uses a default mode baseline of brain activity, for comparisons of the neural basis of cognition and aging. Research on this theory has assessed differences in brain activity between young and old, and between healthy controls and individuals with AD, APOE4 and mild cognitive impairment (Wallack, 2006).

In criticism of Reserve theories, Satz et al. (2010) point out that neither brain reserve nor cognitive reserve have been adequately addressed in terms of their construct validity (i.e., convergent and discriminant validity) as single or multiple forms of reserve(s). Satz et al. (2010) acknowledge that Siedlecki et al. (2009) attempted to establish the construct validity of cognitive reserve, finding that lifetime experience (the targeted form of cognitive reserve), overlapped with two out of three non-targeted forms - these being fluid ability and executive functioning. Satz et al. (2010) propose methods to assess construct validity in order to verify factors that contribute towards reserve capacity. However, to date, it appears that when it comes to reserve theories, we continue to be dealing with *a priori* concepts and *a priori* groupings of variables (Satz et al., 2010).

Empirical Evidence in Support of Reserve Theories

The terms and accompanying theoretical underpinnings of reserve theories, are often used to conceptualise neurocognitive changes accompanying Alzheimer's Dementia, but additionally have been used in reference to neurocognitive changes accompanying HIV induced dementia, bipolar mood disorder, schizophrenia, depression, TBI and MTBI (e.g., Barnett et al., 2006; Collins et al., 1999; Fairnpour et al., 2003; Heilbronner et al., 2009; Kesler, Adams, Blasey, & Bigler, 2003; Shuttleworth-Edwards, Border et al., 2004, Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007a; Stern, 2009; Stern et al., 2003). Research findings in support of the passive reserve models include no cognitive impairment having been shown by 10 to 40% of individuals with neurophysiological markers of AD on

autopsy, and that brains of those without dementia, compared with those with dementia, are of a relatively larger volume with greater numbers of neurons (e.g., Barnett & Sahakian, 2008; den Heijer et al., 2006; Katzman et al., 1988; Mortimer, 1997). The fact that a substantial number of individuals who evidence the neuropathological criteria of AD, but do not manifest the clinical expression of AD, provides support that brain reserve has an important role in the clinical expression of AD (Mortimer, 1997). Additionally larger brains have been found to maintain better functioning for a longer time frame, in crack cocaine addicts (Di Sclafani et al., 1998).

A multitude of general research findings empirically support the active reserve models, whereby proxy measures such as IQ, occupational factors, educational level, educational attainment in combination with size of head circumference, and literacy, physical, social and intellectual or cognitively stimulating activities, provide support for lower levels of these proxy measures serving as risk factors for memory decline, AD, neuropsychiatric disorders or overcoming TBI (e.g., Baltes & Gutzmann, 1995; Barnett & Sahakian, 2008; Barnett et al., 2006; Katzman, 1993; Katzman et al., 1988; Kesler et al., 2003; Klein, Houx, & Jolles, 1996; Manly, Toradji, Tang, & Stern, 2003; Mortimer, Snowdon, & Markesbery, 2003; Ostrosky-Solis, 2004; Ropacki & Elias, 2003; Sánchez, Rodríguez, & Carro, 2002; Scarmeas & Stern, 2003; Wilson, Barnes, & Bennett, 2003). Moreover, Lee (2003) reviewed several studies in support of cognitive reserve, that provide sound and mounting evidence that genetic variation accounts for a portion of cognitive variation. Imaging studies in support of active cognitive reserve models, have assessed variability between the young and old in networks underlying task performance (e.g., Scarmeas et al., 2003; Stern, 2009; Stern et al., 2003, 2005).

Education, in particular, is considered to enhance cognitive reserve by improving the brain's structure and functioning, that then promotes protection against deleterious effects in the aging process (e.g., Coffey, Saxton, Ratcliff, Bryan, & Lucke, 1999; Dawson, Batchelor, Meares, Chapman, & Marosszeky, 2007; Friedman, 2003; Katzman, 1993; Mortimer & Graves, 1993). Furthermore, physical activity in early life (age 15 to 25 years) is associated with less cognitive decline, particularly on information processing speed, among men but not women of average age 74.9 years (Dik, Deeg, Visser, & Jonker, 2003). In support of a behavioural brain reserve, meta-analyses of studies have found less cognitive decline and a reduction in risk for developing AD for high mental

activity levels compared with low mental activity levels (Valenzuela, 2008; Valenzuela & Sachdev, 2006). However, not all studies support the reserve theories. For example, one study found that education, intelligence and creativity did not buffer against cognitive decline amongst those with high levels of brain atrophy (Christensen et al., 2007). A study involving brain training computerised programs three times a week for six weeks, found no improvement in cognitive functioning (Lowry, 2010a). In respect of MTBI specifically, Dawson et al. (2007) did not find age, preinjury alcohol consumption, preinjury marijuana use, prior neurological damage, or emotional distress postinjury, to be significantly associated to PTA duration following MTBI.

Research in support of reserve theories following MTBI in general, has found a significant negative association between IQ and longer PTA duration, and that cognitive dysfunction is a predisposing risk factor for a further MTBI event (e.g., Dawson et al., 2007; Teasdale & Engberg, 1997). Although several studies (see Chapter 5) provide support for the reserve theories, in terms of older and cognitively vulnerable contact athletes evidencing greater impairment than noncontact sport controls, few studies have extrapolated the concept of a Reserve theory directly as a means of interpreting their findings, and accounting for the different outcomes and variability found among athletes on neuropsychological testing following MTBI (e.g., Collins et al., 1999; Heilbronner et al., 2009; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007a; Stern et al., 2003). Collins et al. (1999) conducted a study illustrating the cumulative effects of multiple MTBI events coupled with a learning disorder, in that athletes presenting with both conditions evidenced impaired scores on tests of processing speed and executive functioning, and that these scores were comparatively poorer than scores of athletes with histories of prior MTBI events, but with no learning disorder. Therefore, the presence of a learning disorder in conjunction with MTBI is considered to lower brain reserve capacity (Collins et al., 1999).

Heilbronner et al. (2009) explain the application of cognitive reserve to chronic traumatic encephalopathy (CTE) among boxers as follows. While high exposure to boxing is not sufficient to cause CTE, cumulative boxing related injuries, combined with age related neuronal loss, could account for late life dementia, experienced by some retired professional boxers. Accordingly, boxers who halt their boxing careers may have some

neuronal loss, but may not evidence signs of CTE while a critical percentage of functional neurons remains. However, the combination of neuronal reduction resultant of several concussive and subconcussive traumas, increasing age, and possibly some other health issues (such as hypertension, diabetes and alcohol abuse), may cause clinical signs of dementia at an earlier age in former professional boxers than in non boxers (Heilbronner et al., 2009). Therefore, Cognitive Reserve theory is useful in explaining why CTE tends to emerge after a boxing career has ended (Heilbronner et al., 2009; Jordan, 1993).

Studies on MTBI among rugby players providing support for the Brain Reserve theory, have found that in comparison with control participants, that the cumulative effects of rugby-related MTBI (i) reduce visuomotor processing skills, particularly among older national rugby players compared with schoolboy rugby players and among rugby forwards versus backs, (ii) cause substantially more pronounced impairment, particularly in information processing speed and memory among older, cognitively vulnerable and experienced rugby players, and (iii) result in impaired performance on attentional tasks and visuomotor processing speed among university rugby players (e.g., Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008). In Shuttleworth-Edwards and Whitefield's (2007a) review of potential deleterious outcomes from sport-related concussion, the Brain Reserve model was used to explain the variability and inconsistencies among athletes sustaining MTBI of similar severity, and yet evidencing different outcomes. Shuttleworth-Edwards and Whitefield (2007a) propose that athletes who are prone to sustaining MTBI, or are highly susceptible to the deleterious outcomes of MTBI, are those who initially possess reduced cerebral capacity as a result of factors that include low IQ, a learning disability, and a neurological or psychiatric disorder.

Shuttleworth-Edwards and Whitefield (2007a) point out that most athletes do not evidence directly observable chronic disability following a single incident of MTBI, despite the possibility of continuing to suffer from a reduced brain reserve capacity. However, although 10 to 30% of athletes are considered to suffer chronic outcomes as a result of MTBI, the effects of such are diluted within group analyses, in which most athletes appear to not suffer such consequences. Therefore, the potential exists for failing to find significance when effects do actually exist (Type II error), and this is further

compounded by group numbers being small in most studies, or when inappropriate samples are selected. For example, Stephens et al. (2005) investigated neuropsychological impairment among a young group of athletes aged 13 to 16 years, that in terms of the Reserve model, any effects would be subtle or subclinical during this developmental stage (Shuttleworth-Edwards & Whitefield, 2007a).

Ellemberg et al. (2009) provide the following reasons for the discrepancies among literature pertaining to recovery from MTBI in support of the reserve theories. On the basis that neurocognitive and symptom recovery usually occurs within two weeks, and yet based on empirical findings from brain imaging studies, electrical response abnormalities, metabolic imbalances and impaired oxygen consumption persist for several months following MTBI, Ellemberg et al. (2009) propose that following MTBI, functional recovery takes places rapidly, in that brain plasticity permits compensatory mechanisms in the form of functional reorganisation or adopting new strategies. Therefore, athletes may perform in the normal range on neuropsychological testing, one to two weeks post MTBI. Any extremely subtle residual cognitive deficits are not able to be detected on neuropsychological assessment, despite the prolonged stage of neuronal recovery, that is evidenced by imaging and electrophysiological studies (Ellemberg et al., 2009).

3.7.6 A Combined Reserve Theory

It is clear that the Brain Reserve and Cognitive Reserve theories overlap one another and are inextricably bound, in that the structural and functional aspects they respectively account for, cannot be empirically demarcated in explaining the interindividual differences in outcome from brain pathology, notably MTBI. Furthermore, a combination of the theories appears acceptable, in that no one theory has been adequately addressed in terms of its construct validity as single reserve or multiple forms of reserves (Satz et al., 2010). With this in mind, this thesis will use a combination of these theories under the umbrella of a combined Reserve theory, and will bring together the differences between reserve theories, as discussed below.

Firstly, the combined Reserve theory will be used to take into account the Cognitive Reserve theory that envisions cognitive processing and assumes a physiological basis for

the brain to mediate these processes in terms of brain network or anatomical variability. Therefore, the physiological basis for the brain as a protective factor against pathology in terms of differences in the quantity of neural substrate that include anatomic measures such as head circumference, brain volume, dendritic branching or synaptic counts assumed by the brain reserve is taken into account (Stern, 2009). Secondly, the combined Reserve theory will take into account the Brain Reserve presupposition of a fixed or critical threshold, at which point functional impairment occurs. While the Cognitive Reserve theory does not presuppose a fixed point at which functional impairment occurs, in that individuals differ on the basis of the intact software remaining in the neural substrate (Stern, 2009), the combined theory will consider that at the time data collection commenced, on the basis of cognitive reserve, individuals will differ in terms of the intact software remaining in the neural substrate. However, due to subconcussive and concussive events sustained, at a critical point of brain insult, it is assumed that the software remaining would no longer be capable of maintaining optimal function, and deficits in cognitive processing would emerge on neurocognitive testing intraindividually. Thirdly, it will be assumed that cognitive stimulation, exercise and life experiences are associated with increased cognitive reserve, in the forms of increased neurogenesis, neuronal plasticity and increased brain volume, that contribute towards interindividual variability in brain volume increase (Stern, 2009). Fourthly, variables descriptive of life experience or serving as factors that contribute to reserve include: socioeconomic status, literacy, level of education, occupational attainment, leisure activities, and IQ (Stern, 2009). These variables are assumed to be associated with both brain physiology and cognitive processing in the combined Reserve theory. Finally, Satz (2002) points out that the two reserve concepts overlap, and reserve can be construed as a multidimensional entity, and therefore a combined Reserve theory implicitly construes reserve as a multidimensional entity.

In this study, therefore, if the concepts of brain reserve and cognitive reserve are taken together in understanding the impact of multiple concussive and subconcussive injuries sustained through rugby prior to the study, and over one rugby season of the study, then it can be conceptualised that rugby players are not a homogenous group, and that different individuals will respond in differing ways to these injuries according to their reserve. Various vulnerability factors, that would synergistically lower the threshold level or reserve capacity that each rugby player possesses vary according to a number of

factors, including but not limited to: genetic endowment (e.g., brain size, neuronal density, general intelligence), level and quality of education, age, level of neuronal attrition (through aging or previous brain injuries), adaptive or compensatory behaviours, neuropsychiatric disorders, and environmental factors such as alcohol abuse (e.g., Satz, 1993; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007a). Accordingly, the greater an individual's reserve, the more the individual's brain can withstand concussive or subconcussive insult, and consequently the neuropsychological damage would appear less. In juxtaposition, the lower an individual's reserve, the less the individual's brain can withstand concussive or subconcussive insult, and consequently neuropsychological damage would appear greater. Furthermore, Shuttleworth-Edwards, Border et al. (2004) indicate that individual outcomes can be assessed in terms of examining the distribution of scores and standard deviations between groups, or comparing the percentage of deficits between the rugby and equivalent control groups - so overcoming reliance on group comparisons of the quasi-experimental form.

Based on these constructs and taking into account that other rugby-related MTBI studies have evaluated and interpreted findings in terms of Reserve theory as an appropriate heuristic model, the basic tenets of a combined reserve model will inform the theoretical underpinning for this study. Specifically, in respect of sport-related MTBI, Shuttleworth-Edwards, Border et al. (2004) propose three essential suppositions of reserve capacity that account for vulnerability to or protection from neuropsychological effects of MTBI. These suppositions serve to delineate the theoretical basis adopted for this study:

(i) There are individual differences in reserve capacity that protect the individual or cause the individual to be more vulnerable to the risk of clinically detectable functional impairment

(ii) A premorbid vulnerability factor lowers the brain reserve capacity, rendering the individual to an increased risk of functional impairment

(iii) Combined premorbid vulnerability factors, cumulatively or in conjunction, lower the brain reserve capacity further, increasing risk of functional impairment for the individual.

3.8 OVERVIEW

This chapter provided a detailed background of MTBI in terms of its definition and related terms, followed by a description of the mechanisms, biomechanisms and neurophysiological consequences of MTBI. The outdated means of rating MTBI severity were briefly discussed, and the neuropsychological consequences of MTBI in terms of the neurocognitive consequences and signs and symptoms were discussed in greater detail. MTBI resolution, specifically the acute and chronic stages of MTBI resolution, the relationship between neurocognitive and symptom recovery, and the cumulative and chronic traumatic brain injury aspects were covered. Factors affecting MTBI prognosis were discussed, in particular the Reserve theory that pertains to the theoretical underpinning of this study.

CHAPTER FOUR: EPIDEMIOLOGY OF MILD TRAUMATIC BRAIN INJURY IN TEAM SPORTS

This chapter begins with a description of team contact sports associated with a high risk for mild traumatic brain injury (MTBI), including ice hockey, soccer, American football, Australian football, rugby league and rugby union, the latter being dealt with more extensively being the sport under study in this thesis. A description of the nature of these various sports provides the background to the relative epidemiology of rugby related MTBI in comparison with these other sports. A brief overview of the incidence of MTBI in sport will be presented, following which the epidemiology of MTBI in rugby union is discussed and comparisons are drawn between the other team sports described. Knowledge of MTBI epidemiology facilitates the development of adequate preventative measures for reducing the incidence of sport-related MTBI. Preventative measures for sport-related MTBI are briefly discussed at the end of the chapter.

4.1 TEAM SPORTS WITH A HIGH RISK FOR MTBI

In addition to team sports, MTBI occurs in individual sports, including boxing, the martial arts, cycling, horse riding, sky diving, hang gliding, auto racing and motorcycle racing (e.g., Broshek, Brazil, Freeman, & Barth, 2004; Heilbronner & Ravdin, 2004; Ravdin, Barr, Jordan, Lathan, & Relkin 2003; Koh, Cassidy, & Watkinson, 2003; Mendez et al., 2005; Nicoll & Coleman, 1996; Pieter & Zemper, 1998; Tommasone & Valovich McLeod, 2006). Rugby is a team sport, and accordingly the incidence of MTBI in rugby (section 4.2.4) will involve comparisons with other high-risk team sports, rather than high-risk individual sports.

The sports that will now be described, in order to portray their inherent risk for MTBI, are ice hockey, soccer, American football, Australian football, rugby league and rugby union.

4.1.1 Ice Hockey

Ice hockey is played on an oval ice rink with goalnets at either end. Two teams each have six players on the rink during play (Belasco, undated). The object of the game is to score more goals than the other team by hitting a puck, which can travel at speeds of 160 km/h

or more, into the goal net (e.g., Caselli, Gagne, & Kaplan, 2002). The risk of injury is an integral part of ice hockey, which is a fast game involving high speed collisions with the ice, boards around the rink, other players, the puck or a hockey stick (e.g., Biasca, Wirth, & Tegner, 2002; Lovell, Echemendia, & Burke, 2004; Solomon, Johnston, & Lovell, 2005). Over time players began to wear more protective clothing to avoid injury, however the incidence of injury and MTBI has increased in parallel to the improved design of elbow and shoulder pads, inducing more aggressive play and a sense of being invincible (e.g., Biasca et al., 2002; Molsa, Kujala, Nasman, Lehtipuu, & Airaksinen, 2000; Tegner & Back, 2000).

4.1.2 Soccer

Soccer, commonly referred to as football, is played between two teams. Each team consists of a goalkeeper and 10 outfield players. Soccer is played on a pitch, with goalnets at either end. The aim of the game is to score more goals than the opposing team (Soccer Training Guide, undated). Globally, 265 million soccer players are at risk for MTBI due to blocking, tackling, and impact of the ball to the head (e.g., Bailes & Cantu, 2001; Fédération Internationale de Football Association [FIFA], 2007).

A unique feature of soccer is heading the ball, a move that requires the player to strike the ball with the frontal aspect of the head while bracing the neck muscles to minimise the head's acceleration (e.g., Bailes & Cantu, 2001; Sheldon, 2003; Rutherford et al., 2003). Heading the ball is used in both defensive and offensive play, with approximately 12 to 32 headers occurring in a match (Rutherford et al., 2003). As early as 1961 it was posited that heading the ball may result in concussive or subconcussive brain trauma. However, no studies have conclusively supported this assumption (e.g., Barnes et al., 1998; Bernhardt, 2009; Boden, Kirkendall, & Garrett, 1998; Kirkendall & Garrett, 2001; Rutherford et al., 2003; Salinas, Webbe, & Devore, 2009). Accordingly, head to head, head to ground, and head to body contact are the frequent cause of MTBI in soccer (Matser et al., 2004).

4.1.3 American Football (Gridiron)

American football or Gridiron, evolved in America and Canada from other forms of football. It differs in many ways from other forms of football, for example there are grid-like pattern of lines on the field, a helmet and shoulder pads are worn, forward passes are allowed, and it is possible to score both with and without possession of the ball. Each team comprises eleven players, and the game is played on a field with crossbar goalposts at either end. It is estimated that more than 1.5 million people participate in American football annually from recreational to professional levels of play. American Football League is the amateur league and National Football League is the professional league (e.g., Oriard, undated).

4.1.4 Australian Football

Australian football, commonly termed Aussie rules, is a football variant developed in Australia. It is played on an oval field with four goalposts at each end. Australian football comprises two teams of 18 players and 3 substitutes. There are no offside rules and players can move freely around the field, usually in opposing pairs. Players may run with, kick or handball the ball forward, but throwing is disallowed. Players may tackle each other and may run and step up on another player to gain height. MTBI usually occurs from player to player or player to ground contact. More than 385 000 Australian athletes play Australian football (Hinton-Bayre & Geffen, 2004; Hoskins, Chiro, & Pollard, 2003).

4.1.5 Rugby League

Rugby league is played on a field with goals at either end, and comprises two teams, each with 13 players and 4 substitutes. The oval ball must be carried or kicked forward although forward throws are disallowed, and tackling is an integral part of the game. The object of the game is to carry the ball over the try line in order to score a try worth four points, and then to kick the ball over the goals (e.g., Department of Sport and Recreation, 2010; Gibbs, 1993; Oxford University Women's Rugby football Club, undated).

4.1.6 Rugby Union

A history of rugby union is provided, in order to differentiate it from other forms of football. Player positions and the form of play for rugby union provide a comprehensive backdrop to the physicality of the game, and the risk it incurs for sustaining MTBI. Derivative forms of rugby union will be briefly described.

History and Evolution of Rugby Union

Rugby derived from early forms of football played in Britain and Europe. Various forms of football have been played over centuries, despite football having been banned by nineteen English and European Monarchs between 1314-1527. The English 1835 Highways Act banned football on public grounds where games were frequently played and on highways; however, various schools around England continued to play football (Rugby Football History, undated).

According to popular legend, rugby was developed in the English town of Rugby (Australian Rugby Union Limited, 2009; Cuddon, 2004; History of Rugby Union, 2010; Rugby School, 2009; Smith, 1999; Stuart, undated). In 1876, based on the testimony of an unknown third party, Matthew Bloxam (a former Rugbeian) attributed the birth of rugby to Webb Ellis (History of Rugby Union, 2010; Rugby Football History, undated). According to Bloxam, in 1823 a 16-year old Rugby schoolboy, William Webb Ellis, decided to carry and run with the football through the opposing team in order to score, instead of kicking the ball (e.g., Australian Rugby Union Limited, 2009; Cuddon, 2004; History of Rugby Union, 2010; Rugby Football History, undated; Rugby School, 2009; Smith, 1999; Stuart, undated). However, in 1895 the Old Rugbeian Society quashed Bloxam's assertion, because during Webb Ellis's time, most forms of football involved ball handling and no standard football rules existed (History of Rugby Union, 2010). Nevertheless, as a tribute to Ellis, the Rugby World Cup trophy is named the William Webb Ellis Trophy (Rugby Football History, undated; Smith, 1999).

During the 1890s, controversy led to a division between English players in the south and north, because the northern clubs wanted their players paid for loss of income while playing rugby. However, the Rugby Football Union (RFU) voted against compensation for players in 1893 (History of Rugby Union, 2010; Rugby Football History, undated;

Smith, 1999). As a result, in 1895, approximately 20 clubs withdrew from the RFU to form the Northern Rugby Union (NRFU), and the NRFU rules consequently evolved to form those of what is now known as rugby league (e.g., History of Rugby Union, 2010; Rugby Union Rules, undated; Smith, 1999). In 1995, by which time rugby league was already a professional sport, the International Rugby Board (IRB) finally permitted rugby union to become a professional sport worldwide (e.g., Australian Rugby Union Limited, 2009; Cuddon, 2004; History of Rugby Union, 2010; Rugby Union Rules, undated; Smith, 1999; Stuart, undated).

International and South African Rugby Development

During the 1860s to 1880s, British settlers to the Australasian and Southern African regions introduced rugby to these areas. The British colonists first introduced rugby union to the South African Cape Colony in 1875, and thereafter to other parts of South Africa. The first South African rugby club was formed in 1876. In 1883 the Western Province Rugby Football Union was the first South African rugby union to be formed, and in 1889 the South African Rugby Board (SARB) was founded. In 1992, following the apartheid era, the nonracial South African Rugby Union (SARU), and SARB, merged to form the South African Rugby Football Union (SARFU). In 2005 it changed its name to the South African Rugby Union (SARU) (e.g., Currie Cup, 2010; Rugby Union, 2010).

The British rugby team (England) first toured South Africa in 1891, and South Africa first toured the British Isles in 1906-1907. In 1921 the South African rugby team (Springboks) first toured Australia and New Zealand, and the New Zealand (All Blacks) and Australian (Wallabies) rugby teams first toured South Africa in 1928 and 1933, respectively (Australian Rugby Union Limited, 2009; Smith, 1999). Hereafter, the international Rugby World Cup, the Tri-Nations series, and the Super-14 tournaments were established. These tournaments, together with the South African Currie Cup, are briefly reviewed, because several rugby players in this study participated in these international and national matches.

Rugby World Cup Tournament

The Rugby World Cup tournament, organised by the International Rugby Board (IRB), was first played in 1987, and is held every four years. It is the premier competition among men's national rugby union teams, with 20 qualifying national teams competing in a host

country over one month's duration. The winning team is awarded the William Webb Ellis Cup (Rugby World Cup, 2010). In South Africa, rugby union for women was launched in 2001, and a Women's Rugby World Cup is now held, in which twelve countries participate (Shuttleworth-Edwards & Whitefield, 2007a).

The Tri Nations Test Series

Following both the Rugby World Cup and professionalisation of rugby in 1995, South Africa, New Zealand and Australia Rugby (SANZAR) was formed by the rugby boards of these three countries in 1996 (SANZAR, 2009). SANZAR set up both the Tri Nations Test Series (similar to Europe's Six Nations competition) and the Super 12 tournament between their countries (Super 14, 2010). The Tri Nations Test Series is an annual event in which these nations contest one another in rugby union. The first Tri Nations Test Series was held in 1996 (History of Rugby Union, 2010; Tri Nations, 2010).

The Super 14 Tournament

In 1986 the South Pacific Championship was launched in the southern hemisphere, with two teams from Australia, one from Fiji and three from New Zealand participating. In 1992 this competition was renamed the Super 6. In 1993 the Super 10, following South Africa's readmission to the sporting arena, replaced the Super 6. The Super 10 comprised two teams from Australia, four teams from New Zealand, one team from Samoa, and three teams (including the Sharks rugby team) from South Africa. In 1996 SANZAR launched the Super 12 tournament that featured five New Zealand, four South African and three Australian teams. In 2006 SANZAR extended the competition and renamed it the Super 14 (Australian Rugby Union Limited, 2009; History of Rugby Union, 2010; Super 14, 2010). The Super 14, the largest rugby union championship in the southern hemisphere, comprises four Australian, five New Zealand and five South African teams, including the Sharks rugby team from KwaZulu-Natal (History of Rugby Union, 2010).

The Currie Cup: South Africa's National Rugby Competition

When the first team from the British Isles toured South Africa in 1891, they brought with them the golden cup donated by Sir Donald Currie. Sir Donald Currie requested that the cup be presented to the South African team that played the best and most spirited game, after which it was to be a floating trophy for inter-provincial championships in South Africa (SA Rugby, 2009). In the early years provincial winning teams were not awarded

the cup, and due to the two World Wars tournaments were not held annually. However, it was first awarded in 1939. Since 1968 the Currie Cup has been played annually. Currently the Currie Cup rugby tournament involves 14 provincial or sub-provincial South African teams (Currie Cup, 2010; SA Rugby, 2009).

Player Positions in Rugby Union

A rugby union team comprises 15 players, including eight forwards (the pack) who are numbered 1 to 8, and seven backs (the backline), who are numbered 9 to 15. Seven substitute players are allowed in major tournaments (Rugby Union Rules, undated).

The eight forwards include a hooker supported by a loosehead prop and a tighthead prop (front-row forwards), two locks (second-row forwards), two flanks and the eighth man (back-row forwards). The forwards are generally physically strong and larger than the backs, but as the game of rugby has evolved and become more competitive, the back row forwards tend to be faster, more agile and more able to quickly break away from the remaining forwards in order to support the backs with tactical running manoeuvres. The forwards are responsible for gaining and retaining possession of the ball, particularly after the set pieces of the scrum or lineout, and passing it to the backs (e.g., Cuddon, 2004; Rugby Union Rules, undated).

The seven backs include a scrum half, fly half, inside centre, outside centre, a left wing, a right wing and a fullback. The scrum half and fly half are called the halfbacks and the remaining backline the three-quarter backs. The scrum half is responsible for taking the ball following a scrum or lineout and passing it out to the remaining backs. The backs, reliant on their speed and agility, run the ball forward into their opponents' half, in an attempt to place it across the goal line to score a try, although the forwards are also permitted to score (e.g., Cuddon, 2004; Rugby Union Rules, undated).

Form of Rugby Union Play

Rugby Union is played on a marked grass field measuring up to 70 by 100 metres, with H-shaped goalposts at either end of the field. The in-goal area behind each set of goalposts measures 10 to 22 metres. Two teams play rugby union using an oval shaped rugby ball (Rugby Union Rules, undated). A rugby match is played over two halves of 40-minute duration each, however extra time may be added for stoppages or injury time

(e.g., Rugby Union Rules, undated). An on-field referee and two assistant linesmen adjudicate a rugby match (e.g., Cuddon, 2004; Rugby Union Rules, undated).

The essence of rugby is to kick, carry, pass and then ground the ball in order to score points. A try, worth five points, is scored when a player grounds the ball with his hand in the opponents' in-goal area. Following an awarded try, a member from the scoring team kicks for conversion by drop kicking or place kicking the ball over the goalpost's crossbar, which if successful is worth two points. During play, three points can also be earned by a drop goal or penalty goal (e.g., Cuddon, 2004; Rugby Union Rules, undated).

In an attempt to score a try, the team with the ball advances towards the opponents' in-goal area by kicking the ball forward or passing the ball backwards or laterally from one player's hands to another, so that the player catching the ball can run or kick the ball forwards. Simultaneously, the defending team attempts to take possession of the ball by tackling the ball carrier or by a process termed a 'turn over'. These attempts at gaining the ball continue until a rule is violated, or the ball moves out of play, or a player scores either a try or a goal (Cuddon, 2004; Rugby Union Rules, undated). A rugby tackle entails an opponent grabbing the ball-carrying player, usually around the waist or thighs (tackling above the shoulders is forbidden) and bringing him to ground, whereupon the tackled player must pass or release the ball (Cuddon, 2004; Rugby Union Rules, undated). Often following a tackle, a ruck or maul may form. A ruck is formed when the ball is on the ground and one or more players from the respective teams close around the ball while on their feet and in close physical contact. A maul involves the same process as a ruck, but while the ball being held by a player, and ends when the ball goes to ground or emerges from the maul (Cuddon, 2004).

If a player kicks the ball out of play, a member of the opposing team throws the ball back into play, usually via a lineout (Cuddon, 2004). A lineout entails the forwards from each team lining up in parallel, at right-angles to the touchline where the ball went out, and contesting for the ball when it is thrown in straight between the two lines of players. Two team members may lift a player in order to increase his chance of grabbing the ball in the lineout (e.g., Cuddon, 2004; Rugby Union Rules, undated).

If a player mistakenly throws or knocks the ball forward, the referee may order a scrum at that spot. A scrum is formed by the eight forwards from each team (interlocking close to the ground first as team packs of three rows binding together and then as two packs with the opposing teams' front row players' heads interlocking) for the ball to be thrown by the scrum half of the non-penalised team, along the ground and between the two front rows of the team packs. While the two sets of forwards try to push each other backwards, the scrum participants try to hook or heel the ball back through their scrum in order for their team scrum half or eighth man to take the ball and move it out to the backs (e.g., Cuddon, 2004; Scrum (rugby), 2010; Scrum (rugby union), 2010). Due to the physical nature of the scrum, there is a high risk of injury, particularly to the front row players. Therefore a number of rules apply for safety reasons, such as no twisting of one's body, pulling the opposition or collapsing the scrum, and the back row must stay bound until the ball is no longer in the scrum (Scrum (rugby), 2010; Scrum (rugby union), 2010).

Thus, rugby is an extremely physical sport that can involve collisions (e.g., between players physically competing for ball ownership, between players and the ball, and between players and the ground when tackled or jumping high for the ball following a kick) and close physical contact in the scrums, rucks and mauls. The tackling manoeuvre is the most common cause of rugby injury, particularly for the tackled player and among the rugby backs, followed by the rucks and mauls (e.g., Best, 2006; Brooks et al., 2005; Kemp, Hudson, Brooks, & Fuller, 2008; Jakoet & Noakes, 1998; Kerr et al., 2008; Schneiders, Takemura, & Wassinger, 2009; Shuttleworth-Edwards, Border et al., 2004).

Other forms of abridged rugby exist, that appear far less physical than rugby union, and will now be discussed.

Abridged Forms of Rugby Union

Abridged forms of rugby based on rugby union rules include rugby sevens, rugby tens and touch rugby. Rugby sevens has seven players per team, each half is played for seven minutes and it is a fast and wide-open form of rugby (Ombac Rugby, 2010). The IRB holds eight annual events in the IRB Sevens World Series and South Africa hosts one annual event in George. The IRB Rugby World Cup Sevens, for male national sevens teams, is held every four years, with the first being held in 1993 in Scotland, and for which the winning team is awarded the Melrose Cup (Rugby World Cup Sevens, 2010).

Rugby tens also follows the same basic rugby union rules, but has ten players per team and each half is played for ten minutes (RFU, 2010). Rugby Tens (also known as Xs or ten-a-side) originated in Malaysia. The team consists of five forwards and five backs but, unlike rugby union and rugby sevens, the IRB has not published rules for this rugby variant (Rugby tens, 2010).

Touch rugby began in Australia in the 1960s, as a pre-rugby warm-up game. A team consists of up to 14 players with only six on the field. Each half is usually ten minutes duration, although players can vary the length of play. The game is usually played on half a rugby pitch. The rules are similar to those of rugby, but preclude kicking, tackling, scrumming, lineouts, rucks or mauls, and it is therefore a game of running and ball handling. When the ball carrier is touched by an opponent, the ball carrier has to stop, return to the mark where touched, and perform a rollball. A rollball involves rolling the ball backwards along the ground, so that a player in the opposing team can pick it up. A touchdown earns one point (International Touch Rugby, 2008).

Having described the nature of various high-risk contact team sports, and with a more detailed exposé on rugby union, the epidemiology of TBI, MTBI and specifically MTBI in rugby union in comparison with these other team sports will be discussed.

4.2 EPIDEMIOLOGICAL INCIDENCE OF MTBI

The difficulty in establishing epidemiological statistics is that differing sources, terminology and methodologies are used for calculating prevalence, incidence rates and risks, and often these figures are based on small sample sizes, resulting in overinterpretation of results (e.g., Corrigan, Selassie, & Orman, 2010; Knowles, Marshall, & Guskiewicz, 2006). Prevalence refers to the proportion of individuals in the population who have an injury at a specific time, while incidence refers to the number of new injuries during a specific time period. Epidemiological incidence should ideally be “the number of injured athletes divided by the number of athletes at risk” (Knowles, Marshall, & Guskiewicz, 2006, p. 207), while the incidence rate should be “the number of injuries divided by the number of athlete-exposures” (Knowles at al., 2006, p. 207). However, the literature is unclear on how incidence figures, risk of injury and the prevalence results are derived. They will therefore be reported in this chapter according to the manner in which

they are reported in the literature. Initially the overall incidence of TBI and MTBI will be provided in order to contextualise the incidence of sport-related MTBI.

4.2.1 Epidemiology of TBI in General

Overall, in industrialised countries, TBI is the most common cause of death and disability in young adults, from which 180-250 people per 100 000 die or are hospitalised annually (Behan et al., 2008).

In the USA 1.7 million TBI cases attended emergency departments (EDs) annually between 2002 and 2006, of which almost 80% were not hospitalised, and TBI played a contributing role in 30.5% of all injury-related deaths (e.g., CDC, 2010; Faul, Xu, Wald, & Coronado, 2010; Kelly, 2010). Almost half of these ED visitors were children aged below 15 years, due to this age group becoming more active in sport and due to increased public awareness that TBI is treatable (e.g., CDC, 2010; Faul et al., 2010; Kelly, 2010; NCIPR, 2009). The CDC report included data only from EDs, and therefore the figures are a conservative estimate of the true incidence of TBI in the USA (e.g., CDC, 2010; Faul et al., 2010; Kelly, 2010). The prevalence of Americans living with a residual disability following TBI hospitalisation is 3.2 million (Corrigan et al., 2010). Severe forms of TBI have been evidenced in both recreational and organised sport, and 68 fatalities or injuries of a catastrophic nature were reported among USA high school sports between 1983 and 1997 (Bailes & Cantu, 2001).

In the United Kingdom, the Fife Headway Group (2009) estimate that 1 million people attend an ED annually as a result of TBI, of which approximately 85% of cases are MTBI, 10% moderate TBI, and 5% severe TBI. Half a million people in the UK, aged 16-75 years, live with long-term disabilities resultant of TBI (Fife Headway Group, 2009).

4.2.2 Epidemiology of MTBI in General

Approximately 75 to 90% of total TBIs are MTBIs (e.g., Anderson & Murata, 2009; Bay & McLean, 2007; Bazarian et al., 2005; Crippen, 2009; de Kruijk, Leffers, Menheere et al., 2002; Department of Veteran Affairs, 2009b). The true incidence of MTBI is uncertain as it is often unrecognised, unreported or excluded from statistics, and it is

estimated that 25% of cases do not seek medical attention (e.g., Bay & McLean, 2007; Petchprapai & Winkelman, 2008; Shah, Bazarian, Mattingly, Davis, & Schneider, 2004). Non-recognition of MTBI is supported by findings of an anonymous survey of patients admitted to an ED for MTBI, in which 88.6% of respondents did not recognise that they had suffered MTBI, and 28.2% were engaging in at least one activity that posed a high risk for repeat MTBI (Delaney et al., 2005).

In relation to the USA there is a wide discrepancy in the incidence of MTBI, varying between 200 to 503.1 cases per 100 000 and ranging from 1 million to 3.8 million cases per annum (e.g., Bazarian et al., 2005; Bender et al., 2004; Biotech Week, 2009; Jennett & Frankowski, 1990; Langlois et al., 2006; Petchprapai & Winkelman, 2008; Thurman & Guerrero, 1997).

MTBI is particularly common among individuals in their teens and twenties, is twice as common for males than for females (although this gender difference decreases with age), and although MTBIs may be caused from falls, traffic accidents or violent acts, they are often caused through sport (e.g., NCIPR, 2009; Peloso, von Holst, & Borg, 2004; Terrell, 2004; van der Naalt, 2001). Incidence of MTBI in sport will now be discussed

4.2.3 Epidemiology of MTBI in Sport

Most sports, whether individual or team sports, have a risk for MTBI as a result of high-speed contact with the ground, equipment, objects including balls, as well as with other athletes' bodies and heads (e.g., Bailes & Cantu, 2001; Dvorak, McCrory, Aubry, Molloy, & Engebretsen, 2009; Guskiewicz et al., 2000). It is estimated that 3 to 25% of total TBIs are sport-related, that MTBI accounts for over 75% of all sport-related brain injury, and worldwide 5 to 19% athletes are at risk for sustaining MTBI annually (e.g., Anderson, Schnor, Schroll, & Hein, 2000; Matser et al., 2004; Manko, 2003; McManus, 2006; Sallis, Sunshine, & Simon, 2001; Pretz, 2007).

In the USA, the overall incidence rate of sport and recreation-related MTBI has been estimated to range between 300 000 and 3.8 million persons annually (e.g., Langlois et al., 2006; Terrell, 2004). The high-risk sports have been identified as boxing, football, ice hockey, men's lacrosse, wrestling, rugby and soccer (e.g., Bernhardt, 2009; Covassin et

al., 2003; McGuine, 2006; Terrell, 2004). Over a 16-year period (1988/1989 - 2003/2004), a significant 7% annual increase in MTBI incidence for 15 sports is reported to indicate an improvement in MTBI recognition (e.g., Hootman, Dick, & Agel, 2007).

With reference to USA minors in general and based on data surveys between 2001-2005, one study reported 207 830 sport-related TBIs were treated at a particular ED of which 135 000 cases were aged 5-18 years (CDC, 2007), while another study reported 251 000 sport-related MTBIs were sustained by 8 to 19 year olds over this period (Bakhos, Lockhart, Myers, & Linakis, 2010). Younger, junior athletes playing contact sports are twice as likely to sustain MTBI than their older, senior counterparts (e.g., Cantu, 1992; Iverson et al., 2004a; CDC, 2007; McCrory, Berkovic et al., 2000; McManus, 2006).

Females are considered more vulnerable than males for sustaining sport-related MTBIs, particularly during games as opposed to during practice, with football being the lead cause for males and soccer for females in the USA (e.g., Covassin et al., 2003; Gessel, Fields, Collins, Dick, & Comstock, 2007; McKeever & Schatz, 2003; Powell & Barber-Foss, 1999). Bernhardt (2009) reports that studies of high school athletes have found incidence rates of MTBI per 1 000 exposures as follows: 0.59 for football (boys), 0.25 for wrestling (boys), 0.18 for soccer (boys), 0.23 for soccer (girls), 0.09 for field hockey (girls), 0.11 for basketball (boys), and 0.16 for basketball (girls). Direct impact to the head causes more than half sport-related MTBIs, particularly as a result of rotational and angular forces (e.g., Barth et al., 1989; Mendez et al., 2005).

Research of sport-related MTBI incidence for the UK appears limited or outdated with more focus on general injuries rather than MTBI specifically (e.g., Kerr et al., 2008; Lindsay, McLatchie, & Jennett, 1980; Nicoll & Coleman, 1996). Statistics from South Africa's General Household survey, carried out in July 2008 (The Times Newspaper, Thursday September 3, 2009) reported South Africa's latest estimate of population as 48.7 million people. Extrapolating that MTBI occurs in approximately 500 cases per 100 000 individuals (Bazarian et al., 2005), of which over 75% are sport-related brain injuries (Anderson et al., 2000; McManus, 2006), then it could be approximated that over 182 625 cases of sport-related MTBI occur annually in South Africa.

Overall, incidence figures of sport-related MTBI are grossly under-estimated for several reasons. These include varied definitions and criteria for MTBI resulting in non-recognition, non-reporting by athletes and sport personnel, non-referral of at least 50% of MTBI cases, and inadequate surveillance (e.g., Bailes & Cantu, 2001; Bender et al., 2004; Bernhardt, 2009; Bigler, 2008; Covassin et al., 2003; Cunningham, 2007; Delaney et al., 2005; Delaney, Al-Kashmiri, Drummond, & Correa, 2008; Field et al., 2003; Gerberich et al., 1983; Kaut et al., 2003; Kelly et al., 1991; Kontos et al., 2004; LaBotz et al., 2005; Lovell, Bradley et al., 2003; Martineau et al., 2007; McCrea, Hammeke et al., 2005; McCrea et al., 1997; Pretz, 2007; Roux et al., 1987; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards, Noakes et al., 2008; Shuttleworth-Edwards & Radloff, 2008; Tommasone & Valovich McLeod, 2006; Valovich McLeod, Heil, McVeigh, & Bay, 2006).

4.2.4 Epidemiology of MTBI in Rugby Union

Rugby union is an extremely popular sport worldwide, second only to soccer, and is played in over 150 countries by an estimated four million athletes of all ages, although most are males (e.g., Best, 2006; Finch, Best, McIntosh, Chalmers, & Eime, 2002; Kerr at al., 2008; Kohler, 2004; MacQueen & Dexter, 2010; Micheli & Riseborough, 1974; Nicol et al., 2010; Shuttleworth-Edwards, Noakes et al., 2008; Shuttleworth-Edwards, Smith et al., 2008). Due to the number of bodily collisions associated with tackling, rucks, mauls, scrums, speed of play, competitiveness, and the relatively large build of these athletes, rugby is considered a dangerous sport (e.g., Briscoe, 1985; Hillis, McIntyre, Maclean, Goodwin, & McKenna, 1994; Jakoet & Noakes, 1998; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007a; Wekesa, Asembo, & Njororai, 1996). In order to provide an idea of the physicality of this collision sport, the overall incidence of injuries is discussed below.

Epidemiology of General Rugby Injuries

General injuries are more common among senior than junior divisions of rugby and the incidence increases among youths as age increases (Bottini, Poggi, Luzuriaga, & Secin, 2000; Brooks et al., 2005; McIntosh et al., 2010; Nicol et al., 2010). Therefore, in terms of 1 000 player game hours, rugby injury rates have been reported as: 10.8 for males and 22.5 for females at high school; 16.9 for males and 17.1 for females at collegiate level; 52

for premier club male players; 91 for male professional rugby players; and 37.5 for female world cup players (e.g., Brooks et al., 2005; Kerr et al., 2008; MacQueen & Dexter, 2010; Nicol et al., 2010; Schick, Molloy, & Wiley, 2008; Schneiders et al., 2009). General injury incidence rates for rugby are higher than those of other forms of football among high school athletes (e.g., Briscoe, 1985; Junge, Cheung, Edwards, & Dvorak, 2004; Nathan et al., 1983; MacQueen & Dexter, 2010; Shuttleworth-Edwards, Border et al., 2004). At adult non-professional level it would appear rugby and Australian football share a similar injury incidence (e.g., Braham, Finch, McIntosh, & McCrory, 2004; Gabbe, Finch, Wajsweiner, & Bennell, 2002; McMahon, Nolan, Bennett, & Carlin, 1993; Orchard, Wood, Seward, & Broad, 1998; Shawdon & Bruckner, 1994; Stevenson, Finch, Hamer, & Elliott, 2003).

Professional rugby union players evidence up to 50% more injuries than rugby league players, and the high number of injuries sustained over the 1995 and 2003 Rugby World Cups indicate that superior skill, experience and fitness of professional rugby union players are not necessarily protective factors against injury (e.g., Best, McIntosh, & Savage, 2005; Garraway, Lee, Hutton, Russell, & Macleod, 2000; Gibbs, 1993; Jakoet & Noakes, 1998; Shuttleworth-Edwards, Border et al., 2004).

The tackling manoeuvre is the most common cause of rugby injury in rugby league and rugby union, and rugby union tackles account for almost 33 to 62.1% of male injuries and 63.6% of female injuries. The manoeuvre is more hazardous for the tackled player than the tackler and for backs versus forwards, followed then by the ruck and maul forms of play (e.g., Best, 2006; Bird et al., 1998; Brooks et al., 2005; Collins, Micheli, & Yard, 2008; Finch et al., 2002; Gabbett, 2003; Kemp et al., 2008; Garraway & MacLeod, 1995; Jakoet & Noakes, 1998; Kerr et al., 2008; McIntosh, 2005; Nathan et al., 1983; Nicol et al., 2010; Schick et al., 2008; Schneiders et al., 2009; Shuttleworth-Edwards, Border et al., 2004).

Regarding position of play, injuries are more prevalent among the forwards, particularly among the hookers and locks, versus the backs (e.g., Bird et al., 1998; Bottini et al., 2000; Brooks et al., 2005; Davies & Gibson, 1978; Lingard, Sharrock, & Salmond, 1976; Jakoet & Noakes, 1998; Nathan et al., 1983; Seward, Orchard, Hazard, & Collinson, 1993; Sharp, Murray, & Macleod, 2001; Shuttleworth-Edwards, Border et al., 2004;

Shuttleworth-Edwards & Radloff, 2008). The reason for this is that the forwards are in more situations of contact and are usually taller and heavier than the backs, although this profile is beginning to change among elite players with forwards attaining higher muscularity (Duthie, Pyne, & Hooper, 2003; Brooks et al., 2005; Nathan et al., 1983). Cervical spinal injuries, among the most serious rugby injuries, are common among front row forwards from scrummaging and from tackles (e.g., MacQueen & Dexter, 2010; Quarrie, Cantu, & Chalmers, 2002; Silver, 1984).

Head and face injuries account for 14 to 27% of all rugby injuries (Best, 2006). The high incidence of neck and head injuries for rugby union varies between 25 to 52% which represents a substantially higher incidence than found in ice hockey, rugby league, Australian football and soccer (e.g., Agel, Dompier, Dick, & Marshall, 2007; Junge et al., 2004; MacLeod, 1993; Myers, 1980; Nathan et al., 1983; Roux et al., 1987; Roy, 1974; Ryan & McQuillan, 1992; Seward et al., 1993; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards, Noakes et al., 2008; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007a).

Epidemiology of Rugby-related MTBI

The definition of MTBI, the methodology of data collection, and the reporting measures pertaining to MTBI incidence and epidemiology vary widely between sport studies. Therefore comparisons between the studies and sports are somewhat impeded.

Apart from two rugby union studies in which the incidence of MTBI was relatively uncommon (e.g., Bottini et al., 2000; Durkin, 1977), MTBI is considered one of the most common rugby union injuries (Bird at al., 1998; Nathan et al., 1983; Roux et al., 1987). Among rugby studies, MTBI accounts for 11 to 35.9% of total injuries (e.g., Collins, Micheli, & Yard, 2008; Kohler, 2004; McIntosh, 2005; McIntosh et al., 2009, 2010; Myers, 1980; Nathan et al., 1983; Nicol et al., 2010; Roux et al., 1987; Shuttleworth-Edwards, Smith et al., 2008; Sparks, 1981). This is comparable to the other sports, in which of total injuries MTBI accounts for 2 to 20% for ice hockey and soccer (e.g., Agel et al., 2007; Bailes & Cantu, 2001; Biasca et al., 2002; Cantu, 2001; Kirkendall & Garrett, 2001; Powell & Barber-Foss, 1999; Tegner, & Lorentzon, 1996), 24% for American football (Maroon et al., 2000; Powell & Barber-Foss, 1999), and 1.8 to 8.5% for rugby league (King, Hume, Milburn, & Gianotti, 2009; King, 2010; Seward et al., 1993).

Two reviews of male high school team sports (that among other sports included rugby union, football, soccer and ice hockey), found ice hockey had the highest incidence of MTBI, however at a professional level it was reported that ice hockey and rugby shared a similar incidence, and one review reported the MTBI incidence for professional ice hockey as 6.5 per 1 000 player games and 9.05 per 1 000 player games for professional rugby (e.g., Koh et al., 2003; Tommasone & Valovich McLeod, 2006). Sport-related MTBI incidences reported by Cassidy et al. (2004) are given as up to 5.0 per 1 000 game hours and 17.1 per 1 000 athlete exposures for rugby union compared with ice hockey (17.6 per 1 000 game hours), rugby league (8.0 per 1 000 game hours), Australian football (3.3 per 1 000 athlete hours), American football (3.3 per 1 000 athlete exposures) and soccer (1.3 per 1 000 game hours). This suggests that ice hockey and rugby league have higher MTBI incidences than rugby union.

Generally, the incidence of MTBI among rugby union increases as age, level and competency in play increases, and therefore incidences will be discussed below in terms of level of play (Bathgate, Best, Craig, & Jamieson, 2002; Jakoet & Noakes, 1998; Lingard et al., 1976; Nathan et al., 1983; Seward et al., 1993; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards, Noakes et al., 2008; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Jordan et al., 1993).

Findings for high school level indicate seasonal MTBI incidence rates of 4 to 22% for rugby union (e.g., Junge et al., 2004; Nathan et al., 1983; Roux et al., 1987; Shuttleworth-Edwards, Noakes et al., 2008). In three studies, the schools monitored via correspondence rather than by the researchers directly or that were not strongly motivated to monitor MTBI, evidenced lower MTBI rates of 4% and 12% compared with schools strongly involved in MTBI management, which evidenced rates of 14% and 22% (Roux et al., 1987; Shuttleworth-Edwards, Noakes et al., 2008; Sparks, 1981). Other findings for high school rugby include a history averaging 2.3 (0-7 range) MTBI incidents per rugby player (see review in Shuttleworth-Edwards, Border et al., 2004). Junge et al. (2004) reported MTBI incidence rates of 1.45 per 1 000 hours exposure (6.9 per 1000 game hours) for rugby union compared to the lower MTBI incidence for high school soccer of 0.18 per 1 000 athlete exposures (e.g., Koh et al., 2003; Tommasone & Valovich McLeod, 2006).

Findings for university level rugby union indicate a seasonal MTBI incidence rate of 3 to 24.6% (e.g., Marshall & Spencer, 2001; Shuttleworth-Edwards, Noakes et al., 2008; Shuttleworth-Jordan et al., 1993; Wills & Leatham, 2001). Marshall and Spencer (2001) found a MTBI incidence rate of 3.8 per 1000 athlete exposures (11.3 per 100 player seasons) among university rugby players.

Findings for club level rugby union indicate a seasonal MTBI incidence rate of 5 to 30% (Shuttleworth-Edwards, Noakes et al., 2008; Wills & Leatham, 2001), which is higher than the 10.1% seasonal incidence for Italian club soccer players (Broglio et al., 2010). Among Australian nonprofessional rugby players the seasonal MTBI incidence was 18% or (7.97 per 1 000 game hours) and those wearing protective headgear were at a reduced risk of sustaining MTBI (Hollis et al., 2009). Another study found headgear and mouthguard use associated with a lower risk of MTBI among 13 English premiership rugby union clubs over three seasons, with a MTBI incidence of 4.1 per 1 000 game hours (Kemp et al., 2008).

Overall, the seasonal incidence among high school level (4 to 22%) and university level (3 to 24.6%) for rugby union is higher than that reported in American football, being 3 to 18% for comparative levels of play (e.g., Barr & McCrea, 2001; Dick, 2003; Echemendia et al., 2001; Collins, Grindel et al., 1999; Erlanger, Kaushik et al., 2003; Gerberich et al., 1983; Guskiewicz, McCrea et al., 2003; Guskiewicz et al., 2000; Macciocchi et al., 1996; Maroon et al., 2000; McCrea, 2001; McCrea et al., 1997, 1998, 2002, 2003; McCrea, Hammeke et al., 2005; Powell & Barber-Foss, 1999). However, a survey by Langburt, Cohen, Akhthar, O'Neil and Lee (2001) found a comparatively higher MTBI incidence of 47.2% for high school American football players, however only 52% of surveys were returned which could account for skewed results.

Findings for the professional level indicate a seasonal MTBI incidence rate of 20 to 23% for rugby union, compared with 2 to 8% for NFL professional ice hockey and 8% for professional rugby league (e.g., Biasca et al., 2002; Hinton-Bayre, Geffen, & Friis, 2004; Kohler, 2003; Shuttleworth-Edwards, Noakes et al., 2008). Between 1985 and 2000, rugby demonstrated the highest incidence of MTBI among team male professional sports with an incidence of 9.05 per 1 000 player games (Tommasone & Valovich McLeod, 2006). In contrast Brooks et al. (2005) reported a MTBI incidence of 4.4 per 1 000 player

hours, being more frequent among backs versus forwards - i.e., 4.9 versus 4.0 per 1 000 player hours - among professional English professional rugby players.

For American football, Rochester, Deutsch, Nicholas and Lowery (2006) found 52% of MTBIs occurred during games, versus 48% during practices. In contrast Sebastianeli (2006) found that more than double the number of MTBIs occur in college American football practices than in games, as a result of more athletes being on the field, skills mismatch and physical mismatch. However, there appears to be no rugby union studies directly comparing MTBI incidences between games and practices. The only study found reporting MTBI incidence rates for female rugby union players is that of Schick et al. (2008), in which the incidence rate was 0.73 per 1 000 player hours during the 2006 Women's Rugby World Cup.

In summary, ice hockey, rugby league and rugby union have the highest incidence of MTBI among the contact team sports. At adult and professional level, which is the level of play that this study focuses on, rugby union evidences the highest incidence of MTBI among all the contact team sports.

Having described the nature of the various contact team sports, followed by the epidemiology of MTBI in rugby union in comparison with these other team sports, it is clear that the incidence of MTBI is extremely high among these sports, notably rugby union. It is, therefore, imperative that preventative measures for reducing the incidence of sport-related MTBI be implemented, and these measures will be discussed.

4.3 PREVENTATIVE MTBI MEASURES

The primary goal of MTBI prevention is to reduce its incidence and the associated adverse neuropsychological sequelae that may impact negatively on scholastic or work performance and attendance, and on health care costs (Collins, Lovell, & Mckeag, 1999; Kontos et al., 2004; McManus 2006). Preventative measures that may help reduce sport-related MTBI or the negative consequences, and which will be briefly discussed, include training sport coaches about the recognition and management of MTBI and in implementing comprehensive MTBI protocols, educating athletes and parents about MTBI, encouraging athletes to wear mouthguards and helmets for sports where such measures have been found effective, reviewing sports' rules, and the need for coaches to

train athletes in ways that reduce the risk of MTBI (e.g., Sportex, 2009; Tommasone & Valovich McLeod, 2006).

4.3.1 Training Sports Coaches and Management

Not all sports coaches and management recognise or manage sport-related MTBI appropriately. Studies in Australia and the USA show that a number of coaches and sport managers cannot identify MTBI symptoms, would allow players to return to sport while symptomatic or neurocognitively impaired, have inconsistent and subjective approaches to MTBI management, and do not follow standardised MTBI management procedures (e.g., Broglio et al., 2010; Elbin, Stiller-Ostrowski, & Kontos, 2009; McManus, 2006; Notebaert & Guskiewicz, 2005; Valovich McLeod, Schwartz, & Bay, 2007). It is therefore critical to educate or make it mandatory for sports coaches to recognise and manage MTBI (e.g., Snow, 2010). Accordingly, comprehensive MTBI protocols that should be introduced for contact sports include: (i) sideline evaluation protocols to establish whether or not an athlete is concussed and should be removed from play, (ii) neuropsychological testing, involving baseline measures against which post MTBI measures can be assessed, and (iii) protocols that coaches and medical staff should abide by, following MTBI. These include the athlete's removal from sport, referring an athlete for medical evaluation if MTBI is suspected, ensuring the athlete's abstinence from sport until recovered, and reintroducing the athlete to sport through step-wise return to play measures once the physician has cleared the athlete as symptomatically and neurocognitively recovered (e.g., Cantu, 2001; Powell, 2004; McCrory et al., 2009).

4.3.2 Educating Athletes and Parents about MTBI

The diagnosis and management of MTBI is a challenge in that athletes and parents often do not recognise MTBI or its symptoms and misunderstand the consequences of MTBI (e.g., Broglio et al., 2010; Meehan & Bachur, 2009; Provvidenza & Johnston, 2009; Silver et al., 2009). For example, studies pertaining to rugby union found only half of the first team players were aware of return to play guidelines, almost half the parents were unaware of return to play guidelines, 17% of parents had no knowledge of or would not recognise MTBI, and 4% were unaware of the risks of premature return to play following MTBI (e.g., Sullivan et al., 2009; Sye, Sullivan, & Mc Crory, 2006). Athletes who are

educated about the physiology, neurocognitive deficits, signs and symptoms of MTBI, and long-term consequences of MTBI if not properly managed, are more likely to recognise and voluntarily report MTBI, heed medical advice and suffer fewer negative MTBI outcomes (e.g., Borg et al., 2004; Cunningham, 2007; Kontos et al., 2004; Provvidenza & Johnston, 2009; Quarrie, Gianotti, Hopkins, & Hume, 2007).

Similar programs to the "Lystedt Law" signed in Washington in May 2009 should be introduced among sporting bodies, whereby there is collaboration in formulating guidelines and informational forms to educate athletes, parents and coaches on brain injury; where parents and athletes are required to sign informed consent acknowledging the risk of sport-related MTBI; where athletes suspected of sustaining MTBI are removed from play, and clearance from a licensed health care provider is required prior to return to play (Brain Injury Association of Washington, 2009). A proactive stance in New Zealand involved Brain Injury NZ disseminating information about MTBI in sport and supplying the Sideline MTBI Checklist for MTBI identification, with the symptoms listed and advice on MTBI management provided (e.g., Gianotti & Hume, 2007). Rugby injury prevention and concussion management education programs, effectively reduced MTBI incidence in New Zealand rugby union by 10%, between 2003 and 2005 (e.g., King, Hume, Milburn, & Gianotti, 2009; Quarrie et al., 2007). In 2009 the New Zealand Rugby League (NZRL) distributed RugbySmart to all rugby clubs for coaches and referees to hold information sessions (Brain Injury New Zealand, 2009).

4.3.3 Athletes Wearing Mouthguards

Wearing mouthguards may reduce the risk of both intraoral and concussive injuries (Bailes & Cantu, 2001). Although not scientifically validated, the correct fit and type of mouthguard may decrease the severity of orofacial injuries and MTBI, in reducing the force transmitted by a blow to the jaw (Sports Dentistry Online, 2009). Wearing mouthguards is recommended for ice hockey, baseball and karate to prevent orofacial and dental injury (e.g., Kujala et al., 1995; McCrory et al., 2009). Nathan, Goedeke and Noakes (1983) proposed that wearing mouthguards be compulsory for rugby players to reduce the incidence of MTBI, and in New Zealand the practice is compulsory for all rugby players below international level (Junge et al., 2004). Two systematic reviews, one on rugby union, found no strong evidence that mouthguards reduce MTBI risk

(Cusimano, Nassiri, & Chang, 2010; Hamilton, Meeuwisse, McCrory, & Dvorak, 2009). Furthermore, studies have not found significant differences in MTBI incidence rates between wearing a specific type of mouthguard versus other types of mouthguards in rugby and football; nor between neurocognitive scores post MTBI for those athletes wearing mouthguards and those not doing so (e.g., Barbic, Pater, & Brison, 2005; Mihalik et al., 2007). In contrast, the decrease in MTBI incidence among football players correlated with the increased use of high performance, layered mouthguards (Rochester et al., 2006).

4.3.4 Athletes Wearing Headgear

Controversy exists about the merits and demerits of wearing protective headgear. While the outer case of helmets may spread the impact force over a greater surface area and the inner lining dissipates the acceleration forces, protective headgear may actually be hazardous in that the increased surface surrounding the head may increase the force of angular acceleration, and poorly fitting headgear may provide an additional risk factor for sustaining MTBI (e.g., Bailes & Cantu, 2001; Barth et al., 1989; Bernhardt, 2009; Delaney et al., 2008). Furthermore, protective gear can result in an increase in injures, including MTBI, as a result of risk compensation whereby the athlete in belief of injury resilience embarks on more dangerous forms of play (McCrory et al., 2009). Benson, Hamilton, Meeuwisse, McCrory and Dvorak (2009) reviewed 51 studies and found evidence that helmets reduce head injury in bicycling, skiing and snowboarding, and full facial protection may reduce MTBI severity in ice hockey. However, the effect on MTBI risk was inconclusive. Padded head gear in rugby union does not reduce MTBI or head injury incidence and compliance is a limitation (McIntosh et al., 2009). Cusimano and colleagues (2010) in a systematic review of rugby union studies found limited evidence of headgear effectively reducing neurologic injuries. In contrast, soccer players wearing headgear experienced less symptoms and fewer repeat MTBIs in one year compared to those who did not (Delaney et al., 2008). Furthermore, American football athletes wearing the new Revolution helmet had a 31% decreased risk of MTBI relative to standard helmets, and a 2.3% decreased absolute risk of MTBI (Collins et al., 2006). The CISG panel producing the 3rd International MTBI in Sport consensus statement recognises that biomechanical studies show that headgear reduces impact forces, but that this has not necessarily transferred into a reduced MTBI incidence. The CISG panel

recommends the use of protective helmets in alpine sports, cycling, equestrian and motor sports (McCrory et al., 2009).

4.3.5 Review of Sports Rules

Rules of sports should be reviewed as a means of preventing MTBI (Anderson, 1996). Rugby union is associated with severe cervical spinal injuries, and rules of rugby were changed in New Zealand, Britain, Australia and South Africa between 1979 and 1990 in order to reduce the incidence of spinal cord injuries (e.g., Noakes & Jakoet, 1995; Rotem et al., 1998; Roux et al., 1987; Scher, 1997, 1987). In 2005, the spearing tackle in American football that resulted in MTBI and catastrophic cervical spine injuries was made illegal. Discouraging American football players from leading with their heads has led to a significant reduction of severe head and cervical injuries (e.g., Bernhardt, 2000, 2009; Pelletier, 2006). The CISG panel producing the 3rd International MTBI in Sport consensus statement, recognises the importance of rule changes when particular aspects of play are dangerous, and viewed rule enforcement as critical in reducing MTBI risk (McCrory et al., 2009). Accordingly, sporting authorities such as IRB, FIFA and SARU, need to remain vigilant regarding on-field positions at risk for MTBI, and to evaluate which manoeuvres or forms of play increase the risk for MTBI, and to modify rules of play accordingly.

4.3.6 Responsibility of Coaches

Coaches need to teach athletes sound sport techniques and strategies, for example improved tackling and falling techniques that reduce injuries and MTBI, particularly among player positions and ages at most risk (e.g., Nathan et al., 1983; Powell, 2001). Since finding that almost twice the force is required to concuss a fixed head than that required to concuss a mobile head, and that the degree of head rotation on impact may correlate with MTBI severity, it has been proposed that strengthening the athlete's neck muscles may help increase head stabilisation to prevent MTBI or whiplash injury (e.g., Anderson, 1996; Sports Dentistry Online, 2009). Pre-season neck strengthening exercises are recommended for rugby hookers, particularly at schoolboy level, as these players are at risk of injury in the scrummage (Nathan et al., 1983). Coaches should see that their players are match fit, especially after breaks from the sport, and ensure that players are

not overly ‘psyched-up’ to win matches, in that this may explain high injury incidence, particularly in the more elite teams (Nathan et al., 1983). Coaches and referees need to ensure that sport rules are adhered to, remind players that these rules are in place for their safety, and should encourage fair play. Furthermore violence - as opposed to competitiveness - in sport, needs to be discouraged (McCrory et al., 2009). This is an important factor in rugby, considering that measures of aggressiveness among professional rugby players have been associated with unsanctioned intention to cause pain to other rugby players (Maxwell & Visek, 2009).

4.4 OVERVIEW

This chapter reviewed the team sports that have a high incidence of MTBI, including ice hockey, soccer, American football, Australian football, rugby league and rugby union. The incidence of TBI and MTBI in general were discussed as a background for the general overview of sport-related MTBI. The epidemiology of rugby-related MTBI specifically, was treated and compared with the incidence of MTBI in other high-risk sports. The high incidence of sport-related MTBI needs to be reduced and appropriate preventative measures were discussed accordingly.

CHAPTER FIVE: ASSESSMENT AND MANAGEMENT OF MILD TRAUMATIC BRAIN INJURY IN SPORT

The focus of this chapter is on the medical and neuropsychological assessment and management of sport-related mild traumatic brain injury (MTBI). Medical assessment will be discussed in terms of sideline evaluation, medical evaluation and investigations including balance testing, biomarkers, neuroimaging and electrophysiological evaluation. This will be followed by a discussion of the general medical management of MTBI and decisions regarding return to play.

As discussed in Chapter 3, neuropsychological consequences, including neurocognitive deficits and symptom complaints, occur following MTBI. Therefore the main focus of this chapter will be on the neuropsychological assessment of sport-related MTBI sequelae. Aspects of neuropsychological assessment that will be reviewed, include general assessment issues and neurocognitive assessment tests typically used in sport-related MTBI. Traditional paper and pencil tests commonly used in sport-related MTBI neurocognitive assessment will be discussed, including the traditional Wechsler Memory Scale - Third Edition (WMS-III) Visual Reproduction and Verbal Paired Associates subtests, which are used in this study. Commonly used computerised tests will then be discussed, including the Immediate Post-concussion Assessment and Cognitive Testing (ImPACT) program, that is also used in this study. Finally, the neuropsychological assessment of postconcussive symptoms will be covered in terms of the traditional and computerised tests, notably the ImPACT symptom scale used to measure symptoms commonly occurring in sport-related MTBI.

5.1 MEDICAL ASSESSMENT AND MANAGEMENT OF SPORTS MTBI

The assessment and management of MTBI is multidisciplinary and integrates medical evaluation and neuropsychological assessment (Echemendia & Cantu, 2003). Based on clinical and research evidence, assessment in multiple domains is advocated, and includes clinical assessment (incorporating sideline assessment, balance testing and neuroimaging in certain instances) and the assessment of neurocognitive functioning and symptom reports (e.g., Guskiewicz et al., 2001; Iverson, 2007; McCrea, Barr et al., 2005; McCrea et al., 2003; Mrazik et al., 2000; Oliaro, Anderson, & Hooker, 2001; Peterson et al.,

2003). Consequently, medical management will first be discussed, followed by neuropsychological management. Sideline evaluation assesses neuropsychological responses immediately following an impact, suspected to have caused a MTBI at a sports event. The instruments employed have generally been devised by or had input from neuropsychologists, for use by sports coaches, first aid attendants, physicians, or other health professionals such as nurses or physiotherapists, and are not formal psychometric instruments (e.g., Shuttleworth-Edwards, 2008). Sideline evaluation is typically the first assessment immediately following MTBI, and therefore will be discussed first, under the broad rubric of overall medical management.

5.1.1 Sideline Evaluation of MTBI

As with TBI, treatment of athletes with MTBI begins at the time of impact (Stiver & Manley, 2008). Following an impact, should an athlete exhibit any MTBI characteristics on-field, then spinal injury should be ruled out and the athlete safely removed from the field, following which the athlete should be medically evaluated and assessed using a sideline evaluation protocol (McCrory et al., 2009). Sideline evaluation is a crucial aspect of MTBI assessment. It is used to ensure that an athlete with MTBI is not immediately returned to play and placed at risk of sustaining a further MTBI. Ideally, coaches or medical personnel should use one of the sideline evaluation protocols designed to assess the presence of MTBI at the time of injury, notably the Standardized Assessment of Concussion (SAC), the Sport Concussion Assessment Tool 2 (SCAT2) and Sideline ImPACT. These are described below.

The SAC is a measure of mental status that assesses orientation, concentration, immediate memory and delayed recall (out of a possible score of 30), and includes questions relating to LOC and PTA, and tests of movement and coordination, with administration taking 5 to 10 minutes (e.g., Barr & McCrea, 2001; CogState Sport, 2010; Guskiewicz et al., 2004; Randolph et al., 2006; McCrea, 2001). Compared with various immediate MTBI assessment techniques, SAC has the largest neurocognitive effect size and is 80% sensitive to MTBI (Valovich McLeod, 2009). Although SAC is sensitive to MTBI detection within 15 minutes of injury and is useful in classifying concussed athletes from nonconcussed controls, it cannot discern subtle deficits 48 hours postinjury, and is not a

substitute for a comprehensive neuropsychological assessment (e.g., Barr & McCrea, 2001; Collins, Grindel et al., 1999; McCrea et al., 2002).

The SCAT2 was updated from the SCAT to its present form by the CISG panel at the Third International Conference on Concussion in Sport in 2008, and is the most recent sideline evaluation tool (e.g., McCrory, Johnston et al., 2005; McCrory et al., 2009). It incorporates several test forms. These include the rating of 22 symptoms, physical signs (LOC and balance problems), the GCS rating, 5 Maddock's questions, a cognitive assessment using 5 orientation questions from the SAC, four alternate forms of a list of 5 words for immediate and delayed memory scores, four alternate forms of four series of digits backwards and a trial for giving the names of the months in reverse order, and balance and coordination examinations - that in combination form an overall score (see McCrory et al., 2009). A baseline preinjury assessment on SAC2 is recommended for the comparison of postinjury scores. However, definitive cutoff scores are not available, and SCAT2 is not designed to be a stand-alone measure to diagnose or monitor MTBI recovery or make return to play decisions (McCrory et al., 2009).

The Sideline ImPACT (distinct from the ImPACT computerised program) is a touch screen palm-held device for on-field evaluation. This instrument contains details of the athlete's MTBI history and previous ImPACT assessment results. Taking about five minutes to administer, Sideline ImPACT provides a brief mental status examination, in which the athlete repeats five words over three trials, answers ten orientation questions, repeats four series of digits backwards, following which the athlete is asked to repeat the five words from the earlier trial. The device also can evaluate observed signs and the athlete's reported symptoms, and can record concussion details such as the point of impact and details of the athlete's helmet. The Sideline ImPACT provides a total score out of a possible 34. It allows the tester to compare initial sideline signs and symptoms with previous ImPACT assessments and across different injuries recorded on Sideline ImPACT (ImPACT, 2004).

If MTBI is suspected, the athlete should not be left alone in the first few hours following injury, and needs to be monitored for signs indicating deterioration. Furthermore, the athlete should not return to play on the day of injury, although some adult athletes can do so when in the care of a team physician experienced in MTBI (McCrory et al., 2009). A

conservative approach to same day return to play is required for athletes younger than 18 years of age (e.g., McCrory et al., 2009). However, non-elite athletes, particularly younger athletes, are often at a disadvantage in not having access to the same resources as elite athletes, e.g., the presence of trained medical personnel during practice and matches or sideline preparedness, and as a result may be managed less conservatively (e.g., Putukian, Aubry, & McCrory, 2009). This is supported by Shuttleworth-Edwards, Noakes et al. (2008), who found more MTBI incidents among elite rugby players than among non-elite rugby players, with this associated with vast discrepancies in the management of MTBI across the school, university, club and provincial levels of rugby. Findings suggested that 5 to 10% of schoolboy, and as many as 20% of club and university rugby players, were not receiving adequate medical MTBI management. These authors recommend that all levels of rugby have a proactive approach to MTBI monitoring and management. This includes appropriate on-field assessment, the systematic logging of MTBIs for medical follow-up, a medical physician being present at all matches where possible, and incorporating pre- and post- MTBI neuropsychological assessment (Shuttleworth-Edwards, Noakes et al., 2008).

5.1.2 Specific Medical Evaluation of MTBI

Specific medical evaluation and management of MTBI does not fall within the neuropsychology ambit, and will not be overly detailed. However, immediately following injury, it is necessary for the physician to rule out any neurologic emergency, systemic trauma or spinal injury, and the assessment of multiple domains is required (e.g., AAN, 1997; Crippen, 2009; Department of Veteran Affairs, 2009a; Hinton-Bayre & Geffen, 2004; Hovda et al., 1999; Johnston, McCrory, Mohtadi, & Meeuwisse, 2001; McCrory et al., 2009). Urgent specialist neurological referral should be made in cases of altered consciousness, seizures, weakness or numbness of extremities, slurred speech, unusual behaviour, an increasingly worsening headache, disorientation to person or place, repetitive emesis, double vision, asymmetrical pupils or progressive deterioration on neurological examination (Anderson & Murata, 2009; Department of Veteran Affairs, 2009a). The medical officer can use the algorithms for medical management purposes - in the 2009 Va/DoD Guidelines for concussion - as these are specific to individual symptoms and are based on initial presentation of symptoms and follow-up of persistent symptoms (e.g., Anderson & Murata, 2009; Department of Veteran Affairs, 2009a).

Athletes with medically low risk injuries, such as mild to moderate headaches, dizziness and nausea, need not be hospitalised if monitored according to an instruction sheet provided. Medical attention should then be sought if a severe headache, unabated nausea and vomiting, confusion or strange behaviour, convulsions or a watery discharge from the ear or nose develop (Crippen, 2009). No specific medical treatment is required for most MTBI cases, although non-sedative analgesics for pain control are sometimes indicated (e.g., Anderson & Murata, 2009; Bernhardt, 2009; Department of Veteran Affairs, 2009a). Medication may be prescribed for ongoing symptoms such as anxiety or sleep disturbances, however it is essential that athletes are not taking medication when a return to play decision is made, as the medication may mask symptoms (McCrory et al., 2009). Clinical evidence reveals that progesterone in combination with Vitamin D has a significant positive effect on functional and morphological recovery following moderate to severe TBI, although it is unknown if this treatment has the same success in treating MTBI (e.g., Cekic & Stein, 2010).

Specific medical investigations may in certain circumstances be conducted, including formal balance testing, biomarkers, or neuroimaging and electroencephalography, and these will be discussed below. Although neuroimaging and electroencephalography are not currently routinely used for MTBI assessment, these instruments will be briefly reviewed in that they may have promising future clinical application in sport-related MTBI (e.g., Davis, Iverson, Guskiewicz, Ptito, & Johnston, 2009; Gaetz & Bernstein, 2001; Lovell, Collins, & Fu, 2003).

Formal Balance Testing and Biomarker Evaluation

A number of concussion management programs based on clinical or research evidence advocate balance testing assessment such as postural stability (Guskiewicz et al., 2001; Iverson, 2007; McCrea, Barr et al., 2005; McCrea et al., 2003; Mrazik et al., 2000; Oliaro et al., 2001; Peterson et al., 2003). Balance testing is proposed for diagnosing MTBI and for making return to play decisions following sport-related MTBI. This is because balance testing allows assessment of the athlete's physical capabilities and cortical neuronal functioning at rest and during tasks, and can indicate if the athlete has returned to pre-injury levels of functioning (e.g., Thompson, 2006). The Balance Error Scoring System has shown good concurrent validity and good test reliability as a clinical measure of balance impairment post MTBI (e.g., Davis et al., 2009; Guskiewicz, 2004).

The use of biomarkers for assessing MTBI, is a recent science. Although cerebral spinal fluid and serum markers, such as S-100B, myelin basic protein and neuron specific enolase, have identified MTBI in numerous studies, evidence for clinical use is uncertain (Begaz, Kyriacou, Segal, & Bazarian, 2006; Davis et al., 2009; de Boussard et al., 2005; Department of Veteran Affairs, 2009a; Iverson, Zasler, & Lange, 2007; Lima, Simao Filho, Abib, & Poli de Figueiredo, 2008; McCrory et al., 2009; Stalnacke, Tegner, & Sojka, 2003, 2004; Townend & Ingebrigtsen, 2006). In Europe the absence of significant cranial injury and serum S-100-B level measures less than 0.1 µg/L within four hours of injury have been used to indicate that scanning is not warranted. However, this specific serum test is neither approved nor usually unavailable in USA clinical settings (Burgess et al., 2010). Furthermore, a recent study did not find heading the ball among soccer players associated with biomarker evidence (Zetterberg et al., 2007).

Neuroimaging and Electrophysiological Evaluation

Although not routinely used for sport-related MTBI, Neuroimaging in the forms of magnetic resonance imaging (MRI) or computerised tomography (CT) may be conducted if an intracerebral structural lesion or contusion is suspected, with MRI being the more sensitive measure (e.g., Aubry et al., 2002; Bigler & Orrison, 2004; Crippen, 2009; Department of Veteran Affairs, 2009a; Johnston, Ptito, Chankowsky, & Chen, 2001; McCrory et al., 2009; Mittl et al., 1994). Investigation of protracted MTBI sequelae may include the use of neuroimaging measures such as functional magnetic resonance imaging (fMRI), positron emission tomography (PET), single photon emission computed tomography (SPECT). Electrophysiological measures such as the processed electroencephalogram (EEG) are used to provide information on the chronic pathophysiological effects of MTBI (Bigler & Orrison, 2004). Functional imaging is valuable in detecting cognitive impairment typical to MTBI, including impairment in working memory and information processing. It assists in understanding when the brain recovers from MTBI, and shows promising future clinical application in sport-related MTBI (e.g., Davis et al., 2009; Gaetz & Bernstein, 2001; Lovell, Collins, & Fu, 2003;).

Although beyond the scope of this study, several MTBI studies have used various neuroimaging and electrophysiology modes, and have provided compelling findings (e.g., Alexander, 1995; Ashtekar & Patankar, 2009; Baker & Hutchinson, 2008; Baker & Patel

2000; Bigler & Orrison, 2004; Bernhardt, 2009; Crippen, 2009; Dupuis, Johnston, Lavoie, Lepore, & Lassonde, 2000; Ford & Khalil, 1996a, 1996b; Gaetz & Bernstein, 2001; Georgetown, 2009; Haydel et al., 2000; Hofman et al., 2001; Holli et al., 2009; Ingebrigsten, Romner, & Kock-Jensen, 2000; Jeret et al., 1993; Johnston, Ptito et al., 2001; Kesler et al., 2000; Lavoie, Dupuis, Johnston, Leclerc, & Lassonde, 2004; Lowry, 2010b; Marshall, Marshall, & Klauber, 1991; Mandel, 1989; Mendez et al., 2005; Mittl et al., 1994; Mower, 2010; Pontifex, O'Connor, Broglio, & Hillman, 2009; Thompson, Sebastianelli, & Slobounov, 2005; Tysvaer et al., 1989).

A few pertinent neuroimaging and electrophysiological studies pertaining to MTBI follow. Holli et al. (2009) found MRI revealed differences in the textures between selected segments of the corpus callosum, particularly in the splenium area, among MTBI patients, with 96 to 98% accuracy in differentiating MTBI from non-MTBI groups. PET and SPECT have revealed hypometabolism in the frontal and temporal lobes during working memory tasks and at rest, which correlate with decreased memory function following MTBI (Mendez et al., 2005). SPECT findings have correlated with cognitive deficits, and with chronic postconcussive symptoms four weeks and one year post MTBI (e.g., Bigler & Orrison, 2004; Jacobs, Put, Ingels, & Bossuyt, 1994, 1996). However, controversy surrounds SPECT in that it correlates poorly with the clinical presentation and MRI and CT findings. Abnormal images have been related to mood, headaches and other variables, and SPECT requires an injectable radioactive substance and is seldom used for children (e.g., Mitchener et al., 1997; Lovell & Collins, 2002; Rees, 2003).

The advantage of fMRI in assessing the indirect effects of neuronal activity on the blood's volume, flow and oxygen saturation is that unlike SPECT and PET, there is no radiation exposure (e.g., Bigler & Orrison, 2004; Jantzen, Anderson, Steinberg, & Kelso, 2004). fMRI is suitable for investigations in both the acute and chronic phases post MTBI and is commonly used as an adjunct to neurocognitive testing for validating testing and monitoring recovery (Lovell & Collins, 2002; McAllister et al., 2001). Lovell, Pardini et al. (2007) conducted a study in which at the first post MTBI assessment, athletes demonstrated significantly weaker performance on ImPACT relative to baseline and controls, and neurophysiological changes for three brain networks were identified on fMRI for short-term delayed memory and visuomotor tasks. Hypoactivity in the posterior parietal circuit on fMRI was associated with impaired neurocognitive performance on

memory tasks, in addition to both cognitive symptoms and particularly somatic symptoms. The extent of activation in this area was associated with symptom recovery. Hyperactivity on the Network 1 on fMRI was predictive of delayed recovery, as measured at a later follow-up. Several other studies employing fMRI post MTBI found evidence of areas of brain activation that correlate with working memory, memory tasks, sensorimotor tasks particularly motor sequencing tasks, and postconcussive symptoms (e.g., Jantzen et al., 2004; McAllister et al., 1999; Mendez et al., 2005).

Preliminary data garnered from an ongoing study on sport-related MTBI employing transcranial Doppler ultrasound, indicates neurocognitive impairment improves over time in association with altered cerebrovascular functioning (Tegeler et al., work in progress). Magnetic Source Imaging (MSI) has been found superior to either abnormal MRI or EEG readings in detecting MTBI patients with postconcussive symptoms (Mendez et al., 2005).

EEG readings in combination with balance testing can indicate if an athlete has returned to pre-injury levels of functioning, and can be used for making return to play decisions (Thompson et al., 2005). EEG and Evoked Potentials (EP) differences have classified 100% of the controls and 52 to 74% of suspected MTBI cases correctly (Ford & Khalil, 1996a, 1996b). Promising results have been shown in MTBI studies utilising Event Related Potentials (ERP) and Evoked Potentials (EP), in which both ERP and EP represent the averaged EEG signal in response to a given stimulus, and with ERPs representing cognitive processing and EPs primary sensory pathway processing (e.g., Baker & Hutchinson, 2008; Mendez et al., 2005). Gaetz and Bernstein's (2001) review of MTBI studies found ERPs appearing to be more sensitive to MTBI than EPs, and proposed that an assessment battery including neuropsychological testing, EEG, EP and ERP be developed. ERP P300 amplitude effects have been found associated with decreased processing speed, reduced attentional-cognitive processes, failures in control of action monitoring during a modified flanker task, and the severity of PCS following sport-related MTBI (e.g., Baker & Hutchinson, 2008; Dupuis et al., 2000; Lavoie et al., 2004; Pontifex et al., 2009). ERPs have demonstrated deficits in auditory processing in symptomatic and asymptomatic athletes following MTBI (Gosselin, Theriault, Leclerc, Montplaisir, & Lassonde, 2006). ERP appears more sensitive than neuropsychological testing. This is because ERPs revealed significant differences between athletes with a

history of MTBI averaging 3.4 years prior and athletes without MTBI histories, whereas ImPACT testing revealed no significant differences between these two groups (Broglio, Pontifex, O'Connor, & Hillman, 2009).

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 (e.g., Hofman et al., 2001; Kesler, Adams, & Bigler, 2000).

The general management of MTBI will now be discussed.

5.1.3 General Medical Management of MTBI

The general medical management of MTBI should include ongoing monitoring of postconcussive symptoms for 30 days post MTBI. Early intervention should include education and information on symptoms and recovery, injury prevention, sleep hygiene, relaxation techniques, graded exercises and limiting alcohol, tobacco and caffeine intake (e.g., Anderson & Murata, 2009; Department of Veteran Affairs, 2009a; Kohler, 2004).

Although the Department of Veteran Affairs (2009a) recommend that return to work, studies, duties and leisure activities should take place as soon as possible with a gradual resumption or a progressive return as tolerated, McCrory et al. (2009) consider physical and cognitive rest as the “cornerstone” of MTBI management. The injured athlete should rest and limit activities requiring mental exertion during the recovery period (McCrory et al., 2009). However, the need for rest and avoiding exercise following MTBI requires further validation. For example, no significant differences in posttraumatic complaints at two weeks to six months post MTBI were found between those on full bedrest for six days and those not on bedrest, apart from bedrest being associated with significantly less dizziness over the study duration (de Kruijk, Leffers, Meerhoff, Rutten, & Twijnstra, 2002). Although high levels of activity post MTBI have shown a negative impact on symptom and neurocognitive recovery that significantly affect reaction time and visual memory, moderate levels of activity are associated with better symptom and neurocognitive recovery (e.g., Leddy et al., 2010; Masjerske, Mihalik et al., 2008; Satz et al., 1997; Willer & Leddy, 2006). Current recommendations are that once the athlete

recovers, only then can a graded program of exertion or stepwise return to play program be introduced, and once asymptomatic on exertion then a return to play decision can be taken (McCrory et al., 2009).

5.1.4 Return to Play Decisions

The most critical decision in MTBI management is determining if and when an athlete can return to sport practice and competition. Premature return to sport before complete metabolic recovery can have enduring or even dire neuropsychological consequences for the athlete (e.g., Bender et al., 2004; Collins, Echemendia, & Lovell, 2004). On the other hand, the athlete should not be restricted from sport unnecessarily, particularly in view of the pressure for professional or talented athletes to return to competition expediently (e.g., Bender et al., 2004; Collins et al., 2004; Pretz, 2007).

Several widely varying systems devised in the past to grade MTBI severity and provide return to play guidelines according to severity, were discussed in Chapter 3 (section 3.5). However, these guidelines, ranging from being overly strict to overly lenient, were inapplicable to all age groups, not empirically supported or universally accepted, and were ultimately abandoned (e.g., Aubry et al., 2002; Bernhardt, 2009; Cantu, 1998b; Field et al., 2003; Guskiewicz et al., 2004; Kelly & Rosenberg, 1998; Kohler, 2004; Maroon et al., 2000; McCrory et al., 2004; Mendez et al., 2005; Peloso, Carroll et al., 2004; Pretz, 2007; Purcell, 2009; Wilner, 2010). Currently a complex model is required for making return to play decisions, in which MTBI assessment and management is reliant on a multidisciplinary approach including neuropsychological testing (Echemendia & Cantu, 2003). An individualised approach to MTBI management is now the standard, where there is appreciation of the variability in MTBI sequelae and recovery duration among individuals, acknowledgement of differing symptom presentation, and realisation that MTBI can present with or without apparent cognitive deficits (e.g., Echemendia et al., 2001; Guskiewicz et al., 2004).

The current criteria for return to play are that postconcussive symptoms must have fully resolved, the athlete must be completely asymptomatic at rest and specifically asymptomatic on or following exertion, e.g., following provocative testing such as sit-ups, push-ups, and jogging which elevate the heart rate and blood pressure (Bernhardt,

2009). Moreover, the athlete must have no remaining neurocognitive deficits resultant of the MTBI. This should preferably be established through postconcussive neurocognitive testing involving comparison of postconcussive scores with the individual's preinjury scores (e.g., Bernhardt, 2009; Guskiewicz et al., 2004). Comparing an athlete's preinjury and postinjury symptom and neurocognitive test scores assists with safe management. Once postconcussive symptoms and neurocognitive dysfunction are absent, the sports physician can make a clinical decision on returning the athlete to sport (e.g., Aubry et al., 2005; Bender et al., 2004; Bernhardt, 2009; Cantu, 2001; Collie, Darby, & Maruff, 2001; Collie, Makdissi et al., 2006; Collins, Echemendia, & Lovell, 2004; Grindel et al., 2001; NCAA, 2002). The sports physician's decision to allow a previously concussed athlete to return to sport participation is challenging and complex, in that several sources of information need to be considered, one being the postinjury neuropsychological assessment (e.g., Echemendia, Herring, & Bailes, 2009; Hinton-Bayre & Geffen, 2002).

With regard to rugby union specifically, International Rugby Board regulations stipulate a three-week abstinence from rugby following MTBI, unless the adult athlete is symptom free and declared fit following an assessment by a qualified and recognised neurological specialist who records this in a written report (International Rugby Board, 2010). Therefore, neuropsychological assessment plays a major role in determining if the rugby player is neurocognitively recovered and asymptomatic, and can return to sport within the three-week period. In contrast, not all athletes recover neurocognitively or symptomatically within three weeks, in which case neuropsychological assessment can preclude return to play.

Following recovery from MTBI, the athlete is reintroduced to physical activity via a graded program of exertion in a stepwise progression, in which the athlete can progress to the following step of more intense exercise if asymptomatic on exertion at a particular step. Progress towards return to play should take a week if the athlete remains asymptomatic at each step (Mc Crory et al., 2009).

In some instances an athlete is advised to retire from sport. Quigley's rule proposes that an athlete refrains from sport following three MTBI incidents (Barth et al., 1989). However, an individual sustaining a single MTBI may be left with permanent neurocognitive deficits and postconcussive symptoms that prohibit return to sport, while

an athlete sustaining multiple MTBIs may evidence no obvious neurocognitive deficits or postconcussive symptoms (Heilbronner et al., 2009). Therefore, decisions on retirement should not be based on the number of MTBIs, but on symptom duration, vulnerability to MTBI and findings from a comprehensive neuropsychological evaluation (e.g., Cantu, 2003; Echemendia & Cantu, 2003; Heilbronner et al., 2009).

Overall, MTBI management advocates the assessment of multiple domains, including the medical ones discussed above. One form of assessment particularly sensitive to detecting impairments in cognitive, emotional and behavioural domains resulting from MTBI and which is useful in monitoring the athlete's recovery, is neuropsychological assessment, which will be discussed below (Guskiewicz et al., 2001; Hinton-Bayre & Geffen, 2004; Iverson, 2007; Johnston et al., 2001; McCrea, Barr et al., 2005; McCrea et al., 2003; Mrazik et al., 2000; Oliaro et al., 2001; Peterson et al., 2003).

5.2 NEUROCOGNITIVE ASSESSMENT IN SPORTS MTBI

While medical assessment can detect signs of neurological dysfunction and a neuroimaging assessment can detect neurological structural damage, neither form of assessment can detect subtle neurocognitive deficits arising from MTBI (e.g., Collins, 2003). Neuropsychological assessment appears to be the most appropriate tool for investigating subtle neuropsychological changes resultant of MTBI, and is being more frequently used as a key component towards the multi-tiered management of sport-related MTBI, and upon which recovery can be monitored and individualised return to play decisions can be made (e.g., Aubry et al., 2002; Collins, 2003; Cremona-Meteyard & Geffen, 1994; Guskiewicz et al., 2004; Lovell & Collins, 2002; Meehan & Bachur, 2009; McCrory, Johnston et al., 2005; McCrory et al., 2009; Mendez et al., 2005; Moser et al., 2007; Podell, 2004; Purcell, 2009).

Neuropsychology focuses on understanding the relations between brain and behaviour, and neuropsychological assessment is aimed at quantifying changes in brain function following brain insult and for identifying preserved functions (e.g., Echemendia et al., 2009; Kozora & Gerber, 2004; Levin et al., 1989). Neuropsychology is a relatively new psychology discipline, that developed after World War II, when the need arose to assess soldiers who had suffered TBI (Echemendia et al., 2009). Since the latter half of the

1980s, the value of neuropsychological assessment in sport-related MTBI research and management has been recognised (e.g., Echemendia et al., 2009; Lovell & Collins, 2002; Maroon et al., 2000; Mendez et al., 2005). Sport-related MTBI research initially focused on boxing, but expanded to cover other contact sports including American football, Australian football and soccer. In the 1990s neuropsychological assessment was used by the Pittsburgh Steelers (NFL) for making return to play decisions and subsequently was used in other professional sports such as auto racing and ice hockey (e.g., Lovell, Collins, Pardini, Parodi, & Yates, 2005; Mendez et al., 2005; Pellman, Lovell, Viano, Casson, & Tucker, 2004). Over the past ten years neuropsychological assessment has become an integral part of both the assessment and management of sport-related MTBI in several sports (Pretz, 2007).

Several authors consider neuropsychological assessment to be the most sensitive measure in detecting the subtle signs of brain dysfunction. Some authors acknowledge that this form of assessment considers a broad range of neurocognitive, emotional and psychosocial factors possibly affecting recovery (e.g., Butler, Forsythe, Beverly, & Adams, 1993; Casson et al., 1984; Heilbronner et al., 2009; McLatchie et al., 1987; Ross, Casson, Siegel, & Cole, 1987). Neuropsychological assessment plays a crucial role in detecting neurocognitive impairment, charting recovery, and assisting with return to play decisions following MTBI. This is especially relevant considering that neurocognitive recovery can precede or follow symptom recovery, or in some instances the athlete is asymptomatic but experiences either a delayed onset of symptoms or a delayed resolution of neurocognitive deficits (e.g., Aubry et al., 2002; Barr & McCrea, 2001; Field et al., 2003; Lovell, Collins, Iverson et al., 2004; McCrory, Johnston et al., 2005; McCrory et al., 2009; Mendez et al., 2005; Shuttleworth-Edwards & Whitefield, 2007a).

Sport-related neuropsychological assessments are particularly useful when establishing neuropsychological preinjury or baseline levels of functioning (see section 5.3.1 below), against which postinjury deficits can be quantitatively and objectively measured and compared (e.g., Echemendia, Putukian, & Phillips, 1997; Heilbronner et al., 2009; McCrory, Makdissi, Davis, & Collie, 2005; Schatz, 2010; Weight, 1998). As “one of the cornerstones” (Aubry et al., 2002, p. 8; McCrory, Johnston et al., 2005, p. 51) of MTBI evaluation, contributing significantly valuable information to both injury understanding and management of the athlete, neuropsychological assessment - in conjunction with

other clinical evidence - helps with making the final medical return to play decision (McCrory, Johnston et al., 2005; McCrory et al., 2009). Therefore, neuropsychological testing is not a single means of MTBI diagnosis and assessment, but is a complementary instrument in the assessment and management of MTBI (e.g., Echemendia & Cantu, 2003; Ellemberg et al., 2009; Randolph et al., 2005).

5.2.1 General Assessment Issues

General assessment issues in sport-related MTBI neuropsychological assessment, include factors relating to test characteristics, test conditions, the role of neuropsychologists, and levels of neuropsychological testing. These will be discussed below.

Test Characteristics

Characteristics of a test are an important assessment issue and include factors such as the type of test (discussed in more detail below with reference to traditional and computerised forms of tests), selection of appropriate tests, and issues relating to validity, reliability, and applicable test norms. In order for a neuropsychological test to be of value, it needs to evidence positive detection of impairment in a valid and reliable manner, in a person who is actually impaired (Barr & McCrea, 2001).

Validity refers to the test instrument measuring a particular cognitive domain it is purported to measure, and this requires several empirical studies to provide evidence that it is valid (e.g., Lezak et al., 2004; Rosnow & Rosenthal, 1996). However, neuropsychological tests seldom measure one cognitive domain, and appear open to different interpretations. For example, the Visual Reproduction test of the WMS-R and WMS-III, may be less a measure of memory than a measure of visuospatial analysis and reasoning, incorporating a visual construction component (Lezak et al., 2004). One aspect of determining validity of a computerised test, is to correlate the test scores with a traditional non-computerised test, in order to assess the underlying construct the computerised test is purported to measure (Iverson, Lovell, & Collins, 2005). However, the current range of neuropsychological tests have been criticised for their lack of sensitivity to subtle changes in neurocognitive functions in the presence of brain injury, and have been referred to as “surrogate markers” (Loosemore, Knowles, & Whyte, 2007, p. 6).

Reliability is the ability for a test to consistently measure a particular cognitive domain with consistency across a number of test occasions (Rosnow & Rosenthal, 1996). Therefore, the reliability or regularity with which a test produces the same repeat score under similar test conditions, can only be ascertained using control participants who are unimpaired in the cognitive area being assessed (Lezak et al., 2004). Tests need to be reliable but not to the extent that they are insensitive to the effects of MTBI (Ellemberg et al., 2009). In repeat assessments, such as in serial postconcussive testing, potential sources of error may cause instability in scores and affect reliability. These include changes in scores due to extraneous variables, other than changes in the variable being measured, or improved performance as a result of prior exposure to the test - known as practice effects. Some tests have practice effect sizes that are equivalent to or exceed the effects of MTBI when measured on these tests (Ellemberg et al., 2009). Using different forms of the same test may overcome practice effects to a limited extent, however these effects can still occur when alternate forms of a test are used (Franzen, Frerichs, & Iverson, 2004).

Sample characteristics and demographic variables can impact significantly on test performance, and therefore a number of neuropsychological tests are demographically normed according to variables such as gender or age, in order to assist test interpretation (e.g., Lezak et al., 2004). Several studies among athletes have demonstrated gender differences in neuropsychological performance, including females performing better than males on verbal initiation, processing speed, mental tracking, visuoperceptual attention and concentration, and perceptual motor and verbal fluency tasks. Therefore certain tests require separate norms for males and females (e.g., Barr, 2003; Putukian, Echemendia, & Mackin, 2000; Ryan, Atkinson, & Dunham, 2004).

Overall, however, few studies have specifically investigated the sensitivity, specificity, predictive value, practice effect sizes, standard error of measurement and reliability of neuropsychological tests in sports populations (Ellemberg et al., 2009).

Test Conditions

It is important that the athlete is provided with test conditions conducive to optimal performance. These include a comfortable physical environment free of distractions and for the examiner to be non-time pressured, reassuring, and to administer the test in a

proficient and consistent manner according to standardised procedures at each test occasion. The length of testing should not be arduous or taxing on the testee (e.g., Barr, 2001; Kozora & Gerber, 2004; Franzen et al., 2004; Lezak et al., 2004).

The Role of Neuropsychologists

The professional role of a neuropsychologist is to administer neuropsychological tests and interpret the results (Collie & Maruff, 2003; Shuttleworth-Edwards, 2008; Shuttleworth-Edwards & Border, 2002; Shuttleworth-Edwards & Whitefield, 2007a; Shuttleworth-Edwards & Whitefield, 2007b). Echemendia et al. (2009) propose that although neuropsychologists are specifically trained to administer neuropsychological tests, those used in the sports settings are uncomplicated and a neuropsychologist can designate a non-psychologist, who is proficiently trained to administer such tests. However, there is potential for misuse of neuropsychological tests when this occurs (Shuttleworth-Edwards, 2008; Shuttleworth-Edwards & Border, 2002; Shuttleworth-Edwards & Whitefield, 2007a, 2007b). This is of particular concern with tests that generate automated reports, in that results are not "absolute qualities" (Shuttleworth-Edwards, 2008, p. 483) and cannot be interpreted in isolation. Shuttleworth-Edwards (2008) points out that valid interpretation of test data requires (i) contextualisation of variables such as the athlete's gender, age, language, race, intellectual potential and education, (ii) considering test-taking conditions, psychometric test properties, nature of injury, comorbid physical and psychological conditions, and (iii) evaluating patterns that may indicate artifacts, malingering and functional as opposed to organic pathology (Shuttleworth-Edwards, 2008). Either one of these factors, or a combination of them may result in false positive or a false negative diagnosis, and subsequent mismanagement (Shuttleworth-Edwards, 2008). Echemendia at al. (2009) posit that interpretation differs fundamentally from administration and requires understanding of complex interactions amongst test data, sources of error, psychometric properties, domains of functioning, practice effects, test variables and intraindividual variables. Therefore a neuropsychologist, serving as consultant to the team physician, should manage the interpretation, particularly on medico-legal grounds (Echemendia et al., 2009). In South Africa (Magoke, 2004) and the USA (Guskiewicz et al., 2004; Moser et al., 2007) psychologists are required to supervise neuropsychological testing for clinical purposes, and in South Africa a registered psychologist is required to interpret and report on computerised neuropsychological tests (Shuttleworth-Edwards, 2008).

Forms of Neuropsychological Testing in Sport-Related MTBI

The form of neuropsychological testing in sport-related MTBI, and which pertains to this study, involves baseline testing and postconcussive follow-up testing, and is now discussed. A further form of neuropsychological testing used with chronic and unremitting MTBI sequelae, entails comprehensive testing, that is not directly related to this study, and will therefore be mentioned only briefly. Neuropsychological evaluation has taken place in various forms within the research context, and will be discussed in detail as relevant background for the current research.

Baseline neuropsychological testing involves testing an athlete preinjury, usually before or at the start of the sport season. Baseline testing helps overcome the significant variability between individuals associated with using standard norms for comparisons. Baseline test scores provide an individual measurable level against which the individual's post MTBI test scores can be compared, and allow the clinician access to quantitative neuropsychological data when making return to play decisions (e.g., Echemendia et al., 2009; Heilbronner et al., 2009).

Postconcussive follow-up neuropsychological testing involves testing an athlete postinjury for comparisons of postinjury scores with baseline scores, in order to detect functions that are impaired and spared. Echemendia and colleagues (2001, 2003) caution that although the athlete is required to be both asymptomatic and to perform at or above the athlete's own baseline level on neurocognitive testing before returning to sport, test measures are susceptible to practice effects, and may require that the athlete exceeds baseline performance, the level of which needs to be ascertained from empirical studies. In the absence of baseline testing, Aubry et al. (2002) suggest performance variability on computerised testing might be the solution to MTBI diagnosis, while in the ImPACT (2004) manual it is suggested that postinjury scores are compared with gender and age stratified normative scores. Currently there are two approaches to follow-up testing. The first approach involves testing concussed athletes at certain intervals postinjury, and the second approach involves testing only when the athlete is asymptomatic (e.g., Guskiewicz, 2004; McCrory, Johnston et al., 2005; McCrory et al., 2009). While the second approach may be more practical, in that the athlete is withheld from play based on symptom reports, the first approach yields valuable data on neurocognitive recovery (Guskiewicz, 2004).

Comprehensive neuropsychological testing is conducted when permanent neurocognitive damage is suspected, e.g., symptoms persist, an athlete has suffered multiple MTBI events, or scores have not returned to baseline levels after several weeks. This assessment takes several hours and usually the neuropsychologist assesses estimated premorbid intelligence and multiple domains of cognitive functioning in addition to emotional and psychological functioning, in order to recommend further neurological investigation or to place a moratorium on return to play (e.g., Shuttleworth-Edwards, 2008; Randolph, 2001).

The types of neuropsychological tests commonly used in sport-related MTBI research, and in the clinical setting will now be discussed. Traditional pencil and paper tests are initially described, followed by a review of four computerised tests commonly used.

5.2.2 Traditional Pencil and Paper Neurocognitive Assessment

Originally neuropsychologists used traditional paper and pencil tests. Tests or test batteries that appeared sensitive to the effects of MTBI evolved, particularly those measuring the cognitive abilities of attention, concentration, processing speed, learning, memory, verbal fluency, auditory processing and planning (e.g., Collins, Echemendia, & Lovell, 2004; Guskiewicz et al., 2004; Iverson et al., 2004a; Macciocchi et al., 1996). Traditional neuropsychological evaluation of an athlete took the neuropsychologist several hours and was costly (e.g., Echemendia et al., 2009; Lovell & Collins, 2002). Furthermore, many traditional tests were designed for a single test occasion, and for measuring gross cognitive deficits, whereas sport MTBI testing requires multiple test occasions and measures sensitive to mild cognitive deficits (Collie et al., 2001). Moreover, a restricted range of scores with floor and ceiling effects, often limited traditional tests in terms of test-retest reliability (Collie et al., 2001). However, in order to provide a platform for discussion of MTBI sequelae within contact sport, a description of selected traditional tests that appear sensitive to particular neurocognitive effects of MTBI and have been commonly used for the assessment of sport-related MTBI, follows. These tests are grouped according to the main neurocognitive functions they are purported to measure.

A Test of Immediate Auditory Attention and Concentration

Digit Span (digits forwards and digits backwards)

The various forms of the WAIS (Wechsler, 1945, 1981, 1997) include the Digit Span test, in which the participant is read progressively longer series of numbers and then recalls these numbers in either a forward or backward sequence. The test measures attention, concentration, and immediate auditory memory recall (Oliaro, Guskiewicz, & Prentice, 1998). The Digit Span has revealed improved test-retest performance within a six-month interval among well-educated young adults and therefore appears subject to practice effects (e.g., Lovell & Collins, 1998; Oliaro et al., 1998; Putukian, Echemendia, & Mackin, 2000; Shuttleworth-Edwards, Smith et al., 2008; Strauss, Sherman, & Spreen, 2006). However, Ellemberg et al. (2009) report relatively small practice effect sizes (.10 to .45), and high reliability (r = .80 to r = .91).

Some studies have found that the Digit Span does not differentiate those with and without MTBI in the acute stage postinjury (e.g., Guskiewicz et al., 1997; Leininger et al., 1990; Ruff et al., 1989). However, the Digit Span appears sensitive to measuring attention difficulties following MTBI, in that one study found four out of six athletes revealed deficits on the Digit Span, with resolution between 2 to 4 weeks post MTBI. In addition, Digits Backwards was more sensitive, although not significantly so, to the effects of previous MTBI in the direction of poorer performance in comparison to controls (Hatfield et al., 2004).

Digit Span Forwards is a measure of sustained attention, immediate attention, immediate auditory span and mental tracking. Following MTBI, athletes may recall the digits correctly but in the incorrect order (e.g., Brooks, 1984; Lezak et al., 2004; Shuttleworth-Jordan et al., 1993). However, some studies have found no significant impairment on digits forwards post MTBI, although there is some evidence of not benefitting from practice effects to the same extent as controls in the acute stage postinjury (e.g., Gentilini et al., 1985; Shuttleworth-Jordan et al., 1993).

Digit Span Backwards is considered a measure of short-term memory or working memory (e.g., Brooks, 1984; Lezak et al., 2004). The test has revealed a small but more pronounced effect than the Digit Span Forwards among those suffering MTBI (e.g., Barr, 2003; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Jordan et al., 1993; Strauss

et al., 2006). Shuttleworth-Edwards, Smith et al. (2008) found Digits Backwards particularly sensitive to the effects of rugby-related MTBI, in that the rugby players performed marginally better than controls at preseason, but the controls performed significantly better than the rugby players postseason. Shuttleworth-Jordan et al. (1993) found that by the third month postinjury, the MTBI rugby group had not recovered fully, in that they did not evidence the degree of practice effects as the control group on Digits Backwards.

Tests of Visuomotor Speed

Digit Symbol Substitution Test (DSST)

The various forms of the WAIS (Wechsler, 1945, 1981, 1997) include the Digit Symbol Substitution Test (DSST) that appears to have reliable discriminant validity and screening potential between controls and those with mild forms of neurocognitive dysfunction or diffuse damage associated with MTBI (e.g., De Monte, Geffen, May, & McFarland, 2004; Erlanger et al., 1999; Frencham et al., 2005; Shuttleworth-Edwards, 2002; Shuttleworth-Edwards & Radloff, 2008). The DSST is a measure of visuoperceptual processing, incorporating visual attention, concentration, visual shifting, visuomotor coordination, memory, response speed and copying speed (e.g., Joy, Kaplan, & Fein, 2004; Lezak et al., 2004). The test is prone to practice effects (e.g., Barr, 2003; De Monte, Geffen, & Kwapil, 2005; Hinton-Bayre et al., 1997; Maddocks & Saling, 1996; Makdissi et al., 2001). The DSST reliability coefficients range from $r = 0.74$ to $r = 0.82$ (Wechsler, 2002).

Several studies have found the DSST reveals significant differences between those with and without MTBI (e.g., Dicker & Maddocks, 1988; Hinton-Bayre et al., 1999; Macciocchi et al., 1996; Maddocks & Saling, 1996; Matser et al., 1999; McCrory, Ariens et al., 2000; Preece & Geffen, 2007; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008).

Shuttleworth-Edwards and Radloff (2008) demonstrated residual effects of MTBI among rugby players from school through to adult national level, in the form of significantly poorer performance than controls on DSST, particularly among the more vulnerable rugby forwards. However, not all sport-related studies have demonstrated DSST as a sensitive measure of MTBI, particularly in the chronic stage post MTBI (e.g., Guskiewicz

et al., 2002; Hinton-Bayre et al., 1997, 2004; Jordan, Matser et al., 1996; Maddocks et al., 1995; Makdissi et al., 2001; Matser et al., 1998, 2001; Rutherford et al., 2005).

Symbol Digits Modalities Test (SDMT)

The Symbol Digit Modalities Test (SDMT) (Smith, 1973, 1991) requires visual perceptual and psychomotor problem solving skills, and measures attention (in the forms of visual scanning and tracking) and visuomotor processing speed (Barth et al., 1989; Iverson, Lovell, & Collins, 2005; Sheridan et al., 2006). The SDMT is the inverse of the DSST, and is not significantly affected by the variables of age, education level or income (Sheridan et al., 2006). Reliability ranges between $r = .72$ and $r = .80$, with practice effect sizes ranging between .10 and .20 (Ellemberg et al., 2009). In several studies, the SDMT has revealed statistical differences between those with and without MTBI (e.g., Collins, Lovell, & Mckeag, 1999; Echemendia et al., 2001; Hatfield et al., 2004; Hinton-Bayre et al., 1999; Macciocchi et al., 1996). However, Barth et al. (1989), found that their control group evidenced practice effects on the SDMT which was not evidenced by the MTBI group, who maintained their preseason baseline levels at the 24 hour postinjury test interval. Thereafter, the MTBI group displayed recovery between 24 hours and day 5, with further recovery between the day 5 and day 10 post MTBI (Barth et al., 1989).

Tests of Information Processing

The Paced Auditory Serial Addition Test (PASAT)

The Paced Auditory Serial Addition Test (PASAT) (Gronwall, 1977; Gronwall & Wrightson, 1981) involves the rapid presentation of auditory numeric material for complex cognitive manipulation, requiring a high degree of immediate memory recall, verbal memory, attention, concentration information processing, and executive functioning (e.g., Barth et al., 1989; Levin, Mattis et al., 1987). Ellemberg et al. (2009) report high reliability ($r = .80$ to $r = .90$), and substantial practice effect sizes (.40 to 1.30), for this test. Several studies have found the PASAT reveals significantly poorer performance or a lack of benefit from practice effects by those with MTBI, in comparison to those without MTBI (e.g., Barth et al., 1989; Brooks et al., 1999; Leininger et al., 1990; Levin, Mattis et al., 1987; Macciocchi et al., 1996; Webbe & Ochs, 2003; Witol & Webbe, 2003). Numerous other studies, however, have not found PASAT sensitive to the effects of MTBI, for example amongst boxers, Australian football league, soccer, and rugby athletes (e.g., Abreau, Templer, Schuyler, & Hutchison, 1990; Brooks, Kupsik et

al., 1987; Cremona-Meteyard & Geffen, 1994; Downs & Abwender, 2002; Farace et al., 2003; Jordan, Matser et al., 1996; Macciocchi et al., 2001; Maddocks & Saling 1991, 1996; Matser et al., 1998, 1999, 2001; McLatchie et al., 1987).

Stroop Colour Word Test

The Stroop Colour Word Test (Golden, 1978), in which the participant is required to quickly read colour names printed in ink of different colours over a number of trials, measures selective attention, processing speed, cognitive flexibility, response inhibition and executive function, but has evidenced practice effects (e.g., Killam et al., 2005; Oliaro et al., 1998; Randolph et al., 2005; Spreen & Straus, 1998). Several versions of the test have evolved, and therefore exact reliability figures are difficult to establish. However, a Dutch study among healthy adults ages 24 to 81 years found declines in performance related to older age and lower levels of education, that supported the reserve hypothesis in relation to executive functioning (Van der Elst, Van Boxtel, Van Breukelen, & Jolles, 2006). The Stroop test has been found sensitive to subtle impairment following MTBI (e.g., Jordan, Matser et al., 1996; Killam et al., 2005; Putukian et al., 2000; Wilberger, Haag, & Maroon, 1991). However, a number of studies have found the Stroop test not sensitive to the effects of sport-related MTBI (e.g., Guskiewicz et al., 1997, 2002; Hatfield et al., 2004; Matser et al., 1998, 1999, 2001; Ravdin et al., 2003; Rutherford et al., 2005).

Trail Making Test (TMT)

The Trail Making Test (TMT) (Reitan, 1955; Reitan & Wolfson, 1985), consisting of Parts A and B, involves sequential problem solving and the need to keep two pieces of information in mind simultaneously, and overall is a measure of sustained attention and concentration, cognitive flexibility, orientation, problem-solving, working memory, visuospatial capacity and information or visuomotor processing speed (e.g., Barth et al., 1989; Kaste et al., 1982; Oliaro et al., 1998; Reitan & Wolfson, 1985; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Jordan et al., 1993). TMT A requires the athlete to search for and draw connecting lines between the circled numbers 1 to 24 as quickly as possible (Randolph et al., 2005). TMT B, has three alternate forms, and requires the athlete to connect alternating numbers and letters as quickly as possible. Reitan (1955, 1958) found the TMT sensitive to the effects of diffuse brain injury. Reliability ranges between $r = .45$ and $r = .72$, with practice effect sizes between .20 and .73, and correlates

highly with the Stroop test (e.g., Ellemberg et al., 2009; Oliaro et al., 1998). The practice effects in TMT A and B, are possibly due to the test involving novel tasks with a speeded component (e.g., Lezak et al., 2004; Lovell & Collins, 1998; Shuttleworth-Edwards, Smith et al., 2008; Spreen & Straus, 1998; Strauss et al., 2006).

Several studies have shown that the TMT is sensitive to the effects of sport-related MTBI. Shuttleworth-Jordan et al. (1993) found preseason performance by a nonconcussed rugby group on the TMT A and B was significantly poorer than that of the controls, with the controls revealing a significant practice effect postseason on TMT A and B, whereas the nonconcussed rugby group revealed less ability to benefit from practice with improvement on only TMT B among the rugby backs, but not the rugby forwards postseason. For the concussed rugby group, poorer performance at preseason and three days post injury was revealed on the TMT A and B. There was recovery on the TMT A and B one month post injury, and in terms of the extent of practice effects as the matched control group at two months post injury, suggesting that the TMT has a strong practice effect that masks changes in the variable being measured (Shuttleworth-Jordan et al., 1993). Shuttleworth-Edwards and Radloff (2008) investigated the residual effects of MTBI amongst rugby players from school through to adult national level, finding significantly poorer performance by the rugby players than controls on TMT A and B. Other studies have found the TMT sensitive to the subtle and acute effects of sport-related MTBI in comparison with controls (e.g., Echemendia et al., 2001; Hatfield et al., 2004; Macciocchi et al., 1996). Collins et al. (1999) found the TMT B was sensitive to athletes with a history of two or more MTBI events.

In contrast, Leininger et al. (1990) found no significant differences between the scores of the MVA-related MTBI group and control group on TMT B, suggesting that this subtest lacks sensitivity to differentiating MTBI from non-MTBI individuals. The TMT was not found sensitive to the effects of sport-related MTBI in some studies, possibly as a result of practice effects (e.g., Barth et al., 1989; Field et al., 2003; Guskiewicz et al., 1997; Jordan, Matser et al., 1996; Heilbronner et al., 1991; Macciocchi et al., 2001; Makdissi et al., 2001; Matser et al., 1998, 1991; Moser et al., 2005; Porter & Fricker, 1996, 2003; Putukian et al., 2000; Wilberger et al., 1991).

Controlled Oral Word Association Test (COWAT)

The Controlled Oral Word Association Test (COWAT) (Benton & Hamsher, 1978), consists of three one-minute trials in which the participant is given a different letter of the alphabet and has to give as many words as possible, beginning with a particular letter, in a minute. Although it is a test of verbal fluency, it also tests processing speed and executive functioning (e.g., Benton, Hamsher, & Sivan, 1989; Randolph et al., 2005). A meta-analysis revealed that among individual test instruments used to assess MTBI, the COWAT revealed the largest effect size (Mathias et al., 2004). Reliability for the COWAT ranges between $r = .70$ and $r = .88$, with practice effect sizes ranging between .30 and .52 (Ellemberg et al., 2009). Several studies have found the COWAT reveals significant differences between those with and without MTBI (e.g., Echemendia et al., 2001; Levin, Lippold et al., 1987; Raskin, Mateer, & Tweeten, 1998; Ravdin et al., 2003). Other studies have found the COWAT not sensitive to the effects of MTBI (e.g., Barr, 2003; Collins, Grindel et al., 1999; Field et al., 2003; Guskiewicz et al., 2002; Leininger et al., 1990; Matser et al., 1998, 1999, 2001; Pellman et al., 2004; Rutherford et al., 2005).

Tests of Verbal Memory

Hopkins Verbal Learning Test (HVLT)

Shapiro, Benedict, Schretlen and Brandt (1999), have revised the Hopkins Verbal Learning Test (HVLT), that was based on the California Verbal Learning Test. The HVLT is a test of verbal learning and verbal memory, and has six forms, each of 12 words. One list of 12 words is read and then the athlete's free recall is tested, following which 24 words are read and the athlete indicates if any words were from the original list. This test appears to have no practice effects (Oliaro et al., 1998). Reliability for the HVLT is $r = .78$, with practice effect sizes ranging between .24 and .30 (Ellemberg et al., 2009). Studies have found the HVLT reveals significant differences between those with and without MTBI (e.g., Echemendia et al., 2001; Preece & Geffen, 2007). In contrast, Guskiewicz et al. (1997) found no differences on the HVLT between controls and MTBI athletes - 1, 3, 5 and 10 days postinjury.

Mattis-Kovner Verbal Learning and Memory Test (list learning)

The Mattis-Kovner Verbal Learning and Memory Test developed by Mattis and Kovner in 1978, requires that after reading a list of 20 animal-related words, the testee recalls as

many words as possible. Up to eight trials are provided, with the examiner reminding the testee of words not recalled. Multiple choice type questions are used in trials 4 and 8, and after a 20-minute delay (Ruff et al., 1989). No studies on the reliability of this test could be located. Two studies have found the test showing significantly poorer scores for MTBI patients seven days post injury and with significant gains one month post injury, compared with controls (e.g., Levin, Mattis et al., 1987; Ruff et al., 1989).

Selective Reminding Test (SRT)

The Selective Reminding Test (SRT) (Hannay & Levin, 1985) consists of a list of 12 unrelated words being read to the participant, who is then required to recall as many words as possible, after which any missed words are repeated and the participant has to immediately recall the list again until the list can be recalled on 3 consecutive trials (up to a maximum of 12 trials). Thirty minutes after this, the participant is asked to recall the list (delayed condition). Although this test appears to have been used mainly in studies involving patients with severe TBI or Alzheimer's Dementia (Lezak et al., 2004), it has also been used in a study on sport-related MTBI. Hatfield et al. (2004) found that five of six ice hockey athletes with MTBI evidenced initial verbal memory decrements on the long-term storage and total recall for the SRT, with recovery ranging from 5 to 164 days.

WMS Verbal Paired Associates

A detailed description of this subtest is provided in Chapter 7, section 7.2.1. The WMS Verbal Paired Associates (VPA), developed by Wechsler in 1981, has gone through several revisions. The version used in this study is the WMS-III (Wechsler et al., 1998b). Verbal Paired Associate learning is assumed to study episodic and semantic memory, although hard-paired learning does not measure episodic memory selectively (Elwood, 1997). Reliability for the WMS-III VPA I and II for ages 18 to 34 years, ranges from r = .82 to r = .932 (Wechsler, 2002). The WMS-III VPA has been criticised for the recognition task seeming too easy, not adequately reflecting delayed auditory memory, and having a limited range of scores (Riley & Zellinger, 2000). Burton, Ryan, Axelrod, Schellenberg and Richards (2003) tested the VPA in a clinical sample, and were unable to find empirical support for the viability of a learning dimension between the immediate and delayed indices. The normative data on the WMS-III VPA, suggestive of ceiling effects, has limited this subtest's usefulness (Uttl, Graf, & Richter, 2002). Therefore, part of the rationale for change from the WMS-III to WMS-IV is that the VPA subtest had

floor and ceiling effects, making it difficult to detect association memory impairment. Another easy word pair has been added to the WMS-IV, which now has 4 learning trials of 10 words, instead of 8 words (Pearson Clinical Assessment, 2009).

Brooker (1997) found patients with early onset mild dementia scored significantly lower than MTBI patients on both the WMS-R VPA I and II. Frencham et al. (2005), in their meta-analytic review, reported impaired memory performance - including for Paired Associate Learning - within 3 months of MTBI. MTBI patients with both traumatic and non-specific MRI lesions in comparison with controls, showed significant deficits in immediate and delayed recall on the WMS-III Verbal Paired Associates I and II, respectively (Kurca et al., 2006). There are few sport-related MTBI studies using VPA. However, Shuttleworth-Edwards, Border et al. (2004) found performance on the WMS Verbal Paired Associates (Hard) tasks and delayed recall tasks, to be sensitive to the diffuse brain damage among rugby players relative to field hockey controls, at both professional and school levels. In addition, Ackerman (2000) found the WMS Verbal Paired Associates (Hard) delayed recall task sensitive to diffuse brain damage among school rugby players relative to noncontact sport controls, with evidence of greater verbal learning and memory impairment for rugby forwards than rugby backs.

Using a test similar to VPA, Pettersen and Skelton (2000) used two sets of eight word pairs (Jones, 1974), of which three pairs were abstract and five pairs were concrete. They found performance on the immediate condition and then the delayed condition 24 hours later, did not differ between controls and rugby athletes with prior MTBI.

Tests of Visual Memory

Benton Visual Retention Test for Geometric Designs

The Benton Visual Retention Test for geometric designs (Benton, 1974), requires the testee to look at and copy ten geometric designs. Following this a series of similar designs are shown for 10 seconds, which the testee has to draw from memory (Ruff et al., 1989). This test assesses visual perception and memory, and visuospatial constructional ability, with test-retest reliability being $r = .75$ (Benton, undated). Two studies have found that the test reveals significant differences between those with and without MTBI seven days postinjury (e.g., Levin et al., 1987; Ruff et al., 1989).

The Rey Complex Figure Test (RCFT)

The Rey Complex Figure Test (RCFT) (Rey, 1941) has both a copy and memory portion, similar to the WMS-III Visual Reproduction subtest. The RCFT is a measure of visuospatial ability, recall and memory, and visuospatial constructional ability, with test-retest reliability ranging from $r = .76$ to $r = .89$ (Meyers & Meyers, 2008).

Several studies have found the RCFT reveals significant differences between those with and without MTBI (e.g., Jordan, Matser et al., 1996; Leininger et al., 1990; Matser et al., 1998, 1999, 2001; McLatchie et al., 1987; Raskin et al., 1998). Other studies have not found the RCFT sensitive to the effects of MTBI (e.g., Brooks, Kupshik et al., 1987; Rutherford et al., 2005; Webbe and Ochs, 2003; Witol & Webbe, 2003).

WMS Visual Reproduction

A detailed description of this subtest is provided in Chapter 7, section 7.2.1. The WMS Visual Reproduction subtest (VR) (Wechsler, 1981), has undergone several revisions until the form used in this study - the WMS-III (Wechsler et al., 1998b). Dikmen, Heaton, Grant and Temkin (1999) found the reliability for a former VR subtest (WMS Form 1) was relatively low ($r = .62$), and reliability for the WMS-III VR I and II for ages 16 to 54 years, ranged from $r = .65$ to $r = .72$ (Wechsler, 2002). Tulsky (2003) proposed that a number of WMS-III subtests had not been sufficiently validated or tested in clinical populations, and issues have been raised about the clinical utility of the VR subtest. Part of the rationale for change from the WMS-III to WMS-IV is that the VR scoring is time consuming and complicated, and the VR was not sensitive in detecting visual deficits in younger participants. Consequently the scoring has been simplified and an additional memory subtest has been added to the WMS-IV version (Pearson Clinical Assessment, 2009).

Brooker (1997) found patients with early onset mild dementia scored significantly lower than MTBI patients on the WMS-R VR I and II. However, Ross, Casson, Siegel and Cole (1987) found scores were in the abnormal range for 11 of the 13 former and active boxers who were administered the VR subtest. Shuttleworth-Edwards, Border et al. (2004) found performance on the WMS VR sensitive to the diffuse brain damage among rugby players relative to field hockey controls, at both professional and school levels. Matser and colleagues (1998, 1999) found the WMS VR sensitive to the effects of MTBI among

soccer players. However, in a later study by Matser et al. (2001), the sensitivity of VR to MTBI was not demonstrated.

Tests of Executive Functioning

Some neurocognitive tests (e.g., COWAT, TMT and PASAT) are considered sensitive to impairments in executive functioning and in differentiating individuals with and without MTBI (e.g., Brooks et al., 1999). However, executive functions refer to several dimensions of human behaviour, and tests commonly used to assess executive functioning (e.g., WCST, Verbal Fluency and Stroop) should be regarded as measures of specific executive function processes, rather than being measures of overall executive functioning and frontal lobe functioning (e.g., Alvarez & Emory, 2006; Egger, De Mey, & Janssen, 2007).

Having discussed traditional neuropsychological assessment, including selected pencil and paper tests used in sport-related MTBI studies, and which pertain to the assessment of neurocognitive domains typically impaired as a result of MTBI, computerised neuropsychological assessment will now be discussed.

5.2.3 Computerised Neuropsychological Assessment

Some of the limitations of traditional tests have been surmounted by computerised neuropsychological test batteries, which have several advantages (e.g., Lovell & Collins, 2002). Computerised tests are designed for baseline testing of large numbers of athletes at the same time, and are less time-consuming, less labour intensive, cheaper, and provide automated scoring. Computerised tests that assess several cognitive functions, can objectively measure mild cognitive deficits and response times accurately. Postinjury test scores are automatically compared with preinjury baseline scores, with changes reflecting in the automated scoring and ideally reflecting significant changes. Computerised tests are designed for serial assessment over several test occasions, and incorporate multiple forms of randomised test items to help minimise practice effects and improve reliability (e.g., Collie, Maruff, McStephen, & Darby 2003; Iverson, Lovell, & Collins, 2003; Lovell & Collins, 2002; Pretz, 2007; Schatz, 2010; Schatz & Zillmer, 2003).

Computerised testing has limitations, being less flexible and interactive than a one-on-one assessment. Computerised testing does not take verbal responses into account, particularly spontaneous verbal responses, and therefore cannot measure verbal functioning or auditory memory (e.g., Schatz. & Browndyke, 2002; Schatz & Zillmer, 2003). Accordingly, Schatz and Zillmer (2003) view computerised testing as a sophisticated screening tool, rather than a tool that comprehensively evaluates cognitive abilities. Despite practice effects being minimised through randomised test items allowing for several alternate forms, practice effects are still a threat, even when using alternate forms, in that the athlete becomes familiar with a test format and procedure (e.g., Collie et al., 2004). One solution is to test athletes twice at baseline, and use the second test as the optimum baseline (Collie et al., 2004; Makdissi et al., 2001). However, Erlanger, Feldman and Barth (2001), in their study of reaction times, found evidence of the statistical phenomenon known as regression to the mean. They found that athletes performing fast at the first test slowed towards the group mean at the second test, and athletes performing slow at the first test, quickened towards the group mean at the second test. Therefore athletes performing quickly on reaction time at baseline, would slow at the next test occasion regardless of sustaining MTBI or not, and consequently these authors used the multiple regression statistical technique to overcome this obstacle.

Computerised tests usually employ Reliable Change Indices (RCIs) in cognitive testing, which denote statistical differences between an individual's test scores on different test occasions. RCIs are considered to be statistically valid in medical settings for clinicians to make meaningful interpretations of changes (e.g., Collie, Maruff, Makdissi et al., 2003). Collie and colleagues (2004) point out that several computerised tests employ the standard reliable change index (RCI) to denote a significant change in scores between test occasions, which does not account for confounding variables including practice effects. They suggest that computerised tests use modified RCIs to overcome this difficulty, as further explained by Hinton-Bayre et al. (1999). Adjusted RCIs are calculated to control for practice effects, whereby the predicted postinjury score equates the athlete's baseline score plus the mean practice effect demonstrated by the normative sample. This allows the clinician to observe the meaningful change that has occurred (Parsons, Notebaert, Shields, & Guskiewicz, 2009). However, Sosnoff et al. (2007) argue that mean changes are accounted for by computerised testing, but intraindividual variability is not accounted for.

Several versions of computerised neuropsychological programs have been developed for MTBI assessment since the late 1990s. These have been based on neurocognitive functions that traditional tests have revealed as sensitive to the neuropsychological effects of MTBI, e.g., attention, memory, reaction time and processing speed, and some include an assessment of symptoms associated with MTBI (Podell, 2004; Sosnoff et al., 2007). The following four commonly used programs will be briefly described: Automated Neuropsychological Assessment Metrics (ANAM), CogSport (CogState), Headminder Concussion Resolution Index (CRI) and Immediate Postconcussion Assessment and Cognitive Testing (ImPACT), which is used in this study (e.g., Aubry et al., 2002; Sosnoff et al., 2007).

ANAM (Automated Neuropsychological Assessment Metrics)

The Automated Neuropsychological Assessment Metrics (ANAM), measures a variety of cognitive effects, ranging from radiation exposure to TBI (Bleiberg, Garmoe, Halpern, Reeves, & Nadler, 1997; Bleiberg. Kane, Reeves, Garmoe, & Halpern, 2000; Jones, Loe, Krach, Rager, & Jones, 2008; Reeves, Thorne, Winter, & Hegge, 1989). Bleiberg, Halpern, Reeves and Daniel (1998) proposed that ANAM be used in assessing MTBI. The ANAM battery of tests has changed over time, and currently the ANAM sports medicine battery (ASMB) for MTBI surveillance and management includes the following indices: continuous performance test, simple reaction time, procedural reaction time, code substitution, code substitution - delayed, Sternberg memory procedure, mathematical processing, spatial processing, and matching to sample (Cernich, Reeves, Sun, & Bleiberg, 2007). The simple reaction time and continuous performance indices are sensitive to MTBI (Bleiberg et al., 2004). The ASMB reveals adequate concurrent validity in measuring similar constructs measured by traditional tests, for example the COWAT, Digit Symbol, HVLT, PASAT, Stroop Colour Word Test and TMT A and B, in addition to postconcussive symptoms (Bleiberg et al., 2000; Schatz & Putz, 2006; Woodard et al., 2002). Use of the RCI, developed for the test, has revealed high specificity to MTBI, but low sensitivity (72.6% but with 27.5% false positives). However, the mathematical processing subtest revealed 100% sensitivity to MTBI with no false positives (e.g., Cernich et al., 2007; Parsons et al., 2009). Test-retest improvements after a four-month interval have been found among adolescents in processing efficiency on ANAM, and it was advocated that the practice effects needed to be addressed (e.g., Cernich et al., 2007; Daniel et al., 1999). Studies using ANAM have found female and

male high school athletes revealed impairments on reaction time and processing speed for up to six days, and memory impairment up to ten days post MTBI (Sim, Terryberry-Spohr, & Wilson, 2008). Fifty-five percent of athletes diagnosed with MTBI have demonstrated a decrease in the ANAM composite score within 48 hours post MTBI (Guskiewicz, Mihalik et al., 2007). Bleiberg and colleagues (2004) conducted a prospective study using ANAM. They found cognitive impairment in those suffering boxing-related MTBI, in comparison to controls from the same baselined population, one to two days post MTBI, with recovery occurring between three to seven days post MTBI.

CogState Sport

CogState Sport (previously termed CogSport), a computerised web-based test battery, uses a pack of playing cards as the visual stimulus to assess cognitive changes, and includes a symptom checklist (e.g., CogState Sport, 2010; Schatz & Zillmer, 2003). The test is available in 17 different languages. Preseason baseline testing is required, against which postinjury measures can be compared. The emailed report provides scores for four cognitive domains: psychomotor processing speed, visual attention, visual learning and memory, and verbal learning and memory (CogState Sport, 2010). The test measures fluctuations in attention and performance variability as indicators of MTBI, which the test developers propose is more sensitive than performance changes on traditional tests (Makdissi et al., 2001). CogState Sport tasks are reported as reliable by the test developers (CogState Sport, 2010). Its measures have been found to correlate with the DSST and TMT A and B, but not Digit Symbols. However, CogState Sport's own two different memory measures were found to correlate (e.g., Collie, Darby, & Maruff, 2001; Schatz & Putz, 2006). No studies on the specificity or sensitivity of CogSport are available. Practice effects have been evidenced over a brief test interval and the use of playing cards might influence outcomes, particularly as some persons may have had more exposure to playing card games (e.g., Collie, Maruff, Darby, & McStephen, 2003).

CogSport is primarily used for return to play decisions (Schatz & Putz, 2006). Ninety two percent of Australian Rules football players with MTBI, who were tested on CogState, were able to return to play within a week (Makdissi et al., 2001). Apart from amateur boxers whose contests were stopped by the referee, none of the other boxers participating in several bouts over a week evidenced cognitive impairment on CogState Sport (Moriarity et al., 2004). No correlations were found between cognitive impairment on

CogState Sport and self-reported lifetime concussion history, or with exposure to heading the soccer ball among Norwegian professional soccer players (Straume-Naesheim, Andersen, Dvorak, & Bahr, 2005).

CRI (Headminder Concussion Resolution Index)

Headminder Concussion Resolution Index (CRI) is a computerised online test battery, comprising multiple alternate forms, and is designed to assess cognitive functions associated with sport-related MTBI. It includes a symptom checklist in addition to a questionnaire pertaining to demographic information, and medical and sport-playing histories. Preseason baseline testing is required, against which postinjury measures can be compared. It comprises six subtests measuring reaction time, information processing speed and visual recognition, from which three factors are derived: simple reaction time, complex reaction time and visual scanning/psychomotor speed. The test has been found valid and reliable in measuring cognitive performance (psychomotor speed and information processing) in a heterogeneous group of athletes between the ages of 13 and 35 years following sport-related MTBI (Erlanger, Feldman et al., 2003). CRI evidences 78.6% sensitivity to MTBI (e.g., Broglio, Macciocchi, & Ferrara, 2007b; Ellemberg et al., 2009). CRI reveals adequate concurrent validity in measuring similar constructs measured by traditional tests, for example TMT A and B, and the WAIS III Digit Span and Digit Symbol (Erlanger, Saliba et al., 2001). Within 48 hours of MTBI athletes, tested on the CRI, demonstrated impairment on simple reaction time across all tasks, and were less accurate on the cued response time compared to baseline performance and controls (Sosnoff et al., 2007). However, when controlling for mean reaction time, the group differences were eliminated and response time variability was shown to increase for athletes with MTBI. The authors propose that MTBI is responsible for slowed reaction time, but not elevated intraindividual variability, and therefore mean RT and RT variability are two separate neurocognitive mechanisms.

ImPACT (Immediate Post-concussion Assessment and Cognitive Testing)

The Immediate Post-concussion Assessment and Cognitive Testing (ImPACT) computer based test program is described in detail in Chapter 7, section 7.2.1. In summary, the ImPACT assessment battery has undergone development since 1994 and was released for research purposes in 1998. The first version of ImPACT had a unitary memory composite. ImPACT version 2.0 was released in 2002, with an additional component,

Design Memory. ImPACT version 3.0 was released in 2004 with both percentile and Reliable Change Index (RCI) scores incorporated (Iverson, Lovell, & Collins, 2010).

ImPACT randomises test item presentation, such that version 3.0 has five alternate forms. It includes five neurocognitive test modules, providing composite scores for the neuropsychological functions typically affected by MTBI. These are Visual Memory, Verbal Memory, Visual Motor Speed, and Reaction Time, and Impulse Control that is a validity measure. Additionally, ImPACT has a Postconcussion Symptom Scale comprising 22 common MTBI symptoms (Iverson et al., 2003).
ImPACT is available in 13 different languages, including Afrikaans, Czech, English, Finnish, French, German, Italian, Mandarin, Norwegian, Portuguese, Russian, Spanish and Swedish (ImPACT, 2010).

Shuttleworth-Edwards, Whitefield-Alexander, Radloff, Taylor and Lovell (2009) have empirically validated the American neurocognitive normative data for ImPACT as being appropriate for English speaking males in South Africa. However, clinically relevant effect sizes were revealed for higher symptom scores for the South African rugby players in comparison with American athletes, that would need to be taken into account for clinical interpretation purposes. The important finding of this cross-cultural norming study is the finding of equivalence for the ImPACT neurocognitive composite norms between the USA normative sample and the South African sample of males, including non-white males who have received an advantaged education and are proficient in English (Shuttleworth-Edwards, Whitefield-Alexander, & Radloff, in press). Kontos, Elbin, Covassin, and Larson (2010), recently found that African Americans and White Americans did not differ significantly on ImPACT version 2.0 neurocognitive and symptom composites at baseline. However, the African Americans evidenced a significant decline from baseline performance on at least one neurocognitive composite, and scored lower on the visual motor speed composite, 7 days post MTBI. Therefore, ImPACT offers sound construct validity with cultural equivalence for baseline testing, but norms for post MTBI assessment require further research (Kontos et al., 2010).

ImPACT reveals adequate concurrent validity in measuring similar constructs measured by traditional tests, including the SDMT that was found to measure the same underlying construct as the ImPACT Visual Motor Speed and Reaction Time composites, the Brief

Visuospatial Memory Test, and the TMT A and B. However, ongoing validation of ImPACT is imperative for clinical inferences to be derived from testing (e.g., Iverson, Franzen et al., 2003; Iverson, Lovell, & Collins, 2005). Furthermore, the Impact Verbal Memory, Visual Memory and Visual Motor Speed composites have revealed sensitivity to MTBI, and adequate test-retest reliability (Iverson et al., 2004b; Iverson, Lovell, & Collins, 2003, 2005; Schatz, Pardini, Lovell, & Collins, 2006).

ImPACT shows 79.2 - 81.9% sensitivity to MTBI in terms of neurocognitive impairment or increased symptom reports, and 62.5% sensitivity if the symptom scale is excluded (e.g., Broglio, Macciocchi et al., 2007b; Schatz et al., 2006). In contrast, a few studies have failed to reveal this sensitivity of ImPACT to the long-term neuropsychological effects of MTBI (e.g., Broglio, Ferrara, Piland, Anderson, & Collie, 2006; Collie, McCrory, & Makadissi, 2006; Iverson, Brooks, Lovell, & Collins, 2006; Pontifex et al., 2009). These particular findings are suggestive of ImPACT being more sensitive to neurocognitive changes immediately following MTBI, rather than subtle chronic changes that may persist. However, cognitive testing that requires executive or cognitive control (i.e., flanker task response accuracy) has been found more sensitive than ImPACT in eliciting long-term neurocognitive impairment resultant of MTBI (Pontifex et al., 2009).

In respect of limitations, the ImPACT program should not be administered immediately after a match or practice session, as maximal exercise prior to ImPACT administration negatively affects the immediate and delayed verbal memory scores (Covassin, Weiss, Powell, & Womack, 2007). Other possible limitations of ImPACT are i) the Visual Memory test designs have abstract rather than the 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 is memory recall (e.g., Lezak, 1995). Therefore, traditional neuropsychological tests that incorporate these crucial aspects might reveal greater sensitivity to MTBI than ImPACT.

An overall comparison of three of these computerised neurocognitive batteries has been made. Schatz and Putz (2006) investigated the construct validity between Digit Symbols, TMT A and B and three of the above computerised programs (ANAM was not included), and found significant correlations for complex reaction time between ImPACT and

CogSport and between ImPACT and the CRI, but not between CogSport and the CRI. There appeared to be no other correlations between these three computerised programs, for the measures of simple reaction time, memory and processing speed.

Overall criticism of MTBI neuropsychological measures is that subtle MTBI cognitive deficits are not always identified (Baker & Hutchinson, 2008). Randolph et al. (2005) literature review (1990-2004) of traditional and computerised neuropsychological tests, found that none of the tests reviewed met all the psychometric criteria to warrant their clinical use in the management of sport-related MTBI. This suggested that neuropsychological tests currently used, require further sensitivity, reliability and validity studies in assessing MTBI (e.g., Randolph et al., 2005, 2006). Furthermore, some authors doubt that neurocognitive recovery follows symptom recovery. This is on the grounds that, neurocognitive impairment in the absence of symptoms one week post MTBI, has not been demonstrated in a significant number of concussed athletes. Therefore, these authors are not in favour of neurocognitive testing while the athlete is symptomatic, because neurocognitive testing adds nothing to return to play decisions (e.g., Belanger & Vanderploeg, 2005; McCrea et al., 2003; McCrory, Johnston et al., 2005; Randolph et al., 2005, 2006). Randolph et al. (2005, 2006) propose that neurocognitive assessment requires conservative interpretation, and that the clinical examination and the use of standardised symptom checklists should be used for monitoring recovery from MTBI (Randolph et al., 2006). In contrast, Lovell (2006) argues that athletes should not be asymptomatic prior to neuropsychological testing, on the grounds that (i) the ImPACT test led to a 26% improved diagnostic yield compared with the evaluation of symptoms alone in differentiating concussed athletes from nonconcussed athletes, and that (ii) neuropsychological assessment contributes towards the management of athletes during the early stage of recovery (e.g., Lovell, 2006; Van Kampen et al., 2006).

In view of the above arguments, and in light of recommendations that athletes should be both neurocognitively recovered and asymptomatic in order to return to play, symptom measures as a means of monitoring MTBI recovery will now be discussed.

5.3 SYMPTOM ASSESSMENT IN SPORTS MTBI

The symptoms that commonly occur following MTBI were detailed in Chapter 3. This section will focus on the assessment of symptoms, initially in terms of assessment issues pertinent to the monitoring of symptoms, following which the more commonly used traditional pencil and paper and computerised neuropsychological symptom measures will be described, and related findings elucidated.

5.3.1 General Assessment Issues

The assessment of self-reported symptoms (SRS) is one aspect of sport-related MTBI (Piland, Ferrara, Macciocchi, Broglio, & Gould, 2010). It is well documented that SRS are unreliable, and that athletes tend to over-report symptoms preinjury, and under-report symptoms postinjury (Field et al., 2003). Not all SRS measures involve baseline testing, which is problematic if they are the only diagnostic measure postinjury, particularly considering that symptoms occur in healthy persons and other conditions (e.g., Bigler, 2008; Binder, 1986; Gouvier et al., 1992; Iverson & Lange, 2003). Piland et al. (2010) propose that a SRS preinjury assessment assists in determining symptom resolution postinjury, which will now be discussed.

Baseline Testing

Athletes are known to over-report preinjury symptoms (e.g., Field et al., 2003; Piland et al., 2010). For example, Piland and colleagues (2010) provide baseline SRS reports in nonconcussed individuals from five studies. The studies indicate the percent of individuals assessed who report on particular symptoms at the preinjury baseline assessment. The ranges are as follows: headache (35 to 59%), nausea (12 to 42%), difficulty balancing (9 to 11%), difficulty concentrating (19 to 77%), drowsiness (31 to 39%), fatigue (30 to 89%), feeling in a fog (12 to 14%), feeling slowed down (23 to 28%), and trouble falling asleep (23 to 75%) (e.g., Chan, 2001; Iverson & Lange, 2003; Piland et al., 2010; Piland, Motl, Ferrara, & Peterson, 2003; Piland et al., 2006). Piland et al. (2010) did not find significant gender differences in SRS, but showed that the most common SRS at baseline (fatigue, headache, difficulty concentrating, drowsiness and trouble falling asleep) were also among those most frequently reported at post MTBI assessments (e.g., Collins, Iverson et al., 2003; Piland et al., 2003). A pertinent finding

from the Piland et al. (2010) study is the importance for the clinician to examine pre-existing variables when conducting SRS baseline testing. This is because their results demonstrate a higher level of baseline symptoms among those who had participated in a physical workout, had an orthopaedic injury, were ill, fatigued, or had a history of MTBI compared with those who had not. Other studies lend further support to MTBI history influencing SRS, and show that athletes with a history of three or more MTBIs have an increase in baseline SRS (Collins et al., 1999; Gaetz et al., 2000; Guskiewicz et al., 2000; Iverson et al., 2004a).

Postconcussive Follow-up Testing

An athlete needs to be asymptomatic before embarking on a graded exertion program and return to play. However, athletes are frequently motivated to return to play and therefore tend to under-report symptoms post MTBI (e.g., Broglio, Macciocchi, & Ferrara, 2007a; Lovell et al., 2004; McDaniel, 2010; Pretz, 2007). This needs to be taken into account when postinjury and preinjury scores are compared and therefore the clinician is required to differentiate between the baseline symptoms and postinjury symptoms that are attributable to the recent MTBI event (Piland et al., 2010). An important factor when conducting MTBI follow-up testing also relates to the findings of Pellman et al. (2006), in which professional athletes report fewer symptoms when evaluated day 1 and day 3 postinjury, which may relate to job security (Ellemberg et al., 2009). Furthermore, gender differences have been found, with female athletes reporting significantly more symptoms post MTBI compared with male athletes (Broshek et al., 2005).

5.3.2 Pencil and Paper Assessment of MTBI Symptoms

Symptom Check Lists generally require a yes or no response to symptoms being present, while Symptom Scales are typically summative and incorporate Likert-type scales that may permit capture of symptom duration, intensity and severity. Symptom Scales provide a superior means of symptom assessment, in that (i) an athlete is more apt to report symptoms on a graded scale than on a yes or no checklist, and (ii) bias is reduced when pre- and post- injury measures can be compared (e.g., Guskiewicz et al., 2004; Halstead & Walter, 2010; Piland et al., 2010). The following symptom measures appear in the literature. However, given the dearth of literature pertaining to symptom measures in

MTBI, it is not known how commonly these scales are used following sport-related MTBI.

Graded Symptom Checklist (GSC)

The National Athletics Trainers' Association recommended the Graded Symptom Checklist (GSC) be used in the USA (Ellemberg et al., 2009; Guskiewicz et al., 2004). The GSC has been found 89% sensitive to MTBI in one study, and in another study it was found reliable in identifying MTBI symptoms in brain injured 6 to 18 year olds, compared with controls (Grubenhoff, Kirkwood, Gao, Deakyne, & Wathen, 2010; Valovich McLeod, 2009).

Head Injury Scale (HIS)

The Head Injury Scale (HIS) formerly comprised 16 symptoms, but Piland et al. (2003) found the three-factor model (somatic, neurobehavioural and cognitive) provided a good fit to scores from the 16-item symptom scale, such that when modified to include a 9-item scale, the three-factor model fit was excellent and construct validity was established. Therefore, the HIS is now a 9-item summative 7-point Likert-type measure of the overall duration of MTBI related symptoms over a 24-hour period (Peterson et al., 2003; Piland et al., 2003, 2010). Two day test-retest reliability has been established, internal consistency demonstrated, and the HIS 9-item stability was $r = 0.85$ (Piland et al., 2010). Factorial validity supported the latent relationships of the cohesive constellation of cognitive, somatic and neurobehavioural symptoms in the absence of confounding variables (Piland et al., 2010). Mrazik et al. (Personal Communication Chris Paniak, September 21, 2007) found the original HIS sensitive to classifying 90% athletes with MTBI correctly, but revealed a 10% false positive rate.

Postconcussion Rating Scale (PCRS)

The Postconcussion Rating Scale (PCRS) consists of 21 symptoms modified by Lovell and Collins (1998), and endorsed by the CISG panel at the First International Conference on Concussion in Sport in 2001 (Aubry et al., 2002). Pretz (2007) found a significant difference between the genders in symptom reports on the PCRS, in that males evidenced a higher symptom score than females at baseline, which the author believed was due to the lingering effects of previously unreported MTBI events.

The Rivermead Post Concussion Symptoms Questionnaire (RPQ)

King, Crawford, Wenden, Moss and Wade (1995) developed the Rivermead Post Concussion Symptoms Questionnaire (RPQ). This questionnaire is a 16-item summative 0-4 point Likert-type measure, of the overall duration of MTBI related symptoms over a 24-hour period. The RPQ does not tap into a single underlying construct. Three symptoms headache, dizziness and nausea, measure one construct, and the remaining 13 symptoms measure a separate construct. Each construct has shown good external construct validity and good test-retest reliability, in measuring TBI symptoms three months postinjury (Eyres, Carey, Gilworth, Neumann, & Tennant, 2005). There is also a nine-item version of this test. The nine symptoms were shown to load partially on three factors, cognitive, emotional and somatic (Potter, Leigh, Wade, & Fleminger, 2006), and also on three factors, mood and cognition, general somatic and visual somatic (Hermannn et al., 2009). This test is also used to measure symptoms of depression (e.g., Herrmann et al., 2009; Stalnacke, 2009). However, MTBI patients have shown a greater degree of symptoms reports on the RPQ than TBI patients three months postinjury (Sigurdardottir, Andelic, Roe, Jerstad, & Schanke, 2009). Furthermore, cognitive symptoms were more prominent than physical and behavioural symptoms, on the RPQ, at 3, 6 and 12 months post MTBI (Roe et al., 2009).

5.3.3 Computerised Assessment of MTBI Symptoms

Although it is indicated that the ANAM sports medicine battery (ASMB) has a self-report symptom section, there appears to be no research pertaining directly to this instruments' symptom measure. Therefore, only the CogState Sport, CRI and ImPACT symptom measures will be described.

CogState Sport Symptom Checklist

No research could be located pertaining directly to CogState Sport's symptom measure and reliability studies. However, studies reporting on the symptom checklist have been carried out on Australian footballers. Within 11 days of MTBI, based on whether athletes reported any symptoms on the 14-item symptom checklist, they were divided into symptomatic or asymptomatic groups. The symptomatic group reported an average 3.98 symptoms post MTBI. Headache was the most common symptom. Symptoms resolved between 2 -240 hours (mean 60.6 hours). The symptomatic group slowed on reaction time

(simple, choice and complex) but did not differ on TMT B or DSST scores, in comparison with the asymptomatic MTBI group or footballer controls. The symptomatic group declined significantly on CogState Sport tests of motor function and attention versus baseline testing, whereas the asymptomatic group showed impairment on a test of divided attention only (Collie, Makdissi et al., 2006). Another study, finding simple reaction time impaired in six concussed footballers, reported headache as the most common symptom. Headaches began six hours post MTBI in four athletes and persisted for up to four days in three athletes. Otherwise, fatigue/lethargy was the most persistent symptom. Other symptoms reported were blurred vision, confusion, dizziness and nausea. All symptoms resolved within four days (Makdissi et al., 2001). In elite senior, junior and community football players, post MTBI symptoms lasted an average 48.6 hours, with headaches lasting up to 60 hours in some athletes. Symptoms recovered parallel to recovery on DSST and TMT B scores, however cognitive impairments on CogState Sport tests were found two to three days following symptom recovery (Makdissi et al., 2010).

CRI (Headminder) Neurophysiologic Symptom Scale

Articles relating to this instrument and SRS are limited. Erlanger et al. (2001) reported that athletes experiencing MTBI were monitored on the CRI Neurophysiologic Symptom Scale, and 77% evidenced neurophysiologic symptoms within 1 to 2 days of injury and symptom resolution occurred within 15 days, with the CRI appearing effective in multiple test administrations over extended and short time intervals. Erlanger et al. (2003) found a concordance between MTBI severity, CRI neurocognitive scores and subjective neurophysiologic symptom reports.

ImPACT Symptom Scale

The ImPACT symptom scale was originally developed as the Post-Concussion Scale for the Pittsburgh Steelers MTBI management program in the late 1980s, and variants have since been adopted by the National Football and Hockey Leagues, and has been used in several secondary schools and colleges, and incorporated into the Impact program (Lovell, Iverson, Collins, Podell, Johnston, Pardini et al., 2006). The Post-Concussion Scale, i.e., the ImPACT Symptom Scale, is a 22-item summative 7-point Likert-type measure of the severity of symptoms experienced on the day of testing (Lovell et al., 2006).

Normative data for the ImPACT symptom scale were derived from a sample of 894 high school and 1,295 university students, of which most were healthy at the time of assessment. Some 115 high school and university athletes were assessed at both two and four days post MTBI. Differences on the symptom scale were attributable to gender (females tended to report more symptoms than males) and self-reported history of learning difficulties (those with learning difficulties tended to report more symptoms than those without). Accordingly, norms were stratified according to high school/university, gender and learning status. The natural distribution of scores for these groups tended to be skewed, but were assigned as uniform percentile ranks for the classification ranges of low-normal, normal, unusual, high and very high. The RCIs were calculated at the .80 and .90 confidence level. Reliable change methodology, that relies on the standard error of the differences in scores, was used for interpreting change following MTBI. However, postconcussive symptoms change rapidly over a short duration, in that following MTBI there is a dramatic change from baseline of increased symptom reports, followed by a rapid improvement, and therefore the reliable change methodology has limitations in clinical practice. It is proposed that at the second postconcussion follow-up, if an athlete's post MTBI symptom score decreases by 10 or more points, then improvement can be assumed. However, should the symptom score increase by 2 or more points, this should alert the clinician because athletes seldom obtain progressively worse symptom scores over the follow-up post MTBI test-retest interval (Iverson, Lovell, & Collins, 2010).

The ImPACT symptom scale evidenced high to very high internal consistency reliability among healthy students (secondary school and college) and athletes with MTBI (r = .88 to .94, and r = .93, respectively), with females endorsing more symptoms than males at baseline. The most frequent symptoms (endorsed by 60 to 79% of sample athletes post MTBI) were: headache, fatigue, feeling slowed down, feeling mentally foggy, drowsiness, difficulty concentrating, and dizziness. The least frequent symptoms endorsed by less than 25% of the athletes post MTBI were nervousness, feeling more emotional, numbness or tingling, sadness and vomiting. Athletes were assessed within 72 hours, between 4 to 8 days, and between 7 to 30 days post MTBI, and although considerable variability in symptom reports was evidenced at each test interval, there was a linear reduction in total symptoms across the three test occasions, and most athletes were asymptomatic relative to baseline at the third test occasion (Lovell et al., 2006).

Addition of the symptom scale has increased the sensitivity of the ImPACT test battery to MTBI from 62.5% to 79.2% (Broglio et al., 2007b). The many MTBI studies employing ImPACT, which includes the symptom scale composite, have evidenced an increased score on the symptom composite following MTBI, compared to baseline testing or compared with controls (e.g., Broglio, Macciocchi, & Ferrara, 2007b; Collins, Grindel et al., 2005; Collins, Field et al., 2003; Collins, Iverson et al., 2003; Iverson et al., 2004b; Lovell et al., 2006). Recently, Lau, Lovell, Collins, and Pardini (2009) found a migraine headache and reduced reaction time associated with longer MTBI recovery duration, which they consider might support symptom clusters having predictive value in diagnosing and managing MTBI. Schatz, Neidzwski, Moser and Karpf (2010) found female athletes endorsed more symptoms at baseline and the endorsement of more baseline symptoms was also associated with decreased visual memory scores.

5.4 OVERVIEW

Overall, the aim of this chapter was to provide a detailed background on the management and assessment of sport-related MTBI in two sections. These would incorporate (i) medical assessment and management that included sideline evaluation, medical evaluation (including specific investigations of balance testing, biomarkers, neuroimaging and electrophysiological evaluation), general medical management of MTBI and decisions regarding the return to play of sport, and (ii) neuropsychological assessment and management. Neuropsychological assessment was discussed in terms of neurocognitive assessment that covered general assessment issues and traditional paper and pencil and computerised tests commonly used in sport-related MTBI neurocognitive assessment. Following this, the neuropsychological assessment of postconcussive symptoms was covered in terms of the traditional and computerised measures used to assess typical MTBI symptoms. Neuropsychological assessment not only informs return to play decisions, but assists with the medical management of the athlete, in ensuring continued cognitive and physical rest, and tracking of recovery. Knowledge of the various measures used to assess sport-related MTBI neurocognitive sequelae and resultant symptoms, helps interpret the various findings of sport-related MTBI research, that will be discussed in the next chapter.

CHAPTER SIX: SPORT-RELATED MILD TRAUMATIC BRAIN INJURY RESEARCH

This chapter reviews the neuropsychological findings and limitations of sport-related mild traumatic brain injury (MTBI) research. The first section reviews findings of sport-related MTBI research pertaining to the cognitive functions of attention and concentration, information and visuomotor processing speed, reaction time, memory, and executive functioning, in that these functions are most commonly impaired as a result of MTBI. The second section reviews findings of sport-related MTBI research pertaining to symptom presentation. The third section involves a comprehensive review of neuropsychological studies on MTBI in rugby union. Following this, the methodological limitations of neuropsychological research in sport will be examined. The aforementioned topics serve as a background for the rationale and hypotheses for the present study, which are presented at the end of the chapter.

Pertinent literature pertaining to neuropsychological studies on MTBI in sport among the team sports of ice hockey, soccer, American football, Australian football, rugby league, and rugby union has been reviewed, including those studies employing the ImPACT test battery and WMS-III Verbal Paired Associates and Visual Reproduction measures used in this study. The review is extensive, but is not exhaustive. Where possible, high school, collegiate or university, club and professional levels of play are differentiated, however some studies include several levels or do not differentiate between the levels of play among their samples. When describing findings pertaining to the acute and chronic conditions of MTBI, as per the earlier discussion (Chapter 3, section 3.6.3) this refers to the effects that generally resolve within three months postinjury (acute) or following three months postinjury (chronic) (e.g., Barth et al., 1989; Bernstein, 2002; Frencham et al., 2005; Schretlen & Shapiro, 2003; Segalowitz et al., 2001; Shuttleworth-Edwards & Radloff, 2008; Van der Naalt, 2001; Vanderploeg et al., 2005).

6.1 NEUROCOGNITIVE FINDINGS FOR SPORT-RELATED MTBI

The cognitive functions typically demonstrating impairment following MTBI, including sport-related MTBI, are: diminished attention, immediate memory or working memory skills (Echemendia et al., 2001; Erlanger et al., 2003; Frencham et al., 2005; Gentilini et

al., 1985; Killam et al., 2005; King, 1997; Raskin et al., 1998; Webbe & Ochs, 2003; Witol & Webbe, 2003), reduced information processing speed (e.g., Barth et al., 1989; Echemendia et al., 2001; Hinton-Bayre et al., 1997, 1999; Hinton-Bayre & Geffen, 2004; Mathias et al., 2004; Peterson et al., 2003; Webbe & Ochs, 2003), decreased psychomotor or visuomotor processing speed (e.g., Covassin et al., 2008; Erlanger, Feldman et al., 2003; Shuttleworth-Edwards & Radloff, 2008), slowed reaction time (Broshek et al., 2005; Collins, Field et al., 2003; Erlanger et al., 2003; Gentilini et al., 1989; Hinton-Bayre et al., 1999; Maddocks & Saling, 1996), impaired learning and memory (e.g., Binder et al., 1997; Collins, Field et al., 2003; Echemendia et al., 2001; Erlanger et al., 2003; Field et al., 2003; Frencham et al., 2005; Guskiewicz et al., 2001; Hugenholtz et al., 1988; Leininger et al., 1990; Lovell & Collins, 1998; Lovell, Collins, Iverson et al., 2003, 2004; Matser et al., 1998, 1999, 2001; Pellman, Lovell et al., 2004; Stuss et al., 1985; Webbe & Ochs, 2003), and those pertaining to executive functioning including decreased decision making speed (Erlanger et al., 2003; Hinton-Bayre et al., 1999) or reduced ability to plan and switch between mental sets (Matser et al., 1999).

Commonly documented neurocognitive functions impaired as a result of MTBI were delineated in Chapter 3. The review of specific *sport-related* MTBI studies (which is the purpose of this chapter) will be presented in order, according to the following functional parameters: attention and concentration, information processing and visuomotor processing speed, reaction time, memory, and executive functioning. Where possible, findings relating to acute MTBI will be presented before those pertaining to chronic MTBI.

6.1.1 Impairments in Attention and Concentration

For acute MTBI, Moser et al. (2005) found recently concussed high school athletes performed worse on attention and concentration measures than athletes with no MTBI history. Among mixed gender college athletes compared with controls, Echemendia et al. (2001) found significant impairment on traditional attention and concentration measures (the battery also included the Digit Span, HVLT, and Stroop tests) two hours post MTBI, and on the divided attention measures 48 hours post MTBI. However, these impairments were not reported one week post MTBI. Recently concussed collegiate ice hockey players exhibited the greatest impairment for immediate memory, that measures attention,

compared with athletes not recently concussed or with no MTBI history (Killam et al., 2005). Belanger and Vanderploeg (2005) in their meta-analysis of sport-related MTBI studies did not find a statistically significant effect size for the attention function.

For chronic MTBI, although no significant neurocognitive differences at baseline were revealed between previously concussed and nonconcussed amateur ice hockey players, aged 15 to 18 years, poorer performance approaching significance was revealed by the previously concussed group for the Digits backwards attention measure (Hatfield et al., 2004). Poorer performance on measures of attention has been associated with the number of prior MTBI events (e.g., Matser et al., 2001). Further evidence supporting the cumulative effects of MTBI on attention was found among soccer and rugby players, aged 13 to 16 years, who had not sustained MTBI within three months, and scores on the test of Performance Covert Attention Shift Accuracy reduced by 1.5% for each previous MTBI incident (Stephens, Rutherford, Potter, & Fernie, 2010).

In summary, attention appears to be affected by MTBI in both the acute and chronic phases. Divided attention appears to be a more sensitive measure within 48 hours of MTBI, although attention deficits on specific measures have been found in the chronic phase.

6.1.2 Reduced Speed of Information Processing and Visuomotor Processing

Among American football athletes, Wilberger et al. (1991) found that 75% of high school athletes evidenced information processing deficits at 24 hours, 61% at one month and 55% at three months post MTBI, while at college level Macciocchi et al. (1996) found athletes failed to show improvement within five days of MTBI on visuomotor and information processing tasks, to the extent that controls did. The latter finding concur with those of Barth et al. (1989) and McCrea et al. (2003), who found subtle differences for information processing among USA football players, which returned to baseline level within 5 to 10 days post MTBI. In contrast, a faster recovery on information processing speed was reported by Echemendia et al. (2001), who found significantly impaired performance on the measures of information processing speed 48 hours post MTBI among male and female college athletes, compared with controls, although this was not evidenced two hours and one week post MTBI. Among professional rugby league

players, Hinton-Bayre et al. (1997) also found information processing speed impaired within 48 hours post MTBI. However, Hinton-Bayre et al. (1999) found that 80% at 1 to 3 days and 35% at 1 to 2 weeks post MTBI, revealed information processing speed impairment, with recovery to baseline level taking 3 to 5 weeks. Hinton-Bayre and Geffen (2002) found impaired information processing speed (measured on the Speed of Comprehension, DSST and Symbol Digit tests) the most reliable cognitive indicator of MTBI, days two and ten postinjury, among 175 concussed rugby league players. Impairment appeared unrelated to LOC, symptom duration or MTBI history in the previous year, however impairment appeared associated with PTA at day two postinjury (Hinton-Bayre & Geffen, 2002). Peterson et al. (2003) found information processing speed and composite balance measures remained impaired up until ten days post MTBI, compared with controls.

On the ImPACT test, scores on the Visual Motor Speed composite were significantly reduced for athletes with a migraine, and reduced for those with a headache compared to those not reporting these symptoms within six days post MTBI (Mihalik et al., 2005). High school athletes reporting subjective fogginess versus no fogginess had significantly reduced scores on the Visual Motor Speed composite within 6.8 days post MTBI (Iverson et al., 2004b). In contrast, Lovell, Collins, Iverson, Johnston and Bradley (2004) found high school athletes' visual motor speed slowed slightly and not significantly within 36 hours of MTBI, and then improved significantly on baseline performance six days post MTBI.

Both a learning disorder and a history of MTBI have been independently associated with lowered baseline performance on information processing speed among American football players (Collins, Grindel et al., 1999). In respect of the chronic, residual effects of MTBI, rugby players at school level through to adult national level, showed significantly poorer performance than control participants on tests of visuomotor processing measured on the DSST and TMT A and B (Shuttleworth-Edwards & Radloff, 2008). Matser et al. (2001) found poorer performance on measures of visuomotor processing associated with the number of prior MTBI events.

The Symbol Digit, DSST and Speed of Comprehension Test are frequently used to measure information processing speed (Hinton-Bayre at al., 1999). The DSST is sensitive to the effects of MTBI within the first 24 hours, however practice effects have been found for the second test occasion, that need to be considered in order not to assume recovery erroneously (e.g., De Monte, Geffen, & Kwapil, 2005; De Monte et al., 2006; De Monte, Geffen, May, & McFarland, 2004; Makdissi et al., 2001). Erroneous assumption of recovery on traditional measures, is supported by findings of Makdissi et al. (2001), in which 5 of the 6 athletes' scores on the DSST and all 6 athletes' scores on the TMT B improved on baseline scores within 72 hours of MTBI, however reaction time scores on computerised testing were impaired. Additionally, Makdissi et al. (2010) found Australian football players' scores on DSST and TMT B recovered within the time of symptom resolution (average 49 hours post MTBI), however neurocognitive deficits on computerised testing were found among 35% of players 2 to 3 days post MTBI.

In summary, information processing deficits are more apparent within the first 48 hours post MTBI, with a faster recovery between 5 to 14 days, and a gradual recovery thereafter, although deficits appear to persist beyond three months. Information processing deficits have been shown to differentiate athletes with and without a history of MTBI. Practice effects on the measures used, need to be considered in order not to inadvertently assume recovery.

6.1.3 Reaction Time Deficits

Sport-related MTBI research demonstrates that reaction time is sensitive to the acute effects of MTBI (e.g., Collie, Makdissi et al., 2006; Collins, Field et al., 2003; Cremona-Meteyard & Geffen, 1994; Iverson et al., 2004b; Maddocks & Saling, 1996; Majerske et al., 2008; Makdissi et al., 2001, 2010; Sosnoff et al., 2007). The negative effects of MTBI on reaction time have been revealed in studies among American football players (Collins, Field et al., 2003; Iverson et al., 2004b), and Australian football players (e.g., Collie, Makdissi et al., 2006; Cremona-Meteyard & Geffen, 1994; Maddocks & Saling, 1996; Makdissi et al., 2001, 2010). Gender differences for reaction time have been revealed. Broshek et al. (2005) found female athletes demonstrated a significantly greater decline in simple and complex reaction times relative to baseline, and in comparison with male athletes post MTBI.

Sosnoff et al. (2007), found within 48 hours of MTBI that athletes revealed impaired reaction times on the CRI computerised test, compared with control participants. Makdissi et al. (2001), reported reduced simple reaction time within 72 hours of MTBI in Australian footballers, compared with control participants who improved on the measure. Maddocks and Saling (1996) reported reduced choice reaction time within 5 days of MTBI in Australian footballers versus baseline and in comparison with control participants. Collie, Makdissi et al. (2006) found impaired reaction time more prevalent among symptomatic versus asymptomatic athletes within 11 days of MTBI. With respect to being symptomatic, studies using ImPACT found that high school and collegiate athletes reporting a headache, migraine, fogginess, and/or increased symptoms, performed significantly worse on reaction time measures than those not reporting these symptoms following MTBI. Furthermore, 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 (Collins, Field et al., 2003; Iverson et al., 2004b; Lau et al., 2009; Mihalik et al., 2005). Majerske et al. (2008) also found significantly slowed reaction time post MTBI in high school athletes was associated with both high symptom reports and activity levels. In contrast, Lovell, Collins, Iverson et al. (2004) found high school athletes' reaction time only slowed slightly and not significantly within 36 hours of MTBI, and improved significantly on baseline performance six days post MTBI.

Cremona-Meteyard and Geffen, 1994 found no differences between Australian football players who did and did not sustain MTBI on overall reaction time, except the MTBI group showed little benefit, compared with the non-MTBI group, on the reaction time responses to cued targets in an expected location i.e., directed visuospatial attention, within two weeks of MTBI. One year later, the MTBI football players' reaction time had improved overall, but the deficit on the reaction time responses to cued targets remained. Although Iverson, Brooks, Collins and 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 MTBI events at baseline testing, Covassin et al. (2008), found that reaction time appeared sensitive to the cumulative effects of MTBI, in that athletes with a prior MTBI history performed significantly worse on the reaction time composite than athletes with no prior MTBI history, when tested five days post MTBI.

Limited evidence of impaired reaction time in association with the chronic effects of MTBI has been found in studies of Australian football, soccer and ice hockey (Cremona-Meteyard & Geffen, 1994; Downs & Abwender, 2002; Gaetz et al., 2000). Gaetz et al. (2000) found among junior ice hockey players that those with a history of three or more MTBIs versus those with no history of MTBI, performed worse on visual stimuli reaction time tasks measured on ERPs at least six months post MTBI. Downs and Abwender (2002), assessing the effects of heading the ball in soccer, found reduced reaction time on the Continuous Performance Test for male and female soccer players compared with swimmers.

In summary, although limited evidence suggests that reaction time may be sensitive to the chronic and cumulative effects of MTBI, the implication for the present study based on the literature, is that reaction time is particularly sensitive to the acute effects up until eleven days of MTBI, particularly when athletes are symptomatic and therefore the ImPACT Reaction Time composite should prove a sensitive measure of compromised cognitive performance in concussed rugby players.

6.1.4 Impairments in Memory

Memory in General

Belanger and Vanderploeg (2005), in their meta-analyses of acute neuropsychological outcomes following sport-related MTBI, found that within 24 hours and in comparison with controls, the largest overall effect sizes included global functioning, delayed memory and memory acquisition, in comparison to other functions, such as executive and attention functions, that were not present on testing following 7 days postinjury, apart from delayed memory that remained impaired at day 7.

In respect of sport-related MTBI tested on the ImPACT computerised program, for the previously combined visual and verbal memory composite measure, Lovell, Collins, Iverson et al. (2004) found deficits on the ImPACT memory composite and increased symptom reports within 36 hours of MTBI, despite athletes having previously demonstrated resolution of symptoms within 15 minutes of injury. Among high school athletes, Lovell, Collins, Iverson et al. (2003) found deficits on the ImPACT memory composite lasting up to a week post MTBI, despite symptom resolution within four days.

High school athletes reporting a headache versus no headache, and fogginess versus no fogginess, performed significantly worse on the ImPACT memory composite approximately 6.8 days following MTBI (Collins, Field et al., 2003; Iverson et al., 2004b). In addition, those athletes reporting headache revealed significantly more symptoms and had a strong likelihood of having experienced on-field amnesia (Collins, Field et al., 2003). Furthermore, Mihalik et al. (2005) found athletes reporting a migraine performed significantly worse on the verbal and visual memory composites than those with a headache or without a headache, within six days post MTBI.

In relation to chronic cumulative effects of MTBI on memory performance, De Beaumont et al. (2009), found chronic cognitive deficits in episodic memory (in addition to slow motor execution on a diadochokinesia task) among former hockey and football players, who had sustained their last sport-related MTBI more than 30 years previously. This shows the potential for cognitive and motor aberrations in late adulthood even after only one or two MTBIs (De Beaumont et al., 2009).

Verbal Memory

Verbal memory was the cognitive ability demonstrating the greatest initial impairment in 15 to 18 year old ice hockey athletes following MTBI, with substantial variation in recovery in the younger concussed athletes (Hatfield et al., 2004). Echemendia et al. (2001) found significantly impaired performance on the measures of working memory and verbal learning two hours post MTBI, and on working memory, verbal learning and verbal memory 48 hours post MTBI, among male and female college athletes compared with controls. However, no significant differences between the two groups were found one week postinjury. Echemendia et al. (2001) point out that baseline scores for the two groups were equivalent on the HVLT learning index, and at the 48 hour test interval controls benefitted from practice effects, but the MTBI group did not.

Several sport-related MTBI studies have used the WMS Logical Memory and Verbal Memory tests, with some studies revealing relatively poorer performance by soccer and rugby athletes in the acute and chronic phases post MTBI (e.g., Matser et al., 1998, 1999; Petersen & Skelton, 2000; Shuttleworth-Edwards, Border et al., 2004). However, other studies have not found poorer performance by athletes following MTBI (e.g., Matser et al., 2001; Wilberger et al., 1991).

Verbal memory deficits appear age related. Field et al. (2003) found more prolonged memory impairment on the HVLT among high school athletes compared with collegiate athletes post MTBI, in comparison with controls. The high school athletes evidenced significant impairment at 24 hours on the total words, and at day 7 on total score, whereas the college athletes revealed significant impairment at 24 hours on the total words and delayed recall, at day 5 on total words, but with no significant differences revealed at day 7. However, in another study, verbal memory scores remained significantly lowered compared to baseline performance at 14 days post MTBI, for both high school and collegiate American football athletes (McClincy et al., 2006).

Verbal memory appears sensitive to the cumulative effects of MTBI, in that athletes with a prior MTBI history performed significantly worse on the verbal memory composite than athletes without a prior MTBI history, when tested 5 days post MTBI (Covassin et al., 2008).

Visual Memory

Among high school athletes, visual memory deficits were revealed more than 5 days post MTBI on the Benton Visual Spatial Memory Test-Revised, which measures visual memory for geometric designs. There were significantly poor scores at 24 hours and at day 3 on the total figures and delayed recall, at day 5 on total figures, and scores approaching significance on day 7 on total figures (Field et al., 2003).

Pellman, Lovell et al. (2004) found that NFL athletes did not show significant overall neurocognitive impairment on the NFL test battery in the first 48 hours following MTBI. However, a subset demonstrating on-field memory deficits revealed visual memory deficits within 48 hours of injury, with significantly poor performance on the immediate and delayed memory conditions of the Benton Visual Retention Test-Revised.

Both visuoperceptual organisation and visual memory can be assessed via copying tasks of complex figures, e.g., the Rey Complex Figure Test (RCFT) and the WMS-III Visual Reproduction tasks. Significant chronic visual memory deficits, as a result of sport-related MTBI, have been found among professional soccer and rugby players, compared with controls, on the RCFT, WMS Digit Symbol Incidental Recall, and WMS Visual

Reproduction subtests (e.g., Matser et al., 1998, 1999; Shuttleworth-Edwards, Border et al., 2004) - although not in Matser et al.'s (2001) study.

In summary, most studies have found verbal and visual memory deficits within the first week to two weeks post MTBI, particularly within 48 hours. Both visual memory and verbal memory appear sensitive to the cumulative effects of MTBI. For example, within 5 days of MTBI those with a history of prior MTBI revealed poorer verbal memory performance, than those who did not have a prior MTBI history. Studies have also found chronic visual and verbal memory impairment among soccer and rugby players.

6.1.5 Executive Dysfunction

Belanger and Vanderploeg (2005) in their meta-analyses of neuropsychological outcomes following sport-related MTBI, found the smallest overall effect sizes for attention and executive functioning, in comparison with other functions, such as memory and global cognitive ability.

Several studies have revealed deficits on the Trail Making Test, which is considered sensitive to impairments in executive functioning, in addition to information processing impairments (e.g., Collins et al., 1999; Echemendia et al., 2001; Hatfield et al., 2004; Macciocchi et al., 1996; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Jordan et al., 1993). For example, fifty percent of 15 to 18 year old ice hockey players revealed acute MTBI effects on the TMT in measuring psychomotor speed, sequencing, and the ability to alternate between two sets of information (Hatfield et al., 2004). Among male university athletes, a learning disorder and a history of MTBI, were found independently associated with lowered baseline performance on tasks measuring executive functioning, such as TMT B and SDMT (Collins et al., 1999).

There are few studies on sport-related MTBI and executive functioning specifically. Ellemberg, Leclerc, Couture and Daigle (2007) found female soccer players remained impaired on planning (Tower of London Task), decision-making (complex reaction time), and inhibition and flexibility (Stroop Test) six to eight months following MTBI. In contrast, a study on chronic neuropsychological consequences of both soccer and rugby among UK schoolboys, found no significant effects for executive functioning (Stephens

et al., 2010). Pontifex et al. (2009), however, found an association between a history of MTBI and cognitive control, measured by ERPs and on a modified flanker task.

In summary, findings indicate that impairment in executive functioning, particularly aspects measured by the TMT and SDMT, occurs in the acute phase. Persistent executive functioning impairment is revealed in the chronic phase, as measured by the Tower of London test, Stroop test, and modified flanker task.

6.1.6 Summary of Neurocognitive Findings for Sport-related MTBI

Overall, most neuropsychological studies on sport-related MTBI have found reduced attention skills, processing speed, reaction time and memory impairments, and some studies have found executive dysfunction in the acute stage post MTBI. In a meta-analysis of sport-related MTBI (Belanger & Vanderploeg, 2005), the largest overall effect sizes included delayed memory and memory acquisition, in comparison to other functions, such as attention and executive functions, and therefore memory appears a neurocognitive function commonly affected by sport-related MTBI. Overall, most of these forms of neurocognitive impairment appear to resolve within 10 days, however chronic deficits in the domains of attention and concentration, information processing and visuomotor processing speed, reaction time, memory, and executive functioning have also been revealed, and suggest persistent cognitive decline as a result of concussive and subconcussive trauma.

6.2 SYMPTOM FINDINGS FOR SPORT-RELATED MTBI

Several MTBI studies mention the presence of symptoms, but do not elaborate or specify symptoms present, and few studies named the symptom measure used (e.g., Guskiewicz et al., 2005; Hatfield et al., 2004; Macciocchi et al., 1996, 2001; Putukian et al., 2000; Ruff et al., 1989).

Pertinent findings of four reviews on symptoms and symptom measures follow. Most of the widely used symptom scales have not been empirically scrutinised or methodically developed, despite their use in symptom detection and in guiding return to play decisions (Alla et al., 2009). Gioia, Schneider, Vaughan and Isquith (2009) reviewed five symptom

scales, and found that most scales revealed concurrent validity in discriminating nonconcussed and concussed groups. Eckner and Kutcher (2010) found no single symptom scale demonstrated superiority over the others. Valovich and McLeod (2009) found only 14 articles provided self-reported symptoms (SRS) outcomes, and in the studies measuring both symptom and neurocognitive MTBI effects, SRS revealed the greatest changes of all outcome variables, and revealed the largest effects at both the immediate and follow-up MTBI assessments. This was despite practice effects being an unlikely issue for SRS measures. Review of the literature also indicates the development and validation of new symptom scale measures (e.g., Andersson, Emanuelson, Olsson, Stalhammar, & Starmark, 2006; Randolph et al., 2009; Van Dyke, Axelrod, & Schutte, 2010).

With regard to baseline SRS, female soccer players, aged 8 to 24 years, with a history of at least one MTBI, reported significantly more symptoms and demonstrated poorer performance on ImPACT measures than males (Colvin et al., 2008). For soccer players in comparison with noncontact sport controls, Abreau et al. (1990) found that soccer players reported significantly more SRS, however, Jordan, Green, Galanty, Mandelbaum and Jabour (1996) found no significant differences, between these two groups on SRS, although increased cognitive and somatic SRS were associated with the number of prior MTBI events.

In respect of acute postconcussive SRS, headache is the symptom most commonly reported post MTBI. Other symptoms frequently reported include concentration difficulties, dizziness, confusion, balance problems, lethargy, fatigue, being mentally foggy, memory problems, nausea, nervousness, sleep disturbances, and visual disturbances (e.g., Barbic et al., 2005; Collins, Lovell, Iverson, Ide, & Maroon, 2006; Erlanger, Kaushik et al., 2003; Gessel et al., 2007; Guskiewicz et al., 1997; Guskiewicz et al., 2000; Hinton-Bayre et al., 1999, 2004; Iverson et al., 2004b; Lovell et al., 2005; Macciocchi et al., 1996, 2001; Makdissi et al., 2001; McCrory & Johnston, 2002; Moser et al., 2007; Shuttleworth-Jordan et al., 1993). High school and collegiate American football players reported significantly more symptoms day 3 post MTBI, that resolved day 8 post MTBI in line with neurocognitive recovery. However, the QEEG findings revealed significantly abnormal brain activity day 8, suggestive of physiological recovery being attained after observed neurocognitive and symptom recovery (McCrea, Prichep,

Powell, Chabot, & Barr, 2010). Among American football players post MTBI, there were no differences in the extent of symptom reports between those players with and without a prior history of MTBI (including those players with a prior history of three or more MTBIs), however somatic complaints tended to be higher for players who sustained a repeat MTBI in comparison to their initial MTBI (27.5% versus 18.8%) (Pellman, Viano et al., 2004).

In relation to chronic postconcussive symptoms, Gaetz et al. (2000) conducted a study on the cumulative effects of MTBI and chronic symptoms at least 6 months postinjury, among junior ice hockey players, using SRS and event-related potential recordings. Findings revealed significantly longer P3 latency periods in hockey players who had sustained three or more MTBIs, compared to those who had never sustained a MTBI. Furthermore, the P3 latency correlated significantly with the variables, memory problems and taking longer to think. Aggregate scores for duration, frequency, and intensity of symptoms revealed a significantly higher report on the headache, memory and thinking symptoms by the group who had three or more MTBIs, compared with the group who had never sustained a MTBI. Overall, the study provided support of the cumulative negative effects of MTBI among ice hockey players (Gaetz et al., 2000). In contrast, Killam et al. (2005) studied the enduring residual effects of MTBI among ice hockey, field hockey, lacrosse and soccer athletes (mean age 22.1 years). They found no significant differences for SRS on the Postconcussion Syndrome Checklist between those who had sustained MTBI within two years, and those who had sustained MTBI more than two years previously. Studies investigating chronic symptom complaints found that former professional soccer players report a significant number of persistent symptoms in the form of headache, dizziness, irritability, concentration and memory difficulties, with a significant proportion evidencing neuropsychological impairment and abnormalities on EEG and CT investigations (Tysvaer, 1992; Tysvaer et al., 1989). Among retired football players, those with a history of multiple MTBIs, reported significant memory changes and appeared to experience more symptoms of depression (Guskiewicz et al**.,** 2005; Guskiewicz, Marshall et al., 2007).

No significant differences in SRS have been found between soccer heading versus non-heading players (e.g., Putukian et al., 2000). However, Salinas et al. (2009) found soccer

players aged 9 to 15 years reported significantly more on headache, neck pain, dizziness, tinnitus, and balance difficulties following heading, than in the absence of heading. With regard to expectations of MTBI symptoms, Ferguson et al. (1999) conducted a study among male amateur college and high school athletes, aged 19 to 21, of whom more than half played hockey and the remainder played American football, rugby, football lacrosse and soccer. The athletes each completed a 30-item symptom questionnaire. Although SRS rates did not differ between those who had and who had not sustained a MTBI in the previous 6 months, the athletes who had sustained a sport-related MTBI in the previous 6 months underestimated the incidence of symptoms preinjury. In contrast, the athletes who had not sustained a sport-related MTBI in the previous 6 months, overestimated the anticipated degree of change in symptom presence postinjury compared with preinjury. The authors concluded that symptom expectation has a significant effect on the perception of postconcussion symptoms (Ferguson et al., 1999).

In summary, it appears that athletes involved in contact sport, report more baseline symptoms compared with controls, and support exists for the effects of cumulative MTBI on increased symptom reports. Following MTBI, headache is the most common symptom, and studies have observed an increase in reports of the symptoms concentration difficulties, dizziness, confusion, balance problems, fatigue, being mentally foggy, memory problems, nausea, nervousness, sleep disturbances, and visual disturbances. In comparison to neurocognitive deficits, symptoms revealed the greatest change on all outcome variables, and also demonstrated the largest effects at both the immediate and follow-up MTBI assessments.

6.3 NEUROPSYCHOLOGICAL STUDIES SPECIFIC TO RUGBY UNION

Despite a high MTBI incidence among rugby union players and considering that rugby is played in several countries, it appears that only eight studies have been published on the neuropsychological effects of MTBI in rugby. Of these, four are South African studies (e.g., Shuttleworth-Edwards, Border, Reid, & Radloff, 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith & Radloff, 2008; Shuttleworth-Jordan, Puchert, & Balarin, 1993), one is an American study on female rugby players (Farace, Ferree, Hollier, Barth, & Shaffrey, 2003), two are Canadian studies (Pettersen & Skelton,

2000; Thornton, Cox, Whitfield, & Fouladi, 2008), and one is an Australian study (Gardner, Shores, & Batchelor, 2010). Some authors suggest that minimal neuropsychological deficits persist following MTBI (Binder, 1997; Binder et al., 1997; Satz, 1997, 2001). However, the eight studies on rugby-related MTBI provide support of acute (e.g., Shuttleworth-Jordan et al., 1993) and chronic neuropsychological deficits among rugby players (e.g., Farace et al., 2003; Gardner et al., 2010; Pettersen & Skelton, 2000; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Jordan et al., 1993; Thornton et al., 2008). The one study investigating both acute and chronic effects of MTBI in rugby players (Shuttleworth-Jordan et al., 1993) will be discussed first, following which the remaining studies investigating the chronic effects of MTBI will be presented.

6.3.1 A Study of Acute and Chronic Effects of Rugby-related MTBI

The only one published study investigating the acute (and chronic) effects of rugby-related MTBI, was carried out in South Africa, and follows.

Shuttleworth-Jordan et al. (1993)

The Shuttleworth-Jordan et al. (1993) study involved university athletes aged 18 to 25 years. Sixty rugby players from the top 5 rugby teams, with no prior moderate to severe MTBI or more than one MTBI in the previous 3 years, were assessed preseason, and 20 randomly selected general rugby participants were assessed postseason. Five rugby players sustained MTBI during the season of which four, completing all testing, formed the MTBI group for separate analyses. Of 25 noncontact sport control athletes assessed preseason, 10 were matched with the 5 concussed players (forming the control group for separate analyses), and the 15 remaining control participants were assessed postseason as controls for the general rugby group. The study investigated (1) the preseason and postseason differences on neurocognitive test scores (Denckla Finger Tapping, Digit Span, Digit Supraspan and TMT) between general rugby players and noncontact sport controls, and (2) differences on repeated measures for scores on these same neurocognitive tests, were conducted between rugby players sustaining MTBI and matched noncontact sport controls, at preseason and one week, and one, two, and three

months postinjury. Post MTBI symptoms were reported for the MTBI rugby group. The MTBI incidence among rugby players was 8.33%, and under-reporting was suspected.

Findings for the preseason baseline revealed impairments for the rugby group in comparison with the controls, on hand motor dexterity, new verbal learning, information processing and working memory (measured on the Purdue Pegboard, TMT A and B, and Digit Span backwards relative to the Digit Span forwards). Findings for the control group at postseason versus preseason, revealed a significant practice effect on TMT A and B, Digit Supraspan A and B, Purdue Pegboard (preferred and non-preferred hand), and Finger Tapping (non-preferred hand). However, findings for the general rugby group at postseason versus preseason, revealed less ability to benefit from practice, with improvement on only TMT B and Finger Tapping (non-preferred hand). Comparisons between positions of play for the general rugby group, showed that the rugby forwards revealed no significant improvements relative to the rugby backs, suggestive of increased vulnerability for the rugby forwards.

For analyses purposes, the MTBI rugby group consisted of only four rugby players, in that the fifth player was concussed late in the season. The MTBI rugby group, compared with the control group, revealed significant differences and differences approaching significance in the direction of poorer performance by the rugby players preseason and 3 days post injury, on all measures (hand motor dexterity, new verbal learning, immediate auditory attention, information processing and working memory). The control group demonstrated benefit from practice at the second test interval. No significant differences were found between the MTBI rugby and control groups at the following test intervals. The MTBI rugby group revealed increasing recovery on all measures, but only matched the control group in terms of practice on the TMT A and B and Digit Span forwards at the second month postinjury. However, by the third month the MTBI rugby group still did not match the control group in terms of practice on Digit Span backwards, Digits Difference, Digit Supraspan A and B, and Finger Tapping (preferred and non-preferred hands), with this being suggestive of incomplete neurocognitive recovery.

The study investigated self-reported symptoms in the MTBI rugby group. Three days post MTBI, all five rugby players reported headaches, three players reported nausea and visual disturbances, two players reported poor attention and concentration, anxiety, insomnia,

fatigue, and vomiting, and one player reported limb weakness, loss of appetite, sensitivity to noise, restlessness, clumsiness, and speech problems. One month postinjury, symptoms were markedly reduced among five players, and two players reported mild headaches and mild problems with attention and concentration, and one player reported mild fatigue and blurred vision. Two months postinjury, symptoms were not overly reduced among the remaining four players, and two players reported occasional mild headaches, fatigue, problems with attention and concentration, and restlessness. Three months postinjury, no symptoms were reported (Shuttleworth-Jordan et al., 1993).

The study provides support of chronic deficits among rugby players at baseline testing, indicative of concussive and subconcussive effects, particularly on hand motor dexterity, new verbal learning, information processing and working memory. These deficits were compounded during the rugby season, and shown as a reduced ability to benefit from practice to the extent of the controls, particularly on the information processing and hand motor dexterity tasks. These same neurocognitive deficits were mirrored and were noticeably pronounced for the MTBI group at the first postinjury follow-up, and thereafter a lack of benefit from practice to the extent of the controls was revealed for repeated tests.

There were several limitations to the study. Not all nonconcussed rugby participants tested preseason were retested postseason, and a preseason and postseason repeated measures design would have strengthened the findings. The control sample for the nonconcussed rugby group was small, as was the sample of MTBI players (five reduced to four). Neurocognitive measures for the fifth player were excluded from neurocognitive analyses, although his symptoms were reported at two post MTBI test intervals. The need to have ten matched controls for five concussed players is questionable. While the sample was drawn from a university population providing some control for age and intellectual capacity, age and IQ were not formally controlled for. Nevertheless, the study provides indications of chronic deficits, and information on the presence and recovery of acute MTBI neurocognitive deficits and symptoms in rugby players. It is pertinent that rugby players were asymptomatic three months post MTBI, yet in terms of practice effects on neurocognitive testing, the MTBI group did not attain the benefit of practice that the controls did. This suggests that the MTBI rugby players were not fully recovered.

6.3.2 Studies of Chronic Effects of Rugby-related MTBI

The seven published rugby studies researching only the chronic effects of rugby-related MTBI are now reviewed.

Pettersen and Skelton (2000)

Based on research findings that glucose enhances cognition, particularly memory (Craft, Murphy, & Wenstrom, 1994; Gonder-Frederick et al., 1987; Parsons & Gold, 1992), Pettersen and Skelton (2000) undertook a study that assessed the effects of glucose on memory according to MTBI history. Twenty university rugby players and 4 similar team sport athletes participated, of which 12 with a history of MTBI in the previous 10 years formed the MTBI group (mean age 19.9 years, education 15.3 years), and 12 without a history of MTBI formed the control group (mean age 19.6 years, education 14.6 years). Over three consecutive mornings, participants were baseline blood-glucose tested, ingested a beverage sweetened with either glucose or non-glucose (placebo), were cognitively assessed, and their blood-glucose re-tested. The battery of tests included the Listening Span (LS), WMS-R Logical Memory Scale (LMS), a paired verbal associates test, Auditory Consonant Trigrams (CCC), SDMT, and four ANAM subtests. Findings revealed that the MTBI group scored significantly lower than the non-MTBI group on SDMT delayed, LMS delayed, LS, and CCC. This indicates a cumulative effect of MTBI on long-term declarative memory. However, glucose intake improved the MTBI group's performance on the measures of declarative memory (due to their faster glucose uptake). In contrast, glucose intake did not enhance the control group's performance on the measures of declarative memory (Pettersen & Skelton, 2000). From the study it appears that diminished declarative memory may be a residual effect of rugby-related MTBI, however the authors were unable to provide convincing evidence for the relationship between glucose metabolism and memory. Limitations of the study were a small sample size, no noncontact sport control group, and between the comparative groups there was no control for IQ or years of sport participation, and the control for education was weak.

Farace et al. (2003)

Based on the assumption that long-term cognitive deficits as a result of sport-related MTBI are rare, and if present are due to extraneous factors such as age or premorbid IQ, Farace et al. (2003) conducted a study on 54 female university rugby players aged 17 to

22 years. Of the 54 players, 29 had a history of one or more MTBIs. The athletes all had average or above average estimated IQ scores. Measures included the brief Standardized Assessment of Concussion (SAC), TMT A and B, and PASAT. No differences were found between players with or without a history of MTBI on the PASAT or SAC, however those with a history of MTBI made a significantly greater number of errors on the TMT A, than the players with no history of MTBI (Farace et al., 2003). This finding is suggestive of MTBI causing chronic neurocognitive deficits in attention and concentration, working memory, and particularly information and visuomotor processing. A limitation of this study was no noncontact sport control group was used, and if it had, then the findings of Shuttleworth-Jordan et al. (1993), that rugby players evidenced a significantly poorer performance than controls on TMT A, might have been replicated.

Gardner et al. (2010)

The Gardner et al. (2010) study involved active adult male rugby union players. Thirty-four rugby players with a history of three or more self-reported MTBIs (not sustained in the previous three months) formed the MTBI group, and 39 players without a history of MTBI formed the non-MTBI group. Both groups were equivalent for gender, age, estimated IQ, education, years playing sport, and rugby position. Tests included a traditional measure, the WAIS-III Processing Speed Index (comprising digit symbol-coding and symbol search), and the computerised ImPACT program. Significantly lower performance on both the traditional Processing Speed Index and the computerised ImPACT Visual Motor Speed composite, was evidenced by the MTBI group versus the non-MTBI group. This suggests that a history of multiple MTBIs among rugby players has a detrimental effect on processing speed. Although the MTBI group reported almost 73% more symptoms than the non-MTBI group, the difference was not significant. The traditional WAIS-III Processing Speed Index and ImPACT visual motor speed measures appear sensitive to the chronic effects of MTBI (Gardner et al., 2010).

The authors failed to point out the significant difference between the groups on the Impulse Control composite, with significant impulsivity in the MTBI group, which indicates that this measure may be sensitive to the effects of MTBI. This study supports findings of other rugby studies, in which processing speed is negatively affected by MTBI (e.g., Farace et al., 2003; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Jordan et al., 1993). A limitation of the study was no noncontact sport control group was used.

Shuttleworth-Edwards, Border et al. (2004)

This study compared the preseason assessment of three rugby groups (national open, national under-21, and schoolboy levels) totalling 92 rugby players, with appropriate noncontact sport control groups totalling 76 athletes. The comparative groups were equivalent for IQ, education and age, apart from the under-21 national rugby group having significantly lower scores on these variables than the control group. According to age, IQ estimates were measured on the WAIS or SAWAIS Picture Completion and Comprehension subtests. Additionally, schoolboys were administered the National Adult Reading Test. All groups were assessed on: SAWAIS Digit Span (forwards and backwards) and DSST, TMT A and B, WMS Associate Learning and Visual Reproduction subtests, Digit-Symbol Incidental Recall (Immediate and Delayed), Words-in-One–Minute (Unstructured Verbal Fluency Test and Structured Verbal Fluency Test-'S' Words), Sequential Finger Tapping Test, and the Postconcussion Symptom Questionnaire. School participants were also assessed on the WAIS-III Letter-Number Sequencing subtest and the STROOP Neuropsychological Screening Test. Scores were rated as none, mild, or moderate/severe deficits, according to whether scores fell within one, less than two, or greater than two standard deviations from the norm, respectively. The 31-item postconcussion questionnaire was scored according to answers such as never, sometimes or often.

Findings for the two adult rugby groups showed greater deficits in information processing speed, verbal fluency, memory, and hand motor dexterity compared with the control groups. In contrast, none of the rugby players revealed deficits on the sequential Finger Tapping task, however 10% of the controls did. Of relevance to the present study is, 23% of the national open rugby players and 5% of the controls, evidenced deficits on the WMS Visual Reproduction-delayed. In addition, 19% of the national open rugby players and none of the controls, evidenced deficits on the WMS Associate Pairs, while 11% of the under-21 rugby players and none of the controls, evidenced deficits on the WMS Associate Pairs hard-immediate condition.

For the schoolboys, only one trial of hand motor function supported greater deficits for the rugby group versus the control group. Contrary to the hypothesis, schoolboy rugby backs versus forwards revealed marginal deficits across verbal fluency and visual memory measures, possibly due to rugby positions being more flexible at school level,

and the MTBI incidence was marginally lower for the forwards versus backs. Among schoolboy rugby players, 4% of forwards versus 26% of backs and 4% of forwards versus 21% of backs, revealed deficits on the WMS Visual Reproduction immediate and delayed conditions, respectively. In the absence of normative standards, schoolboy group comparisons on the WMS Associate Pairs were not made. The school rugby subgroup with learning difficulties had a higher estimated IQ score than the control subgroup with learning difficulties, and yet 67% of this rugby subgroup, but virtually none of the control subgroup, evidenced cognitive deficits. This concurs with the findings of Collins, Grindel et al. (1999), that following MTBI, learning disabled football players revealed significantly higher cognitive deficits than non-learning disabled football players.

In respect of symptoms, SRS were substantially evident for the two adult rugby groups versus control groups, and for the forwards versus the backs. The rugby groups reported 20 to 30% more neurobehavioural symptoms than the control groups. Furthermore, both adult rugby groups, particularly the forwards versus the backs, reported attention and memory difficulties that were cross validated on the objective tests, e.g., speed of information processing as a measure of sustained attention. However, the open national rugby players reported less on the key physical and cognitive postconcussion symptoms (e.g., headache, limb weakness and visual disturbances) compared with the control participants. This was attributed to possible under-reporting in order avoid exclusion from sport. Schoolboy rugby players reported more symptoms than controls, although the schoolboy rugby SRS were less prevalent than for the adult rugby players.

Overall, the study found overwhelming support that rugby players show more neuropsychological impairment than noncontact sport athletes, and the impairment is substantially more pronounced among older, more experienced and cognitively vulnerable rugby players. This is particularly the case in the areas of information processing speed (experienced by 30 to 40% of rugby participants) and memory (experienced by 47% rugby participants). The study supports the possibility of permanent neurocognitive dysfunctions on both SRS symptoms and objective neurocognitive testing for rugby players versus noncontact sport controls, for adult rugby forwards versus rugby backs, and particularly in older professional rugby players. The latter is postulated as being due to more prolonged exposure to concussive and subconcussive events, and therefore greater neuronal attrition in terms of Reserve theory (e.g., Shuttleworth-

Edwards, Border et al., 2004). The study provides further support that adult rugby players experience deficits in the areas of information processing, and on the WMS measures of verbal and visual memory. Methodologically the study was strong given that there was control for age, education and IQ across groups, and the inclusion of equivalent noncontact sports controls. However, there were still limitations of the study in terms of the relatively small sample numbers, and the exclusive use of paper and pencil tests without any computerised testing that may have provided the opportunity for more fine testing of response times. While there was a fairly comprehensive battery of paper and pencil tests, the Bonferroni Correction was not applied, thereby increasing the risk of Type 1 error (i.e., the possibility that some of the identified differences were due to chance).

Shuttleworth-Edwards and Radloff (2008)

The Shuttleworth-Edwards and Radloff (2008) study lends further support to the findings that rugby players show greater neuropsychological impairment than noncontact sport athletes. The study compared the preseason assessment of three rugby groups (national open, national under-21, and schoolboy levels) totalling 124 rugby players with appropriate noncontact sport control groups, totalling 102 athletes. The total rugby sample was divided to compare differences between forward players (n = 71) and backline players (n = 53), in that the forwards are considered more exposed than the backs to concussive and subconcussive trauma (Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008). Rugby versus control groups, and rugby forwards versus rugby backs, were statistically equivalent for estimated IQ, level of education, age and performance on the Denckla Finger Tapping (preferred and non-preferred hand) tests. Rugby players had a significantly higher percentage of two or more MTBIs versus the control participants, but there was no significantly higher percentage of two or more MTBIs between the rugby forwards versus the rugby backs.

The study assessed chronic visuomotor processing deficits among rugby players, as a result of both reported and unreported concussive events and assumed subconcussive incidents, from prior rugby participation. Data were collected following a four to five month break from rugby, on the basis that any acute MTBI effects would have resolved within three months. Three tests of visuomotor processing speed implemented individually, included the DSST and TMT A and B. Findings for the total rugby group

versus the total control group, revealed significantly poorer performance for the rugby group on all three measures, and a highly significant difference in mean scores for the Processing Speed Factor. For the rugby forwards versus the rugby backs, there was a significant difference on the DSST in the direction of poorer performance for the rugby forwards. However, there were no significant differences on TMT A and B or on the mean Processing Speed Factor scores. This was possibly because the study involved a greater number of schoolboy and under-21 rugby players than adult rugby players (n = 98 versus *n* = 26), and the effects would be less obvious in that rugby positions are not entrenched at school level (Shuttleworth-Edwards & Radloff, 2008). Nevertheless, the study clearly shows that the cumulative effects of rugby-related MTBI and inferred subconcussive trauma, impacts significantly on visuomotor processing skills. Again, as with the previous study (Shuttleworth-Edwards, Border et al., 2004), this was a methodologically strong study, given that there was control for age, education and IQ across groups, and the inclusion of equivalent noncontact sports' controls. There was also control for hand motor speed. Further strengths of this study were the large sample numbers, and the analysis was limited to a single cognitive function, in order to circumvent the risk of Type 1 error due to multiple comparisons. The only limitation was the exclusive use of paper and pencil tests without any computerised testing, that may have provided the opportunity for finer testing of response times.

Shuttleworth-Edwards, Smith et al. (2008)

This was a further study investigating the chronic effects of concussive and subconcussive injury on neuropsychological functioning, but with the overlay of additive effects from a season of rugby participation. The study involved both preseason and postseason assessments of 27 university rugby players and 18 noncontact sport controls. The two groups were equivalent on race, language and estimated IQ. Compared with the control group, the rugby group had a history of significantly more MTBIs, and a lower mean age and educational level.

Preseason IQ estimates were established on the WAIS-III Vocabulary and Picture Completion subtests for all participants. Two cognitive functions, 'memory' and 'attention', were tapped at both test intervals. Memory was measured on the ImPACT verbal and visual memory composites. Attention was measured on the ImPACT visual motor speed composite, TMT A and B, and Digit Span (forwards and backwards). The

directional hypothesis assumed the rugby group would perform worse than the control group, therefore one-tailed *t*-tests were used. For purposes of alpha adjustment, the Bonferroni Correction was applied to the level of significance, according to the two cognitive functions being investigated (memory and attention). Findings for Digits backwards revealed that the rugby players performed marginally better than the controls at preseason and the controls performed significantly better than the rugby players at postseason. Independent *t*-test comparisons between groups revealed no significant differences for tests measuring the functions of either memory or attention at preseason. Postseason, significantly lower scores were revealed for the rugby players versus the controls on the ImPACT Visual Motor Speed and TMT A, and differences approaching significance on the TMT B. The ImPACT Visual Motor Speed and TMT A and B, as tests of attention involving speeded visuomotor tracking tasks, appear sensitive to the effects of MTBI in comparison with the Digit Span. In respect of the memory function postseason, differences approaching significance were revealed on the ImPACT Verbal and Visual memory measures, in the direction of the rugby group performing worse than the control group.

Repeated measures analyses revealed that the rugby players' performance remained relatively stable between the two test intervals, suggestive of the absence of practice effects and implicating neurocognitive vulnerability. In contrast, significant effects for the control group revealed improvements, suggestive of practice effects, on the TMT A and B and Digits backwards. Overall, the controls' improved performance on six of the seven measures (except Digits forwards) at postseason versus preseason, was suggestive of benefit from practice. However, the rugby players were unable to benefit from practice to the extent of the controls, which purports a learning decrement. The ImPACT Visual Motor Speed composite appeared less vulnerable to practice effects over the seven month test retest interval, compared with the traditional measures employed in the study (Shuttleworth-Edwards, Smith et al., 2008).

Limitations of the study were a small sample size, non-equivalence on age and education, the controls volunteered on an ad hoc basis and participated in a variety of sports, and use of the Bonferroni Correction, while controlling for Type 1 error, may in turn have led to Type II error (i.e., the masking of significant results). Nevertheless, this study indicates that the rugby players revealed lowering in the attentional tasks involving visuomotor

speed postseason, and revealed a mild visual memory reduction postseason. The relatively stable scores of the rugby group over the two test intervals, in comparison with controls, suggests a reduced ability for rugby players to benefit from practice on attentional tasks, that have been found to reveal test-retest improvements. Overall results suggest that concussive and subconcussive events may render rugby players cognitively vulnerable.

Thornton et al., 2008

This Canadian study investigated the association between self-reported MTBI exposure and neurocognitive functioning and symptom complaints among male and female rugby players. The sample included 80 competitive younger rugby players (mean age 26.43 years) and 31 recreational/retired older rugby players (mean age 39.3 years, with 16 retired from rugby and 15 involved in recreational rugby). Exclusion criteria included: aged over 66 years, neurological illness or a serious non-sport TBI. On the basis of self-reported grades 2 and 3 MTBI history, three groups were derived: no heavy concussions, 1-2 heavy concussions, and 3 or more heavy concussions. Although athletes had been tested on IQ measures (Vocabulary and CCFT), these were excluded from analyses and not reported on. Three neurocognitive components were statistically derived as dependent variables: information processing speed and long-term memory, working memory, and executive skills. Measures included: WAIS-III Digit Symbol and Symbol Search; WMS-III Visual Reproduction II, Digit and Visual memory span, and Logical Memory II; TMT A and B combined, the Rey Auditory Verbal Learning Test (trials 1-5), Computational Span, Listening Span, and Spatial Span, and the first 64 cards of the WCST. SRS were assessed using the 9-item Postconcussive Symptom Checklist. Minimal differences in neuropsychological functioning were evidenced across the three concussion exposure groups, although Listening Span was significantly reduced for the 3 heavy concussions group versus the 1-2 heavy concussions group, but not versus the no heavy concussions group.

The no heavy concussions group reported less on total symptoms and on memory complaints or distress, compared to the 3 or more heavy concussions group, and the same pattern emerged when females were excluded from the analyses. When controlling for health, age and gender, grades 2 and 3 concussions were reported to have a cumulative,

dose-response effect on self-reported symptoms among retired and recreational athletes, in comparison to competitive athletes (Thornton et al., 2008).

Limitations of the study were that a noncontact sport control group was not used; and there was no control for IQ or level of education, and the control for age appears weak. A major limitation of the study is that the grading of MTBIs has not been empirically supported. Nevertheless, the study adds to research findings, that increased MTBI exposure is associated with increased postconcussive symptom reports, although it does not support the notion of persistent neurocognitive deficits in association with cumulative MTBI.

Summary of Findings for the Rugby Studies

The rugby union studies above, from high school through to adult and national levels of play, provide support of persistent neurocognitive deficits in information or visuomotor processing speed measured on the DSST, TMT A and B, and ImPACT Visual Motor Speed composite (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), lowering in performance for rugby players on attentional tasks with visuomotor processing speed aspects post-season (Shuttleworth-Edwards, Smith et al., 2008), and indications of increased vulnerability on visuomotor processing speed for rugby forwards versus rugby backs and in association with the number of years exposed to concussive and subconcussive events (Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008).

Memory deficits, measured on traditional tests, are shown in the forms of impaired learning (Shuttleworth-Jordan et al., 1993), long-term declarative memory (Pettersen & Skelton, 2000) verbal memory and visual memory (Shuttleworth-Edwards, Border et al., 2004). The finding of practice effects is an outstanding feature of the rugby studies, particularly on the traditional measures. The practice effects appear more prominent among the control groups than among the rugby groups, both in terms of chronic deficits and in the acute stage post MTBI. This suggests neurocognitive vulnerability among the rugby players who are unable to benefit from learning (e.g., Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Jordan et al., 1993).

In summary of symptom reports in rugby studies, persistent self-reported symptoms have been shown in the rugby studies that investigated chronic symptom presentation (e.g., Gardner et al., 2010: Thornton et al., 2008; Shuttleworth-Edwards, Border et al., 2004). Adult rugby players report substantially more symptoms than schoolboys, and more so in the forwards than in the backs (Shuttleworth-Edwards, Border et al., 2004).

In the only study that investigated acute MTBI impairment and recovery, Shuttleworth-Jordan et al. (1993) reported that four university rugby players evidenced significant deficits on all neurocognitive measures three days post MTBI compared with controls, revealing impairments in hand motor dexterity, new verbal learning, immediate auditory attention, information processing and working memory. No significant differences were found between the MTBI rugby and control groups at the test intervals that followed. However, at three months post injury, the MTBI rugby group had not recovered to the level of the controls in terms of practice effects on all measures, suggesting incomplete neurocognitive recovery. This was despite no symptoms being present three months post MTBI. It is uncertain when neurocognitive recovery occurred but, in terms of significant findings, this would have occurred at some point between the day three and one month test intervals. Symptom reports were markedly high among the five rugby players three days post MTBI, were comparatively less for these five rugby players at one month and for the remaining four rugby players at two months post MTBI, and absent three months post MTBI. Although symptom reports were high at the day three post MTBI interval and lowered at the one month post MTBI interval, symptom recovery is relatively unknown since no comparative preseason symptom assessment was conducted.

As indicated in the above review, a number of the sport-related MTBI studies, including the rugby studies, are beset with methodological limitations that weaken their findings. It appears pertinent, therefore, to provide a comprehensive survey of possible methodological limitations that need to be taken into account when designing a new study in this area.

6.4 METHODOLOGICAL ISSUES FOR RESEARCH ON MTBI IN SPORT

Essentially, good methodology for research within the sports MTBI arena should strive to enhance the internal and external validity of studies (e.g., Macciocchi & Barth, 2004).

Therefore, the pertinent methodology issues of internal and external validity, experimental designs, and statistical matters are discussed below.

6.4.1 Internal Validity

Internal validity is the extent to which a study establishes a casual relationship between the factor being explored and the outcome, i.e., establishing that the change in the dependent variable is produced exclusively by the independent variable, as opposed to extraneous variables (e.g., Campbell & Stanley, 1966; Huitt, Hummel, & Kaeck, 1999; Macciocchi & Barth, 2004; Slack & Draugalis, 2001). Internal validity is the *sine qua non* of any study, based on logic rather than statistics (e.g., Campbell & Stanley, 1966; Slack & Draugalis, 2001). Threats to internal validity include the following extraneous variables:

History

History is a threat when unique factors or experiences affect the participants pre-morbidly and confound results (e.g., Huitt et al., 1999; Macciocchi & Barth, 2004; Slack & Draugalis, 2001). Examples of these variables include age, IQ, education, and premorbid medical and psychiatric history. Therefore participants should be screened for comorbid conditions that might influence their neurological functioning, and controls should be built into the design for these influential variables (e.g., Jordan, 2000).

Maturation

Maturation is a threat when unique factors or experiences affect the participants between test intervals and confound results (e.g., Huitt et al., 1999; Macciocchi & Barth, 2004; Slack & Draugalis, 2001). Examples of these variables include aging, fatigue or reduced motivation to participate in the study (e.g., Huitt et al., 1999).

Testing

Testing is a threat when changes in test scores occur not as a result of the independent variable (e.g., MTBI recovery), but as a result of repeat testing on the same measure (e.g., Campbell & Stanley, 1966; Slack & Draugalis, 2001). This is particularly relevant to MTBI research when using repeated measures, and in terms of practice effects, that were discussed in Chapter 5.

Instrumentation

Instrumentation is a threat when measurement methods, the administration of instruments or administers of instruments are changed. This is because these changes affect results, rather than actual changes in the participants' performance (e.g., Campbell & Stanley, 1966; Huitt et al., 1999).

Statistical Regression

Statistical regression is a threat when participants are selected based on having extreme scores on a performance measure, because the scores at a second test occasion tend to move towards the population mean (e.g., Huitt et al., 1999; Slack & Draugalis, 2001). This is particularly relevant to MTBI research using repeated measures.

Selection

Selection is a threat when participants are not randomly assigned to groups (Macciocchi & Barth, 2004; Slack & Draugalis, 2001). In quasi-experimental research typically used in MTBI studies, participants are not randomly assigned to groups, on the basis that the experimental group had or has MTBI and the control group does not. If groups are functionally equivalent at baseline testing, e.g., age, IQ, gender and education, this assists in overcoming selection bias. This is because differences in performance on measures for the dependent variable are more likely to be attributed the independent variable, e.g., MTBI (e.g., Huitt et al., 1999).

Experimental Attrition

Experimental attrition is a threat when participants withdraw from a study and differences between groups may be erroneously attributed to the independent variable, when the differences are due to the loss of participants from a group (e.g., Huitt et al., 1999; Slack & Draugalis, 2001). This has relevance for a study that includes repeat testing of dependent measures, such as when prospectively investigating postconcussion outcomes in the sports arena.

Selection Interactions

Selection interactions occur when any two or more of the above extraneous variables interact and threaten internal validity.

6.4.2 External Validity

External validity refers to the extent to which findings from a study apply to other population groups, measurement instruments and settings (e.g., Dimitrov & Rumrill, 2003; Huitt et al., 1999). For example, findings relating to MTBI among primary school soccer players may not be extrapolated to what one could expect in relation to MTBI among adult rugby players. Four factors typically affect external validity, and will now be discussed.

Interaction

An interaction may occur between participant selection bias, and the independent variable (e.g., Dimitrov & Rumrill, 2003; Huitt et al., 1999). Non-randomly selected participants and their specific characteristics may influence performance in a manner that does not represent other population groups (Huitt et al., 1999). In sports studies, it is not always possible to acquire a randomly selected control group (e.g., Shuttleworth-Edwards, Smith et al., 2008), and this needs to be considered when evaluating the outcome).

Pretesting

Pretesting participants can result in their reacting or performing differently at the next test occasion, on the basis of test experience and not on the basis of the independent variable being studied. Therefore, findings from studies using pretests, are difficult to generalise (Huitt et al., 1999). This element clearly has relevance for sports studies where repeat testing is involved, however within the sports MTBI study context, this element can be used to identify differential pretest influences on subsequent test outcome between contact and noncontact sport comparison groups, or concussed versus nonconcussed comparison groups.

Experimental Setting

The experimental setting can influence participants' performance. This includes the actual test setting or the participants' awareness of being observed (Huitt et al., 1999). The difference in performance then becomes a confounding independent variable, making it difficult to generalise findings to other population groups.

Multiple Interventions

Multiple interventions are a threat to external validity, in that one intervention can affect the participant's performance on the next and following interventions cumulatively (Huitt et al., 1999). This is problematic among several sport-related studies using extensive batteries for testing.

Knowledge of threats to internal and external validity enables researchers to discern the degree to which these variables may be present in studies, and to account for these when analysing findings (Slack & Draugalis, 2001). The research design and methods employed influence the level of confidence in the internal and external validity of studies, and therefore research designs are discussed.

6.4.3 Experimental Designs

Studies investigating sport-related MTBI are limited to quasi-experimental designs (Macciocchi & Barth, 2004). Unlike classic controlled experimental designs, in quasi-experimental designs the participants are not randomly assigned to the experimental or control group, in that the independent variable (e.g., MTBI) is not being manipulated. True experimental designs, help control for threats to internal validity, and are those that employ both (an) experimental group(s) and control group(s), and implement pretest and posttest assessments (Yu & Ohlund, 2010). A number of sport-related MTBI studies have implemented a variety of experimental designs - not limited to true experimental designs - and therefore these designs are discussed below.

Single Group Posttest Design

The single group posttest design involves only the experimental group of participants being assessed at some point following injury, and comparison of scores are made against standardised population norms (e.g., Campbell & Stanley, 1966; Macciocchi & Barth, 2004). This design is particularly vulnerable to threats of internal validity (Macciocchi & Barth, 2004). The participants already belong to a MTBI population, and without a control group, it is difficult to assess if scores that differ from standardised norms are attributable to MTBI or other extraneous variables such as age, education or IQ. Therefore cause and effect cannot be established with certainty. This design has been used in several studies investigating the chronic effects of MTBI among soccer, American

football, Australian football, rugby league and rugby union players (e.g., Farace et al., 2003; Guskiewicz et al., 2000; Hinton-Bayre et al., 2004; Kutner et al., 2000; Maddocks, Saling, & Dicker, 1995; Matser et al., 2001; McCrory et al., 2000; Straume-Naesheim et al., 2005; Thornton et al., 2008).

Comparison Group Posttest Design

The comparison group posttest design involves an experimental group and a control group being assessed at some point following injury, and comparison of scores are made between these groups (e.g., Campbell & Stanley, 1966; Macciocchi & Barth, 2004). This design provides a reduced risk in controlling for premorbid factors as a threat to internal validity than the Single Group design (Macciocchi & Barth, 2004). Ideally, both groups should be matched on gender, age, education, estimated IQ and number of MTBI incidents, in order to establish the causal effect of MTBI. This design has been used in several studies investigating the chronic effects of MTBI among ice hockey, soccer, American football and rugby union players (e.g., Abreau et al., 1990; Collins, Field et al., 2003; Iverson et al., 2004b, 2006; Gardner et al., 2010; Killam et al., 2005; Matser et al., 1998, 1999; Rutherford et al., 2005; Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008; Stephens et al., 2005; Tysvaer & Lochen, 1991; Webbe & Ochs, 2003).

Single Group Pretest Posttest and Single Group Time Series Designs

The single group pretest posttest design involves the experimental group of participants being assessed prior to and at some point following injury, and the difference between test scores is attributed to casual effects of the injury (e.g., Yu & Ohlund, 2010). This design is vulnerable to threats of internal validity, for example maturation, testing, instrumentation and statistical regression (Yu & Ohlund, 2010). This design has been used in few studies investigating the chronic effects of MTBI among ice hockey, American football, Australian football and rugby league players (e.g., Dicker & Maddocks, 1988; Gaetz et al., 2000; Hinton-Bayre et al., 1997; Pellman et al., 2004, 2006).

The single group time series design involves the experimental group of participants being assessed prior to and at several points following injury, and the difference between test scores is attributed to casual effects of the injury. This design is vulnerable to threats of

internal validity, for example maturation, testing (particularly practice effects), and instrumentation. This design has been used in a few studies investigating the chronic effects of MTBI among ice hockey, American football and rugby league players (e.g., Barth et al., 1989; Guskiewicz et al., 2003; Hatfield et al., 2004; Hinton-Bayre et al., 1999; Macciocchi et al., 2001; McClincy et al., 2006; McCrea et al., 2002; Wilberger et al., 1991).

Comparison Group Pretest Posttest Design and Time Series Design

The comparison group pretest posttest design involves one or more experimental groups and one or more control groups, who are assessed prior to and at some point following injury, in order to compare groups, and/or changes in measures (Dimitrov & Rumrill, 2003). Time series designs involve a series of posttest assessments in order to gauge effects such as MTBI recovery. The comparison group pretest posttest and time series designs are frequently used in sport-related MTBI research. These forms of experimental design are not without limitations, which include maturation and testing. Therefore, practice effects are a particular threat. This design has been used in a few studies investigating the acute and/or chronic effects of MTBI among athletes including soccer, American football, Australian football and rugby union players (e.g., Barr, 2003; Collins, Grindel et al., 1999; Collie, Makdissi et al., 2006; Cremona-Meteyard & Geffen, 1994; Field et al., 2003; Guskiewicz et al., 1997; Iverson et al., 2004a; Lovell et al., 2003; Macciocchi et al., 1996; Maddocks & Sailing, 1996; Makdissi et al., 2001; Mihalik et al., 2005; Pettersen & Skelton, 2000; Putukian et al., 2000; Shuttleworth-Jordan et al., 1993).

Non-equivalent Control Group Design

The non-equivalent control group design is a powerful design in which the experimental group and control group are not equivalent on the dependent variable (e.g., MTBI, or extent of exposure to multiple concussive and subconcussive insults), but are matched and, therefore, equivalent on variables such as gender, age, IQ, education, years of sport participation, and in which the groups are assessed prior to and following MTBI, for comparisons (Macciocchi & Barth, 2004). This design has been used in a rugby study investigating the chronic effects of MTBI (Shuttleworth-Edwards, Smith et al., 2008).

While pretest posttest designs provide premorbid data against which postmorbid comparisons can be made, Shuttleworth-Edwards, Border et al. (2004) point out that a

limitation of quasi-experimental group comparisons is that they do not provide for evaluation of individual outcomes that have relevance in terms of Reserve theory. They suggest that this be overcome by examining the between groups distribution of scores and standard deviations, or by comparing the percentage of deficits between the rugby and demographically equivalent control groups (Shuttleworth-Edwards, Border et al., 2004).

6.4.4 Other Methodological and Statistical Issues

It is difficult to draw comparisons between various sport-related MTBI studies for several reasons. Firstly, MTBI as the independent variable has several different definitions, and it is uncertain if the various studies are measuring the same entity. For example, some studies do not provide a definition of MTBI, or include only participants whose MTBIs involved LOC or certain grades of MTBIs, or rely on athletes' self-reports of MTBI history. It is also possible that athletes with MTBI have been included in comparative control groups, due to non-MTBI disclosure.

A further difficulty in drawing comparisons between studies, is that a variety of neuropsychological measures are used to assess a specific neurocognitive function. Additionally, authors often provide different interpretations of what functions one specific test measures. Additionally, studies use different time intervals between that of injury and postconcussion testing, and make it difficult to ascertain when exactly recovery occurred. Furthermore, studies do not generally elaborate on extraneous variables that may occur during test intervals, which might affect test results. These include whether or not the participants have been on bed rest, are involved in sport participation (training or match level), are attending class/lectures/work, are fatigued, or have suffered family or personal problems. On these grounds provided, it is difficult to draw comparisons between the various studies.

Several other methodological issues pertain to sport-related MTBI, making it difficult for general inferences to be drawn, and these are described below.

<u>Lack of or Inadequate Control Groups</u>

Studies that lack control groups or use weak control groups (e.g., non-athletes or contact sport athletes when studying the effects within a contact sport group), or use noncontact

sport control groups that are unmatched with the experimental group, do not allow for adequate comparisons to be made between contact sports groups and demographically equivalent control groups (e.g., Barth et al., 1983; Ruff et al., 1989). Several sport-related MTBI studies lack a control group (e.g., Barnes et al., 1998; Collins, Field et al., 2003; Farace et al., 2003; Gaetz et al., 2000; Guskiewicz et al., 2003, 2005; Hatfield et al., 2004; Hinton-Bayre et al., 1997, 1999; Iverson et al., 2004b; Kutner et al., 2000; Lovell, Collins, Iverson et al., 2004; Macciocchi et al., 2001; Maddocks & Dicker, 1988; Matser et al., 2001; McCrea et al., 2002; McCrory, 2001; McCrory et al., 2000; Mihalik et al., 2005; Pellman et al., 2004, 2006; Straume-Naesheim et al., 2005; Thornton et al., 2008; Wilberger et al., 1991).

Shuttleworth-Edwards and Whitefield (2007a) point out that studies evaluating the relationship of neuropsychological deficits among multiple versus zero MTBI events, and which lack a noncontact sport control group, have two major limitations. Firstly, these studies fail to account for the effects of unrecognised concussive and subconcussive events within the zero MTBI contact sport group, that may result in contradictory outcomes (Shuttleworth-Edwards, Smith et al., 2008). Secondly, although within contact sport group studies have evidenced chronic neuropsychological deficits in association with an increasing number of MTBI events (e.g., Iverson et al., 2004a; Killam et al., 2005), one study did not evidence neuropsychological deficits, in that athletes returned to baseline performance (e.g., McCrory, 2001). Methodologically however, it is unknown if these athletes were genuinely recovered, in that they may have benefited from practice effects, and comparisons with a control group would have helped elucidate this matter (Shuttleworth-Edwards & Whitefield, 2007a). If no control group is used for comparative purposes, the influence of practice effects cannot be assessed in studies using repeated measures.

Practice Effects

A limitation associated with the neuropsychological test-retest condition used in the assessment of sport-related MTBI, is practice effects (Bernstein, 1999; Erlanger, Feldman et al., 2003). Practice effects vary across participant characteristics, tests characteristics, and across test-retest intervals, which makes comparisons of studies difficult (e.g., Erlanger et al., 2003). Significant practice effects have been evidenced on traditional neuropsychological tests, including the PASAT, Stroop Colour and Word tests, and TMT

A and B tests (Erlanger et al., 2003; Spreen & Strauss, 1998). Despite alternate test forms being posited as mitigating practice effects, a number of neuropsychological tests used in assessing sport-related MTBI do not have alternate test forms (Erlanger, Feldman et al., 2003).

Small Sample Sizes

Small sample sizes are problematic in sport-related MTBI research. Two studies (Putukian et al., 2000; Stephens et al., 2005), which both included control groups and refuted the presence of MTBI sequelae, used small samples. Small samples are prone to the Type II error, and methodologically it can be argued that these studies failed to find significant effects, when such effects do exist. Generally, statistical power is less in small samples, and therefore even if differences do exist they fail to be revealed statistically (Shuttleworth-Edwards & Whitefield, 2007a).

Inferences are difficult to make when small samples are used. Several sport-related MTBI studies used small sample groups of 25 or less (e.g., Cremona-Meteyard & Geffen, 1994; Downs & Abwender, 2002; Jordan, Green et al., 1996; Guskiewicz et al., 1997; Iverson et al., 2002; Iverson, Brooks, Lovell & Collins, 2006; Iverson et al., 2004a; Killam et al., 2005; Macciocchi et al., 2001; Maddocks & Dicker, 1988; Maddocks & Saling, 1996; Makdissi et al., 2001; McCrea et al., 2002; Pettersen & Skelton, 2000; Putukian et al., 2000; Rutherford et al., 2005; Shuttleworth-Jordan et al., 1993; Stephens et al., 2005; Webbe & Ochs, 2003).

Lack of Control for Extraneous Variables

The few studies that included adequate size control groups, but refuted the presence of MTBI sequelae, did not provide convincing evidence of such (Shuttleworth-Edwards & Whitefield, 2007a). For example, the studies by Barr (2003) and Guskiewicz et al. (2002) lacked control for the extraneous variables education, IQ and gender. Studies need to control for confounding factors and match comparative groups on extraneous variables, particularly education and vocabulary that are known to affect test performance (Loosemore et al., 2007; Shuttleworth-Edwards, Kemp et al., 2004).

Shuttleworth-Jordan (1996) points out that racial effects on cognitive tests is highly contentious, and that differences on test performance often attributed to ethnic or cultural

differences, are actually the result of differences in educational level. As the socio-cultural gap diminishes, so does differing test performance between races, revealing a fundamentally common neurobehavioural function on cognitive test attainment. Shuttleworth-Jordan (1996) concludes that adjustments to cognitive test scores appear unwarranted among students of an equivalent educational level, but with differing first languages, in that socio-cultural influences are more closely aligned. Therefore, comparative sample groups should rather be matched on educational level, which implies socio-cultural equivalence, rather than being matched on race or first language (Shuttleworth-Jordan, 1996).

Separating the Effects of Concussive and Subconcussive Events

Rutherford et al. (2003) note that a major methodological difficulty is being able to separate the neuropsychological effects of concussive as opposed to subconcussive events, in that a combination of these factors is likely (e.g., Baroff, 1998). Rutherford et al. (2003) point out that studies often include participants who have sustained one or more MTBIs, and failure to control for these events in data analyses confounds the neuropsychological effects of concussive and subconcussive injury (e.g., Abreau et al., 1990; Downs & Abwender, 2002; Matser et al., 1998, 1999; Thomassen, Juul-Jensen, Olivarius, Briemer, & Christensen, 1979; Tysvaer & Lochen, 1991; Webbe & Ochs, 2003). This methodological problem is hampered further by concussive events being under-reported (e.g., Matser et al., 2001).

Under-reporting of MTBI

The under-reporting of concussive events, in addition to the likelihood of the occurrence of subconcussive events that obviates reporting, have major implications for the study of sport-related MTBI (Killam et al., 2005; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008). Therefore, contact sport studies investigating the cumulative effects of concussive and subconcussive injuries, require noncontact sport controls who are less susceptible to these injuries, for comparative purposes (Shuttleworth-Edwards & Radloff, 2008). However, Shuttleworth-Edwards and Radloff (2008) note that studies involving comparative contact and noncontact sports groups have used small sample sizes of 20 to 30 participants (e.g., Matser et al., 1999; Rutherford et al., 2005; Shuttleworth-Edwards, Border et al., 2004).

Normative Data Pertaining to Specific Sports

Neuropsychological normative data for various sports appear non-existent and comparing test scores with normative scores for the general population is inappropriate (Jordan, 2000). A need for sport-related normative data is essential, particularly when repeated measures are used. Therefore, sport-related normative data would take into account the subtle changes in scores post MTBI, regression to the mean at a second test occasion, and practice effects.

Statistical Problems

On the assumption that the null hypothesis is true, statistical hypothesis testing calculates the probability of an occurrence in the sample data having actually occurred, and that the results are not due to chance. The *p*-value or attained significance level is the smallest value of alpha for which the null hypothesis can be rejected. If the null hypothesis is rejected, when it actually should not be rejected, i.e., finding a significant difference between the means of the values being compared, when such difference does not exist, then this is a Type I or alpha error. To help avoid such error, a cut-off value, termed Alpha or level of significance, is used against which the *p*-value is evaluated. The smaller the Alpha value, the less likely is the risk of falsely rejecting the null hypothesis (University of South Africa [UNISA], 1996).

Researchers commonly use the 5% level of significance ($\alpha = 0.05$), indicating that there is a 5% probability that the observed differences in mean scores could have occurred by chance. The more stringent 1% level of significance ($\alpha = 0.01$) indicates that there is a 1% probability that the observed differences in mean scores could have occurred by chance. The stringent 1% level of significance, however, could result in a Type II or beta error, where the null hypothesis is not rejected, when it actually should have been rejected. In other words, not finding a significant difference between the means of the values being compared, when such difference does exist (UNISA, 1996). When a directional hypothesis is assumed (e.g., rugby players will perform more poorly than the control participants on neurocognitive testing due to repetitive subconcussive and concussive incidents), this permits the use of one-tailed tests, i.e., the *p*-values that are usually assigned for two-tailed tests are divided by two (UNISA, 1996). One-tailed tests have been used in previous rugby-related MTBI studies (e.g., Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards, Smith et al., 2008).

In order to reduce the risk of Type 1 error, when multiple measures are used, or the same data set is analysed a number of times, the Bonferroni Correction is used for a more scrupulous level of statistical significance, in order to reduce the risk of Type 1 error. The Bonferroni Correction is defined as a mathematical adjustment originally devised to reduce false significant results, that might arise from multiple statistical analyses on the same data set (Bland & Altman, 1995; Hsu, 1996; Perneger, 1998; Weisstein, 1996).

An explanation of why the Bonferroni Correction is used follows. Research studies usually yield a large amount of data, on which different statistical tests are conducted for comparisons between different sets of values, for the purpose of analyses. If a null hypothesis that is in fact true, was tested using one test at the 0.05 level of significance (Alpha), then the probability of reaching the right conclusion (i.e., not significant) would be 0.95. If several more null hypotheses that are in fact true (n), were tested using one test (0.95^n), or many tests (n) were used to test one null hypothesis that is in fact true (0.95^n), the probability ($1 - 0.95^n$) of reaching the right conclusion (i.e., not significant) in either scenario, decreases proportionately (Bland, 1995). Therefore, due to chance alone, the probability of being wrong (erroneously obtaining significant results, i.e., false positives) increases dramatically when more hypotheses are tested, or an increased number of tests are used on the same data set. The Bonferroni Correction is used to overcome this probability of obtaining false positives. The Correction involves either multiplying the *p*-value by the number of hypotheses or tests carried out on the data, and then measuring the corrected *p*-value against Alpha to determine significance, or dividing Alpha by the number of hypotheses or tests used and measuring the original *p*-value against the modified Alpha (Feise, 2002).

Much criticism has been levelled at use of the Bonferroni Correction, because it increases the probability of the Type II error occurring, and the Type II error is no less erroneous than the Type I error (Bland, 1995; Feise, 2002; Perneger, 1998). Therefore, the Bonferroni Correction may misrepresent what might ordinarily be sound results (Feise, 2002; Perneger, 1998). The misrepresentation of statistical results can have such critical consequences, that it is argued that the Bonferroni Correction is "at best, unnecessary and, at worst, deleterious to sound statistical inference" (Perneger, 1998, p. 1236). He argues that the focus of the Bonferroni method is on all null hypotheses being simultaneously true, which is seldom of interest or use to researchers. Perneger critically points out that

the study-wide error rate is applicable to only the universal null hypothesis (that two groups are identical on all variables), and thus no *a priori* hypothesis is held. However, researchers frequently assume differences between the groups being compared, and are interested in identifying the variables on which the groups differ.

With respect to criticism of the Bonferroni Correction in neuropsychology, it is notable that in neuropsychology clinical practice, several measures are commonly used in a neuropsychological assessment, in order to avoid missing subtle neurocognitive deficits and to assist diagnosis. Paradoxically, in neuropsychology research, when several measures are used, the Bonferroni Correction is often required. This is at the risk of losing statistical sensitivity and possibly incurring a Type II error with little or no significant difference in subtle neurocognitive functioning being found between the groups being compared, when it is most likely that such differences do exist (Shuttleworth-Edwards & Whitefield, 2007a).

While MTBI research is criticised for not taking the Type I error into account (Rutherford et al., 2003), more recently there is equal concern that both general neuropsychological research and MTBI research need also take the Type II error into account (Demakis, 2006; Frencham et al., 2005; Ruff, 2005; Shuttleworth-Edwards & Whitefield, 2007a; Woods et al., 2006). For a statistical adjustment to be appropriate, the statistical power should be neither over- nor under-corrected, thus eliminating the risk of Type I or Type II errors (Johnson & Wichern, 2002).

The most appropriate statistical adjustments for studies investigating subtle neuropsychological effects, appear to be those that (i) arbitrarily set Alpha at .01 to increase stringency when using between 27 to 30 tests (Cysique et al.,2007; Rutherford et al., 2003, 2005; Shuttleworth-Edwards, Smith et al.,2008), and those that used (ii) a the number of overarching cognitive functions, rather than the number of tests, for correction purposes. For example, Matser et al. (1998, 1999) used 27 neurocognitive variables that they classified into eight cognitive functions, and used these eight overarching functions for correction purposes. In addition, Shuttleworth-Edwards, Smith et al. (2008) classified seven neuropsychological test results into the two cognitive functions of attention and memory, and used these two overarching functions for correction purposes.

The neuropsychological effects of sport-related MTBI are subtle (Belanger & Vanderploeg, 2005; Shuttleworth-Edwards, Smith et al., 2008; Shuttleworth-Edwards & Whitefield, 2007a). Therefore, the possibility of incurring a Type II error (false-negative findings) is always pertinent, particularly in studies with small sample sizes (Shuttleworth-Edwards & Whitefield, 2007a). However, no known definitive solutions have been proposed to maintain statistical power in studies employing a number of neuropsychological tests, and investigating the subtle changes in neurocognitive functioning following MTBI. Considering the valid arguments of Perneger (1998), who vehemently criticises the use of Bonferroni Correction in biomedical related research, yet taking into consideration the Bonferroni concern (e.g., Rutherford et al., 2003, 2005), logic implores a compromise. The most compelling compromise appears to be that of using the number of over-arching neurocognitive functions investigated in a study, for the purposes of the Bonferroni Correction (e.g., Matser et al., 1998, 1999; Shuttleworth-Edwards, Smith et al., 2008). This compromise evolved in response to opposing demands of avoiding Type I or Type II errors, and to achieve a balance in the statistical power. The compromise appears to be becoming commonly employed in MTBI research (e.g., Matser et al., 1998, 1999; Shuttleworth-Edwards, Smith et al., 2008).

In summary of the discussion of methodological problems associated with sport-related MTBI studies, a sound study should include (i) a non-equivalent control group design in which the experimental and control groups are matched on variables such as gender, age, IQ, education, years of sport participation, and it should include (ii) pretesting and posttesting, and it should (iii) incorporate relevant and appropriate statistical procedures. Based on an understanding of these methodological problems, the rationale and hypotheses for this study are now addressed.

6.5 RATIONALE AND HYPOTHESES FOR THE PRESENT STUDY

The rationale for the present study will be discussed in terms of what appears to be required for a rugby-related MTBI study, with a focus on the experimental design and statistical issues. The theoretical hypotheses will then be provided.

6.5.1 Rationale for the Present Study

Although slowed reaction time is considered the most typical and meaningful feature of diffuse brain damage and MTBI (e.g., Sosnoff et al., 2007), two meta-analyses of neuropsychological outcomes following MTBI, found memory functions revealed the largest overall effect sizes, in comparison to several other cognitive functions e.g., attention, executive, or motor functions (Belanger et al., 2005; Belanger & Vanderploeg, 2005). Sport-related MTBI studies have used various versions of the WMS verbal and visual memory tests, to test memory impairment. Three studies that did not evidence memory deficits on these measures, both suffered methodological limitations regarding control groups e.g., no control group, a contact sport control group, or a small control group (e.g., Matser et al., 2001; Thornton et al., 2008; Wilberger et al., 1991). Two soccer studies have evidenced chronic memory deficits on these measures (e.g., Matser et al., 1998, 1999). Specifically, a well-controlled rugby study revealed that the WMS Visual Reproduction and Verbal Paired Associates subtests are sensitive to the chronic effects of MTBI (Shuttleworth-Edwards, Border et al., 2004). However, it is unknown if these two traditional tests are sensitive to the acute effects of rugby-related MTBI. Furthermore, no known sport-related MTBI study has used both the WMS Visual Reproduction and Verbal Paired Associates subtests, in conjunction with a computerised neuropsychological test.

On the grounds that traditional tests might reveal greater sensitivity to MTBI than a computerised test alone, and because a computerised test may evidence less practice effects than evidenced on several traditional measures, it was considered applicable that this study employ both the ImPACT computerised test and the WMS-III Verbal Paired Associates and Visual Reproduction subtests. The latter two traditional tests would evaluate verbal and visual memory specifically. The ImPACT test would provide a broader evaluation in measuring the functions typically sensitive to MTBI e.g., reaction time, visual motor speed, lack of impulse control, and including verbal and visual memory.

No sport-related MTBI study appears to have investigated both acute and chronic effects of MTBI, combining both traditional and computerised neuropsychological testing coupled with the study of symptoms, while employing non-equivalent noncontact sport

control groups for comparative purposes, where there is control for the demographic variables of gender, age, education, estimated IQ and length of sport participation. Furthermore, no rugby studies have investigated both the chronic and acute neurocognitive and symptom profiles among rugby players at a top provincial level that includes some national players, (earlier studies being restricted to top team high school, university level, and national adult and under-21 players only), and none have used both traditional and computerised neurocognitive testing and investigated symptom reports among adult rugby players in comparison with non-equivalent noncontact sport control groups. Therefore, it was decided that the current study would investigate both the chronic and acute effects of concussive and subconcussive injury among rugby players at a top provincial level. It would use *both* traditional and computerised measures, and would investigate *both* neurocognitive performance together with symptom reports at (i) baseline and end of season for nonconcussed rugby players and controls to investigate chronic neuropsychological effects, and at (ii) baseline and four follow-up intervals for concussed rugby players to investigate the overlay of acute neuropsychological effects on the chronic neuropsychological effects.

For the purposes of the above investigation, the statistics included independent (cross-sectional) comparisons and dependent (prospective) comparisons. As no differences were assumed between the means of the rugby and control groups on demographic variables (i.e., non-directional null hypotheses), standard *p*-values for two-tailed tests were employed for the demographic statistical analyses. However, with expectation that the rugby groups would perform more poorly than the noncontact sport control groups on neurocognitive and symptom measures (i.e., directional null hypotheses), *p*-values for one-tailed tests were implemented. The Bonferroni Correction was implemented for the neurocognitive measures as a means of added statistical stringency.

6.5.2 Theoretical Hypotheses

According to the rationale for the present study, the following theoretical hypotheses were proposed for (i) rugby players who did not report MTBI during the rugby season (hereafter termed General Rugby group) and their demographically equivalent noncontact sport controls (hereafter termed General Control group), and (ii) rugby players who did report MTBI during the rugby season (hereafter termed Concussed Rugby group) and

their demographically equivalent noncontact sport controls (hereafter termed Matched Controls). The hypotheses are presented first in terms of the indications in respect of neurocognitive measures, followed by the indications in respect of symptom measures:

General Rugby and General Control Groups

Neurocognitive Measures

(i) It was hypothesised that the General Rugby group relative to the General Control group, would demonstrate poorer performance on the *neurocognitive tests* at the *baseline* test interval. This is due to long-term exposure to concussive and subconcussive events historically, implicating chronic or permanent residual effects.

(ii) It was hypothesised that the General Rugby group relative to the General Control group, would demonstrate poorer performance on the *neurocognitive tests* at the *end of season* test interval. This is due to long-term exposure to concussive and subconcussive events historically, and an overlay of unreported concussive and subconcussive events sustained during the 2005 rugby season. However, it was understood that it would not be possible to differentiate between these two sets of past and more recent effects.

(iii) It was hypothesised that the General Rugby group would demonstrate poorer performance on the *neurocognitive tests* at the *end of season* test interval compared to the *baseline* test interval, in support of deleterious neurocognitive effects in the form of either poorer performance, or the absence of a practice effect when it is expected. This is due to long-term exposure to concussive and subconcussive events historically, and an overlay of unreported concussive and subconcussive events sustained during the 2005 rugby season. However, it was understood that it would not be possible to differentiate between these two sets of past and more recent effects.

(iv) It was hypothesised that the General Control group would not demonstrate poorer performance on the *neurocognitive tests* at the *end of season* test interval compared to the *baseline* test interval, and/or that there would be a practice effect when it is expected, in association with a lack of exposure to concussive and subconcussive events historically and during the equivalent 2005 rugby season.

Symptom Measures

(v) It was hypothesised that the General Rugby group relative to the General Control group, would demonstrate higher *symptom reports* on the Symptom Scale at the *baseline* test interval. This is due to long-term exposure to concussive and subconcussive events historically, implicating chronic or permanent residual effects.

(vi) It was hypothesised that the General Rugby group relative to the General Control group, would demonstrate higher *symptom reports* on the Symptom Scale at the *end of season* test interval. This is due to long-term exposure to concussive and subconcussive events historically, and an overlay of unreported concussive and subconcussive events sustained during the 2005 rugby season. However, it was understood that it would not be possible to differentiate between these two sets of past and more recent effects.

(vii) It was hypothesised that the General Rugby group would demonstrate increased *symptom reports* on the Symptom Scale at the *end of season* test interval compared to the *baseline* test interval, in support of deleterious neuropsychological effects. This is due to long-term exposure to concussive and subconcussive events historically, and an overlay of unreported concussive and subconcussive events sustained during the 2005 rugby season. However, it was understood that it would not be possible to differentiate between these two sets of past and more recent effects.

(viii) It was hypothesised that the General Control group would not demonstrate increased *symptom reports* on the Symptom Scale at the *end of season* test interval compared to the *baseline* test interval, in association with a lack of exposure to concussive and subconcussive events historically and during the equivalent 2005 rugby season.

<u>Concussed Rugby and Matched Control Groups</u>

Neurocognitive Measures

(i) It was hypothesised that the Concussed Rugby group relative to the Matched Control group, would demonstrate poorer performance on the *neurocognitive tests* at the *baseline*

test interval. This is due to long-term exposure to concussive and subconcussive events historically, implicating chronic or permanent residual effects.

(ii) It was hypothesised that the Concussed Rugby group relative to the Matched Control group, would demonstrate poorer performance on the *neurocognitive tests* at the *postconcussion* test intervals. This is due to long-term exposure to concussive and subconcussive events historically, and the overlay of acute effects of reported concussive events. However, it was understood that it would not be possible to clearly differentiate between the past and more recent acute effects of the reported concussive events.

(iii) It was hypothesised that the Concussed Rugby group would demonstrate poorer performance on the *neurocognitive tests* at the *postconcussion* test intervals compared to the *baseline* test interval, in support of deleterious neurocognitive effects in the form of either poorer performance, or the absence of a practice effect when it is expected. This is due to long-term exposure to concussive and subconcussive events historically, and the overlay of acute effects of reported concussive events. However, it was understood that it would not be possible to clearly differentiate between the past and more recent acute effects of the reported concussive events.

(iv) It was hypothesised that the Matched Control group would not demonstrate poorer performance on the *neurocognitive tests* at the *postconcussion* test intervals compared to the *baseline* test interval, and/or that there would be a practice effect when it is expected, in association with a lack of exposure to concussive and subconcussive events historically and during the equivalent portion of the 2005 rugby season.

Symptom Measures

(v) It was hypothesised that the Concussed Rugby group relative to the Matched Control group, would demonstrate higher *symptom reports* on the Symptom Scale at the *baseline* test interval. This is due to long-term exposure to concussive and subconcussive events historically, implicating chronic or permanent residual effects.

(vi) It was hypothesised that the Concussed Rugby group relative to the Matched Control group, would demonstrate higher *symptom reports* on the Symptom Scale at the

postconcussion test intervals. This is due to long-term exposure to concussive and subconcussive events historically, and the overlay of acute effects of reported concussive events. However, it was understood that it would not be possible to clearly differentiate between the past and more recent acute effects of the reported concussive events.

(vii) It was hypothesised that the Concussed Rugby group would demonstrate increased *symptom reports* on the Symptom Scale at the *postconcussion* test intervals compared to the *baseline* test interval, in support of deleterious neuropsychological effects. This is due to long-term exposure to concussive and subconcussive events historically, and the overlay of acute effects of reported concussive events. However, it was understood that it would not be possible to clearly differentiate between the past and more recent acute effects of the reported concussive events.

(viii) It was hypothesised that the Matched Control group would not demonstrate an increased report of *symptom scores* on the Symptom Scale at the *postconcussion* test intervals compared to the *baseline* test interval, in association with a lack of exposure to concussive and subconcussive events historically and during the equivalent portion of the 2005 rugby season.

6.6 OVERVIEW

This chapter reviewed the neuropsychological findings of sport-related MTBI research, in terms of the cognitive functions most commonly impaired, including attention and concentration, information and visuomotor processing speed, reaction time, memory, and executive functioning, and symptoms arising as a result of MTBI.

A comprehensive review of neuropsychological studies on MTBI in rugby union was detailed. The methodological limitations of neuropsychological research in sport were treated. The findings of sport-related MTBI and specifically rugby-related MTBI studies, and the methodological pitfalls of these studies, prepared the foundation and motivation for the rationale and hypotheses of the present study. This lays the foundation for the methodology of this study, which is presented in the following chapter.

NEUROCOGNITIVE AND SYMPTOM PROFILES OF CONCUSSED AND NONCONCUSSED PROVINCIAL RUGBY UNION PLAYERS OVER ONE SEASON

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CHAPTER SEVEN: METHODOLOGY

The focus of this chapter is on the methodological procedures employed in this study. Initially the participants in this study are discussed in terms of exclusion criteria and their inclusion in the comparative groups that were derived for the purpose of analysis. Following this, the sample characteristics pertaining to the comparative groups are described, including age, education completed, estimated Full Scale IQ, Verbal (Vocabulary) and Non-Verbal (Matrix Reasoning) IQ subtest scaled scores, primary sport participation and number of prior concussions. The research procedure and research materials employed, including demographic questionnaires, neurocognitive measures and symptom measures, are then addressed. Finally, attention is given to the data processing and analysis, followed by the statistical hypotheses for this study.

7.1 PARTICIPANTS

All tables relating to participants appear at the end of this subsection.

7.1.1 Consent

Approval for the research was obtained from the Rhodes University Ethics committee and from the two Chief Executive Officers of The Sharks Pty Ltd. Participation in this study was voluntary and participants were free to withdraw from the research at any stage of the procedure. Participants were briefed about the research verbally and were each provided with a document outlining basic details of the research (Appendix A) before giving their written, informed consent (Appendix B).

7.1.2 Selection Criteria

Rugby Participants

During 2005, 97 national, provincial and academy male rugby players from the province of KwaZulu-Natal, South Africa, were invited to participate in this study (n = 97). All rugby players were post-school sportsmen. National rugby players consisted of sportsmen who had been selected to play rugby for the country, in the open, under-21 or under-19 age groups for either the 15-aside or 7-aside national rugby teams (n = 22). Provincial

rugby players consisted of sportsmen who had been selected to play rugby for the province, in the open, under-21 or under-19 age groups for either the 15-aside or 7-aside provincial rugby teams (n = 87). Academy rugby players consisted of sportsmen who were undertaking tertiary studies at the Sharks Academy while concentrating on rugby training and included both provincial rugby players and six aspiring provincial rugby players who played club rugby (n = 6).

Control Participants

Initially an ideal noncontact sport control sample of either provincial hockey or cricket players was anticipated. In 2005, the management and coach for the provincial cricket were approached to elicit their participation, but this was not forthcoming. The management for provincial hockey was then approached but as the players were drawn from various clubs throughout the province it was advised that the individual hockey clubs be approached. Individual hockey clubs were approached, but unlike rugby players no clear benefit to the players warranted the recommendation of routine participation in a concussion management program that included neurocognitive baseline assessment as part of club policy. Therefore, comparatively ad hoc volunteer participation was the only option as a means of gaining control participants. An inadequate number of hockey control participants volunteered their participation and thus control participants playing various noncontact sports had to be sought. Hence during 2005 and 2006 the researcher directly approached the hierarchy (management, coaches and captains) of various sports clubs, covering a variety of noncontact sports at national, provincial or club levels, to elicit their support for the study. An agreement was reached at each organisation that their sportsmen would be informed about the study by both the club hierarchy and via written invitation (see Appendix C) in order to encourage participation in a noncontact sport control group. In order to obtain a demographically balanced group of volunteers it was decided to offer payment for participation, in that it was of concern that only those who had transport, or could afford transport to the stadium, would be able to volunteer for testing. Remuneration of R50 was offered for each test session attended, an amount that covered approximately 50% of transport costs. Seventy-three male participants volunteered their participation in the study (n = 73).

7.1.3 Exclusion Criteria

Information pertaining to the first six exclusion criteria detailed below was obtained at the time of baseline testing. Information was elicited via the Immediate Post-concussion Assessment and Cognitive Testing (ImPACT) computer based test program's inbuilt demographic questionnaire that each participant completed. In addition, using a pencil and paper questionnaire, the researcher elicited comprehensive historical and current information pertaining to each participant's health, sport, educational and concussion history, in order to establish which participants should be excluded from this study (see Appendix E). Exclusion criteria, including the associated numbers of participants considered unsuitable for the study on these grounds, were as follows:

History of a Neurological Disorder

This included meningitis, encephalitis, epilepsy and brain surgery for any reason: No rugby or control participants were excluded on these grounds.

Current Diagnosis of a Psychiatric Disorder

This included depression, anxiety, sleep disorders, and Attention Deficit/Hyperactivity Disorder (determined by those who were treated with Ritalin when younger). No rugby or control participants were excluded on these grounds.

History of Alcohol or Substance Abuse

This included a diagnosed history of alcohol or substance *abuse* (prescription or recreational drugs). No rugby or control participants were excluded on these grounds. Additional detailed information was elicited on alcohol and nicotine *use* amongst the rugby and control participants, and is detailed in the Sample Characteristics section below.

Low Estimated Intelligence Quotient (IQ)

This included estimated IQ scores that fell in the borderline or mental retardation ranges of intelligence (Wechsler et al., 1998a), based on estimated Full Scale IQ scores of less than 80 points. Five participants were excluded from the study on these grounds, including one rugby and four control participants (total $n = 5$; rugby $n = 1$; control $n = 4$).

History of Recent Concussions (Controls only)

This included any concussive incident (MTBI) that a control participant had sustained within the previous five years, including those that were actually diagnosed by medical personnel (e.g., medical doctor, registered nurse or First Aid attendant), as well as those reported to have been sustained by the participant that complied with the definition of concussion (MTBI) 'any non-penetrative traumatic force to the brain resulting in the alteration of consciousness (that may or may not include loss of consciousness for a period of less than thirty minutes duration) or a posttraumatic amnesia persisting for less than 24 hours' (ACRM, 1993) and, for purposes of this study, includes a set of signs and symptoms impacting on neuropsychological and functional impairment as result of underlying structural impairment. Three control participants with a history of a diagnosed concussion within the past five years were excluded from the study (n = 3). Three control participants who reported a history of concussion seven, nine and fifteen years previously were included in the study. All rugby players with a history of concussion were included in the study.

History of Moderate to Severe Traumatic Brain Injury (TBI)

This included any moderate to severe TBI incident that a rugby or control participant had sustained at any time in their past, and constituted those TBI incidents that (i) resulted in hospitalisation and (ii) were defined as a non-penetrative traumatic force to the brain, with loss of consciousness exceeding a period of 30 minutes. Two control participants with histories of TBI, caused by motor vehicle accidents with resultant hospitalisation and loss of consciousness *exceeding* 30 minutes duration, were excluded from the study (n = 2).

Potentially Confounding Test-taking Issues

This included the potentially confounding criteria of extreme tiredness and veisalgia (symptoms resulting from excessive alcohol consumption) that might confound results. Three control participants were excluded on these grounds (n = 3). Two of these participants were excluded at the end of season testing (n = 2) and one participant at repeat testing (n = 1).

Additional Miscellaneous Exclusions

This included a number of exclusions for miscellaneous reasons, not specified anywhere else, that might confound results or result in incomplete data for the analyses: One rugby player was concussed at the time of baseline testing (n = 1); one control participant received a concussion during the research period (n = 1); one concussed rugby player received a further concussion during his initial concussion follow-up (n = 1); one concussed player (n = 1) and one control participant (n = 1) did not return for adequate repeat testing (n = 2); seventeen rugby participants (n = 17) and four control participants (n = 4) were absent from end of season testing (n = 21); five injured rugby players did not play rugby during the rugby season (n = 5); two control participants withdrew from the research (n = 2). A summary of all exclusions both at the start and during the study, are provided in Table 7 (at the end of this subsection).

7.1.4 Constitution of the Comparative Subgroups

Rugby Participants

The 97 rugby participants were divided into two subgroups of rugby players for the purposes of this study, including a 'General Rugby group' who had no reported concussions during the rugby season and a 'Concussed Rugby group', who had reported concussions during the rugby season, as follows:

General Rugby group (n = 54)

Twenty rugby players were concussed during the rugby season (n = 20). Removal of the concussed rugby players (n = 20), left a pool of 77 general rugby players (n = 77). Of these 77 general players, one player (n = 1) was totally excluded from all analysis as his estimated IQ score fell in the borderline range of intelligence. This left a pool of 76 general rugby players (n = 76). Twenty-two of these players were excluded from the end of season analysis (n = 22) for the following reasons: Five injured rugby players did not play rugby during the season (n = 5); seventeen rugby players did not attend end of season testing (n = 17). This left a pool of 54 rugby players who made up the General Rugby group (n = 54), for the purposes of an analysis that constituted baseline and end of season testing.

Concussed Rugby group (n = 17)

Of the total 97 rugby players who received baseline testing (*n* = 97), 20 players were concussed during the season (*n* = 20). Of these 20 concussed players three were excluded from the analysis (*n* = 3) for the following reasons: One player returned for only one instead of four postconcussion follow-up testing occasions (*n* = 1); one player received a second concussion during the process of his postconcussion follow-up testing (*n* = 1); one player had a concussion at the time of baseline testing (*n* = 1). This left a pool of 17 concussed rugby players who made up the Concussed Rugby group (*n* = 17), for the purposes of an analysis that involved baseline and four postconcussion testing occasions.

Control Participants

Of the 73 control participants 16 were excluded for the purpose of analysis (*n* = 16) for the following reasons: One sportsman was concussed during the research (*n* = 1); three sportsmen had been diagnosed as concussed within the previous five years (*n* = 3); two sportsmen had a history of moderate to severe traumatic brain injury involving loss of consciousness for more than 30 minutes (*n* = 2); four sportsmen's estimated IQ scores fell in the borderline and mild mental retardation ranges of intelligence (*n* = 4); two sportsmen withdrew from the research as they left the country (*n* = 2); four sportsmen could not be located for their end of season testing (*n* = 4). This left a pool of 57 control participants (*n* = 57) who were divided up into two subgroups. Of these 57 control participants, nine sportsmen had been selected to represent their sport at National level (*n* = 9); twenty-four sportsmen had been selected to represent their sport at a provincial level (*n* = 24); and thirty-three sportsmen competed in their sport at a First league or Club First side equivalent level (*n* = 33). The control group consisting of national, provincial and first league equivalent sportsmen were considered a good match with the rugby group for comparison purposes. The pool of 57 control participants (*n* = 57) were divided up into two subgroups, including a General Control group for comparisons with the General Rugby group, and a Matched Control group for comparisons with the Concussed Rugby group, as follows:

General Control group (for comparisons with the General Rugby group) (n = 37)

Of the 57 control participants 18 sportsmen (*n* = 18) were removed and used for comparison purposes with the concussed rugby players, leaving a pool of 39 sportsmen in the General Control group (*n* = 39). Of these 39 control participants, two sportsmen's end

of season test results were excluded from the analysis (n = 2) as they were suffering with veisalgia at the time of their end of season testing and this was considered to be a potentially confounding variable (n = 37). This left a pool of 37 control participants in the General Control group (n = 37) who were used for comparison purposes with the General Rugby group (n = 54) in an analysis that constituted baseline and end of season testing.

Matched Control group (for comparisons with the Concussed Rugby group) (n = 17)
In order to gain a demographically similar control group, a list of the 57 control participants was drawn up in terms of age, education, WAIS-III estimated IQ scores, race and language and as closely as possible a matching partner on each of these dimensions was chosen for each of the 18 concussed rugby players. These 18 control participants (n = 18) with a spread of similar demographic characteristics to the group of concussed rugby players were approached and agreed to participate in the repeat testing at almost identical postconcussion test intervals to that of the concussed rugby players. One matched control participant was excluded (n = 1) as he was extremely tired at the time of his second repeat test occasion and that was considered to be a potentially confounding variable. This left a pool of 17 control participants in the Matched Control group (n = 17) for comparison purposes with the Concussed Rugby group (n = 17) that involved baseline and four repeat test occasions.

In sum the final samples for analysis consisted of two sets of comparative groups: the General Rugby group (n = 54) and General Control group (n = 37); and the Concussed Rugby group (n = 17) and Matched Control group (n = 17). As indicated, for the purposes of differentiation the control groups will be referred hereafter as the General Control group and the Matched Control group.

7.1.5 Sample Characteristics

Rugby and control groups were compared on a number of common demographic variables that might affect neurocognitive test performance, including age, educational level, estimated Intelligence Quotient (IQ), history of MTBI, race and language (Lezak et al., 2004). Specifically, independent t-test analyses were used to compare the group means for the rugby and control groups in order to ascertain equivalence between groups on the variables of age, educational level, estimated Full Scale IQ and the scaled scores of

two IQ subtests. Chi-square analyses were conducted to compare the distribution between groups for first language and race. Independent *t*-test analyses were used to compare the number of years the sportsmen participated in their primary sport, and the number of prior diagnosed concussions (i.e., those that had occurred prior to the onset of baseline testing) that were reported between the rugby and control groups.

Age, Education, and IQ

In all instances the units of measurement for these variables were calculated at the time of baseline testing. The *age* of each participant was documented in years. The *educational level* of each participant was calculated in years according to the number of grades successfully completed at school (12 being the maximum) and additional years of successfully completed tertiary education at University or College. Research conducted by Shuttleworth-Edwards, Kemp et al. (2004) alert to the general research finding that in addition to *level* of education, *quality* of education has an effect on neurocognitive test performance. Relevant to the South African context is the presence of vastly differing qualities of education offered between the relatively disadvantaged black township schools compared with that available at the relatively advantaged traditionally white schools that can now (since the dismantling of Apartheid) be attended by black and coloured individuals. In South Africa, for official census purposes, a division is made between 'white', 'black' and 'coloured' individuals (coloured persons being those of mixed race), and accordingly a number of psychometric measures used in South Africa have been normed with stratification along the same racial divisions given the vastly differing educational opportunities that have been historically accessible to these various race groups (e.g., Claassen, Krynauw, Paterson, & Mathe, 2001; Shuttleworth-Edwards, Gaylard, & Radloff, in press; Shuttleworth-Edwards, Van der Merwe, van Tonder, & Radloff, in press). Shuttleworth-Edwards, Kemp et al. (2004) found that differences in neurocognitive test performance are minimised between White English first language and Black African first language individuals when exposed to the relatively advantaged quality of education offered by the former white schools. None of the participants in this study were known to be from a background of relatively disadvantaged education.

An estimate of a *Full Scale Intelligence Quotient* (FSIQ), reported in IQ points (standardisation mean = 100), was obtained for each participant by administering and using the raw scores of the Vocabulary and Matrix Reasoning subtests from the Wechsler

Adult Intelligence Scales (WAIS–III) in conjunction with use of the Oklahoma Pre-morbid Intelligence Estimate [OPIE-3 (2ST)] formula (Schoenberg, Scott, Duff, & Adams, 2002). Schoenberg et al. (2002), reported that the OPIE-3 (2ST) formula correlates highly with the WAIS-III FSIQ (p = .897). The OPIE, that neither over nor under estimated predicted IQ, has been found a reliable method in premorbid IQ prediction among clinical samples (Ropacki & Elias, 1999), and demonstrated utility in both normal and neurologically impaired populations (Ropacki & Elias, 1999, 2003; Scott, Krull, Williamson, Adams, & Iverson, 1997; Spreen & Straus, 1998). A summary of the OPIE-3 (2ST) formula is given in Appendix D. In addition to the FSIQ, the Vocabulary and Matrix Reasoning subtest scaled scores were calculated (scaled scores range 1 – 20, standardisation mean = 10). The Vocabulary subtest scaled score was extrapolated as an estimate of general verbal ability as it forms part of the WAIS-III Verbal Scale IQ. Equivalence between groups on verbal ability needed to be ascertained due to the number of different languages spoken by the participants. The Matrix Reasoning subtest scaled score was extrapolated as an estimate of non-verbal ability because it forms part of the WAIS-III Non-Verbal Scale IQ, for which equivalence between groups was ascertained for descriptive purposes. The independent t-test analyses for comparisons for the demographic variables of age, educational level, estimated FSIQ, Vocabulary scaled score, and Matrix Reasoning scaled score across the rugby and control groups are provided in Table 8, at the end of this section.

There were no significant differences between the mean scores of the comparative sports groups for age (General Rugby versus General Control p = .323; Concussed Rugby versus Matched Control p = 1.000), education (General Rugby versus General Control p = .147; Concussed Rugby versus Matched Control p = 1.000), estimated FSIQ (General Rugby versus General Control p = .053; Concussed Rugby versus Matched Control p = .705), and Vocabulary (General Rugby versus General Control p = .147; Concussed Rugby versus Matched Control p = .836). In respect of Matrix Reasoning, there was a significant difference between the mean scores of the General Rugby group and the General Control group in favour of the General Control group (p = .032). The General Rugby group obtained a Matrix Reasoning mean scaled score of 11.19 (SD = 2.04) with scores ranging between 5 - 17, and the General Control group obtained a Matrix Reasoning mean scaled score of 12.43 (SD = 2.43), with scores ranging between 6 - 16. No significant difference was revealed on the Matrix Reasoning mean score between the Concussed Rugby group

and the Matched Control group (p = .514). In respect of the estimated FSIQ, the difference between the General Rugby group and the General Control group was approaching significance in favour of the General Control group (p = .053). The General Rugby group obtained an estimated mean FSIQ of 105.33 (*SD* = 10.06) with estimated scores ranging between 83 - 124, and the General Control group obtained an estimated mean FSIQ of 109.70 (*SD* = 10.96), with estimated scores ranging between 83 - 126.

Concussion History

Concussion history was recorded as the number of previous concussions sustained by each participant. Each participant recorded the number of prior concussions on the ImPACT program's inbuilt questionnaire, and concussion histories were also extensively investigated by the researcher and documented on a pencil and paper questionnaire (See Appendix E.). For the purpose of this study, control participants who reported a prior 'concussion' involving loss of consciousness for more than 30 minutes duration were excluded, as this was considered to delineate mild traumatic brain injury (MTBI) from moderate and severe traumatic brain injury (Kibby & Long, 1996).Thus documented concussions pertained to the milder spectrum of TBI severity, typical of that which occurs in sport-related concussion (Lovell et al., 1999). The independent *t*-test analyses for comparisons for the number of prior concussions across the rugby and control groups are provided in Table 8, at the end of the section.

In respect of the number of concussions, both the General and Concussed Rugby groups revealed a history of significantly more concussions than the General and Matched Control groups (p = .032 and p = .004, respectively). The General Rugby group averaged 1.30 prior concussions (*SD* 1.74) ranging from nil to eight concussions, whereas General Control group reported 0.08 prior concussions (*SD* 0.28) ranging from nil to one concussion. The Concussed Rugby group averaged 3.29 prior concussions (*SD* 4.31) ranging from nil 0 to 16 concussions, whereas the Matched Control group reported nil prior concussions.

Primary Sport Participation

Years of primary sport participation were calculated by taking each participant's age in years at baseline testing and deducting the age in years at which each rugby participant started playing rugby and each control participant started playing his primary sport. The

independent *t*-test analyses for comparisons for the years of primary sport participation across the rugby and control groups are provided in Table 8, at the end of the section. There were no significant differences between the mean scores of the comparative sports groups for length of primary sport participation (General Rugby versus General Control $p = .914$; Concussed Rugby versus Matched Control $p = .440$).

Type of primary sport: Rugby was the primary sport for all the rugby players. The distribution of primary noncontact sports for the General Control group ($n = 37$) follows (see Table 9, at the end of this section): angling ($n = 1$), athletics ($n = 6$), basketball ($n = 1$), bowling ($n = 1$), cricket ($n = 7$), cycling ($n = 2$), field hockey ($n = 8$), indoor hockey ($n = 3$), rowing ($n = 2$), squash ($n = 1$), surfing ($n = 1$), swimming ($n = 1$), tennis ($n = 1$), and waterpolo ($n = 2$). The distribution of primary noncontact sports for the Matched Control group ($n = 17$) follows (see Table 9): athletics ($n= 4$), cricket ($n = 3$), field hockey ($n = 6$), indoor hockey ($n = 1$), lifesaving ($n = 1$), squash ($n = 1$) and touch rugby ($n = 1$).

Language and Race

As indicated above, research on cross-cultural effects on IQ test performance conducted by Shuttleworth-Edwards, Kemp et al. (2004) found that the *level* and particularly the *quality* of education, rather than either first language or race per se, explained differing neurocognitive test performance. Although lowered neurocognitive test performance was found amongst Black African first language individuals exposed to a relatively disadvantaged education offered by the former non-White South African schools, they found minimal differences in neurocognitive test performance between White English first language and Black African first language individuals when exposed to a relatively advantaged quality of education offered by the private and former government White schools. Thus, relevant to the South African context, is that the quality and level of education is affirmed as an important factor influencing neurocognitive test performance, rather than first language or race. As no participants in this study were known to have been exposed to a relatively disadvantaged education, it is considered unlikely that either first language or racial distribution across the groups would affect neurocognitive test performance. However, to further ensure equivalence between groups being compared, Pearson Chi-square analyses were conducted for comparisons of the percentage distribution within groups for both language and race for the General and Concussed Rugby versus the General and Matched Control groups, respectively.

A Pearson Chi-square analysis for comparisons of the percentage distribution within groups for African, Afrikaans and English *first language* for the General and Concussed Rugby versus the General and Matched Control groups, respectively, is provided in Table 10, at the end of this section. There were no significant differences between the percentage distribution within the comparative sports groups for the variable of first language (General Rugby versus General Control $p = .084$; Concussed Rugby versus Matched Control $p = .379$), suggesting equivalence between the groups on the variable of language. The percentage distribution between the General Rugby versus the General Control was approaching significance ($p = .084$), due to there being a higher percentage of African first language sportsmen in the General Rugby group versus the General Control group, however due to none of the participants having been exposed to a relatively disadvantaged education, it is considered that this trend is unlikely to have served to confound the results.

A Pearson Chi-square analysis for comparisons of the *racial distribution* within groups for the General and Concussed Rugby versus the General and Matched Control groups, respectively, is provided in Table 11, at the end of this section. There were no significant differences between the percentage distribution within the comparative sports groups for the variables of race (General Rugby versus General Control $p = .082$; Concussed Rugby versus Matched Control $p = .327$), suggesting equivalence between the groups on the variable of race. The percentage distribution between the General Rugby versus General Control was approaching significance ($p = .082$), due to there being a higher percentage of Black sportsmen in the General Rugby group versus the higher percentage of White sportsmen in the General Control group, however due to none of the participants having been exposed a relatively disadvantaged education it is considered that this trend is unlikely to have served to confound the results.

Use of Alcohol and Nicotine

As indicated above alcohol *abuse* was used as an exclusion criterion. Alcohol *use*, however, was not used as an exclusion criterion because it was not possible to establish a suitable cut-off point. Literature (Macciocchi & Barth, 2004; Matser et al., 2000; Shuttleworth-Edwards & Radloff, 2008) has alluded to alcohol use as a confounding variable in MTBI studies, but research has not suggested a decisive level at which alcohol use can be considered a confounding variable. There is a growing body of literature

relating chronic smoking to brain insult and dysfunction particularly in the middle aged and aging population (Cervilla, Prince, & Mann, 2000; Deary et al., 2003; Durazzo, Gazdzinski, & Meyerhoff, 2007; Elwood et al., 1999; Kalmijn, van Boxtel, Verschuren, Jolles, & Launer, 2002; Launer, Feskens, Kalmijn, & Kromhout, 1996). No known literature has referred to the use of nicotine as a confounding variable in neurocognitive testing of MTBI generally or in neurocognitive testing of rugby players specifically and was therefore (as with alcohol *use)* not used as a formal exclusion criterion for the purposes of this research. However, information about alcohol and nicotine use, elicited at the time of taking the demographic histories, was included as an additional means of describing differences between the rugby and control groups, and could be used for discussion purposes.

Pearson Chi-Square analyses for comparisons of the percentage distribution within groups for *alcohol consumption* and *cigarettes smoked* weekly for the General and Concussed Rugby groups versus the General and Matched Control groups, respectively are provided in Tables 12 and 13, at the end of this section. Significant differences were revealed between the percentage distributions of the General Rugby group versus the General Control group in respect of both alcohol consumption and cigarettes smoked per week (p = .009 and p = < .000, respectively), with the implication of a significantly higher consumption of each of these substances amongst the General Control group. No significant differences were revealed between the percentage distributions of the Concussed Rugby group versus the Matched Control group for the variables of alcohol consumption and cigarettes smoked per week (p = .186 and p = .267, respectively), although trends were in the direction of higher consumption of each of these substances amongst the Matched Control group.

Summary of Sample Characteristics

In sum, between the comparative sports groups no significant differences existed between the means on the variables of age, education, FSIQ, Vocabulary mean scaled score, first language, race and length of primary sport participation, suggesting equivalence between the groups on these variables. A significant difference for the Matrix Reasoning mean scaled score was revealed between the General Rugby group and the General Control group in favour of the General Control group. No significant difference on the Matrix Reasoning mean scaled score, however, was revealed between the Concussed Rugby

group and the Matched Control group. Furthermore, it was revealed that both the General and Concussed Rugby groups had a history of significantly more concussions than the General and Matched Control groups, respectively, which would be anticipated as rugby is a high impact sport with more concussions received than with noncontact types of sport in which concussion incidences are lower. Significant differences for average weekly alcohol consumption and cigarettes smoked were revealed between the General Rugby group and the General Control group with the General Control group consuming more alcohol and smoking more cigarettes than the General Control group. No significant differences for average weekly alcohol consumption and cigarettes smoked were revealed between the Concussed Rugby group and the Matched Control group, despite a trend being revealed that on average the Matched Control group consumed more alcohol and smoked more cigarettes than the Concussed Rugby group.

Table 7

Exclusions From the Total Rugby and Control Groups

Exclusion Categories	Rugby (Original Pool, *n* = 97) (Final Pool, *n* = 71)	Controls (Original Pool, *n* = 73) (Final Pool, *n* = 54)
Neurological disorder	0	0
Psychiatric disorder	0	0
Alcohol or substance abuse	0	0
Low Estimated IQ (<80)	1	4
Prior concussions in the previous five years	N/A	3
Moderate or Severe Traumatic Brain Injury	0	2
Concussion at baseline	1	0
Concussion during research period	0	1
Second concussion while being followed–up	1	0
Inadequate concussion follow-up testing	1	1
Test taking issues	0	2
Absent from end of season testing	17	4
End of season testing invalid (no sport played)	5	0
Withdrew from the research	0	2

Table 8

Comparisons of Demographic Data, Number of Concussions, and Sport Participation for Rugby versus Control Groups

	General Rugby and General Controls					
	Rugby (n = 54)		Controls (n = 37)			
Variable	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Age	22.33	(3.56)	23.14	(4.09)	-0.993	0.323
Education	13.02	(1.14)	13.43	(1.56)	-1.465	0.147
Estimated IQ score[I]	105.33	(10.06)	109.70	(10.96)	-1.962	0.053
Vocabulary	11.30	(2.04)	12.05	(2.91)	-1.462	0.147
Matrix Reasoning	11.19	(2.84)	12.43	(2.43)	-2.177	0.032*
No. prior MTBIs	1.30	(1.74)	0.08	(0.28)	4.195	0.000**
Years of sport participation	13.61	(4.38)	13.73	(6.03)	-0.109	0.914
	Concussed Rugby and Matched Controls					
	Rugby (n = 17)		Controls (n = 17)			
Variable	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Age	22.53	(3.84)	22.53	(3.92)	0.000	1.000
Education	13.18	(1.13)	13.18	(1.33)	0.000	1.000
Estimated IQ score[I]	108.76	(12.90)	110.41	(12.22)	-0.382	0.705
Vocabulary	12.65	(3.46)	12.88	(3.10)	-0.209	0.836
Matrix Reasoning	11.59	(3.12)	12.29	(3.12)	-0.659	0.514
No. prior MTBIs	3.29	(4.31)	0.00	(0.00)	3.150	0.004**
Years of sport participation	14.76	(4.99)	13.35	(5.51)	0.783	0.440

Note. [1]Control for estimated Full Scale IQ established on the basis of WAIS-III Vocabulary and Matrix Reasoning subtests using the OPIE-3 (2ST) Estimation Formula.

* $p < .05$, ** $p < .01$, two-tailed

Table 9

Primary Sport for the Noncontact Sport Control Groups

	Noncontact Sport Controls	
	General Controls (n = 37)	Matched Controls (n = 17)
Primary sport category	*n*	*n*
Angling	1	
Athletics	6	4
Basketball	1	
Bowling	1	
Cricket	7	3
Cycling	2	
Field hockey	8	6
Indoor hockey	3	1
Lifesaving		1
Rowing	2	
Squash	1	1
Surfing	1	
Swimming	1	
Tennis	1	
Touch rugby		1
Waterpolo	2	

Table 10

Language Distribution Within the Rugby and Control Groups

General Rugby and General Controls	*N*	Language			χ^2	*df*	*p*-value
		African[a] %	Afrikaans %	English %			
Rugby	54	22.2	40.7	37.0			
Controls	37	5.4	54.1	40.5	5.0	2	0.084
Total	91	15.4	46.2	38.5			
Concussed Rugby and Matched Controls	*N*	Language			χ^2	*df*	*p*-value
		African[a] %	Afrikaans %	English %			
Rugby	17	11.8	52.9	35.3			
Controls	17	17.6	29.4	52.9	1.9	2	0.379
Total	34	14.7	41.2	44.1			

Note [a] Xhosa and isiZulu

Table 11

Race Distribution Within Rugby and Control Groups

General Rugby and General Controls	*n*	Race			X^2	*df*	*p*-value
		Black %	Coloured %	White %			
Rugby	54	22.2	9.3	68.5			
Controls	37	5.4	8.1	86.5	5.0	2	0.082
Total	91	15.4	8.8	75.8			
Concussed Rugby and Matched Controls	*n*	Race			X^2	*df*	*p*-value
		Black %	Coloured %	White %			
Rugby	17	11.8	11.8	76.5			
Controls	17	17.6	0.0	82.4	2.2	2	0.327
Total	34	14.7	5.9	79.4			

Table 12

Distribution of Alcohol Consumed Within Rugby and Control Groups

General Rugby and General Controls	*n*	Alcohol units (per week) None %	1 – 14 %	15 – 30 %	χ^2	*df*	*p*-value
Rugby	54	22.2	77.8	0			
Controls	37	18.9	64.9	16.2	9.4	2	0.009**
Total	91	20.9	72.5	6.6			

Concussed Rugby and Matched Controls	*n*	Alcohol units (per week) None %	1 – 14 %	15 – 45 %	χ^2	*df*	*p*-value
Rugby	17	17.7	82.4	0			
Controls	17	17.7	64.7	17.7	3.4	2	0.186
Total	34	17.6	73.5	8.8			

** $p < .01$, two-tailed

Table 13

Distribution of Cigarettes Smoked Within Rugby and Control Groups

General Rugby and General Controls	*n*	Cigarettes (per week) None %	1 – 14 %	15 – 125 %	χ^2	*df*	*p*-value
Rugby	54	100	0	0			
Controls	37	59.5	16.2	24.3	26.2	2	<0.000**
Total	91	83.5	6.6	9.9			

Concussed Rugby and Matched Controls	*n*	Cigarettes (per week) None %	1 – 14 %	15 – 105 %	χ^2	*df*	*p*-value
Rugby	17	94.1	5.9	0			
Controls	17	76.5	11.8	11.8	2.6	2	0.267
Total	34	85.3	8.8	5.9			

** $p < .01$, two-tailed

7.2 PROCEDURE

The research was carried out solely by the researcher (with the exception of one excluded concussed rugby player who was tested by the team doctor while on rugby tour). All participants were tested individually at either the rugby stadium medical facilities (familiar to the rugby players) or in rare instances at the researcher's office. While the stadium facility used for testing was relatively quiet, there were occasions when building at the stadium caused some noise, but the participants indicated that this was not disturbing to them. The final section of the ImPACT program requests that participants provide details on factors, including instruction clarity, computer and environmental problems, that might negatively affect test performance. No participant responses suggested any such factors impaired the validity of their test results. Prior to testing, each participant was provided with a clear explanation about what the testing entailed. Each participant was informed that the results were confidential, except that postconcussion test results, in the form of a written report, would be available to the researcher's supervisor and to the doctor treating the concussion. Standardised test instructions were given to each participant at the time of testing, and as the researcher was present during individual testing, any queries could be answered. All participants including both rugby players and control participants, being competitive sportsmen, appeared highly motivated to give of their best during testing and, although unanswered, they often sought affirmation that they had achieved better than their colleagues or than at the time of a previous testing.

Baseline testing was carried out for all research participants in order to obtain information on each participant's neuropsychological functioning as a starting point pre-season and pre-injury, as such measure provided a basis with which future test performance was compared. End of season testing was carried out for all research participants who did not constitute the Concussed Rugby group or the Matched Control group, in order to obtain information on each participant's neuropsychological functioning post-season and possible within-season brain injury (in the form of subconcussive and unreported concussive events for the General Rugby group) and as a means of equivalent testing for the General Control group, for comparative purposes with baseline test performance. Postconcussion testing was carried out for the Concussed Rugby group and the Matched Control group in order to obtain information on each rugby participant's

neuropsychological functioning post-concussion, and as a means of equivalent testing for the Matched Control group, for comparative purposes with baseline test performance.

Baseline Testing

Baseline testing for the rugby players took place at the beginning of the 2005 rugby season, in early January for the senior national and provincial players, and in February to early March for the junior provincial and academy players. Two newly acquired senior provincial players received baseline testing in June. Baseline testing for the control participants took place between January 2005 and June 2006. Ninety minutes were allocated for each participant's individual baseline testing in that this included two tests for the IQ estimate.

End of Season Testing

End of season testing for the rugby players took place in October 2005, and equivalent repeat testing for control participants took place from November 2005 to January 2007 with the same test-retest time interval as the rugby players. The average time between baseline and end of season testing, or equivalent repeat testing, for both the rugby players and control participants was 8.2 months. Forty-five minutes were allocated for each individual end of season testing that did not repeat the two subtests used for an IQ estimate.

Postconcussion Follow-Up Testing

In the case of a concussion, and for the purposes of comparative control testing, postconcussion follow-up testing was administered at four follow-up intervals, with intervals being on average four days following concussion (ranging from 2 – 10 days postconcussion); the interval between the first and second postconcussion follow-up averaging 6 days (ranging from 2 – 9 days); the intervals between the second and third postconcussion follow-up averaging 6 days (ranging from 2 – 8 days); and the interval between the third and fourth postconcussion follow-up averaging 8 days (ranging from 1 – 15 days). There was a considerable attempt by the researcher to conduct the first postconcussion follow-up within two to three days of the concussion, and thereafter to follow-up at weekly intervals, depending on the schedule of the concussed athlete. These time intervals were impossible to maintain as often players did not attend when scheduled and in many instances required re-testing prior to the scheduled time, as neurocognitive

recovery from concussion needed to be ascertained in order for players to participate in important provincial rugby matches. Forty-five minutes were allocated for each individual first and fourth postconcussion follow-up testing, that did not repeat the two subtests used for an IQ estimate. Thirty minutes were allocated for each individual second and third postconcussion follow-up testing, that involved administration of only the ImPACT test battery.

7.2.1 Measures and Their Administration

Broadly the measures for this study consisted of modes of eliciting demographic data, estimating IQ, eliciting symptoms, and cognitive test performance by means of two neurocognitive test batteries including a computerised test and subtests from a traditional pencil and paper test, which together assess multiple aspects of neurocognitive functioning including attention, learning, short-term memory, processing speed, reaction time and impulse control.

More specifically, the measures for this study consisted of (i) a demographic questionnaire, (ii) tests for the assessment of neurocognitive functioning including the ImPACT computer based test (with a demographic section, the ImPACT Symptom Scale, and six neurocognitive test modules), and the traditional pencil and paper version of the Wechsler Memory Scale – Third Edition (WMS-III) Visual Reproduction and Verbal Paired Associates subtests, (iii) the Wechsler Adult Intelligence Scale – Third Edition (WAIS-III) Vocabulary and Matrix Reasoning subtests as a measure of estimated premorbid IQ functioning.

<u>Baseline Testing</u>

At the time of baseline testing the demographic questionnaire and measures were administered in the following order: demographic questionnaire, ImPACT test (Baseline Form), WMS-III Visual Reproduction I, WMS-III Verbal Paired Associates I, WMS-III Visual Reproduction II, WMS-III Verbal Paired Associates II, WAIS-III Vocabulary and WAIS-III Matrix Reasoning subtests. In addition to Verbal Paired Associates 1 being a learning task it acted as an interference task for Visual Reproduction II (Reproduction and Recognition) that followed, translating into an eight minute delay. Following Visual Reproduction II, the Verbal Paired Associates II (Recall and Recognition tasks) was

administered, with Visual Reproduction II acting as an interference task for the two Verbal Paired Associates conditions, which translated into a five minute delay. The usual time lapse between the immediate and delayed conditions, on the standardised WMS-III is 25-35 minutes for both Visual Reproduction (Tulsky, Zhu, & Ledbetter, 2002) and Verbal Paired Associates (Wechsler et al., 1998b). In this study the time lapse between the immediate and delayed conditions of the Visual Reproduction and Verbal Paired Associates subtests was approximately 8 and 5 minutes, respectively. The short time lapse between the immediate and delayed conditions was unavoidable due to (i) time constraints and (ii) the need for ImPACT not to be invalidated by other tests preceding its implementation as ImPACT was also a stand-alone test at the second and third postconcussion test intervals. In other words it was not a solution to administer the WMS before ImPACT as a means of acquiring a longer delayed recall. The short time lapse between the immediate and delayed conditions is likely to provide less challenge being a more lenient mode of testing, and could offer less sensitivity in differentiating scores between the rugby and control groups. Although the shorter time lapse differs from the standard requirements of the test this does not invalidate the comparative indications in the study since both the rugby and control participants were subject to the same altered test conditions.

End of Season Testing

At the end of season testing the ImPACT computerised test (First Postconcussion Form), followed by the same order of WMS-III Visual Reproduction and Verbal Paired Associates subtests were administered. The WAIS-III Vocabulary and Matrix Reasoning subtests were not re-administered in that they were utilised for control purposes only to establish the demographic criteria of the sample. Typically an estimate of IQ is based on a single test occasion only, on the assumption that these tests are not sensitive to the effects of MTBI (Lezak et al., 2004) and test performance is likely to remain stable regardless of neurological insult (Schoenberg at al, 2002).

Postconcussion Follow-up Testing

In the case of a concussion, and for the purposes of comparative control testing, the ImPACT 3.0 battery was administered at each of the four follow-up intervals. The ImPACT 3.0 battery was designed for repeated use over short intervals, and consists of five forms in order to minimise practice effects. The first postconcussion form was

administered at the first postconcussion follow-up testing, the second form at the second follow-up, the third form at the third follow-up and the fourth form at the fourth postconcussion follow-up. The WMS-III has only one version, and therefore in order to reduce the presence of practice effects, and also for the purposes of time economy this test was not administered on every follow-up occasion. The WMS-III Visual Reproduction and Verbal Paired Associates subtests were thus only administered at the first and fourth postconcussion follow-up test intervals, following the ImPACT test and in the same order as that used at baseline and end of season testing.

Details of the various measures employed follow below.

Demographic Questionnaire (see Appendix E)

The demographic questionnaire was administered individually as the first measure at the time of baseline testing only.

The demographic questionnaire was completed with all participants at the occasion of the baseline testing to provide the researcher with information relating to the participant and various exclusion criteria, comprising: i) biographical information such as name, age, date of birth, educational history, etc., ii) medical history including fractures sustained, encephalitis, meningitis, epilepsy, convulsions, and any serious illness/inherited condition, iii) concussion history including TBI in general, iv) psychological history including history of depression, anxiety, sleeping difficulties and Attention-Deficit/Hyperactivity Disorder, and v) recreational history including nicotine and alcohol use.

Immediate Post-concussion Assessment and Cognitive Testing (ImPACT)

ImPACT (Lovell, Collins, Podell, Powell, & Maroon, 2000; Maroon et al., 2000) was administered individually as the first neurocognitive measure at the baseline (following administration of the demographic questionnaire), end of season or postconcussion testing intervals.

ImPACT, the computerised neurocognitive screening test, is specific and sensitive to the assessment of sport induced MTBI (Schatz et al., 2006). Currently four versions of the ImPACT test exist and, as the fourth version was released in 2006 following the inception

of this study, the third version (ImPACT 3.0) was used in this study (see Chapter 5, section 5.2.3 for a description of the ImPACT test battery development). The ImPACT program was loaded onto the researcher's laptop computer to use for testing. ImPACT testing time takes approximately 30 minutes. Participants respond to items using the keyboard and an external mouse. The ImPACT test comprises three sections: (a) Sport and health history, (b) Symptom Scale and (c) Neurocognitive test battery.

ImPACT Subject Profile and Health History Questionnaire

The ImPACT demographic section (Lovell et al., 2000; Maroon et al., 2000), completed only at baseline testing, requires the individual be given a subject ID number that the researcher assigned and from then on is used to access the database for the individual's records. The individual supplies demographic information pertaining to himself (name, date of birth, age, gender, handedness, height, weight, native country, native language, second language), his educational history (years of formal education, whether or not special classes, grade repeats or speech therapy were required and if problems with Attention Deficit/Hyperactivity Disorder were experienced or a learning disability diagnosed), sport details (current sport, position, level of participation and years experience), concussion history (number of diagnosed concussions and dates, number of concussions resulting in each: loss of consciousness, confusion, retrograde amnesia, anterograde amnesia, and total number of games missed as a result of concussion), and medical history (history of brain surgery or meningitis, treatment for headaches, migraines, epilepsy, psychiatric conditions or substance/alcohol abuse).

Symptom Measure: ImPACT Symptom Scale

The ImPACT computerised testing program includes a Symptom Scale (Lovell et al., 2000; Maroon et al., 2000) that has been shown to be sensitive to the effects of MTBI (Lovell et al., 2003; Lovell & Collins, 1998). The Symptom Scale is a self-report inventory that consists of 22 items. Participants subjectively rate the severity of symptoms experienced at the time of testing on a 7-point Likert scale that ranges from 0 = not experienced to 6 = severe. The 22 concussive symptoms include: headache, nausea, vomiting, dizziness, balance problems, 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). Being a computerised test, the symptom scores are automatically computed and generated in the print-out report.

ImPACT Neurocognitive Test Battery

ImPACT 3.0 (Lovell et al., 2000; Maroon et al., 2000) consists of six modules measuring aspects of neurocognitive functioning that previous research has shown to be sensitive to the effects of sports related MTBI, including tests of attention, learning, memory, processing speed and reaction time (Collins, Field et al., 2003; Lovell, Collins, Iverson et al., 2003; Schatz et al., 2006). Aspects taken from these six modules contribute to the five composite scores of Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time and Impulse Control.

The description and application of each module is cited directly from the ImPACT Test website (2007c):

> **Module 1** *(Word Memory)*
>
> This module evaluates attentional processes/verbal recognition memory and utilizes a word discrimination paradigm. Twelve target words are presented for 750 milliseconds on the computer screen. This word list is presented twice to facilitate learning of the list. At the end of the second presentation of the list, the subject is tested for recall via the presentation of the 24-word list that is comprised of 12 target words and 12 non-target words that have been chosen from the same semantic category as the target word. For example, the word "ice" is a target word, while the word "snow" represents the non-target word. The subject responds by mouse-clicking the "yes" or "no" buttons on the screen. Individual scores are provided both for correct "yes" and "no" responses. In addition, a total percent correct score is provided. There are five different forms of the word list.
>
> *Delay Condition: Following the administration of all other test modules (approximately 20 minutes), the subject is again tested for recall via the same method described above. The same scores that are described above are provided for the delay condition.*

Module 2 *(Design Memory)*

This module evaluates attentional processes and visual recognition memory and utilizes a design discrimination paradigm. Twelve target designs are presented for 750 milliseconds on the computer screen. This sequence is presented twice to facilitate learning. At the end of the second presentation of the list, the subject is tested for recognition via the presentation of 24-designs comprised of 12 target designs and 12 non-target designs (target designs that have been rotated in space). Similar to the word recognition task, the subject responds by mouse-clicking the "yes" or "no" buttons on the screen. Individual scores are provided both for correct "yes" and "no" responses. In addition, a total percent correct score is provided. There are five different forms of this task.

Delay Condition: Following the administration of all other test modules (approximately 20 minutes), the subject is again tested for recall via the same method described above. The same scores that are described above are provided for the delay condition.

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 distractor task. The subject is allowed to practice the distractor task prior to presentation of the memory task. The distractor is a choice reaction time test during which the subject is asked to click the left mouse button if a blue square is presented and the right mouse button if a red circle is presented. Once the subject has completed this task, the memory task is presented. For each of the trials of the memory task, a screen is displayed for 1.5 second that has a computer generated random assortment of X's and O's. For each of the trials, three of the X's or O's are illuminated in YELLOW on the screen. The subject is asked to remember the location of the illuminated objects. The X's and O's that are illuminated are randomized by the computer for each trial and for each administration of the test. Immediately after the presentation of the three X's or O's, the distractor task re-appears on the screen. Following the distractor task, the memory screen (X's and O's) re-appears and the subject 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 distractor

task, and number of errors on the distractor task. For each administration of ImPACT, the subject completes four trials.

Module 4 *(Symbol Matching)*

This module evaluates visual processing speed, learning and memory. Initially, the subject is presented with a screen that displays nine well-known symbols (triangle, square, arrow, etc). Directly under each symbol is a number button from 1 to 9. Below this grid, a symbol is presented. The subject is required to click the matching number as quickly as possible and to remember the symbol/number pairings. Correct performance is reinforced through the illumination of a correctly clicked number in GREEN. Incorrect performance illuminates the number button in RED. Following the completion of 27 trials, the symbols disappear from the top grid. The symbols again appear below the grid and the subject is asked to recall the correct symbol/number pairing by clicking the appropriate number button. This module provides an average reaction time score and a score for the memory condition.

Module 5 *(Colour Match)*

This module represents a choice reaction time task and also measures impulse control/response inhibition. First, the subject is required to respond by clicking a red, blue or green button as they are presented on the screen. This procedure is completed to assure that subsequent trials would not be affected by colour blindness. Next, a word is displayed on the screen in the same coloured ink as the word (e.g., RED), or in a different colored ink (GREEN or BLUE). The subject is instructed to click in the box as quickly as possible only if the word is presented in the matching 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 subject is allowed to practice a distractor task, which consists of 25 numbered buttons (5 x 5 grid). The subject is instructed to click as quickly as possible on the numbered buttons in backward order starting with “25”. Once the subject has completed this initial practice task, he/she is presented with three consonant letters

> that are displayed on the screen. Immediately following display of the three letters, the numbered grid re-appears and the subject is instructed to click the numbered buttons in backward order as quickly as possible. After a period of 18 seconds, the numbered grid disappears and the subject is asked to recall the three letters by typing them from the keyboard. 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. Five trials of this task are presented for each administration of the test (ImPACT, 2007c).

Being a computerised test, all the ImPACT individual and composite test scores are automatically computed and generated in the print-out report. Five composite scores, Verbal Memory, Visual Memory, Visual Motor Speed, Reaction Time and Impulse Control, result from these six modules described above. According to the ImPACT 3.0 Clinical Interpretation Manual (ImPACT, 2004), the Impulse Control composite measures the sum of errors during testing (the colour match subtest, and the Xs and Os interference task of counting backwards), and although clinical decisions should not be based on this composite's score, in that it is a validity measure, its inclusion helps interpretation of other composites. The manual states that the Impulse Control composite "provides a measure of errors on testing and is useful in determining test validity…Scores above 20 should be viewed as invalid" (ImPACT, 2004, p. 32). It is unclear in the manual whether high Impulse Control composite scores invalidate an entire test profile, or in what context this applies. It would appear likely that this refers to protocols of individuals who are baseline tested in large groups, and who may not have applied themselves appropriately for reasons other than compromised neurocognitive functioning, such as poor motivation or fatigue. This problem would not apply to the present research context, as each rugby and control participant was tested individually and was under the careful observation of the researcher throughout the test procedure, so that those who suffered extreme tiredness or veisalgia were identified and excluded. From the description of the Impulse Control composite it is clear that this composite taps important aspects of cognitive functioning including level of attention, processing instructions and impulsivity that are typically highly sensitive to diffuse brain injury). Thus, for the purposes of this study it was decided that test protocols that contained Impulse Control composite scores in excess of

20 should be retained as a useful construct on which to compare the rugby and control participants. Furthermore, no research studies using ImPACT as a test measure could be found whereby Impulse Composite scores above 20 were explicitly used as an exclusion criterion. The ImPACT 3.0 Clinical Interpretation Manual (ImPACT, 2004), details the specific contribution of the various module's subtests to the five composite scores, that for ease of reference are summarised in Table 14, at the end of this section.

Table 14

ImPACT Test 3.0 Description of the Computations of the Composite Scores

Composite Scores	Contributing scores
Verbal Memory	Average performance on: • Word Memory (module 1) • Symbol Match (module 4) • Three Letters (module 6)
Visual Memory	Average performance on: • Design Memory (module 2) Total percent correct • X's and O's (module 3) Total correct (memory)
Visual Motor Speed	Average performance on: • Interference task X's and O's (module 3) Total correct/4 • Interference task Three Letters (module 6) Average counted correctly x 3)
Reaction Time	Average performance on: • Interference task X's and O's (module 3) Average correct RT • Symbol Match (module 4) Average Correct RT/3 • Colour Match (module 5) Average Correct RT
Impulse Control	Adding: • Interference task X's and O's (module 3) Average incorrect • Color Match (module 5) Total incorrect

Note. The summary table is an updated version of a similar Table (Iverson et al., 2002, ImPACT Version 2.0 Manual)

Validity of ImPACT

Validity refers broadly to the degree of accumulated evidence which supports a test measuring that aspect which the test developers and users claim it to measure (Lezak et al., 2004). Therefore, validity of ImPACT refers to this neurocognitive test battery being a valid measure of neurocognitive and neurobehavioural effects ofsport-related concussion. No studies have been found specifically relating to the validity of the

ImPACT version 3.0, the version used in the present study, but several studies have established the validity of ImPACT versions 1.0 and 2.0. As ImPACT version 3.0 is broadly the same test as ImPACT versions 1.0 and 2.0, but with refinements and improvements on previous versions, this indicates that version 3.0 is similarly, a valid measure of the neurocognitive and neurobehavioural effects of sport-related concussion. Iverson, Lovell and Collins' 2002 research, involving 120 high school and college athletes who were baseline tested and tested within three days of concussion, studied several validity measures of ImPACT version 1.0, (Iverson, Lovell, & Collins, 2002a).

Criterion validity, the degree with which a test correlates with some outcome variable or criterion, was determined by examining the sensitivity of the composite scores to the effects of concussion, with concussed athletes performing significantly worse on the ImPACT version 1.0 Memory and Reaction Time composites and reporting significantly more symptoms compared with their baseline performance (Iverson, Lovell, & Collins, 2002a). *Convergent validity*, a high correlation between tests measuring the same construct, was examined by Iverson et al. (2002a), by correlating specific items from the ImPACT version 1.0 Symptom Scale with the composite scores post injury, with findings of medium to high correlations (*r* .53 to *r* .83) between the Total Symptom composite and selected individual symptoms; between the Memory Composite and the symptoms of: poor memory (*r* = -.40), poor concentration (*r* = .40), balance problems (*r* = -.27), light sensitivity (*r* = -.32) but with no correlations on the remaining symptoms; between the Processing Speed composite and vomiting (*r* = -.19); and no correlation between the Reaction Time composite and individual symptoms (Iverson et al., 2002a). *Divergent validity*, a low correlation between tests measuring different constructs, was also examined in the Iverson et al. (2002a) study, through an intercorrelation matrix of the preseason and post injury ImPACT composite scores, whereby it was determined that the different composites appeared to measure different aspects because the correlations were small and the composite scores did not have much shared variance. At preseason only the Reaction Time and Processing Speed composites were significantly correlated (*r* = .35). Post injury the Memory composite correlated with Symptoms (*r* = -.38), Reaction Time (*r* = -.27) and Processing Speed (*r* = .35); and Reaction Time correlated with Processing Speed (*r* = .32) (Iverson et al., 2002a).

Construct validity, seeing whether a test measures the construct it is designed to measure, was studied by Iverson, Lovell and Collins (2005) with investigation of the relationship between ImPACT version 2.0 and the Symbol Digit Modalities Test (SDMT) as measures of attention and processing speed. The retrospective study involved 72 amateur athletes who were tested within 21 days of concussion. Findings were that the decreased performance on the SDMT correlated significantly with Processing Speed (r = .70) and Reaction Time (r = .60), suggesting that they measure a similar underlying construct. Iverson, Franzen et al. (2003) found the construct validity the ImPACT Version 2.0 memory composites correlated highly with the Brief Visuospatial Memory Test total score (r = .50 for both), and the ImPACT Visual Motor Speed composite correlated with TMT A (r = -.49), TMT B (r = .56) and SDMT (r = -.70). Schatz and Putz (2006) found the ImPACT Version 2.0 complex Reaction Time correlated with TMT A (r = -.64), TMT B (r = .44) and WAIS-R Digit Symbol (r = -.46), and the Processing Speed composite correlated with TMT B (r = -.51) and Digit Symbol (r = .53). Furthermore, significant correlations for complex Reaction Time were found between ImPACT and CogSport (r = .66), and ImPACT and HeadMinder (r = .41) (Schatz & Putz, 2006).

The *diagnostic utility* or sensitivity of ImPACT version 2.0 was explored by Schatz et al. (2005) who assessed the probability of the test correctly classifying whether or not a concussion had occurred. The investigation compared the composites scores of 72 concussed high school athletes (tested within 72 hours of concussion) with 66 high school athletes with no history of concussion. By combining the sensitivity of the Symptom composite with the Visual Memory, Processing Speed and Impulse Control composites, 85.5% of cases were correctly classified: 82% of the concussed group and 89% of the control group were correctly classified, with the sensitivity of ImPACT being 81.9% and the specificity 89.4%, suggesting that ImPACT is a useful tool in the neurocognitive and neurobehavioural assessment of concussion. However, it should be noted that (i) the concussed athletes were mostly male football players and the control athletes mostly younger, females involved in noncontact sports, and (ii) the symptom scale accounted for a major portion of variance in differentiating the concussed athletes from the nonconcussed athletes (Randolph et al., 2006). Overall, however, the above studies indicate that the ImPACT neurocognitive test battery, has good criterion validity, convergent validity, divergent validity, construct validity and diagnostic utility as a measure of the neurocognitive and neurobehavioural effects of sport-related concussion.

ImPACT's *discriminative validity* was demonstrated in Lovell, Collins, Iverson et al.'s (2003) study in which the ImPACT battery separated age-matched concussed and nonconcussed athletes individuals, demonstrating robust statistical differences and a large effect size for the memory composite score (1.21), resulting in a 26% improved diagnostic yield than if relying on reported symptoms alone (Lovell, 2006). However, this study was criticised in that the data reported were for one index of one version of ImPACT on which the concussed and nonconcussed athletes differed at the baseline evaluation with no comparisons being made postinjury (Randolph et al., 2006).

Reliability of ImPACT

Reliability refers to the stability of the relative rankings of individual scores (Theisen, 1997), and in essence would assume that repeat testing under the same conditions would reflect the absence of meaningful score changes. Reliability in neurocognitive assessment is viewed as the stability of responses over test occasions, and the test-retest reliability coefficient is often considered the coefficient of stability (Theisen, 1997). The Pearson *r* correlation coefficient is usually used to evaluate test-retest reliability as high reliability scores may be obtained despite substantial changes in test scores between test intervals. A participant's score may change substantially on retesting but such change will have little effect on the reliability coefficient if the participants maintain their same relative rank order (Theisen, 1997).

In a study on the reliability and stability of *ImPACT 1.0* with retest intervals averaging 14 days (range 7-21 days) and 18.5 days (range 2-7 days), involving 49 nonconcussed amateur athletes, the Parametric and Nonparametric test-retest Pearson correlation coefficients between all test occasions (test 1 and test 2, test 2 and test 3, test 1 and test 3 intervals) ranged from .40 – .54 on the Memory Composite, .76 – .86 on the Processing Speed composite, and .62 - .71 on the Reaction Time composite (Iverson, Lovell, Collins, & Norwig, 2002). A study on the stability of *ImPACT 2.0* test-retest scores, with a retest interval of approximately one week, involving 56 nonconcussed young adult athletes, was used to calculate the reliable change confidence intervals (RCI), calculated at the .80 confidence level. The study found no significant differences on the Verbal Memory, Visual Memory, Reaction Time or Total Symptom composites, but found a significant difference on the Processing Speed (Visual Motor Speed) composite ($p = < .003$),

resulting in a correction to the latter's RCI, following which the Pearson test-retest correlation coefficients ranged from .65 to .86 for the same five composite scores, and the researchers concluded that these relatively modest test-retest reliability coefficients were comparable or superior to a number of traditional neurocognitive tests, including the Wechsler Memory Scale – Third Edition (Iverson, Lovell, & Collins, 2003).

Notwithstanding these positive observations concerning the reliability of the ImPACT test, Randolph et al. (2005), provided a challenging article in which they argued that no computerised neuropsychological tests, including ImPACT, had been shown to demonstrate adequate test-retest reliability (i.e. reliability close to .9) for a test interval exceeding months or more than a year; while Broglio, Ferrara, Macciocchi, Baumgartner and Elliott (2007), acknowledging .60 a minimum adequate test-retest reliability, found no computerised neuropsychological tests index scores, including those of ImPACT, exceeded a good test-retest reliability (.75) for a test interval of 45 days. However, substantial challenge can be brought to bear in terms of the validity of the Broglio et al. (2007) study in evaluating test-retest reliability for the ImPACT test, in that several neurocognitive screening instruments were used at one two hour sitting providing a serious potentially confounding test stimulus interference factor, and moreover it was evident that there was an unusually high attrition rate over the test-retest period. In a more recent study (Schatz, 2010), *ImPACT 3.0* has been shown to demonstrate adequate test-retest reliability for baseline assessment two years apart by 95 collegiate athletes, with intraclass correlation coefficient estimates for visual memory (.65), visual motor speed (.74) and reaction time (.68), however verbal memory (.46) and symptom scale (.43) reflected greater variability. Only a small percentage of athletes revealed significant change on the Reliable Change Index over this period on the neurocognitive composite scores (0 to 6%) and symptom scale (5 to 10%), indicating that baseline assessments for adults need not be repeated every year and can be conducted every two years (Schatz, 2010).

Finally, in respect of gender, there was one study in which females revealed higher mean scores on the verbal memory composite. The natural distribution of scores for both males and females were assigned as uniform percentile ranks for the classification ranges of impaired, borderline, low average, average, high average, superior and very superior (Iverson et al., 2010).

Practice Effects and ImPACT

Practice effects refer to the improvement in performance on each subsequent test administration due to familiarity with the test material, learning and positive carryover resultant from previous exposure to the task (Theisen, 1997). As practice effects are those that result in enhanced test performance due to repeated testing, usually but not exclusively on the same test instrument, the ImPACT 3.0 program is specifically designed for multiple repeat testing following MTBI. In order to minimise practice effects, it consists of five alternate forms with a randomised presentation of stimuli. Tests that involve a large speed component with a single solution is a relatively unfamiliar and unrehearsed response mode, yet once attained is easily conceptualised and likely to show practice effects (Lezak et al., 2004).

Speed is an important factor on the ImPACT test (e.g., Reaction Time and Visual Motor Speed Composites) but this was taken into account when the test was normed and the Reliable Change Index (RCI) introduced. As indicated above, a study on the stability of ImPACT 2.0 test-retest scores, with a retest interval of approximately one week found no significant differences on Verbal Memory, Visual Memory, Reaction Time or Total Symptoms, but a significant difference on the Processing Speed (Visual Motor Speed) composite (Iverson, Lovell, & Collins, 2003), thus minimal practice effects were demonstrated for athletes taking the test on two occasions (Lovell, 2006). However, a reliable change method that corrects for the presence of practice effects was used and it was considered that this practice effect observed on the Processing Speed composite might disappear over a longer test-retest period (Iverson, Lovell, & Collins, 2003). In another study mentioned above, on ImPACT 1.0 with retest intervals averaging 14 days (range 7-21 days) and 18.5 days (range 2-7 days) no practice effects on the composite scores was indicated and thus there was no need to adjust the composite score reliable change indices (Iverson, Lovell, Collins, & Norwig, 2002). In a study on concussed athletes followed up for one week postconcussion, the age-matched controls' Memory composite scores did not increase with three repeat testing opportunities indicating, on the basis of this research, that the Memory composite is not particularly prone to practice effects (Lovell, Collins, Iverson et al., 2003).

The Wechsler Memory Scale – Third Edition (WMS-III)

Two subtests (Visual Reproduction and Verbal Paired Associates) of the The Wechsler Memory Scale – Third Edition (WMS-III) (Wechsler et al., 1998b) were administered individually as the second neurocognitive measure at the time of baseline, end of season or first postconcussion and fourth postconcussion testing intervals.

Visual Reproduction I and II

Visual Reproduction, one of the eleven WMS-III subtests, has been present in some form in each of the previous versions of the WMS and has undergone considerable content revision (Wechsler et al., 1998b)**.** The current form now has five pages of designs, with the first page being a new addition, implemented to extend downward the floor of this subtest. The second to fourth pages remain unchanged from previous versions. The fifth page is new and is a modification of the original WMS designs. In addition to these revisions, two tasks have been added to Visual Reproduction II: a recognition task (48 items) and a discrimination task (7 items), the latter of which was not used in this study due to testing time constraints. A copy task was also added to help evaluate if the examinee's performance is negatively affected by motor difficulties, but the copy task was not included in this study (e.g., Wechsler et al., 1998b).

In the current WMS-III version, Visual Reproduction is an optional subtest, whereas its scores contributed to the memory indexes in previous versions of the scale and Visual Reproduction I previously contributed to the Visual Immediate Index. The administration procedure remains much the same, apart from the following modifications: The examinee now draws all designs in a Visual Reproduction Response booklet, instead of on two perforated pages taken from the Record Form. In Visual Reproduction II the recall administration procedure remains the same as for the WMS-R – the examinee is requested to draw the designs he remembers following a time delay. In the recently added recognition task, the examinee is shown 48 designs, one page at a time, and asked to identify whether or not each design corresponds with one of those previously drawn. The recall scoring criteria no longer relies on the all-or-nothing WMS-R criteria, but has been significantly revised so that partial credit can be earned for that drawn (Wechsler et al., 1998b).

For *Visual Reproduction I,* the immediate condition, each design is shown for 10 seconds and once the page is turned to show a blank page, the examinee is asked to draw the design from memory. This is done for each of the five designs. This is a visually based test. Scoring criteria is presented in 38 pages of the WMS-III Administration and Scoring Manual (Wechsler et al., 1998b), where very precise instructions are given according to the examinee's attempts at replicating the designs, with the maximum scores permissible as follows: Design A = 10 points, Design B = 10 points, Design C = 18 points, Design D = 34 points and Design E = 32 points (Maximum Total = 104 points) (Wechsler et al., 1998b).

For *Visual Reproduction II*, the delayed condition, the examinee is asked to draw from memory and in any order, the designs learned in the immediate condition. For the recognition task the examinee is shown 48 designs one at a time and identifies those learned in the immediate condition (Wechsler et al., 1998b). The introduction of the recognition task allows examiners to make comparisons between the delayed recall and recognition tasks (Tulsky et al., 2002). Scoring criteria is the same as per Visual Reproduction 1 (Wechsler et al., 1998b). The Visual Reproduction Percent Retention score is calculated as the VR II recall total score divided by the VR I recall total score multiplied by 100.

Verbal Paired Associates I and II

Verbal Paired Associates, one of the eleven WMS-III subtests, has been present in some form in each of the previous versions of the WMS and has undergone considerable content revision (Wechsler et al., 1998b). The current form's eight word pairs are a complete replacement of the word pairs in the former WMS-R version. WMS-III word pairs are unrelated and novel e.g., "truck-arrow", thus replacing the former version's easily acquired and over learned word associations. The imagery- based WMS-III word pairs were derived by taking into account age acquisition, reading year levels and number of syllables. The scores from the delayed recall condition can be contrasted with the scores from the recognition condition (Wechsler et al., 1998b).

In the current WMS-III version, as with previous versions, the Verbal Paired Associates subtest forms part of the overall test and is not an optional subtest. Verbal Paired Associates I contributes to the Auditory Immediate and Immediate Memory Primary

Indexes. Verbal Paired Associates II contributes to the Auditory Delayed and General Memory Primary Indexes and its recognition component to the Auditory Recognition Delayed Primary Index. The administration procedure remains much the same, apart from the following modifications: The Verbal Paired Associates I now has four trials and Verbal Paired Associates II recall condition precedes the recognition condition (Wechsler et al., 1998b).

For *Verbal Paired Associates I,* the immediate condition, eight word pairs are read to the examinee, following which the examiner orally provides the first word of each pair and the examinee is required to provide the corresponding word. There are four trials of the same list, but the order of word pairs varies. This is an oral-based test. Scoring permits one point for each correct response and no point for an incorrect response (Maximum Total = 32 points) (Wechsler et al., 1998b). The Verbal Paired Associates First Recall score is the Verbal Paired Associates I list A score (the first trial of VPA I).

For *Verbal Paired Associates II*, the delayed condition consists of a single recall trial where usually the examiner orally provides the first word of each pair and the examinee is required to provide the corresponding word from memory (Wechsler et al., 1998b). For this study, however, the Kaplan modification was used whereby the second word of each pair was presented and the examinee was required to provide the first word of each pair, in order to assess true learning of the word association as opposed to passive learning of phonetic associations (Milberg, Hebben, & Kaplan, 1996; Lezak et al., 2004). For the recognition task the examinee is read a list of 24 word pairs and asked to indicate if each pair is a new word pair or a pair he was asked to remember (Wechsler et al., 1998b). The introduction of the recognition task allows examiners to make comparisons between the delayed recall (usually a 25-35 minute delay) and recognition tasks (Tulsky et al., 2002). Scoring criteria is the same as per Verbal Paired Associates 1 (Maximum Total = 8 points) (Wechsler et al., 1998b). The Verbal Paired Associates Percent Retention score is calculated as the Verbal Paired Associates II total recall score divided by the VPA I list D (the fourth trial of VPA I) multiplied by 100.

Validity of WMS-III

As defined above, *validity* refers broadly to the degree of accumulated evidence that supports a test measuring that aspect which the test developers and users claim it to

measure (Lezak et al., 2004). In a study comparing the *convergent* validity of the WMS-III and WMS-R, involving 207 adults with retest intervals ranging from two to twelve weeks, the auditory presented materials showed higher correlations than did the visually presented materials, due to changes introduced to the WMS-III for the visually presented memory subtests. The correlation coefficients between the two forms of WMS were: .72 between the WMS-III Auditory Immediate Index and the WMS-R Verbal Memory Index; .36 between the WMS-III Visual Immediate Index and the WMS-R Visual Memory Index; and .62 between the WMS-III Immediate Memory Index and the WMS-R General Memory Index (Tulsky et al., 2002).

In Cohen's 1997 study involving 86 adolescents who were administered the WMS-III and Children's Memory Scale (CMS) in a counterbalanced design, with the two test intervals ranging between 2 to 12 weeks, the WMS-III Working Memory Index correlated highest with the corresponding CMS Attention/Concentration Index (p = .68); followed by the WMS-III auditory indexes that correlated with the corresponding CMS indexes (WMS-III Auditory Immediate and CMS Verbal Immediate, p = .74; WMS-III Auditory Delayed and CMS Verbal Delayed, p = .65); followed by the WMS-III and CMS visual indexes (WMS-III Visual Immediate and CMS Visual Immediate, p = .55; WMS-III Visual Delayed and CMS Visual Delayed, p = .26), providing evidence of *convergent validity* that, apart from the Visual Delayed Indexes, suggests the WMS-III and CMS measure similar constructs. The WMS-III Working Memory Index had lower correlations with the other CMS Indexes, ranging from .21 for the Verbal Recognition Delayed to .48 for the Learning Index, providing evidence of *divergent* validity for the WMS-III. On versions of WMS predating WMS-III, Visual Reproduction (VR) I and II were found to load on a factor with purported non-verbal memory tests (Heilbronner, Buck, & Adams, 1989) confirming findings of a former (1985) study that had also found VR II loaded more heavily on the visual memory factor than on the visual-perceptual-motor factor, and Wong and Gilpin (1993) found the WMS-R VR clustered in a visual memory cluster rather than a verbal memory cluster (Stoddard, 2007), suggesting that through the development of the WMS, VR has evolved as a more valid test of visual memory. Burton et al. (2003) performed a confirmatory factor analysis of the WMS-III using results pertaining to a clinical sample (n = 281) which supported that the test loads on four factors, namely auditory memory, visual memory, working memory and learning factors.

With respect to the WMS-R and *discriminant validity*, Brooks (1976) reported poor performance on WMS-R Logical Memory and Paired Associates by severe TBI individuals compared with control individuals, with posttraumatic amnesia duration being predictive of the degree of memory impairment (Tulsky, 2002). Reid and Kelly (1993) studied WMS-R profiles for individuals with closed head injuries and found coma severity unrelated to WMS-R scores, but increased forgetting rates on the WMS-R Visual Reproduction subtests correlated with the longer the duration of posttraumatic amnesia. Findings of the same study were that group performance on the WMS-R indexes showed impaired performance on the delayed recall indexes, borderline performance on the General Memory and Verbal Indexes, and low average performance on the attention/concentration and visual memory indexes. The WMS-III has been shown to have discriminant validity for assessing memory deficits in a range of populations, for example, individuals with traumatic brain injury, alcoholism, attention Deficit/Hyperactivity Disorder, schizophrenia and mental retardation, to name a few (Stoddard, 2007; Tulsky et al., 2002).

The above studies suggest that the WMS-III, broadly the same test as the former WMS versions, is a valid neurocognitive measure of verbal memory with good convergent validity between the WMS-R and WMS-III auditory presented materials. As discussed above, although Cohen's 1997 study of convergent validity between WMS-III and the Children's Memory Scale (CMS), reported low convergent validity between the WMS-III Visual Immediate and CMS Visual Immediate ($p = .55$) and particularly the WMS-III Visual Delayed and CMS Visual Delayed ($p = .26$), of importance is that on the WMS-R, the VR subtests were shown to cluster in a visual cluster rather than a verbal cluster (Wong & Gilpin, 1993); increased forgetting rates on VR correlated with the longer the duration of posttraumatic amnesia in individuals with closed head injuries (Reid and Kelly, 1993)**;** and overall factor analysis of the WMS-III supports the test loads on four factors including auditory memory and visual memory (Burton et al., 2003). Thus, the WMS-III VPA and VR subtests used in this study are considered relatively valid neurocognitive measures of verbal memory and visual memory, respectively.

Reliability of WMS-III

As defined above, *reliability* refers to the stability of the relative rankings of individual scores (Theisen, 1997), and in essence would assume that repeat testing under the same

conditions would reflect the absence of meaningful score changes. A study on the *reliability* of WMS-III (Tulsky et al., 2002), with a retest interval averaging 35.6 days (with a range of 2 to 12 weeks), involving 141 participants aged 16 - 54 years, revealed reliability coefficients that represented a sound improvement in reliability of scores compared to the reliability of the WMS-R, and adequate reliabilities above .70, with Verbal Paired Associates 1 averaging .93 and Verbal Paired Associates II averaging .83. It was anticipated that with relatively longer test-retest intervals, smaller gains in retest performance might be expected. The test-retest correlations for Visual Reproduction I Recall averaged .79, Visual Reproduction II Recall averaged .77 and Visual Reproduction II Recognition averaged .75.

Practice Effects and WMS-III

As defined above, practice effects refer to the improvement in performance on each subsequent test administration due to familiarity with the test material, learning and positive carryover resultant from previous exposure to the task (Theisen, 1997). The improved general test performance on repeated test occasions are of particular concern in memory testing, because repeated testing on the same test results in learning the material by all individuals except seriously memory-impaired patients (Lezak et al., 2004). Thus, practice effects are of importance with memory tests such as the WMS-III. Although no known study of practice effects on WMS-III has been carried out, studies on practice effects pertaining to its predecessor, WMS-R are available as follows.

Thiesen (1997) carried out a study on practice effects pertaining to a prorated version of the WMS-R, involving the Verbal Paired Associates (VPA) and Visual Reproduction (VR) subtests, amongst other measures assessing memory. Testing took place over four test occasions with retest intervals averaging two weeks, and involved 64 adult undergraduate psychology students. The criterion selected for meaningful change in scores was a conservative effect size of .70. Findings revealed meaningful effect sizes on VPA I. The greatest increase in score change was evidenced at the first retest interval, whereas at the third and fourth retest intervals increases in score were of a smaller magnitude. Relatively small effect sizes occurred for VPA II, VR I and VR II. It was concluded that the absence of significant change across retest intervals was due to a ceiling effect rather than the absence of practice effects (Theisen, 1997).

Finally, Kaufman (2003) reported that experience, novelty, motor speed, retest interval, subtest reliability and the nature of the task contribute to practice effects, and in relation to the WMS-R discussed the following: For three age groups, retest gains after an interval of over a month, on the WMS-R Verbal Memory Scale averaged 13 points in contrast to gains of 8 points on the WMS-R Visual Memory Scale. When comparing this with retest gains in the WAIS-R IQ test, the WMS-R Visual Memory practice effect was commensurate with the WAIS-R Performance IQ gain, but the WMS-R Verbal Memory gain was much larger than the WAIS-R Verbal IQ gain. Kaufman (2003) concluded that with verbal memory subtests, it is likely that adults remember specific word associations and facts which assist recall when retested more than a month later (Kaufman, 2003).

Wechsler Adult Intelligence Scale – Third Edition (WAIS-III)

Two subtests (Vocabulary and Matrix Reasoning) of the Wechsler Adult Intelligence Scale – Third Edition (WAIS-III) (Wechsler et al., 1998a) were administered individually to acquire an IQ estimate at the time of the baseline testing only.

Central to neurocognitive evaluation is change in neurocognitive functioning as a result of brain injury (Schoenberg et al., 2002). In order to establish equivalence between groups in terms of IQ level, estimates of premorbid levels of intellectual functioning were made. Ample evidence exists that intellectual functioning is known to affect neurocognitive performance (Lezak et al., 2004; Macchiocci & Barth, 2004) and the two are highly correlated, thus between group equivalence in premorbid intellectual functioning was employed to reduce threat to the internal validity of this quasi-experimental concussion research (Macchiocci & Barth, 2004).

The Wechsler Adult Intelligence Scale – Third Edition (WAIS – III) Vocabulary and Matrix Reasoning subtests were administered. Estimated Full Scale IQ scores were calculated using the Schoenberg et al. (2002) OPIE – 3 (2ST) formula (See Appendix D). While Schoenberg et al.'s research did not investigate Vocabulary as a substitute measure of the Verbal Scale IQ or Matrix Reasoning as a substitute measure of the Non-Verbal (Performance) Scale IQ, for purposes of this study the Vocabulary subtest scaled score was used as a measure of verbal ability and the Matrix Reasoning subtest scaled score as a measure of non-verbal ability. Each of these subtests has demonstrated both resistance to neurological insult and reliability (Schoenberg et al., 2002; Wechsler et al., 1998a). As

indicated above, it is not customary to pursue repeat testing in order to establish an estimate of IQ as a control for equivalence between the comparative groups and therefore the estimate of IQ was calculated at baseline only, on the assumption that the derived estimated IQ score would remain stable regardless of neurological insult (Lezak et al., 2004) and over the brief time period of this study. Therefore in the absence of repeat testing on these subtests, practice effects were not an issue with these two WAIS-III subtests.

WAIS-III Vocabulary

Commencing on the fourth word, the Vocabulary subtest consists of a series of 33 words. The examinee orally defines each word that is presented both orally and visually in order to evaluate the individual's expressive vocabulary (e.g., Wechsler et al., 1998a). Although no time limit is imposed, administration is of approximately 15 minutes duration. Discontinuation criteria exist, and individual answers score between 0-2 points according to the word definition the examinee provides.

WAIS-III Matrix Reasoning

Commencing on the fourth item, following the presentation of three sample items, the Matrix Reasoning subtest consists of a series of 26 gridded patterns that the examinee completes by selecting the correct response from five possible choices, in order to evaluate non-verbal reasoning tasks of analogy, classification, serial reasoning and pattern completion (Wechsler et al., 1998a). No time limit is imposed for this subtest. Discontinuation criteria exist, and individual answers score 0 points for an incorrect response and 1 point for a correct response.

7.2.2 Data Processing

Being a computerised test, the generation of ImPACT 3.0 scores was automated. The WAIS–III Vocabulary and Matrix Reasoning subtests and the WMS-III Visual Recognition and Verbal Paired Associates subtests, being pencil and paper test protocols, were scored in accordance with the relevant manuals by the researcher at the time of testing and scores were re-checked by the researcher within a week of administration. These test protocols were then re-scored by the researcher's research assistant and any discrepancies discussed and resolved between the two scorers. Scores were again re-

checked at the end of the data collection period by the researcher. The entire collection of research data was then entered into Excel spreadsheet format by the researcher who triple checked entries and then stored the data in a standardised manner.

7.2.3 Data Analysis

Independent *t*-test analyses were conducted on the group means of the General Rugby sample versus the General Control sample at the baseline and end of season test intervals, and of the Concussed Rugby sample versus the Matched Control sample at baseline and at the four postconcussion follow-up test intervals, to investigate differences in neurocognitive functioning and symptoms reported.

Dependent *t*-test analyses were conducted on the group means of the General Rugby sample and the General Control sample at the baseline versus the end of season test intervals, and of the Concussed Rugby sample and the Matched Control sample at baseline versus the four postconcussion follow-up test intervals, to investigate prospective differences in neurocognitive functioning and symptoms reported.
More specifically, the following statistical analyses were implemented.

<u>Independent Sample Analyses</u>
Independent *t*-test analyses were conducted to compare the group means between the General Rugby sample and the General Control sample at (i) baseline and (ii) end of season test intervals in respect of four ImPACT neurocognitive composite scores (Verbal Memory, Visual Memory, Reaction Time and Visual Motor Speed), the ImPACT Impulse Control composite**,** the WMS-III Visual Recognition and Verbal Paired Associates, and the ImPACT Symptom Scale.

In addition, independent *t*-test analyses were conducted to compare the group means between the Concussed Rugby sample and the Matched Control sample at (i) baseline and (ii) four postconcussion follow-up test intervals in respect of four ImPACT neurocognitive composite scores (Verbal Memory, Visual Memory, Reaction Time and Visual Motor Speed), the ImPACT Impulse Control composite**,** the WMS-III Visual Recognition and Verbal Paired Associates, and the ImPACT Symptom Scale.

Dependent Sample Analyses

Dependent *t*-test analyses were conducted to compare the group means of (i) the General Rugby sample, and (ii) the General Control sample, at baseline versus end of season test intervals in respect of four ImPACT neurocognitive composite scores (Verbal Memory, Visual Memory, Reaction Time and Visual Motor Speed), the ImPACT Impulse Control composite, the WMS-III Visual Recognition and Verbal Paired Associates, and the ImPACT Symptom Scale.

Dependent *t*-test analyses were conducted to compare the group means of (i) the Concussed Rugby sample, and (ii) the Matched Control sample, at baseline versus each of the four postconcussion follow-up test intervals in respect of four ImPACT neurocognitive composite scores (Verbal Memory, Visual Memory, Reaction Time and Visual Motor Speed), the ImPACT Impulse Control composite, the WMS-III Visual Recognition and Verbal Paired Associates, and the ImPACT Symptom Scale.

7.2.4 Significance Level

Statistical hypothesis testing is conducted to calculate the probability that the sample data could have occurred under the assumption that the null hypothesis is true. Due to sampling and measurement error, data is never in complete agreement with a null hypothesis and thus the probability (*p*-value) of the data is evaluated. This *p*-value or attained significance level is the criterion used for rejecting the null hypothesis as it is the smallest value of alpha for which the hull hypothesis can be rejected. If the null hypothesis is rejected when it actually should not be rejected i.e., it is concluded that a difference between the means exists when it does not exist, then this is a Type I error. To help avoid such error a cut-off value, termed Alpha or level of significance, is used against which the *p*-value is evaluated. The smaller the Alpha value, the less likely is the risk of falsely rejecting the null hypothesis. Researchers commonly use the 5% level of significance ($\alpha = .05$), indicating that there is a 5% probability that the observed differences in mean scores could have occurred by chance. The 5% level of significance may be considered too lenient and thus the 1% level of significance ($\alpha = .01$) may be employed, indicating that there is a 1% probability that the observed differences in mean scores could have occurred by chance. The stringent 1% level of significance, however, could result in a Type II error where the null hypothesis is not rejected when it actually

should have been rejected, i.e., it is concluded that a difference between the means does not exist when it does exist. This study uses both the 1% and 5% levels of significance for discussion purposes ($p < .01$ and $p < .05$, respectively). This study considers 5.1 to 10% levels as approaching significance for discussion purposes ($p < .051$ to $p < .10$).

Previous research on rugby-related MTBI (e.g., Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards, Smith et al., 2008) used one-tailed tests when comparing neurocognitive test performance and reported symptoms of rugby groups with noncontact sport control groups. One-tailed tests can be used if directional hypotheses are assumed. In the above studies the directional hypotheses assumed that rugby players would perform more poorly than the control participants on neurocognitive testing and reported symptoms, due to repetitive subconcussive and concussive incidents sustained from playing this contact sport. Drawing on findings of the research reviewed (see Chapter 6) that rugby players generally perform more poorly than the noncontact sport control participants, this study assumed directional hypotheses relating to the mean neurocognitive testing results and symptoms reported. Assuming directional hypotheses permits the use of one-tailed tests, the latter of which allows the *p*-values that are usually assigned for two-tailed tests to be divided by two, one-tailed tests were used for the comparison analyses involving neurocognitive tests and symptom reports. In contrast, no differences were assumed between the means of the rugby and control groups on demographic variables (i.e., non-directional null hypotheses), and therefore standard *p*-values for two-tailed tests were retained for the demographic statistical analyses. This included the number of concussions reported prior to baseline testing, since some studies have revealed unusual contradictory findings in this regard, such as noncontact sport cricket players reporting more concussions than rugby players, due to the under-reporting of concussions in the rugby group for fear of exclusion from the game (Shuttleworth-Edwards, Border et al., 2004). The following symbols are used for the number of tails relevant to the *p*-values: † denotes one-tailed tests, and * denotes two-tailed tests, thus significance values in the tables are represented as follows: † $p < .05$, one-tailed, †† $p < .01$, one-tailed, * $p < .05$, two-tailed, ** $p < .01$, two tailed.

7.2.5 The Bonferroni Correction

The Bonferroni Correction, discussed in Chapter 6, is the statistical adjustment made to compensate for multiple comparisons on the same data set (Bland & Altman, 1995; Hsu, 1996; Perneger, 1998; Weisstein, undated). The Royal College of Surgeons (2007) propose that adjustments need not be made for analysis of a single data set, or for multiple statistical tests in a single study such as the present study, and Perneger (1998) vehemently argues against the necessity of the Bonferroni Correction in biomedical related research. However, in order to avoid criticism such as that levelled at the previous MTBI studies (Rutherford, 2003), the Bonferroni Correction was employed in this study for the neurocognitive comparisons. The Bonferroni Correction increases stringency in the statistical analyses of this study and reduces the risk of Type I error (rejecting the null hypothesis when it is true, thus finding a significant difference between the means when such does not exist).

The Bonferroni Correction has been criticised as being too conservative in setting an unreasonably high standard and thus increasing the risk of a Type II error (Bland & Altman, 1995; Brandt, 2007; Hsu, 1996; Perneger, 1998; Weisstein, 1996). In order to reduce the risk of incurring a Type II error (failing to reject the null hypothesis when it is false, thus not finding a significant difference between the means when such does exist), the present study employed a more appropriate use of the Bonferroni Correction, that has been used in other MTBI studies (Matser et al., 1998, 1999; Shuttleworth-Edwards, Smith et al., 2008). These researchers (Matser et al., 1998, 1999; Shuttleworth-Edwards, Smith et al., 2008) calculate a Bonferroni Correction towards greater stringency on the number of neurocognitive functional modalities, rather than on the number of neurocognitive tests used which would unacceptably increase the potential for Type II error.

If the route of Matser at al. (1998, 1999) and Shuttleworth-Edwards, Smith et al. (2008) were followed, it can be argued that the present study investigates a series of neurocognitive subtests (n = 14) that fall within the two neurocognitive functional modalities of memory and attention (n = 2), as indicated on Table 15, at the end of this section. However, this conceptualisation of the neurocognitive measures cuts across the two test batteries used for the study, and does not concur with the manner in which the

research was conceived (i.e., the use of a computerised test battery together with a traditional pencil and paper test battery).

Accordingly it was proposed that this functional division would render the presentation of the data unwieldy for discussion purposes. Therefore, an alternate conceptualisation for the purposes of Bonferroni Correction was to argue that this study employs two separate types of neurocognitive test batteries including one *computerised* test battery (ImPACT) and one *traditional pencil and paper* memory test (WMS-III) (see table 16, at the end of this section), and to use this as the basis for the Alpha adjustment. *Importantly either mode results in Alpha being divided by two.* Therefore, in order to avoid both over and under stringency, as well as for the conceptual clarity of this thesis in both analysing and tabulating results, the two types of neurocognitive test measures: (i) computer and (ii) traditional pencil and paper measures (n = 2), were used as the two units for the purposes of Alpha adjustment. Accordingly, the alpha adjustment for the directional hypotheses for the neurocognitive comparisons, allowing for the Bonferroni Correction, was as follows: 5% level of significance p = .025; 1% level of significance p = .005.

In this study there was only one Symptom Scale involved in the analyses, the ImPACT Symptom Composite, and thus for the investigation of the *total* symptom composite score it is argued that the Bonferroni Correction was *not* warranted. However, the Symptom Scale comprises 22 symptoms, each treated separately, and arguably the Bonferroni Correction should apply when investigating the effects of each one of the individual symptoms in order to avoid erroneous chance effects due to multiple tests (i.e., Type I error). At the same time, however, were such a correction introduced, the division of Alpha by 22 would incur an unacceptably high risk of missing any potentially significant effects (i.e., Type II error). This is particularly the case with symptom reporting, because the expected effects are subtle, and the problem of missing subtle effects is even more pronounced in the case of a small sample size such as the Concussed Rugby group (n = 17). Furthermore, Anderson & Catroppa (2005) would argue that the need for excessive alpha adjustments is circumvented when a wide range measure, such as the Symptom Scale, is employed. Overall, therefore, in respect of the symptom analyses, it was decided not to use the Bonferroni Correction, either for the investigation of the total symptom score (on the basis that there was only one Symptom Scale used and therefore this was

statistically warranted), nor for the scores for the individual symptom measures (on the basis that division by 22 might obscure clinically relevant insights). However, this decision was based on the understanding that the investigation of outcome in respect of individual symptoms should be considered exploratory due to the high risk of Type I error.

Table 15

Neurocognitive Subtests Pertaining to Two Types of Functional Modalities

Subtest measures	Functional Modality
	Memory
ImPACT measures	
Verbal Memory	
Visual Memory	
WMS-III Measures	
Verbal Paired Associates 1	
Verbal Paired Associates 1st Recall	
Verbal Paired Associates 11	
Verbal Paired Associates % Retention	
Verbal Paired Associates % Recognition	
Visual Reproduction 1	
Visual Reproduction 11	
Visual Reproduction Recognition	
Visual Reproduction % Retention	
	Attention
ImPACT measures	
Visual Motor Speed	
Reaction Time	
Impulse Control	

Table 16

Neurocognitive Subtests Pertaining to the Two Types of Neurocognitive Tests

Subtest measures	Type of Neurocognitive Test
	ImPACT Computerised Test
Verbal Memory	
Visual Memory	
Visual Motor Speed	
Reaction Time	
Impulse Control	
	WMS-III Traditional Pencil and Paper Test
Verbal Paired Associates 1	
Verbal Paired Associates 1st Recall	
Verbal Paired Associates 11	
Verbal Paired Associates % Retention	
Verbal Paired Associates % Recognition	
Visual Reproduction 1	
Visual Reproduction 11	
Visual Reproduction Recognition	
Visual Reproduction % Retention	

7.3 STATISTICAL HYPOTHESES FOR THIS STUDY

As discussed in Chapter 3, neurocognitive and symptomatic effects for this study are delineated into the following time periods: *acute* refers to those effects suffered until three months post injury; and *chronic* refers to those effects suffered more than three months post injury.

Baseline testing was conducted with the objective of analysing persistent cognitive deficits amongst rugby players compared with noncontact sport control participants, as a result of concussive and subconcussive events sustained during former years of play but without the overlay of any acute effects of recent concussive/subconcussive events that would most likely have resolved during the three month period since the last rugby match played in the 2004 rugby season and baseline testing. *End of season* testing excluded the rugby players formally concussed during the 2005 rugby season, in order to establish residual effects of concussive and subconcussive events sustained during former years of play in combination with unreported concussive and subconcussive events sustained during the 2005 rugby season. It is understood that concussive events may be purposely

unreported or unrecognised and thus unreported. It was expected that outcomes for rugby players would be worse than for control participants at baseline testing. It was further expected that adverse outcomes for rugby players would be more evident at end of season testing than at baseline testing due to the possibility of additional effects of unreported concussive and subconcussive injury sustained during the season that would operate synergistically with past effects, and therefore it was not expected that it would be possible to differentiate any such seasonal effects from the effects of prior exposure. It was also anticipated that rugby players would show less improvement than noncontact sport control participants between baseline and end of season testing on tests subject to practice effects, due to their neurocognitive vulnerability. Accordingly, and in view of previous MTBI research findings and the theoretical underpinnings discussed in the introductory chapters the following hypotheses were formulated:

7.3.1 General Rugby and General Control Groups

Neurocognitive Measures:

(i) It was hypothesised that *on the basis of independent t-test analyses* there would be significant differences between the mean scores of the General Rugby group and the noncontact sport General Control group in respect of the *neurocognitive scores* on the ImPACT test and the WMS-III Visual Reproduction and Verbal Paired Associates subtests at the *baseline* test interval in support of poorer performance for the General Rugby group relative to the General Control group, due to long-term exposure to concussive and subconcussive events historically, implicating chronic or permanent residual effects.

(ii) It was hypothesised that *on the basis of independent t-test analyses* there would be significant differences between the mean scores of the General Rugby group and the noncontact sport General Control group in respect of the *neurocognitive scores* on the ImPACT test and the WMS-III Visual Reproduction and Verbal Paired Associates subtests at the *end of season* test interval in support of deleterious neurocognitive sequelae for the General Rugby group relative to the General Control group, due to long-term exposure to concussive and subconcussive events historically, and an overlay of unreported concussive and subconcussive events sustained during the 2005 rugby season.

However, it was understood that it would not be possible to differentiate between these two sets of past and more recent effects.

(iii) It was hypothesised that *on the basis of dependent t-test analyses* there would be significant differences in the mean scores of the General Rugby group in respect of the *neurocognitive scores* on the ImPACT test and the WMS-III Visual Reproduction and Verbal Paired Associates subtests at the *end of season* test interval compared to the *baseline* test interval in support of deleterious neurocognitive effects in the form of either poorer performance or the absence of a practice effect when it is expected. This is due to long-term exposure to concussive and subconcussive events historically, and an overlay of unreported concussive and subconcussive events sustained during the 2005 rugby season. However, it was understood that it would not be possible to differentiate between these two sets of past and more recent effects.

(iv) It was hypothesised that *on the basis of dependent t-test analyses* there would be no significant differences in the mean scores of the noncontact sport General Control group in respect of the *neurocognitive scores* on the ImPACT test and the WMS-III Visual Reproduction and Verbal Paired Associates subtests at the *end of season* test interval compared to the *baseline* test interval in support of poorer performance, and/or that there would be a practice effect when it is expected, in association with a lack of exposure to concussive and subconcussive events historically and during the equivalent 2005 rugby season.

<u>Symptom Measures:</u>

(v) It was hypothesised that *on the basis of independent t-test analyses* there would be significant differences between the mean scores of the General Rugby group and the noncontact sport General Control group in respect of the total symptom and individual *symptom scores* on the Symptom Scale at the *baseline* test interval in support of higher scores for the General Rugby group relative to the General Control group, due to long-term exposure to concussive and subconcussive events historically, implicating chronic or permanent residual effects.

(vi) It was hypothesised that *on the basis of independent t-test analyses* there would be significant differences between the mean scores of the General Rugby group and the

noncontact sport General Control group in respect of the total symptom and individual *symptom scores* on the Symptom Scale at the *end of season* test interval in support of deleterious neuropsychological sequelae for the General Rugby group relative to the General Control group, due to long-term exposure to concussive and subconcussive events historically, and an overlay of unreported concussive and subconcussive events sustained during the 2005 rugby season. However, it was understood that it would not be possible to differentiate between these two sets of past and more recent effects.

(vii) It was hypothesised that *on the basis of dependent t-test analyses* there would be significant differences in the mean scores of the General Rugby group in respect of the total symptom and individual *symptom scores* on the Symptom Scale at the *end of season* test interval compared to the *baseline* test interval in support of deleterious neuropsychological effects. This is due to long-term exposure to concussive and subconcussive events historically, and an overlay of unreported concussive and subconcussive events sustained during the 2005 rugby season. However, it was understood that it would not be possible to differentiate between these two sets of past and more recent effects.

(viii) It was hypothesised that *on the basis of dependent t-test analyses* there would be no significant differences in the mean scores of the noncontact sport General Control group in respect of the total symptom and individual *symptom scores* on the Symptom Scale at the *end of season* test interval compared to the *baseline* test interval, in association with a lack of exposure to concussive and subconcussive events historically and during the equivalent 2005 rugby season.

7.3.2 Concussed Rugby and Matched Control Groups

Neurocognitive Measures:

(i) It was hypothesised that *on the basis of independent t-test analyses* there would be significant differences between the mean scores of the Concussed Rugby group and the noncontact sport Matched Control group in respect of the *neurocognitive scores on* the ImPACT test and the WMS-III Visual Reproduction and Verbal Paired Associates subtests at the *baseline* test interval in support of poorer performance for the Concussed Rugby group relative to the Matched Control group, due to long-term exposure to

concussive and subconcussive events historically, implicating chronic or permanent residual effects.

(ii) It was hypothesised that *on the basis of independent t-test analyses* there would be significant differences between the mean scores of the of the Concussed Rugby group and the noncontact sport Matched Control group in respect of the *neurocognitive scores* on the ImPACT test and the WMS-III Visual Reproduction and Verbal Paired Associates subtests at the *postconcussion* test intervals in support of deleterious neurocognitive sequelae for the Concussed Rugby group relative to the Matched Control group, due to long-term exposure to concussive and subconcussive events historically, and the overlay of acute effects of reported concussive events. However, it was understood that it would not be possible to clearly differentiate between the past and more recent acute effects of the reported concussive events.

(iii) It was hypothesised *on the basis of dependent t-test analyses* that there would be significant differences in the mean scores of the Concussed Rugby group in respect of the *neurocognitive scores* on the ImPACT test and the WMS-III Visual Reproduction and Verbal Paired Associates subtests at the *postconcussion* test intervals compared to the *baseline* test interval in support of deleterious neurocognitive effects, in the form of either poorer performance or the absence of a practice effect when it is expected. This is due to long-term exposure to concussive and subconcussive events historically, and the overlay of acute effects of reported concussive events. However, it was understood that it would not be possible to clearly differentiate between the past and more recent acute effects of the reported concussive events.

(iv) It was hypothesised that *on the basis of dependent t-test analyses* there would be no significant differences in the mean scores of the noncontact sport Matched Control group in respect of the *neurocognitive scores* on the ImPACT test and the WMS-III Visual Reproduction and Verbal Paired Associates subtests at the *postconcussion* test intervals compared to the *baseline* test interval in support of poorer performance, and/or that there would be a practice effect when it is expected, in association with a lack of exposure to concussive and subconcussive events historically and during the equivalent portion of the 2005 rugby season.

Symptom Measures:

(v) It was hypothesised that *on the basis of independent t-test analyses* there would be significant differences between the mean scores of the Concussed Rugby group and the noncontact sport Matched Control group in respect of the total symptom and individual *symptom scores* on the Symptom Scale at the *baseline* test interval in support of higher scores for the Concussed Rugby group relative to the Matched Control group, due to long-term exposure to concussive and subconcussive events historically, implicating chronic or permanent residual effects.

(vi) It was hypothesised that *on the basis of independent t-test analyses* there would be significant differences between the mean scores of the Concussed Rugby group and the noncontact sport Matched Control group in respect of the total symptom and individual *symptom scores* on the Symptom Scale at the *postconcussion* test intervals in support of deleterious neuropsychological sequelae for the Concussed Rugby group relative to the Matched Control group, due to long-term exposure to concussive and subconcussive events historically, and the overlay of acute effects of reported concussive events. However, it was understood that it would not be possible to clearly differentiate between the past and more recent acute effects of the reported concussive events.

(vii) It was hypothesised that *on the basis of dependent t-test analyses* there would be significant differences in the mean scores of the Concussed Rugby group in respect of the total symptom and individual *symptom scores* on the Symptom Scale at the *postconcussion* test intervals compared to the *baseline* test interval in support of deleterious neuropsychological effects. This is due to long-term exposure to concussive and subconcussive events historically, and the overlay of acute effects of reported concussive events. However, it was understood that it would not be possible to clearly differentiate between the past and more recent acute effects of the reported concussive events.

(viii) It was hypothesised that *on the basis of dependent t-test analyses* there would be no significant differences in the mean scores of the noncontact sport Matched Control group in respect of the total symptom and individual *symptom scores* on the Symptom Scale at the *postconcussion* test intervals compared to the *baseline* test interval, in association with a lack of exposure to concussive and subconcussive events historically and during the equivalent portion of the 2005 rugby season.

CHAPTER EIGHT: RESULTS

The results of this study are presented in this chapter. The results pertaining to the General Rugby group and General Control group will be presented in the first section, followed by the results pertaining to the Concussed Rugby group and Matched Control group in the second section. In each case the results will first cover the independent *t*-test comparisons of group means, followed by the dependent *t*-test comparisons of group means. Significant results and the general trends pertaining to each analysis will be highlighted in the text. Tables detailing the means, standard deviations, *t*-statistics and significant effects (*p*-values) for each comparison will be provided. *Tables for all data, and figures for noteworthy data only, will appear at the end of the applicable subsection in which they are first mentioned in the text.*

8.1 GENERAL RUGBY AND GENERAL CONTROL GROUPS

The first section includes reports on the *independent t*-test comparisons between the General Rugby group and General Control group across all neurocognitive measures at baseline and then end of season test intervals, with the composite scores on the ImPACT computerised program being reported first, followed by the scores on the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) paper-and pencil tests. Following this are reports on the independent *t*-test comparisons between the General Rugby group and General Control group across all symptom measures, these being the Symptom Composite score and each of the individual symptoms on the ImPACT program's Symptom Scale, at baseline and then end of season test intervals.

The second section includes reports on the *dependent t*-test comparisons for the General Rugby group and the General Control group across all neurocognitive measures at the end of season versus baseline test intervals, with the composite scores on the ImPACT computerised program being reported first, followed by the scores on the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) paper-and pencil tests. Following this is the report on the dependent *t*-test comparisons for the General Rugby group and the General Control group across all symptom measures, these being the

Symptom Composite score and each of the individual symptoms on the ImPACT program's Symptom Scale, at the end of season versus baseline test intervals.

8.1.1 Independent *t*-test Comparisons

Baseline: Independent *t*-test Comparisons on ImPACT

The independent *t*-test comparisons of group means between the General Rugby group and General Control group on the ImPACT neurocognitive measures at the baseline interval (Table 17) revealed no significant findings. However, findings that were approaching significance were revealed for the Impulse Control measure ($p = .098$), in the direction of the General Rugby group's performance being poorer than that of the General Control group, and for the Reaction Time measure ($p = .067$), in the direction of the General Control group's performance being poorer than that of the General Rugby group. Furthermore, there was no overall trend of one group performing better than the other, although the General Rugby group's performance was poorer than that of the General Control group for the Visual Memory and Visual Motor Speed measures, and the General Control group's performance was poorer than that of the General Rugby group for the Verbal Memory measure, although none were approaching significance.

Baseline: Independent *t*-test Comparisons on WMS-III VPA and VR

The independent *t*-test comparisons of group means between the General Rugby group and General Control group on the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) neurocognitive measures at the baseline interval (Table 18) revealed no significant findings. However, a finding that was approaching significance was revealed for the VPA Percent Retention measure ($p = .075$), in the direction of the General Rugby group's performance being poorer than that of the General Control group. Furthermore, there was no overall trend of one group performing better than the other, although the General Rugby group's performance was poorer than that of the General Control group for a further four of the nine measures (VPA I, VPA First Recall, VPA II and VR Percent Retention). The General Control group's performance was poorer than that of the General Rugby group for four measures (VPA Recognition, VR I, VR II and VR Recognition), although none were approaching significance.

End of Season: Independent *t*-test Comparisons on ImPACT

The independent *t*-test comparisons of group means between the General Rugby group and General Control group on the ImPACT neurocognitive measures at the end of season interval (Table 19) revealed one significant finding for the Impulse Control measure (p = .018), and one finding approaching significance for the Visual Motor Speed measure (p = .094), in the direction of the General Rugby group's performance being poorer than that of the General Control group. Overall, the trend was in the direction of the General Rugby group's performance being poorer than that of the General Control group on three of the five ImPACT measures (including the Verbal Memory measure), but there was a marginally higher score for the Visual Memory measure, and the two groups obtained the same mean score for the Reaction Time measure (M = .057).

End of Season: Independent *t*-test Comparisons on WMS-III VPA and VR

The independent *t*-test comparisons of group means between the General Rugby group and General Control group on the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) neurocognitive measures at the end of season interval (Table 20) revealed one significant finding for the VPA Percent Retention measure (p = .025), and findings approaching significance for three measures, including (i) VPA I (p = .031), (ii) VPA First Recall (p = .036), and (iii) VPA II (p = .058), all in the direction of the General Rugby group's performance being poorer than that of the General Control group. Overall, the trend was in the direction of the General Rugby group's performance being poorer than that of the General Control group for eight of the nine measures (with the exception of VR I).

Baseline: Independent *t*-test Comparisons on the Symptom Scale

The independent *t*-test comparisons of group means between the General Rugby group and General Control group on the ImPACT Symptom Scale at the baseline interval (Table 21) revealed a significant finding for the ImPACT Symptom Composite measure (p = .002), in the direction of the General Rugby group having a higher total symptom score than the General Control group. More specifically, there were significant findings for five of the individual symptom measures in the direction of the General Rugby group having higher scores than the General Control group, including (i) trouble falling asleep (p = .021), (ii) sleeping more than usual (p = .022), (iii) sadness (p = .040), (iv) feeling more emotional (p = .049) and (v) difficulty remembering (p = .037). In addition, there were

findings that were approaching significance for two of the individual symptom measures in the direction of the General Rugby group having higher scores than the General Control group, including (i) headache (p = .057) and (ii) mentally foggy (p = .079). Overall, the General Rugby group obtained higher scores for 17 of the 22 individual symptom measures, and the General Control group obtained a higher score for only one individual symptom measure (difficulty concentrating). Neither group reported symptoms on four symptom measures (nausea, vomiting, balance problems, and sensitivity to noise).

End of Season: Independent *t*-test Comparisons on the Symptom Scale

The independent *t*-test comparisons of group means between the General Rugby group and General Control group on the ImPACT Symptom Scale at the end of season interval (Table 22) revealed a significant finding for the ImPACT Symptom Composite measure (p = .003), in the direction of the General Rugby group having a higher total symptom score than the General Control group. More specifically, there were significant findings for two of the individual symptom measures in the direction of the General Rugby group having higher scores than the General Control group, including (i) fatigue (p = .046), and (ii) trouble falling asleep (p = .048). In addition, there were findings that were approaching significance for two of the individual symptom measures in the direction of the General Rugby group having higher scores than the General Control group, including (i) numbness (p = .078), and (ii) difficulty concentrating (p = .076). Overall, the General Rugby group obtained higher scores for 13 of the 22 individual symptom measures, and the General Control group obtained a higher score for only one individual symptom measure (nervousness). Neither group reported symptoms on eight symptom measures (nausea, vomiting, balance problems, dizziness, sensitivity to light, irritability, slowed down and mentally foggy).

Table 17

Baseline Comparisons on ImPACT Scores for General Rugby versus Controls

	General Rugby (n = 54)		General Controls (n = 37)			
Baseline Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Verbal Memory	87.02	(9.21)	85.43	(10.02)	0.779	0.219
Visual Memory	77.15	(11.48)	79.24	(10.90)	-0.873	0.193
VMS	34.69	(5.40)	35.85	(6.80)	-0.907	0.184
Reaction Time	0.59	(0.07)	0.61	(0.10)	-1.515	0.067
Impulse Control	6.63	(5.50)	5.24	(4.09)	1.306	0.098

Note. No '*p'* values reached significance ($p < .05$, one-tailed), with Bonferroni's adjustment. Visual Motor Speed (VMS).

Table 18

Baseline Comparisons on WMS-III Scores for General Rugby versus Controls

	General Rugby (n = 54)		General Controls (n = 37)			
Baseline Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
VPA 1	9.85	(3.44)	10.35	(3.71)	-0.659	0.256
VPA 1st Recall	10.54	(3.42)	11.05	(3.85)	-0.673	0.252
VPA 11	9.61	(3.18)	10.30	(2.79)	-1.061	0.146
VPA Recognition	99.39	(2.51)	99.22	(3.66)	0.267	0.395
VPA % Retention	10.11	(2.96)	10.95	(2.24)	-1.453	0.075
VR 1	12.98	(2.62)	12.43	(2.54)	0.994	0.162
VR 11	13.69	(3.53)	13.27	(3.29)	0.566	0.287
VR Recognition	11.57	(2.31)	11.32	(2.15)	0.521	0.302
VR % Retention	12.50	(2.60)	12.51	(2.48)	-0.025	0.490

Note. No '*p'* values reached significance ($p < .05$, one-tailed), with Bonferroni's adjustment. Verbal Paired Associates (VPA), Visual Reproduction (VR).

Table 19

End of Season Comparisons on ImPACT Scores for General Rugby versus Controls

	General Rugby (n = 54)		General Controls (n = 37)			
End of season Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Verbal Memory	86.35	(9.66)	87.81	(9.28)	-0.719	0.237
Visual Memory	78.59	(11.44)	77.51	(12.43)	0.427	0.336
VMS	36.18	(5.59)	37.80	(5.93)	-1.330	0.094
Reaction Time	0.57	(0.07)	0.57	(0.08)	0.158	0.438
Impulse Control	7.94	(5.57)	5.49	(5.14)	2.132	0.018†

Note. † $p < .05$, one-tailed, with Bonferroni's adjustment. Visual Motor Speed (VMS).

Table 20

End of Season Comparisons on WMS-III Scores for General Rugby versus Controls

	General Rugby (n = 54)		General Controls (n = 37)			
End of season Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
VPA 1	11.04	(3.38)	12.35	(3.07)	-1.893	0.031
VPA 1st Recall	12.15	(3.18)	13.41	(3.27)	-1.831	0.036
VPA 11	10.28	(2.94)	11.16	(2.01)	-1.594	0.058
VPA Recognition	99.85	(0.76)	100.00	(0.00)	-1.180	0.121
VPA % Retention	10.17	(2.95)	11.24	(1.71)	-2.000	0.025†
VR 1	13.43	(2.30)	12.86	(2.64)	1.078	0.142
VR 11	14.93	(3.37)	15.57	(3.01)	-0.931	0.178
VR Recognition	12.41	(1.88)	12.70	(1.41)	-0.811	0.210
VR % Retention	13.46	(2.44)	14.05	(1.81)	-1.255	0.107

Note. † $p < .05$, one-tailed, with Bonferroni's adjustment. Verbal Paired Associates (VPA), Visual Reproduction (VR).

Table 21

Baseline Comparisons on ImPACT Symptoms for General Rugby versus Controls

	General Rugby (n = 54)		General Controls (n = 37)			
Baseline Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Symptom Composite	3.94	(5.63)	1.03	(2.30)	2.981	0.002††
Headache	0.26	(0.87)	0.03	(0.16)	1.597	0.057
Nausea	0.00	(0.00)	0.00	(0.00)	-	-
Vomiting	0.00	(0.00)	0.00	(0.00)	-	-
Balance problems	0.00	(0.00)	0.00	(0.00)	-	-
Dizziness	0.07	(0.54)	0.00	(0.00)	0.826	0.206
Fatigue	0.20	(0.68)	0.11	(0.46)	0.743	0.230
Trouble falling asleep	0.67	(1.29)	0.19	(0.66)	2.073	0.021†
Sleeping more	0.33	(0.99)	0.00	(0.00)	2.043	0.022†
Sleeping less	0.22	(0.77)	0.05	(0.33)	1.253	0.107
Drowsiness	0.13	(0.73)	0.00	(0.00)	1.081	0.142
Sensitivity to light	0.15	(0.68)	0.08	(0.49)	0.512	0.305
Sensitivity to noise	0.00	(0.00)	0.00	(0.00)	-	-
Irritability	0.31	(1.10)	0.16	(0.69)	0.751	0.227
Sadness	0.24	(0.82)	0.00	(0.00)	1.777	0.040†
Nervousness	0.17	(0.64)	0.11	(0.52)	0.464	0.322
Emotional	0.20	(0.74)	0.00	(0.00)	1.679	0.049†
Numbness	0.09	(0.45)	0.00	(0.00)	1.261	0.106
Slowed down	0.13	(0.62)	0.05	(0.33)	0.682	0.249
Mentally foggy	0.13	(0.55)	0.00	(0.00)	1.428	0.079
Diff concentrating	0.15	(0.68)	0.19	(0.66)	-0.285	0.388
Diff remembering	0.39	(1.11)	0.05	(0.23)	1.812	0.037†
Visual problems	0.09	(0.49)	0.00	(0.00)	1.156	0.126

Note. † $p < .05$, one-tailed. †† $p < .01$, one-tailed. Difficulty (Diff).

Table 22

End of Season Comparisons on ImPACT Symptoms for General Rugby versus Controls

	General Rugby (n = 54)		General Controls (n = 37)			
End of season Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Symptom Composite	1.43	(2.72)	0.11	(0.52)	2.910	0.003††
Headache	0.06	(0.30)	0.03	(0.16)	0.523	0.301
Nausea	0.00	(0.00)	0.00	(0.00)	-	-
Vomiting	0.00	(0.00)	0.00	(0.00)	-	-
Balance problems	0.00	(0.00)	0.00	(0.00)	-	-
Dizziness	0.00	(0.00)	0.00	(0.00)	-	-
Fatigue	0.22	(0.79)	0.00	(0.00)	1.701	0.046†
Trouble falling asleep	0.26	(0.94)	0.00	(0.00)	1.683	0.048†
Sleeping more	0.07	(0.38)	0.00	(0.00)	1.180	0.121
Sleeping less	0.02	(0.14)	0.00	(0.00)	0.826	0.206
Drowsiness	0.02	(0.14)	0.00	(0.00)	0.826	0.206
Sensitivity to light	0.00	(0.00)	0.00	(0.00)	-	-
Sensitivity to noise	0.07	(0.54)	0.00	(0.00)	0.826	0.206
Irritability	0.00	(0.00)	0.00	(0.00)	-	-
Sadness	0.13	(0.67)	0.00	(0.00)	1.167	0.123
Nervousness	0.02	(0.14	0.08	(0.49)	-0.886	0.189
Emotional	0.15	(0.76)	0.00	(0.00)	1.180	0.121
Numbness	0.15	(0.63)	0.00	(0.00)	1.435	0.078
Slowed down	0.00	(0.00)	0.00	(0.00)	-	-
Mentally foggy	0.00	(0.00)	0.00	(0.00)	-	-
Diff concentrating	0.19	(0.78)	0.00	(0.00)	1.444	0.076
Diff remembering	0.02	(0.14)	0.00	(0.00)	0.826	0.206
Visual problems	0.06	(0.30)	0.00	(0.00)	1.117	0.134

Note. † $p < .05$, one-tailed. †† $p < .01$, one-tailed. Difficulty (Diff).

8.1.2 Dependent *t*-test Comparisons

ANOVAs and Dependent *t*-test Comparisons on ImPACT

The repeated measures ANOVAs for the end of season interval versus the baseline interval on the ImPACT neurocognitive measures (Table 23) revealed a significant group by season interaction effect for the Reaction Time measure ($F = 5.69$, $df = 1,89$, $p = .019$). Results also revealed significant effects for season for two measures, including (i) Visual Motor Speed ($F = 17.54$, $df = 1,89$, $p = .000$), and (ii) Reaction Time ($F = 23.74$, $df = 1,89$, $p = .000$) in that there were significant improvements on these measures at the end of season interval versus the baseline interval for both the General Rugby group and General Control group.

Follow-up investigation of the ANOVA effects using dependent *t*-test comparisons of group means for the ImPACT neurocognitive measures at the end of season interval versus the baseline interval (Table 23) revealed the following significant findings for two measures: For the Visual Motor Speed measure, scores for the General Rugby group improved significantly at the end of season interval versus the baseline interval ($p = .001$), and similarly, scores for the General Control group improved significantly at the end of season interval versus the baseline interval ($p = .006$). For the Reaction Time measure (Figure 1), scores for the General Rugby group improved significantly at the end of season interval versus the baseline interval ($p = .024$), and similarly, scores for the General Control group improved significantly at the end of season interval versus the baseline interval ($p = .000$).

Perusal of the *p*-values revealed that the improvement by the General Rugby group appeared more robust (i.e., a stronger *p*-value was yielded) than that of the General Control group for the Visual Motor Speed measure ($p = .001$ versus $p = .006$, respectively), although the group by season interaction effect failed to reach significance ($p = .574$). On the other hand, the improvement by the General Control group appeared more robust (i.e., a stronger *p*-value was yielded) than that of the General Rugby group for the Reaction Time measure ($p = .000$ versus $p = .024$, respectively), and the group by season interaction effect, as well as the season effect, were both significant ($p = .019$ and $p = .000$, respectively). Specifically, the General Rugby group achieved a stronger (i.e., faster) mean score than the General Control group for the Reaction Time measure at the

baseline interval (*M* = .59 versus *M* = .61, respectively), but at the end of season interval this performance was not sustained and the two groups obtained an equivalent mean score (*M* = .57). For the General Rugby group, their poorer performance at the end of season interval versus the baseline interval for the Impulse Control measure was approaching significance (*p* = .058), whereas the General Control group's performance for this measure deteriorated only marginally at the end of season interval versus the baseline interval (*p* = .362), although the group by season interaction effect failed to reach significance (*p* = .350). For the General Control group, improved performance at the end of season interval versus the baseline interval for the Verbal Memory measure was approaching significance (*p* = .060), whereas the General Rugby group's performance for this measure deteriorated marginally at the end of season interval versus the baseline interval (*p* = .302), although the group by season interaction effect failed to reach significance (*p* = .127).

Perusal of trends that were neither significant nor approaching significance, revealed that at the end of season interval versus the baseline interval, the General Rugby group evidenced stronger performance for the Visual Memory measure, whereas the General Control group's performance was poorer for the Visual Memory measure.

ANOVAs and Dependent *t*-test Comparisons on WMS-III VPA and VR

The repeated measures ANOVAs for the end of season interval versus the baseline interval on the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) measures (Table 24) did not reveal any significant group by season interaction effects for any of the measures, but significant effects for season were revealed for six measures, including (i) VPA I (*F* = 38.86, *df* = 1,89, *p* = .000), (ii) VPA First Recall (*F* = 33.23, *df* = 1,89, *p* = .000), (iii) VPA II (*F* = 10.75, *df* = 1,89, *p* = .001), (iv) VR II (*F* = 30.07, *df* = 1,89, *p* = .000), (v) VR Recognition (*F* = 18.66, *df* = 1,89, *p* = .000), and (vi) VR Percent Retention (*F* = 19.04, *df* = 1,89, *p* = .000), in that there were significant improvements on these measures at the end of season interval versus the baseline interval for both the General Rugby group and General Control group.

Follow-up investigation of the ANOVA effects using dependent *t*-test comparisons of group means for the WMS-III neurocognitive measures at the end of season interval versus the baseline interval (Table 24) revealed the following significant findings for six

measures: For the General Rugby group, scores improved significantly at the end of season interval versus the baseline interval for six measures, including (i) VPA I, (ii) VPA First Recall, (iii) VPA II, (iv) VR II, (v) VR Recognition, and (vi) VR Percent Retention ($p = .000$, $p = .001$, $p = .021$, $p = .004$, $p = .014$ and $p = .006$, respectively). Similarly, the dependent *t*-test comparisons of group means for the General Control group on the WMS-III neurocognitive measures at the end of season interval versus the baseline interval (Table 24) revealed that scores improved significantly for these same six measures, including (i) VPA I, (ii) VPA First Recall, (iii) VPA II, (iv) VR II, (v) VR Recognition and (vi) VR Percent Retention ($p = .000$, $p = .000$, $p = .005$, $p = .000$, $p = .000$ and $p = .001$, respectively).

Perusal of the *p*-values revealed that the improvement by the General Rugby group appeared less robust (i.e., a weaker *p*-value was yielded) than that of the General Control group for five of these measures (VPA First Recall $p = .001$ versus $p = .000$, VPA II $p = .021$ versus $p = .005$, VR II $p = .004$ versus $p = .000$, VR Recognition $p = .014$ versus $p = .000$, and VR Percent Retention $p = .006$ versus $p = .001$, respectively), but for the sixth measure (VPA I) improvement appeared equivalent for both groups ($p = .000$ in each case).

Perusal of trends that were neither significant nor approaching significance, at the end of season interval versus the baseline interval, revealed that the improvements by General Rugby group appeared less robust (i.e., a weaker *p*-value was yielded) than that of the General Control group for the remaining three WMS-III measures not mentioned above (VPA Recognition $p = .103$ versus $p = .101$, VPA Percent Retention $p = .451$ versus $p = .250$, and VR 1 $p = .075$ versus $p = .073$). Thus, the overall trend noted in respect of the neurocognitive test comparisons on the WMS-III measures was that there was an improvement in scores for both groups on all nine WMS-III measures at the end of season interval, with comparatively weaker improvement for the General Rugby group than for the General Control group.

ANOVAs and Dependent *t*-test Comparisons on the Symptom Scale

The repeated measures ANOVAs for the end of season interval versus the baseline interval on the ImPACT Symptom Composite only (Table 25) did not reveal a significant group by season interaction effect, although it was approaching significance ($F = 2.84$, *df*

= 1,89, p = .095). However, results revealed a significant effect for season for the Symptom Composite (F = 13.11, df = 1,89, p = .000), in that there was a significant decline in symptom reports at the end of season interval versus the baseline interval for both the General Rugby group and General Control group (Figure 2), with a more robust decline in symptom reports for the General Rugby group than for the General Control group (p = .001 versus p = .009).

Follow-up investigation of the ANOVA effects using dependent *t*-test comparisons of group means for the ImPACT Symptom Scale at the end of season interval versus the baseline interval (Table 25) revealed the following significant findings: For the General Rugby group, scores lowered significantly at the end of season interval versus the baseline interval for the ImPACT Symptom Composite measure (p = .001) and for six individual symptom measures, including (i) trouble falling asleep (p = .025), (ii) sleeping more (p = .023), (iii) sleeping less (p = .031), (iv) irritability (p = .020), (v) mentally foggy (p = .045), and (vi) difficulty remembering (p = .008). In addition, there were findings that were approaching significance for the General Rugby group's lowered scores for two symptom measures at the end of season interval versus the baseline interval, including (i) headache (p = .058) and (ii) sensitivity to light (p = .059). Follow-up investigation of the ANOVA effects using dependent *t*-test comparisons of group means for the General Control group on the ImPACT Symptom Scale at the end of season interval versus the baseline interval (Table 25) revealed significant findings for the ImPACT Symptom Composite measure (p = .009) and for two individual symptom measures, including (i) trouble falling asleep (p = .045) and (ii) difficulty concentrating (p = .045), in the direction of the General Control group having lower symptom scores at the end of season interval versus the baseline interval.

Perusal of trends revealed that the General Rugby group reported symptoms on a total of 14 symptom measures at the end of season interval compared with a total 18 symptom measures at the baseline interval. Although not reaching significance, the General Rugby group achieved higher scores at the end of season interval versus the baseline interval for five individual symptom measures, including (i) fatigue (M = 0.22, SD = 0.79 versus M = 0.20, SD = 0.68, respectively), (ii) drowsiness (M = 0.19, SD =0.14 versus M = 0.13, SD = 0.73, respectively), (iii) sensitivity to noise (M = 0.07, SD = 0.54 versus M = 0.00, SD = 0.00, respectively), (iv) numbness (M = 0.15, SD = 0.63 versus M = 0.09, SD = 0.45,

respectively), and (v) difficulty concentrating (M = 0.19, SD = 0.78 versus M = 0.15, SD = 0.68 , respectively). The General Control group reported symptoms on a total of two individual symptom measures at the end of season interval compared with a total of ten individual symptom measures at the baseline interval. For the only two individual symptom measures reported by the General Control group at the end of the season interval, compared with the baseline interval, the score was lower for the nervousness symptom measure (M = 0.08, SD = 0.49 versus M = 0.11, SD = 0.52, respectively) and of equivalence for the headache symptom measure (M = 0.03, SD = 0.16), although neither reached significance.

Table 23

End of Season Versus Baseline Comparisons on ImPACT Scores for General Rugby and Controls

Test Measures	General Rugby (n = 54)					General Controls (n = 37)					
	Baseline		End of season			Baseline		End of season			Interaction
ImPACT	Mean	(*SD*)	Mean	(*SD*)	*p*- value	Mean	(*SD*)	Mean	(*SD*)	*p*-value	*p*-value
Verbal Memory	87.02	(9.21)	86.35	(9.66)	0.302	85.43	(10.02)	87.81	(9.28)	0.060	0.127
Visual Memory	77.15	(11.48)	78.59	(11.44)	0.181	79.24	(10.90)	77.51	(12.43)	0.166	0.188
VMS	34.69	(5.40)	36.18	(5.59)	0.001 † †	35.85	(6.80)	37.80	(5.93)	0.006 †	0.574
Reaction Time	0.59	(0.07)	0.57	(0.07)	0.024 †	0.61	(0.10)	0.57	(0.08)	0.000 ††	0.019*
Impulse Control	6.63	(5.50)	7.94	(5.57)	0.058	5.24	(4.09)	5.49	(5.14)	0.362	0.350

Note. † $p < .05$, one-tailed, with Bonferroni's adjustment. †† $p < .01$, one-tailed, with Bonferroni's adjustment. * $p < .05$, 2-tailed, for interaction values. Visual Motor Speed (VMS).

Table 24

End of Season Versus Baseline Comparisons on WMS-III Scores for General Rugby and Controls

Test Measures	General Rugby (n = 54)					General Controls (n = 37)					
	Baseline		End of season			Baseline		End of season			Interaction
WMS-III	Mean	(*SD*)	Mean	(*SD*)	*p*- value	Mean	(*SD*)	Mean	(*SD*)	*p*-value	*p*-value
VPA 1	9.85	(3.44)	11.04	(3.38)	0.000††	10.35	(3.71)	12.35	(3.07)	0.000††	0.114
VPA 1st Recall	10.54	(3.42)	12.15	(3.18)	0.001††	11.05	(3.85)	13.41	(3.27)	0.000††	0.284
VPA 11	9.61	(3.18)	10.28	(2.94)	0.021†	10.30	(2.79)	11.16	(2.01)	0.005††	0.672
VPA Recognition	99.39	(2.51)	99.85	(0.76)	0.103	99.22	(3.66)	100.00	(0.00)	0.101	0.629
VPA % Retention	10.11	(2.96)	10.17	(2.95)	0.451	10.95	(2.24)	11.24	(1.71)	0.250	0.712
VR 1	12.98	(2.62)	13.43	(2.30)	0.075	12.43	(2.54)	12.86	(2.64)	0.073	0.978
VR 11	13.69	(3.53)	14.93	(3.37)	0.004††	13.27	(3.29)	15.57	(3.01)	0.000††	0.105
VR Recognition	11.57	(2.31)	12.41	(1.88)	0.014†	11.32	(2.15)	12.70	(1.41)	0.000††	0.290
VR % Retention	12.50	(2.60)	13.46	(2.44)	0.006†	12.51	(2.48)	14.05	(1.81)	0.001††	0.317

Note. † $p < .05$, one-tailed, with Bonferroni's adjustment. †† $p < .01$, one-tailed, with Bonferroni's adjustment. No interaction '*p'* values reached significance ($p < .05$, 2-tailed). Verbal Paired Associates (VPA), Visual Reproduction (VR).

Table 25
End of Season Versus Baseline Comparisons on ImPACT Symptoms for General Rugby and Controls

Symptom Measures	General Rugby (n = 54)			General Controls (n = 37)			Interaction
	Baseline	End of season		Baseline	End of season		
	Mean (*SD*)	Mean (*SD*)	*p*- value	Mean (*SD*)	Mean (*SD*)	*p*- value	*p*- value
Symptom Composite	3.94 (0.49)	1.43 (0.30)	0.001††	1.03 (2.30)	0.11 (0.52)	0.009††	0.095
Headache	0.26 (0.87)	0.06 (0.30)	0.058	0.03 (0.16)	0.03 (0.16)	0.500	-
Nausea	0.00 (0.00)	0.00 (0.00)	-	0.00 (0.00)	0.00 (0.00)	-	-
Vomiting	0.00 (0.00)	0.00 (0.00)	-	0.00 (0.00)	0.00 (0.00)	-	-
Balance Problems	0.00 (0.00)	0.00 (0.00)	-	0.00 (0.00)	0.00 (0.00)	-	-
Dizziness	0.07 (0.54)	0.00 (0.00)	0.161	0.00 (0.00)	0.00 (0.00)	-	-
Fatigue	0.20 (0.68)	0.22 (0.79)	0.451	0.11 (0.46)	0.00 (0.00)	0.080	-
Trouble falling asleep	0.67 (1.29)	0.26 (0.94)	0.025†	0.19 (0.66)	0.00 (0.00)	0.045†	-
Sleeping more	0.33 (0.99)	0.07 (0.38)	0.023†	0.00 (0.00)	0.00 (0.00)	-	-
Sleeping less	0.22 (0.77)	0.02 (0.14)	0.031†	0.05 (0.33)	0.00 (0.00)	0.162	-
Drowsiness	0.13 (0.73)	0.19 (0.14)	0.139	0.00 (0.00)	0.00 (0.00)	-	-
Sensitivity to light	0.15 (0.68)	0.00 (0.00)	0.059	0.08 (0.49)	0.00 (0.00)	0.162	-
Sensitivity to noise	0.00 (0.00)	0.07 (0.54)	0.161	0.00 (0.00)	0.00 (0.00)	-	-
Irritability	0.31 (1.10)	0.00 (0.00)	0.020†	0.16 (0.69)	0.00 (0.00)	0.080	-
Sadness	0.24 (0.82)	0.13 (0.67)	0.230	0.00 (0.00)	0.00 (0.00)	-	-
Nervousness	0.17 (0.64)	0.02 (0.14)	0.051	0.11 (0.52)	0.08 (0.49)	0.393	-
Emotional	0.20 (0.74)	0.15 (0.76)	0.355	0.00 (0.00)	0.00 (0.00)	-	-
Numbness	0.09 (0.45)	0.15 (0.63)	0.268	0.00 (0.00)	0.00 (0.00)	-	-
Slowed down	0.13 (0.62)	0.00 (0.00)	0.064	0.05 (0.33)	0.00 (0.00)	0.162	-
Mentally foggy	0.13 (0.55)	0.00 (0.00)	0.045†	0.00 (0.00)	0.00 (0.00)	-	-
Difficulty concentrating	0.15 (0.68)	0.19 (0.78)	0.284	0.19 (0.66)	0.00 (0.00)	0.045†	-
Difficulty remembering	0.39 (1.11)	0.02 (0.14)	0.008††	0.05 (0.23)	0.00 (0.00)	0.080	-
Visual problems	0.09 (0.49)	0.06 (0.30)	0.210	0.00 (0.00)	0.00 (0.00)	-	-

Note. † $p < .05$, one-tailed. †† $p < .01$, one-tailed. No interaction effects run for individual symptoms, only the Symptom Composite. No interaction '*p*' values reached significance ($p < .05$, 2-tailed).

Figure 1. ImPACT Reaction Time scores at baseline versus end of season for General Rugby and Controls

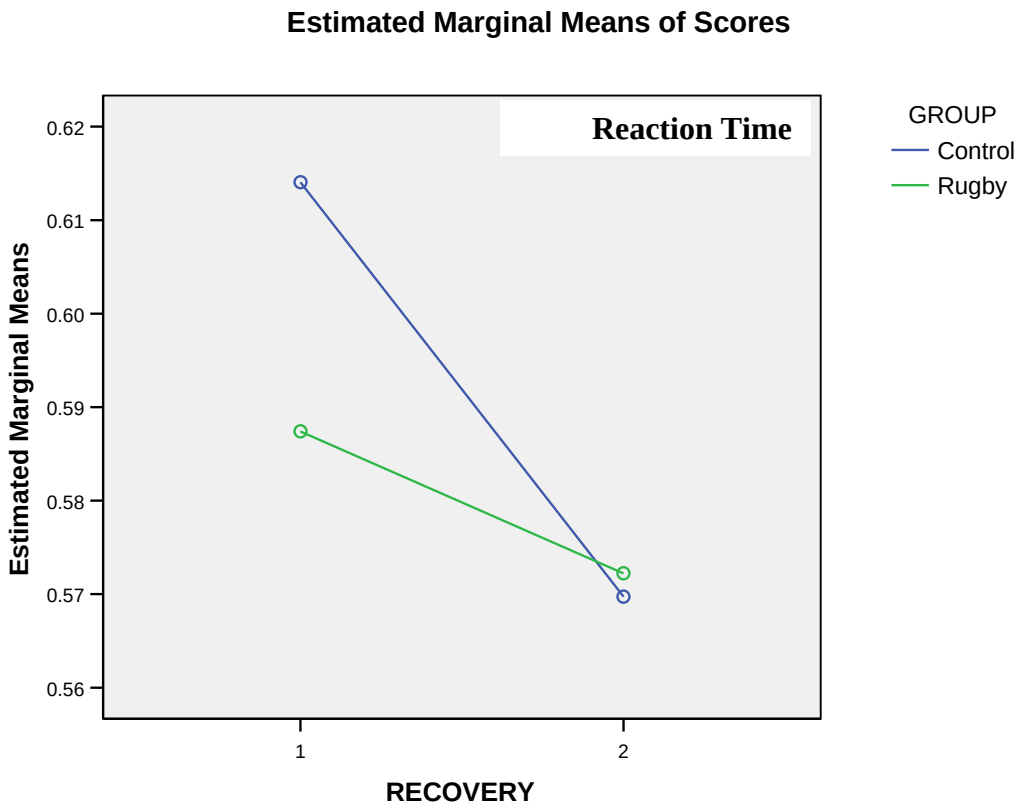
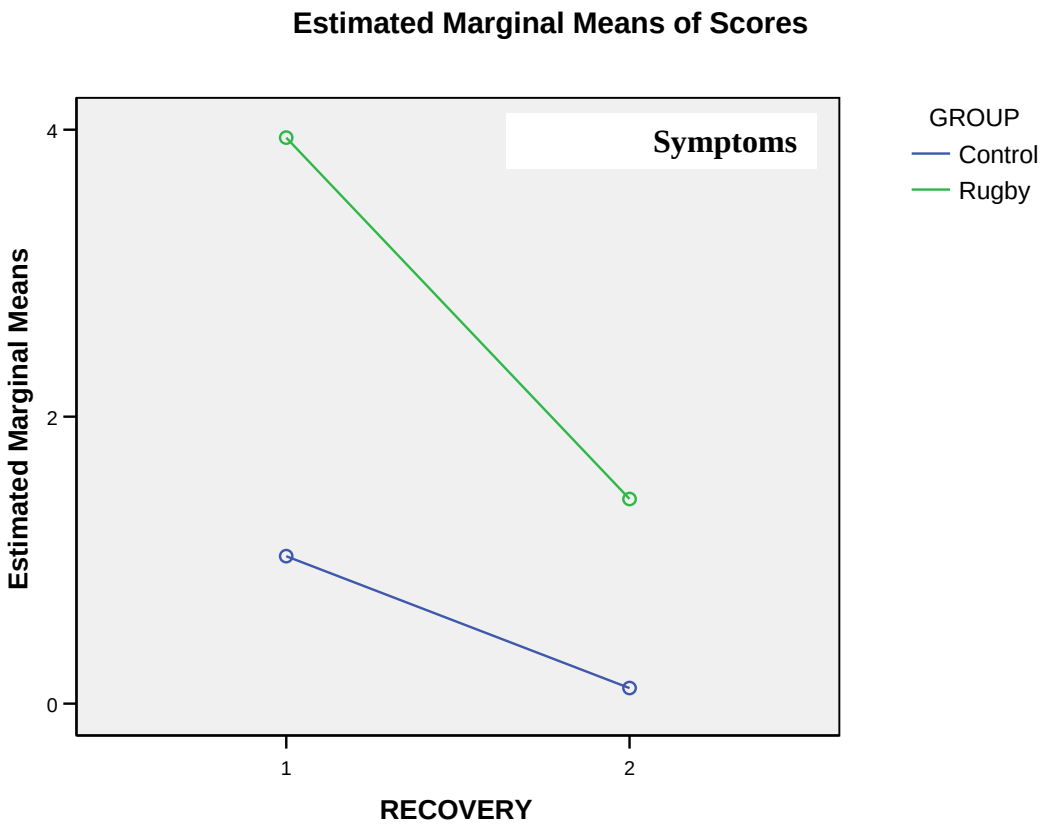


Figure 2. ImPACT Symptom scores at baseline versus end of season for General Rugby and Controls



8.1.3 Synthesis of Results for General Rugby and General Control Groups

Neurocognitive Measures

On the *independent* analyses between the General Rugby group and General Control group no significant differences were revealed on the ImPACT and WMS-III neurocognitive measures at the baseline interval, although for two measures (ImPACT Impulse Control and WMS-III VPA Percent Retention) there were differences that were approaching significance in the direction of poorer performance for the General Rugby group, and a difference approaching significance on the ImPACT Reaction Time measure in the direction of poorer performance for the General Control group. However, at the end of season interval significant differences were revealed for two measures (ImPACT Impulse Control and WMS-III VPA Percent Retention) and differences were approaching significance for four measures (ImPACT Visual Motor Speed and WMS-III VPA I, VPA First Recall and VPA II), all in the direction of poorer performance for the General Rugby group.

On the *dependent* analyses for the General Rugby group and General Control group, the repeated measures ANOVAs revealed both a significant group by season interaction effect and significant effect for season for the Reaction Time measure, in that both groups improved significantly on this measure at the end of season interval, but the improvement for the General Control group was substantially more pronounced. Significant effects for season also revealed improvement by both groups for seven further measures (ImPACT Visual Motor Speed and WMS-III VPA I, VPA First Recall, VPA II, VR I, VR II, VR Recognition and VR Percent Retention), with dependent analyses revealing that the General Rugby group, compared with the General Control group, appeared to make a less robust improvement for five of the WMS-III measures (VPA First Recall, VPA II, VR II, VR Recognition and VR Percent Retention), an equivalent improvement for one WMS-III measure (VPA I), and a more robust improvement for one measure (ImPACT Visual Motor Speed). Both groups obtained poorer scores for the ImPACT Impulse Control measure at the end of season interval versus the baseline interval, with the greater deterioration for the General Rugby group approaching significance. The General Control group's improved performance at the end of season interval on the ImPACT Verbal Memory measure was approaching significance, whereas the General Rugby group's performance deteriorated on this measure.

In summary, taking into account significant results as well as trends, both the independent and dependent analyses for the ImPACT and WMS-III neurocognitive measures revealed a general (albeit not entirely consistent) tendency for the General Rugby group to obtain comparatively poorer scores than the General Control group at each test interval and to make comparatively less pronounced improvement between test intervals on the ImPACT and WMS-III neurocognitive measures compared with the General Control group.

Symptom Measures

On the *independent* analyses between the General Rugby group and General Control group a significant difference was revealed on the ImPACT Symptom Scale in that the General Rugby group obtained a significantly higher Symptom Composite score than the General Control group at both the baseline and end of season intervals. At the baseline interval the General Rugby group, compared with the General Control group, evidenced higher scores for 17 of the 22 individual symptom measures, of which the difference was significant for 5 individual symptom measures (trouble falling, sleeping more, sadness, feeling more emotional and difficulty remembering) and approaching significance for 2 individual symptom measures (headache and mentally foggy). At the end of season interval the General Rugby group, compared with the General Control group, evidenced higher scores for 13 of the 22 individual symptom measures, of which the difference was significant for 2 individual symptom measures (fatigue and trouble falling asleep) and approaching significance for 2 individual symptom measures (numbness and difficulty concentrating).

On the *dependent* analyses for the General Rugby group and General Control group, the repeated measures ANOVAs for the ImPACT Symptom Scale revealed a significant effect for season for the Symptom Composite measure in that there was a significant overall decline in symptom reports at the end of season interval versus the baseline interval for both groups, and the group by season interaction effect on this measure was approaching significance indicating a more robust decline in symptom reports for the General Rugby group than for the General Control group. The dependent analyses for the General Rugby group revealed significantly lower scores for the Symptom Composite measure and for six individual symptom measures (trouble falling asleep, sleeping more, sleeping less, irritability, mentally foggy and difficulty remembering), and lower scores approaching significance for two further individual symptom measures (headache and

sensitivity to light), at the end of season interval versus the baseline interval. However, higher scores for the General Rugby group were revealed on five individual symptom measures at the end of season interval versus the baseline interval, although not significant. In contrast, the dependent analyses for the General Control group also revealed significantly lower scores for the Symptom Composite measure but for only two individual symptom measures (trouble falling asleep and difficulty concentrating) at the end of season interval versus the baseline interval.

In summary, taking into account significant results as well as trends, both the independent and dependent analyses for the ImPACT symptom measures revealed that the General Rugby group evidenced significantly higher symptom reports than the General Control group at both baseline and end of season intervals, and although overall symptom reports at the end of season interval decreased significantly for both groups, the trend was that the General Rugby group obtained higher scores for five individual symptom measures whereas the General Control group obtained no higher scores on any individual symptom measures, at the end of season interval versus the baseline interval.

8.2 CONCUSSED RUGBY AND MATCHED CONTROL GROUPS

The first section includes reports on the *independent t*-test comparisons between the Concussed Rugby group and Matched Control group across all neurocognitive measures at baseline and then first, second, third and fourth follow-up test intervals, with the composite scores on the ImPACT computerised program being reported first, followed by the scores on the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) paper-and pencil tests where applicable (first and fourth follow-up test intervals only). Following this are reports on the independent *t*-test comparisons between the Concussed Rugby group and Matched Control group across all symptom measures, these being the Symptom Composite score and each of the individual symptoms on the ImPACT program's Symptom Scale, at baseline and then first, second, third and fourth follow-up test intervals.

The second section includes reports on the *dependent t*-test comparisons for the Concussed Rugby group and the Matched Control group across all neurocognitive measures at the applicable follow-up test intervals versus the baseline interval, with the

composite scores on the ImPACT computerised program being reported first, followed by the scores on the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) paper-and pencil tests. Following this is the report on the dependent *t*-test comparisons for the Concussed Rugby group and the Matched Control group across all symptom measures, these being the Symptom Composite score and each of the individual symptoms on the ImPACT program's Symptom Scale, at the four follow-up test intervals versus the baseline test interval.

8.2.1 Independent *t*-test Comparisons

Baseline: Independent *t*-test Comparisons on ImPACT

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the ImPACT neurocognitive measures at the baseline interval (Table 26) revealed no significant findings. However, a finding that was approaching significance was revealed for the Impulse Control measure ($p = .061$), in the direction of the Concussed Rugby group's performance being poorer than that of the Matched Control group. Furthermore, there was no overall trend of one group performing better than the other, although the Concussed Rugby group's performance was poorer than that of the Matched Control group for the Verbal Memory and Visual Motor Speed measures; both groups obtained equal mean scores for the Reaction Time measure; and the Matched Control group's performance was poorer than that of the Concussed rugby group for the Visual Memory measure, although none were approaching significance.

Baseline: Independent *t*-test Comparisons on WMS-III VPA and VR

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) neurocognitive measures at the baseline interval (Table 27) revealed no significant findings. However, findings that were approaching significance were revealed for three measures, including (i) VPA I ($p = .062$), (ii) VPA II ($p = .063$), and (iii) VPA Recognition ($p = .090$), all in the direction of the Concussed Rugby group's performance being poorer than that of the Matched Control group. Overall, the trend was in the direction of the Concussed Rugby group's performance being poorer than that of the Matched Control group for seven of the nine measures, with the exception of marginally stronger performance for the VR I and VR Recognition measures.

First Follow-Up Test Interval: Independent *t*-test Comparisons on ImPACT

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the ImPACT neurocognitive measures at the first follow-up interval (Table 28) revealed no significant findings. However, findings that were approaching significance were revealed for two measures, including, (i) Reaction Time ($p = .067$) and (ii) Impulse Control ($p = .076$), both in the direction of the Concussed Rugby group's performance being poorer than that of the Matched Control group. Overall, the trend was in the direction of the Concussed Rugby group's performance being poorer than that of the Matched Control group for four of the five measures, with the exception of marginally stronger performance for the Visual Memory measure.

First Follow-Up Test Interval: Independent *t*-test Comparisons on WMS-III VPA and VR

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) neurocognitive measures at the first follow-up interval (Table 29) revealed no significant findings. However, findings that were approaching significance were revealed for four measures, including (i) VPA II ($p = .041$), (ii) VPA Recognition ($p = .077$), (iii) VR II ($p = .052$) and (iv) VR Percent Retention ($p = .029$), all in the direction of the Concussed Rugby group's performance being poorer than that of the Matched Control group. Overall, the trend was in the direction of the Concussed Rugby group's performance being poorer than that of the Matched Control group, with the exception of marginally stronger performance for the VR I and VR Recognition measures.

Second Follow-Up Test Interval: Independent *t*-test Comparisons on ImPACT

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the ImPACT neurocognitive measures at the second follow-up interval (Table 30) revealed no significant findings. Overall, the trend was in the direction of the Concussed Rugby group obtaining poorer scores than the Matched Control group for four of the five ImPACT measures, although obtaining a marginally stronger score for the Visual Memory measure, although none were approaching significance.

Third Follow-Up Test Interval: Independent *t*-test Comparisons on ImPACT

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the ImPACT neurocognitive measures at the third follow-up interval (Table 31) revealed no significant findings. Furthermore, there was no overall trend of one group performing better than the other, although the Concussed Rugby group obtained marginally stronger scores than the Matched Control group for the Verbal Memory, Visual Memory and Reaction Time measures, although none were approaching significance.

Fourth Follow-Up Test Interval: Independent *t*-test Comparisons on ImPACT

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the ImPACT neurocognitive measures at the fourth follow-up interval (Table 32) revealed no significant findings. Furthermore, there was no overall trend of one group performing better than the other, although the Concussed Rugby group (as with the third follow-up interval) obtained marginally stronger scores than the Matched Control group for the Verbal Memory, Visual Memory and Reaction Time measures, although none were approaching significance.

Fourth Follow-Up Test Interval: Independent *t*-test Comparisons on WMS-III VPA and VR

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) neurocognitive measures at the fourth follow-up interval (Table 33) revealed one significant finding for VPA I ($p = .013$), and findings approaching significance for three measures, including (i) VPA First Recall ($p = .099$), (ii) VPA II ($p = .093$) and (iii) VR Recognition ($p = .050$), all in the direction of the Concussed Rugby group's performance being poorer than that of the Matched Control group. Overall, the trend was in the direction of the Concussed Rugby group obtaining poorer scores than the Matched Control group for seven of the nine measures and equivalent scores for two measures (VPA Recognition and VPA Percent Retention).

Baseline: Independent *t*-test Comparisons on the Symptom Scale

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the ImPACT Symptom Scale at the baseline interval

(Table 34) revealed a significant finding for the ImPACT Symptom Composite measure (p = .021), in the direction of the Concussed Rugby group having a higher total symptom score than the Matched Control group. More specifically, there were significant findings for three of the individual symptom measures in the direction of the Concussed Rugby group having higher scores than the Matched Control group, including (i) nervousness (p = .037), (ii) difficulty concentrating (p = .022), and (iii) difficulty remembering (p = .043). In addition, there was a finding approaching significance in the direction of the Concussed Rugby group having a higher score for the headache symptom measure (p = .077). Overall, the Concussed Rugby group obtained higher scores for 19 of the 22 individual symptom measures, equivalent scores of zero for 2 individual symptom measures, and the Matched Control group obtained a higher score for only one individual symptom measure, slowed down.

First Follow-Up Test Interval: Independent *t*-test Comparisons on the Symptom Scale

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the ImPACT Symptom Scale at the first follow-up interval (Table 35) revealed a significant finding for the ImPACT Symptom Composite measure (p = .001), in the direction of the Concussed Rugby group having a higher total symptom score than the Matched Control group. More specifically, there were significant findings for five of the individual symptom measures in the direction of the Concussed Rugby group having higher scores than the Matched Control group, including (i) headache (p = .001**)**, (ii) fatigue (p = .012), (iii) drowsiness (p = .040), (iv) sensitivity to light (p = .026), and (v) slowed down (p = .005). In addition, there were findings approaching significance for three of the individual symptom measures in the direction of the Concussed Rugby group having higher scores than the Matched Control group, including (i) nausea (p = .077), (ii) sleeping more (p = .077), and (iii) numbness (p = .077). Overall, the Concussed Rugby group obtained higher scores for 18 of the 22 individual symptom measures, equivalent scores of zero for four individual symptom measures, and the Matched Control group did not obtain higher score for any individual symptom measures.

Second Follow-Up Test Interval: Independent *t*-test Comparisons on the Symptom Scale

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the ImPACT Symptom Scale at the second follow-up interval (Table 36) revealed a significant finding for the ImPACT Symptom Composite

measure (p = .043), in the direction of the Concussed Rugby group having a higher total symptom score than the Matched Control group (M = 4.00, SD = 7.96 versus M = 0.47, SD = 1.94, respectively). More specifically, although no significant findings were revealed, there were findings that were approaching significance for five individual symptom measures in the direction of the Concussed Rugby group having higher scores than the Matched Control group, including (i) headache (p = .084), (ii) sensitivity to light (p = .077), (iii) nervousness (p = .079), (iv) mentally foggy (p = .097), and (v) difficulty remembering (p = .089). Overall, the Concussed Rugby group obtained higher scores for 13 of the 22 individual symptom measures, equivalent scores of zero for 8 of the individual symptom measures, and the Matched Control group obtained a higher score for only one individual symptom measure, sleeping less.

Third Follow-Up Test Interval: Independent *t*-test Comparisons on the Symptom Scale

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the ImPACT Symptom Scale at the third follow-up interval (Table 37) revealed a significant finding for only one individual symptom measure, fatigue (p = .044), in the direction of the Concussed Rugby group having a higher score than the Matched Control group. In addition, there were findings that were approaching significance for the Symptom Composite (p = .055), and for two of the individual symptom measures, in the direction of the Concussed Rugby group having higher scores than the Matched Control group, including (i) headache (p = .097), and (ii) sensitivity to light (p = .079). Overall, the Concussed Rugby group obtained higher scores for 9 of the 22 individual symptom measures and equivalent scores of zero for 13 of the individual symptom measures, in that the Matched Control group did not score on any individual symptom measures.

Fourth Follow-Up Test Interval: Independent *t*-test Comparisons on the Symptom Scale

The independent *t*-test comparisons of group means between the Concussed Rugby group and Matched Control group on the ImPACT Symptom Scale at the fourth follow-up interval (Table 38) revealed neither significant findings nor findings that were approaching significance for the Symptom Composite or individual symptom measures. Overall, the Concussed Rugby group reported symptoms on six individual symptom measures and the Matched Control Group reported symptoms on nine individual symptom measures. The Concussed Rugby group obtained higher scores for four of the

individual symptom measures (including, headache, sensitivity to light, difficulty concentrating and difficulty remembering), both groups obtained an equivalent score for one individual symptom measure (mentally foggy), and the Matched Control group obtained higher scores for four individual symptom measures (including, fatigue, sleeping more, irritability and slowed down).

Table 26

Baseline Comparisons on ImPACT Scores for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
Baseline Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Verbal Memory	86.65	(9.68)	87.59	(9.55)	-0.285	0.389
Visual Memory	76.06	(13.15)	75.06	(16.92)	0.192	0.425
VMS	34.34	(6.49)	35.01	(8.80)	-0.251	0.402
Reaction Time	0.59	(0.07)	0.59	(0.08)	0.045	0.482
Impulse Control	8.82	(11.11)	4.35	(3.37)	1.587	0.061

Note. No '*p'* values reached significance ($p < .05$, one-tailed), with Bonferroni's adjustment. Visual Motor Speed (VMS).

Table 27

Baseline Comparisons on WMS-III Scores for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
Baseline Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
VPA 1	9.06	(2.33)	10.41	(2.65)	-1.582	0.062
VPA 1st Recall	9.82	(2.53)	10.35	(2.69)	-0.591	0.280
VPA 11	9.71	(2.49)	10.82	(1.55)	-1.569	0.063
VPA Recognition	99.29	(2.11)	100.00	(0.00)	-1.367	0.090
VPA % Retention	10.18	(2.48)	10.59	(2.35)	-0.497	0.311
VR 1	12.94	(2.36)	12.24	(2.39)	0.868	0.196
VR 11	13.41	(3.37)	14.18	(3.58)	-0.641	0.263
VR Recognition	11.59	(2.55)	11.47	(2.65)	0.132	0.448
VR % Retention	12.41	(2.43)	13.12	(2.26)	-0.878	0.194

Note. No '*p'* values reached significance ($p < .05$, one-tailed), with Bonferroni's adjustment. Verbal Paired Associates (VPA), Visual Reproduction (VR).

Table 28

First Follow-Up Comparisons on ImPACT Scores for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
1st Follow-Up Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Verbal Memory	86.88	(9.20)	88.94	(9.13)	-0.655	0.259
Visual Memory	80.35	(11.75)	77.59	(10.79)	0.714	0.240
VMS	36.70	(5.52)	38.79	(7.57)	-0.918	0.183
Reaction Time	0.61	(0.09)	0.57	(0.06)	1.542	0.067
Impulse Control	6.65	(9.45)	3.18	(2.33)	1.471	0.076

Note. No '*p*' values reached significance ($p < .05$, one-tailed), with Bonferroni's adjustment. Visual Motor Speed (VMS).

Table 29

First Follow-Up Comparisons on WMS-III Scores for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
1st Follow-Up Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
VPA 1	11.12	(3.14)	11.94	(2.79)	-0.808	0.213
VPA 1st Recall	11.94	(2.75)	12.94	(3.27)	-0.965	0.171
VPA 11	10.06	(2.46)	11.29	(1.40)	-1.797	0.041
VPA Recognition	99.04	(2.72)	100.00	(0.00)	-1.460	0.077
VPA % Retention	10.65	(2.64)	10.71	(2.34)	-0.069	0.473
VR 1	13.47	(1.88)	13.18	(2.38)	0.400	0.346
VR 11	13.88	(4.05)	16.00	(3.26)	-1.681	0.052
VR Recognition	12.76	(1.15)	12.59	(2.53)	0.262	0.398
VR % Retention	12.47	(3.13)	14.18	(1.70)	-1.976	0.029

Note. No '*p*' values reached significance ($p < .05$, one-tailed), with Bonferroni's adjustment. Verbal Paired Associates (VPA), Visual Reproduction (VR).

Table 30

Second Follow-Up Comparisons on ImPACT Scores for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
2nd Follow-Up Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Verbal Memory	88.53	(6.64)	89.71	(7.37)	-0.489	0.314
Visual Memory	82.47	(8.90)	78.35	(10.90)	1.207	0.118
VMS	36.44	(7.02)	39.22	(7.47)	-1.119	0.136
Reaction Time	0.59	(0.09)	0.57	(0.13)	0.357	0.362
Impulse Control	5.47	(5.86)	4.41	(2.79)	0.672	0.253

Note. No '*p'* values reached significance ($p < .05$, one-tailed), with Bonferroni's adjustment. Visual Motor Speed (VMS).

Table 31

Third Follow-Up Comparisons on ImPACT Scores for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
3rd Follow-Up Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Verbal Memory	91.00	(6.99)	90.00	(5.90)	0.451	0.328
Visual Memory	82.00	(9.73)	78.18	(12.27)	1.007	0.161
VMS	38.86	(7.58)	39.53	(7.33)	-0.264	0.397
Reaction Time	0.55	(0.09)	0.57	(0.13)	-0.514	0.306
Impulse Control	5.65	(4.12)	5.47	(4.72)	0.116	0.454

Note. No '*p'* values reached significance ($p < .05$, one-tailed), with Bonferroni's adjustment. Visual Motor Speed (VMS).

Table 32

Fourth Follow-Up Comparisons on ImPACT Scores for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
4th Follow-Up Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Verbal Memory	91.00	(6.12)	89.65	(7.08)	0.596	0.278
Visual Memory	80.12	(11.65)	79.12	(15.67)	0.211	0.417
VMS	39.46	(6.54)	39.87	(7.70)	-0.168	0.434
Reaction Time	0.55	(0.08)	0.56	(0.10)	-0.280	0.391
Impulse Control	5.76	(3.60)	5.00	(3.45)	0.633	0.266

Note. No '*p'* values reached significance ($p < .05$, one-tailed), with Bonferroni's adjustment. Visual Motor Speed (VMS).

Table 33

Fourth Follow-Up Comparisons on WMS-III Scores for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
4th Follow-Up Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
VPA 1	12.76	(2.51)	14.47	(1.63)	-2.350	0.013 †
VPA 1st Recall	13.76	(2.68)	14.94	(2.54)	-1.314	0.099
VPA 11	11.18	(1.51)	11.76	(0.97)	-1.351	0.093
VPA Recognition	100.00	(0.00)	100.00	(0.00)	-	-
VPA % Retention	11.06	(1.56)	11.06	(1.85)	-0.000	0.500
VR 1	12.71	(2.23)	13.59	(2.21)	-1.159	0.128
VR 11	15.47	(3.11)	16.65	(2.57)	-1.203	0.119
VR Recognition	12.76	(1.35)	13.47	(1.07)	-1.693	0.050
VR % Retention	14.24	(2.11)	14.53	(1.18)	-0.502	0.310

Note. † $p < .05$, one-tailed, with Bonferroni's adjustment. Verbal Paired Associates (VPA), Visual Reproduction (VR).

Table 34

Baseline Comparisons on ImPACT Symptoms for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
Baseline Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Symptom Composite	7.41	(8.41)	2.12	(5.90)	2.124	0.021†
Headache	0.12	(0.33)	0.00	(0.00)	1.461	0.077
Nausea	0.12	(0.49)	0.00	(0.00)	1.000	0.163
Vomiting	0.12	(0.49)	0.00	(0.00)	1.000	0.163
Balance problems	0.12	(0.49)	0.00	(0.00)	1.000	0.163
Dizziness	0.00	(0.00)	0.00	(0.00)	-	-
Fatigue	0.53	(1.23)	0.29	(0.85)	0.649	0.261
Trouble falling asleep	0.59	(1.37)	0.35	(1.06)	0.560	0.290
Sleeping more	0.47	(1.28)	0.00	(0.00)	1.515	0.070
Sleeping less	0.41	(1.06)	0.12	(0.33)	1.088	0.143
Drowsiness	0.24	(0.75)	0.00	(0.00)	1.289	0.104
Sensitivity to light	0.24	(0.75)	0.00	(0.00)	1.289	0.104
Sensitivity to noise	0.00	(0.00)	0.00	(0.00)	-	-
Irritability	0.47	(1.13)	0.35	(1.06)	0.314	0.378
Sadness	0.35	(0.79)	0.24	(0.97)	0.389	0.350
Nervousness	0.53	(1.18)	0.00	(0.00)	1.852	0.037†
Emotional	0.41	(1.28)	0.24	(0.97)	0.454	0.327
Numbness	0.24	(0.97)	0.00	(0.00)	1.000	0.163
Slowed down	0.06	(0.24)	0.18	(0.73)	-0.632	0.266
Mentally foggy	0.41	(1.06)	0.18	(0.73)	0.753	0.229
Diff concentrating	0.94	(1.71)	0.06	(0.24)	2.103	0.022†
Diff remembering	1.00	(1.84)	0.18	(0.53)	1.776	0.043†
Visual problems	0.06	(0.24)	0.00	(0.00)	1.000	0.163

Note. †$p < .05$, one-tailed. Difficulty (Diff).

Table 35

First Follow-Up Comparisons on ImPACT Symptoms for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
1st Follow-Up Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Symptom Composite	7.41	(8.32)	0.65	(1.50)	3.301	0.001††
Headache	1.00	(1.12)	0.00	(0.00)	3.688	0.001††
Nausea	0.12	(0.33)	0.00	(0.00)	1.461	0.077
Vomiting	0.00	(0.00)	0.00	(0.00)	-	-
Balance problems	0.06	(0.24)	0.00	(0.00)	1.000	0.163
Dizziness	0.24	(0.75)	0.00	(0.00)	1.289	0.104
Fatigue	0.88	(1.54)	0.00	(0.00)	2.368	0.012†
Trouble falling asleep	0.12	(0.49)	0.00	(0.00)	1.000	0.163
Sleeping more	0.35	(1.00)	0.00	(0.00)	1.461	0.077
Sleeping less	0.24	(0.75)	0.00	(0.00)	1.289	0.104
Drowsiness	0.47	(1.07)	0.00	(0.00)	1.817	0.040†
Sensitivity to light	0.76	(1.56)	0.00	(0.00)	2.018	0.026†
Sensitivity to noise	0.06	(0.24)	0.00	(0.00)	1.000	0.163
Irritability	0.29	(0.99)	0.18	(0.73)	0.396	0.348
Sadness	0.00	(0.00)	0.00	(0.00)	-	-
Nervousness	0.00	(0.00)	0.00	(0.00)	-	-
Emotional	0.53	(1.23)	0.12	(0.49)	1.283	0.105
Numbness	0.24	(0.66)	0.00	(0.00)	1.461	0.077
Slowed down	0.82	(1.24)	0.00	(0.00)	2.746	0.005††
Mentally foggy	0.47	(1.07)	0.18	(0.73)	0.939	0.178
Diff concentrating	0.53	(1.23)	0.18	(0.73)	1.018	0.158
Diff remembering	0.24	(0.97)	0.00	(0.00)	1.000	0.163
Visual problems	0.00	(0.00)	0.00	(0.00)	-	-

Note. † $p < .05$, one-tailed. †† $p < .01$, one-tailed. Difficulty (Diff).

Table 36

Second Follow-Up Comparisons on ImPACT Symptoms for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
2nd Follow-Up Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Symptom Composite	4.00	(7.96)	0.47	(1.94)	1.776	0.043†
Headache	0.47	(1.38)	0.00	(0.00)	1.411	0.084
Nausea	0.00	(0.00)	0.00	(0.00)	-	-
Vomiting	0.00	(0.00)	0.00	(0.00)	-	-
Balance problems	0.00	(0.00)	0.00	(0.00)	-	-
Dizziness	0.00	(0.00)	0.00	(0.00)	-	-
Fatigue	0.35	(1.46)	0.00	(0.00)	1.000	0.163
Trouble falling asleep	0.29	(0.99)	0.24	(0.97)	0.175	0.431
Sleeping more	0.00	(0.00)	0.00	(0.00)	-	-
Sleeping less	0.18	(0.73)	0.24	(0.97)	-0.200	0.422
Drowsiness	0.00	(0.00)	0.00	(0.00)	-	-
Sensitivity to light	0.35	(1.00)	0.00	(0.00)	1.461	0.077
Sensitivity to noise	0.00	(0.00)	0.00	(0.00)	-	-
Irritability	0.24	(0.97)	0.00	(0.00)	1.000	0.163
Sadness	0.18	(0.73)	0.00	(0.00)	1.000	0.163
Nervousness	0.41	(1.18)	0.00	(0.00)	1.444	0.079
Emotional	0.24	(0.97)	0.00	(0.00)	1.000	0.163
Numbness	0.00	(0.00)	0.00	(0.00)	-	-
Slowed down	0.12	(0.49)	0.00	(0.00)	1.000	0.163
Mentally foggy	0.41	(1.28)	0.00	(0.00)	1.329	0.097
Diff concentrating	0.24	(0.97)	0.00	(0.00)	1.000	0.163
Diff remembering	0.35	(1.06)	0.00	(0.00)	1.376	0.089
Visual problems	0.18	(0.73)	0.00	(0.00)	1.000	0.163

Note. † $p < .05$, one-tailed. Difficulty (Diff).

Table 37

Third Follow-Up Comparisons on ImPACT Symptoms for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
3rd Follow-Up Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Symptom Composite	2.71	(6.79)	0.00	(0.00)	1.643	0.055
Headache	0.41	(1.28)	0.00	(0.00)	1.329	0.097
Nausea	0.00	(0.00)	0.00	(0.00)	-	-
Vomiting	0.00	(0.00)	0.00	(0.00)	-	-
Balance problems	0.00	(0.00)	0.00	(0.00)	-	-
Dizziness	0.00	(0.00)	0.00	(0.00)	-	-
Fatigue	0.59	(1.37)	0.00	(0.00)	1.768	0.044 †
Trouble falling asleep	0.24	(0.97)	0.00	(0.00)	1.000	0.163
Sleeping more	0.18	(0.73)	0.00	(0.00)	1.000	0.163
Sleeping less	0.00	(0.00)	0.00	(0.00)	-	-
Drowsiness	0.18	(0.73)	0.00	(0.00)	1.000	0.163
Sensitivity to light	0.41	(1.18)	0.00	(0.00)	1.444	0.079
Sensitivity to noise	0.00	(0.00)	0.00	(0.00)	-	-
Irritability	0.00	(0.00)	0.00	(0.00)	-	-
Sadness	0.00	(0.00)	0.00	(0.00)	-	-
Nervousness	0.00	(0.00)	0.00	(0.00)	-	-
Emotional	0.00	(0.00)	0.00	(0.00)	-	-
Numbness	0.00	(0.00)	0.00	(0.00)	-	-
Slowed down	0.18	(0.73)	0.00	(0.00)	1.000	0.163
Mentally foggy	0.00	(0.00)	0.00	(0.00)	-	-
Diff concentrating	0.29	(1.21)	0.00	(0.00)	1.000	0.163
Diff remembering	0.24	(0.97)	0.00	(0.00)	1.000	0.163
Visual problems	0.00	(0.00)	0.00	(0.00)	-	-

Note. † $p < .05$, one-tailed. Difficulty (Diff).

Table 38

Fourth Follow-Up Comparisons on ImPACT Symptoms for Concussed Rugby versus Controls

	Concussed Rugby (n = 17)		Matched Controls (n = 17)			
4th Follow-Up Measures	Mean	(*SD*)	Mean	(*SD*)	*t*-value	*p*-value
Symptom Composite	1.47	(4.63)	1.41	(5.33)	0.034	0.487
Headache	0.29	(0.85)	0.24	(0.97)	0.188	0.426
Nausea	0.00	(0.00)	0.00	(0.00)	-	-
Vomiting	0.00	(0.00)	0.00	(0.00)	-	-
Balance problems	0.00	(0.00)	0.00	(0.00)	-	-
Dizziness	0.00	(0.00)	0.00	(0.00)	-	-
Fatigue	0.00	(0.00)	0.18	(0.73)	-1.000	0.163
Trouble falling asleep	0.00	(0.00)	0.00	(0.00)	-	-
Sleeping more	0.00	(0.00)	0.12	(0.49)	-1.000	0.163
Sleeping less	0.00	(0.00)	0.00	(0.00)	-	-
Drowsiness	0.00	(0.00)	0.00	(0.00)	-	-
Sensitivity to light	0.41	(1.18)	0.18	(0.73)	0.702	0.244
Sensitivity to noise	0.00	(0.00)	0.00	(0.00)	-	-
Irritability	0.00	(0.00)	0.24	(0.97)	-1.000	0.163
Sadness	0.00	(0.00)	0.00	(0.00)	-	-
Nervousness	0.00	(0.00)	0.00	(0.00)	-	-
Emotional	0.00	(0.00)	0.00	(0.00)	-	-
Numbness	0.00	(0.00)	0.00	(0.00)	-	-
Slowed down	0.18	(0.73)	0.24	(0.97)	-0.200	0.422
Mentally foggy	0.18	(0.73)	0.18	(0.73)	0.000	0.500
Diff concentrating	0.18	(0.73)	0.12	(0.49)	0.277	0.392
Diff remembering	0.24	(0.97)	0.12	(0.49)	0.447	0.329
Visual problems	0.00	(0.00)	0.00	(0.00)	-	-

Note. No '*p*' values reached significance ($p < .05$, one-tailed). Difficulty (Diff).

8.2.2 Dependent *t*-test Comparisons

ANOVAs and Dependent *t*-test Comparisons on ImPACT

The repeated measures ANOVAs, for the first, second, third and fourth follow-up intervals versus the baseline interval on the ImPACT neurocognitive measures (Table 39), did not reveal any significant group by season interaction effects for any of the measures, however the group by season interaction effect was approaching significance for the Reaction Time measure ($F = 2.17$, $df = 4,29$, $p = .098$). Results also revealed significant effects for season for two measures, including (i) Visual Motor Speed ($F = 12.10$, $df = 4,29$, $p = .000$) and (ii) Reaction Time ($F = 6.01$, $df = 4,29$, $p = .001$).

Follow-up investigation of the ANOVA effects using dependent *t*-test comparisons of group means for the ImPACT neurocognitive measures at the four follow-up intervals versus the baseline interval (Table 39) revealed the following significant findings and findings approaching significance: For the Visual Motor Speed measure (Figure 3), scores for the Concussed Rugby group improved significantly at the first, third and fourth follow-up intervals versus the baseline interval and approached significance at the second follow-up interval versus the baseline interval at which time the improved performance at the first follow-up interval was not maintained ($p = .017$, $p = .068$, $p = .001$, $p = .000$, respectively), whereas scores for the Matched Control group improved significantly at all four follow-up intervals versus the baseline interval ($p = .000$, $p = .001$, $p = .001$, $p = .000$, respectively). For the Reaction Time measure (Figure 4), scores for the Concussed Rugby group improved significantly at the third and fourth follow-up intervals versus the baseline interval ($p = .009$ and $p = .005$, respectively), whereas scores for the Matched Control group improved significantly at the fourth follow-up interval versus the baseline interval ($p = .023$). For the Visual Memory measure, scores for the Concussed Rugby group improved significantly at the second and third follow-up intervals versus the baseline interval and approached significance at the fourth follow-up interval versus the baseline interval ($p = .022$, $p = .025$, and $p = .072$, respectively), whereas scores for the Matched Control group revealed no improvements that were significant or approaching significance. For the Verbal Memory measure, improved scores for the Concussed Rugby group were approaching significance at the third and fourth follow-up intervals versus the baseline interval ($p = .039$ and $p = .069$, respectively), whereas scores for the Matched Control group revealed no improvements that were significant or approaching

significance. For the Impulse Control measure, improved scores for the Concussed Rugby group were approaching significance at the second and third follow-up intervals versus the baseline interval (p = .062 and p = .067, respectively), whereas scores for the Matched Control group were approaching significance at the first follow-up test interval versus the baseline interval in the direction of improved performance and at the third follow-up interval versus baseline interval in the direction of poorer performance (p = .071 and p = .076, respectively).

Perusal of the p-values revealed that, in addition to the Concussed Rugby group obtaining poorer mean scores than the Matched Control group at every test interval for the Visual Motor Speed measure, the improvement by the Concussed Rugby group appeared less robust (i.e., a weaker p-value was yielded) than that of the Matched Control group for the Visual Motor Speed measure at the first follow-up interval (p = .017 versus p = .000, respectively) and at the second follow-up interval (p = .068 versus p = .001, respectively), whereas their improvements were of equivalence at the third follow-up interval (p = .001), and at the fourth follow-up interval (p = .000). For the Concussed Rugby group, at the first follow-up interval versus the baseline interval, the group's mean score for the Reaction Time measure was 0.2 seconds slower with the difference approaching significance (p = .084), whereas at the same test interval the Matched Control group's mean score was 0.2 seconds faster compared with their baseline score. At the second follow-up interval, the Concussed Rugby group's Reaction Time score was equivalent to baseline performance and then showed an improvement at the third interval that was maintained at the fourth follow-up interval.

Perusal of trends revealed that for the Concussed Rugby group the slowed score for the Reaction Time measure at the first follow-up interval was the only score, amongst all the scores at the four follow-up intervals on the ImPACT neurocognitive measures**,** that was poorer than the baseline scores for any of the ImPACT measures, whereas trends revealed that for the Matched Control group all scores for the various ImPACT neurocognitive measures at the four follow-up intervals were improvements on the group's baseline scores, with the exception of poorer scores at the second, third and fourth follow-up intervals for the Impulse Control measure.

ANOVAs and Dependent *t*-test Comparisons on WMS-III VPA and VR

The repeated measures ANOVAs, for the first and fourth follow-up intervals versus the baseline interval on the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) measures (Table 40) did not reveal any significant group by season interaction effects for any of the measures, however the group by season interaction effect was approaching significance for the VR I measure ($F = 2.81$, $df = 2.31$, $p = .076$). Results also revealed significant effects for season for six measures, including (i) VPA I ($F = 58.18$, $df = 2,31$, $p = .000$), (ii) VPA First Recall ($F = 34.38$, $df = 2,31$, $p = .000$), (iii) VPA II ($F = 10.26$, $df = 2,31$, $p = .000$), (iv) VR II ($F = 8.10$, $df = 2,31$, $p = .001$), (v) VR Recognition ($F = 11.73$, $df = 2,31$, $p = .000$), and (vi) VR Percent Retention ($F = 10.40$, $df = 2,31$, $p = .000$), in that there were significant improvements on these measures at the follow-up intervals versus the baseline interval for both the Concussed Rugby group and the Matched Control group.

Follow-up investigation of the ANOVA effects using dependent *t*-test comparisons of group means for the WMS-III neurocognitive measures at the first and fourth follow-up intervals versus the baseline interval (Table 40) revealed the following significant findings and findings approaching significance: For the VR I measure (Figure 5), where the group by season interaction effect was approaching significance, there were no scores for the Concussed Rugby group that were significant or approaching significance although the score improved marginally at the first follow-up interval versus baseline interval, but at the fourth follow-up interval the score was poorer than at the baseline interval ($p = .221$ and $p = .358$, respectively), however improved scores for the Matched Control group were significant at the fourth follow-up interval versus the baseline interval and approaching significance at the first follow-up interval versus the baseline interval ($p = .001$ and $p = .030$, respectively).

Analyses for the rest of the WMS-III measures revealed a series of significant improvements for both the Concussed Rugby group and Matched Control group: For the Concussed Rugby group, scores improved significantly at the first and fourth follow-up intervals versus the baseline interval for two measures, including (i) VPA I ($p = .002$ and $p = .000$, respectively), and (ii) VPA First Recall ($p = .002$ and $p = .000$, respectively); and scores improved significantly at the fourth follow-up interval versus baseline interval for a further four measures, including (i) VPA II ($p = .002$), (ii) VR II ($p = .024$, (iii) VR

Recognition ($p = .004$), and (iv) VR Percent Retention ($p = .008$). The Concussed Rugby group's improved score on the VR Recognition measure at the first follow-up interval versus the baseline interval was approaching significance ($p = .038$). For the Matched Control group scores improved significantly at the first and fourth follow-up intervals versus the baseline interval for five measures, including (i) VPA I ($p = .013$ and $p = .000$, respectively), (ii) VPA First Recall ($p = .003$ and $p = .000$, respectively), (iii) VR II ($p = .008$ and $p = .000$, respectively), (iv) VR Recognition ($p = .000$ and $p = .001$, respectively), and (v) VR Percent Retention ($p = .004$ and $p = .001$, respectively); and at the fourth follow-up interval versus baseline interval for a further two measures, including (i) VPA II ($p = .005$) and (ii) VR I ($p = .001$). The Matched Control group's improved score on the VR I measure at the first follow-up interval versus the baseline interval was approaching significance ($p = .030$).

Perusal of the p-values at the first and fourth follow-up intervals versus the baseline interval revealed that the Concussed Rugby group appeared to make more robust improvements (i.e., higher *p*-values were yielded) than the Matched Control group on the following four WMS-III VPA measures, including (i) VPA I ($p = .002$ versus $p = .013$, respectively), (ii) VPA First Recall ($p = .002$ versus $p = .003$, respectively), (iii) VPA II ($p = .069$ versus $p = .075$, respectively) and (iv) VPA Percent Retention ($p = .277$ versus $p = .435$, respectively), at the first follow-up interval versus the baseline interval, and on (i) VPA II ($p = .002$ versus $p = .005$, respectively) and (ii) VPA Percent Retention ($p = .064$ versus $p = .119$, respectively) at the fourth follow-up interval versus the baseline interval. However, the Concussed Rugby group appeared to make less robust improvements (i.e., lower *p*-values were yielded) than the Matched Control group on four WMS-III VR measures at the first and fourth follow-up intervals versus the baseline interval, including (i) VR I ($p = .221$ and $p = .358$ versus $p = .030$ and $p = .001$, respectively), (ii) VR II ($p = .309$ and $p = .024$ versus $p = .008$ and $p = .000$, respectively), (iii) VR Recognition ($p = .038$ and $p = .004$ versus $p = .000$ and $p = .001$, respectively), and (iv) VR Percent Retention ($p = .470$ and $p = .008$ versus $p = .004$ and $p = .001$, respectively). Overall, compared with the Matched Control group, the Concussed Rugby group appeared to make slightly more robust improvements on the WMS-III VPA measures and less robust improvements on the WMS-III VR measures. The general trend was that the Matched Control group obtained equivalent or higher mean scores than the Concussed Rugby group for most of the measures at each test interval (23 instances out of

27 instances). The only scores for the Concussed Rugby group that were lower than their baseline scores, although not significant or approaching significance, were for VPA Recognition at the first follow-up interval and VR I at the fourth follow-up interval.

ANOVAs and Dependent *t*-test Comparisons on the Symptom Scale

The repeated measures ANOVAs for the first, second, third and fourth follow-up intervals versus the baseline interval on the ImPACT Symptom Scale (Table 41) revealed a significant group by season interaction effect for the headache symptom measure ($F = 2.92$, $df = 4,29$, $p = .038$), and the group by season interaction effect for the Symptom Composite measure was approaching significance ($F = 2.29$, $df = 4,29$, $p = .084$). Results also revealed significant effects for season for the Symptom Composite measure, ($F = 3.55$, $df = 4,29$, $p = .018$), and for two individual symptom measures, including (i) headache ($F = 3.00$, $df = 4,29$, $p = .034$) and (ii) mentally foggy ($F = 3.02$, $df = 4,29$, $p = .034$), and the effect for season was approaching significance for one individual symptom measure, slowed down ($F = 2.33$, $df = 4,29$, $p = .079$).

Follow-up investigation of the ANOVA effects using dependent *t*-test comparisons of group means for the ImPACT Symptom Scale at the four follow-up intervals versus the baseline interval (Table 41) revealed the following significant findings and findings approaching significance: For the Symptom Composite (Figure 6), scores for the Concussed Rugby group decreased significantly at the third and fourth follow-up intervals versus the baseline interval ($p = .039$ and $p = .007$, respectively), whereas decreased scores for the Matched Control group at the second and third follow-up test intervals versus the baseline interval, were approaching significance ($p = .058$ and $p = .079$, respectively). Follow-up investigation of the significant group by season interaction effect for the headache symptom measure (Figure 7), was further supported by between-subjects effects revealing (i) significant differences between the two groups ($p = .013$) and (ii) significant differences between the testing intervals ($p = .003$), particularly as the Concussed Rugby group obtained a significantly higher score for the headache symptom measure at the first follow-up interval versus the baseline interval ($p = .002$), whereas the Matched Control group did not report symptoms on the headache symptom measure at the baseline and first three follow-up intervals. The significant effect for season for the mentally foggy symptom measure, was further supported by the between-subjects effects revealing significant differences between the testing intervals ($p = .002$), due to the

Concussed Rugby group having higher mean scores on this symptom measure than the Matched Control group, particularly at the baseline and first two follow-up intervals, (*M* = 0.41, *SD* = 1.06, *M* = 0.47, *SD* = 1.07, *M* = 0.41, *SD* = 1.28 versus *M* = 0.18, *SD* = 0.73, *M* = 0.18, *SD* = 0.73, *M* = 0.00, *SD* = 0.00, respectively).

Follow-up investigation of the dependent *t*-test comparisons of group means for the Concussed Rugby group on the ImPACT Symptom Scale at the four follow-up intervals versus the baseline interval (Table 41) revealed these findings that were significant or approaching significance:

First follow-up interval versus baseline interval: The Concussed Rugby group obtained significantly higher scores for two symptom measures, including (i) headache (*p* = .002) and (ii) slowed down (*p* = .009), and significantly lower scores for three symptom measures, including (i) sadness (*p* = .041), (ii) nervousness (*p* = .041), and (iii) difficulty remembering (*p* = .039). The lower scores for two individual symptom measures were approaching significance, including (i) trouble falling asleep (*p* = .095), and (ii) irritability (*p* = .094). *Second* follow-up interval versus baseline interval: No significant findings were revealed for the Concussed Rugby group, however lower scores for four symptom measures were approaching significance, including (i) sleeping more (*p* = .075), (ii) feeling more emotional (*p* = .094), (iii) difficulty concentrating (*p* = .066), and (iv) difficulty remembering (*p* = .079). *Third* follow-up interval versus baseline interval: The Concussed Rugby group obtained significantly lower scores for the Symptom Composite measure (*p* = .039) and three individual symptom measures, including (i) sadness (*p* = .041), (ii) nervousness (*p* = .041), and (iii) difficulty remembering (*p* = .039). The lower scores for four symptom measures were approaching significance, including (i) sleeping less (*p* = .065), (ii) irritability (*p* = .052), (iii) mentally foggy (*p* = .065), and (iv) difficulty concentrating (*p* = .093). *Fourth* follow-up interval versus baseline interval, the Concussed Rugby group obtained significantly lower scores for the Symptom Composite measure (*p* = .007) and six individual symptom measures, including (i) fatigue (*p* = .048), (ii) trouble falling asleep (*p* = .048), (iii) sadness (*p* = .041), (iv) nervousness (*p* = .041), (v) difficulty concentrating (*p* = .045), and (vi) difficulty remembering (*p* = .039). The group's non-report for three individual symptom measures were approaching significance, including (i) sleeping more (*p* = .075), (ii) sleeping less (*p* = .065), and (iii) irritability (*p* = .052).

Follow-up investigation of the dependent *t*-test comparisons of group means for the Matched Control group on the ImPACT Symptom Scale at the four follow-up intervals versus the baseline interval (Table 41) revealed no significant findings, however lower individual symptom scores approaching significance (due to their non-presence except for irritability at the first follow-up interval) were as follows:

First follow-up interval versus baseline interval: The Matched Control group obtained lower scores that were approaching significance for five individual symptom measures, including (i) fatigue (p = .086), (ii) trouble falling asleep (p = .094), (iii) sleeping less (p = .082), (iv) irritability (p = .094), and (v) difficulty remembering (p = .094). *Second* follow-up interval versus baseline interval: The Matched Control group obtained lower scores that were approaching significance for the Symptom Composite (p = .058) and for three individual symptom measures (due to their non-report), including (i) fatigue (p = .086), (ii) irritability (p = .094), and (iii) difficulty remembering (p = .094). *Third* follow-up interval versus baseline interval: The Matched Control group obtained lower scores that were approaching significance for the Symptom Composite (p = .079) and for five individual symptom measures (due to their non-report), including (i) fatigue (p = .086), (ii) trouble falling asleep (p = .094), (iii) sleeping less (p = .082), (iv) irritability (p = .094), and (v) difficulty remembering (p = .094). *Fourth* follow-up interval versus baseline interval: The Matched Control group obtained lower scores that were approaching significance for two individual symptom measures (due to their non-report), including (i) trouble falling asleep (p = .094), and (ii) sleeping less (p = .082).

Perusal of trends revealed that the Concussed Rugby group reported symptoms on 20 symptom measures at the baseline interval compared with 18 symptom measures at the first follow-up interval, 14 symptom measures at the second follow-up interval, 9 symptom measures at the third follow-up interval and 6 symptom measures at the fourth follow-up interval. The Matched Control group reported symptoms on 10 symptom measures at the baseline interval compared with 4 symptom measures at the first follow-up interval, 2 symptom measures at the second follow-up interval, 0 symptom measures at the third follow-up interval, and 9 symptom measures at the fourth follow-up interval.

Table 39

Comparisons on ImPACT Scores for Concussed Rugby and Controls at Baseline versus Four Follow-Up Intervals

Test measures by group	Baseline		1st Follow-Up			2nd Follow-Up			3rd Follow-Up			4th Follow-Up		Interaction	
	Mean	(*SD*)	Mean	(*SD*)	*p- value*	Mean	(*SD*)	*p- value*	Mean	(*SD*)	*p-value*	Mean	(*SD*)	*p-value*	*p-value*
Verbal Mem															
Rugby ($n = 17$)	86.65	(9.68)	86.88	(9.20)	0.467	88.53	(6.64)	0.246	91.00	(6.99)	0.039	91.00	(6.12)	0.069	0.830
Control ($n = 17$)	87.59	(9.55)	88.94	(9.13)	0.232	89.71	(7.37)	0.223	90.00	(5.90)	0.175	89.65	(7.08)	0.161	
Visual Mem															
Rugby	76.06	(13.15)	80.35	(11.75)	0.123	82.47	(8.90)	0.022†	82.00	(9.73)	0.025†	80.12	(11.65)	0.072	0.942
Control	75.06	(16.92)	77.59	(10.79)	0.262	78.35	(10.90)	0.181	78.18	(12.27)	0.198	79.12	(15.67)	0.158	
VMS															
Rugby	34.34	(6.49)	36.70	(5.52)	0.017†	36.44	(7.02)	0.068	38.86	(7.58)	0.001††	39.46	(6.54)	0.000††	0.429
Control	35.01	(8.80)	38.79	(7.57)	0.000 ††	39.22	(7.47)	0.001††	39.53	(7.33)	0.001††	39.87	(7.70)	0.000 ††	
Reaction Time															
Rugby	0.59	(0.07)	0.61	(0.09)	0.084	0.59	(0.09)	0.475	0.55	(0.09)	0.009†	0.55	(0.08)	0.005††	0.098
Control	0.59	(0.08)	0.57	(0.06)	0.148	0.57	(0.13)	0.232	0.57	(0.13)	0.193	0.56	(0.10)	0.023 †	
Impulse Ctrl															
Rugby	8.82	(11.11)	6.65	(9.45)	0.144	5.47	(5.86)	0.062	5.65	(4.12)	0.067	5.76	(3.60)	0.106	0.203
Control	4.35	(3.37)	3.18	(2.33)	0.071	4.41	(2.79)	0.473	5.47	(4.72)	0.076	5.00	(3.45)	0.245	

Note. † $p < .05$, one-tailed, with Bonferroni's adjustment. †† $p < .01$, one-tailed, with Bonferroni's adjustment.
No interaction '*p*' values reached significance ($p < .05$, 2-tailed). Memory (Mem), Visual Motor Speed (VMS), Control (Ctrl)

Table 40

Comparisons on WMS-III Scores for Concussed Rugby and Controls at Baseline versus Four Follow-Up Intervals

Test measures by group	Baseline	1st Follow-Up		4th Follow-Up		Interaction
	Mean (*SD*)	Mean (*SD*)	*p*- value	Mean (*SD*)	*p*- value	*p*-value
VPA 1						
Rugby (n = 17)	9.06 (2.33)	11.12 (3.14)	0.002††	12.76 (2.51)	0.000††	0.375
Control (n = 17)	10.41 (2.65)	11.94 (2.79)	0.013†	14.47 (1.63)	0.000 ††	
VPA 1st Recall						
Rugby	9.82 (2.53)	11.94 (2.75)	0.002††	13.76 (2.68)	0.000††	0.826
Control	10.35 (2.69)	12.94 (3.27)	0.003††	14.94 (2.54)	0.000 ††	
VPA 11						
Rugby	9.71 (2.49)	10.06 (2.46)	0.069	11.18 (1.51)	0.002††	0.545
Control	10.82 (1.55)	11.29 (1.40)	0.075	11.76 (0.97)	0.005 ††	
VPA Recog						
Rugby	99.29 (2.11)	99.04 (2.72)	0.325	100.00 (0.00)	0.094	0.302
Control	100.00 (0.00)	100.00 (0.00)	-	100.00 (0.00)	-	
VPA % Retent						
Rugby	10.18 (2.48)	10.65 (2.64)	0.277	11.06 (1.56)	0.064	0.834
Control	10.59 (2.35)	10.71 (2.34)	0.435	11.06 (1.85)	0.119	
VR 1						
Rugby	12.94 (2.36)	13.47 (1.87)	0.221	12.71 (2.23)	0.358	0.076
Control	12.24 (2.39)	13.18 (2.38)	0.030	13.59 (2.21)	0.001 ††	
VR 11						
Rugby	13.41 (3.37)	13.88 (4.05)	0.309	15.47 (3.11)	0.024†	0.449
Control	14.18 (3.58)	16.00 (3.26)	0.008†	16.65 (2.57)	0.000 ††	
VR Recog						
Rugby	11.59 (2.55)	12.76 (1.15)	0.038	12.76 (1.35)	0.004††	0.339
Control	11.47 (2.65)	12.59 (2.53)	0.000††	13.47 (1.07)	0.001 ††	
VR % Retent						
Rugby	12.41 (2.43)	12.47 (3.13)	0.470	14.24 (2.11)	0.008†	0.109
Control	13.12 (2.26)	14.18 (1.70)	0.004††	14.53 (1.18)	0.001 ††	

Note. † $p < .05$, one-tailed, with Bonferroni's adjustment. †† $p < .01$, one-tailed, with Bonferroni's adjustment
No interaction '*p'* values reached significance ($p < .05$, 2-tailed). Verbal Paired Associates (VPA), Visual Reproduction (VR).

Table 41

Comparisons on ImPACT Symptoms for Concussed Rugby and Controls at Baseline versus Four Follow-Up Intervals

Test measures by Group	Baseline		1st Follow-Up			2nd Follow-Up			3rd Follow-Up			4th Follow-Up			Interaction
	Mean	(*SD*)	Mean	(*SD*)	*p- value*	Mean	(*SD*)	*p- value*	Mean	(*SD*)	*p-value*	Mean	(*SD*)	*p-value*	*p-value*
Symptom Comp															
Rugby ($n = 17$)	7.41	(8.41)	7.41	(8.32)	0.500	4.00	(7.96)	0.111	2.71	(6.79)	0.039†	1.47	(4.63)	0.007††	0.084
Control ($n = 17$)	2.12	(5.90)	0.65	(1.50)	0.121	0.47	(1.94)	0.058	0.00	(0.00)	0.079	1.41	(5.33)	0.360	
Headache															
Rugby	0.12	(0.33)	1.00	(1.12)	0.002††	0.47	(1.38)	0.166	0.41	(1.28)	0.193	0.29	(0.85)	0.166	0.038*
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.24	(0.97)	0.166	
Nausea															
Rugby	0.12	(0.49)	0.12	(0.33)	0.500	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.206
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	
Vomiting															
Rugby	0.12	(0.49)	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.325
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	
Balance Probs															
Rugby	0.12	(0.49)	0.06	(0.24)	0.334	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.368
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	
Dizziness															
Rugby	0.00	(0.00)	0.24	(0.75)	0.108	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.207
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	

Continued on following page

Table 41, ctd. *Comparisons on ImPACT Symptoms for Concussed Rugby and Controls at Baseline versus Four Follow-Up Intervals*

Test measures by Group	Baseline		1st Follow-Up			2nd Follow-Up			3rd Follow-Up			4th Follow-Up			Interaction
	Mean	(*SD*)	Mean	(*SD*)	*p- value*	Mean	(*SD*)	*p- value*	Mean	(*SD*)	*p-value*	Mean	(*SD*)	*p-value*	*p-value*
Fatigue															
Rugby	0.53	(1.23)	0.88	(1.54)	0.235	0.35	(1.46)	0.361	0.59	(1.37)	0.434	0.00	(0.00)	0.048†	0.140
Control	0.29	(0.85)	0.00	(0.00)	0.086	0.00	(0.00)	0.086	0.00	(0.00)	0.086	0.18	(0.73)	0.342	
Tr falling asleep															
Rugby	0.59	(1.37)	0.12	(0.49)	0.095	0.29	(0.99)	0.241	0.24	(0.97)	0.211	0.00	(0.00)	0.048†	0.686
Control	0.35	(1.06)	0.00	(0.00)	0.094	0.24	(0.97)	0.334	0.00	(0.00)	0.094	0.00	(0.00)	0.094	
Sleeping more															
Rugby	0.47	(1.28)	0.35	(1.00)	0.375	0.00	(0.00)	0.075	0.18	(0.73)	0.220	0.00	(0.00)	0.075	0.363
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.12	(0.49)	0.166	
Sleeping less															
Rugby	0.41	(1.06)	0.24	(0.75)	0.303	0.18	(0.73)	0.241	0.00	(0.00)	0.065	0.00	(0.00)	0.065	0.261
Control	0.12	(0.33)	0.00	(0.00)	0.082	0.24	(0.97)	0.326	0.00	(0.00)	0.082	0.00	(0.00)	0.082	
Drowsiness															
Rugby	0.24	(0.75)	0.47	(1.07)	0.248	0.00	(0.00)	0.108	0.18	(0.73)	0.413	0.00	(0.00)	0.108	0.166
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	
Sens to light															
Rugby	0.24	(0.75)	0.76	(1.56)	0.113	0.35	(1.00)	0.272	0.41	(1.18)	0.242	0.41	(1.18)	0.242	0.422
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.18	(0.73)	0.166	

Continued on following page

Table 41, ctd. *Comparisons on ImPACT Symptoms for Concussed Rugby and Controls at Baseline versus Four Follow-Up Intervals*

Test measures by Group	Baseline		1st Follow-Up			2nd Follow-Up			3rd Follow-Up			4th Follow-Up			Interaction
	Mean	(*SD*)	Mean	(*SD*)	*p- value*	Mean	(*SD*)	*p- value*	Mean	(*SD*)	*p-value*	Mean	(*SD*)	*p-value*	*p-value*
Sens to noise															
Rugby	0.00	(0.00)	0.06	(0.24)	0.166	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.325
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	
Irritability															
Rugby	0.47	(1.13)	0.29	(0.99)	0.094	0.24	(0.97)	0.272	0.00	(0.00)	0.052	0.00	(0.00)	0.052	0.740
Control	0.35	(1.06)	0.18	(0.73)	0.094	0.00	(0.00)	0.094	0.00	(0.00)	0.094	0.24	(0.97)	0.375	
Sadness															
Rugby	0.35	(0.79)	0.00	(0.00)	0.041†	0.18	(0.73)	0.228	0.00	(0.00)	0.041†	0.00	(0.00)	0.041†	0.602
Control	0.24	(0.97)	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.00	(0.00)	0.166	
Nervousness															
Rugby	0.53	(1.18)	0.00	(0.00)	0.041†	0.41	(1.18)	0.371	0.00	(0.00)	0.041†	0.00	(0.00)	0.041†	0.130
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	
Emotional															
Rugby	0.41	(1.28)	0.53	(1.23)	0.401	0.24	(0.97)	0.094	0.00	(0.00)	0.101	0.00	(0.00)	0.101	0.348
Control	0.24	(0.97)	0.12	(0.49)	0.166	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.00	(0.00)	0.166	
Numbness															
Rugby	0.24	(0.97)	0.24	(0.66)	0.500	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.206
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	

Continued on following page

Table 41, ctd. *Comparisons on ImPACT Symptoms for Concussed Rugby and Controls at Baseline versus Four Follow-Up Intervals*

Test measures by Group	Baseline		1st Follow-Up			2nd Follow-Up			3rd Follow-Up			4th Follow-Up			Interaction
	Mean	(*SD*)	Mean	(*SD*)	*p- value*	Mean	(*SD*)	*p- value*	Mean	(*SD*)	*p-value*	Mean	(*SD*)	*p-value*	*p-value*
Slowed down															
Rugby	0.06	(0.24)	0.82	(1.24)	0.009††	0.12	(0.49)	0.334	0.18	(0.73)	0.272	0.18	(0.73)	0.272	0.130
Control	0.18	(0.73)	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.24	(0.97)	0.424	
Mentally foggy															
Rugby	0.41	(1.06)	0.47	(1.07)	0.442	0.41	(1.28)	0.500	0.00	(0.00)	0.065	0.18	(0.73)	0.241	0.318
Control	0.18	(0.73)	0.18	(0.73)	0.500	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.18	(0.73)	0.500	
Diff concentr															
Rugby	0.94	(1.71)	0.53	(1.23)	0.176	0.24	(0.97)	0.066	0.29	(1.21)	0.093	0.18	(0.73)	0.045†	0.233
Control	0.06	(0.24)	0.18	(0.73)	0.272	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.12	(0.49)	0.166	
Diff remember															
Rugby	1.00	(1.84)	0.24	(0.97)	0.039†	0.35	(1.06)	0.079	0.24	(0.97)	0.039†	0.24	(0.97)	0.039†	0.246
Control	0.18	(0.53)	0.00	(0.00)	0.094	0.00	(0.00)	0.094	0.00	(0.00)	0.094	0.12	(0.49)	0.334	
Visual Problems															
Rugby	0.06	(0.24)	0.00	(0.00)	0.166	0.18	(0.73)	0.272	0.00	(0.00)	0.166	0.00	(0.00)	0.166	0.368
Control	0.00	(0.00)	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	0.00	(0.00)	-	

Note. † $p < .05$, one-tailed. †† $p < .01$, one-tailed. * $p < .05$, 2-tailed, for interaction values. Composite (Comp), Problems (Probs), Trouble (Tr), Sensitivity (Sens), Concentrating (Concentr), Remembering (Remember), Difficulty (Diff).

Figure 3. ImPACT Visual Motor Speed scores at baseline versus four intervals for Concussed Rugby and Controls

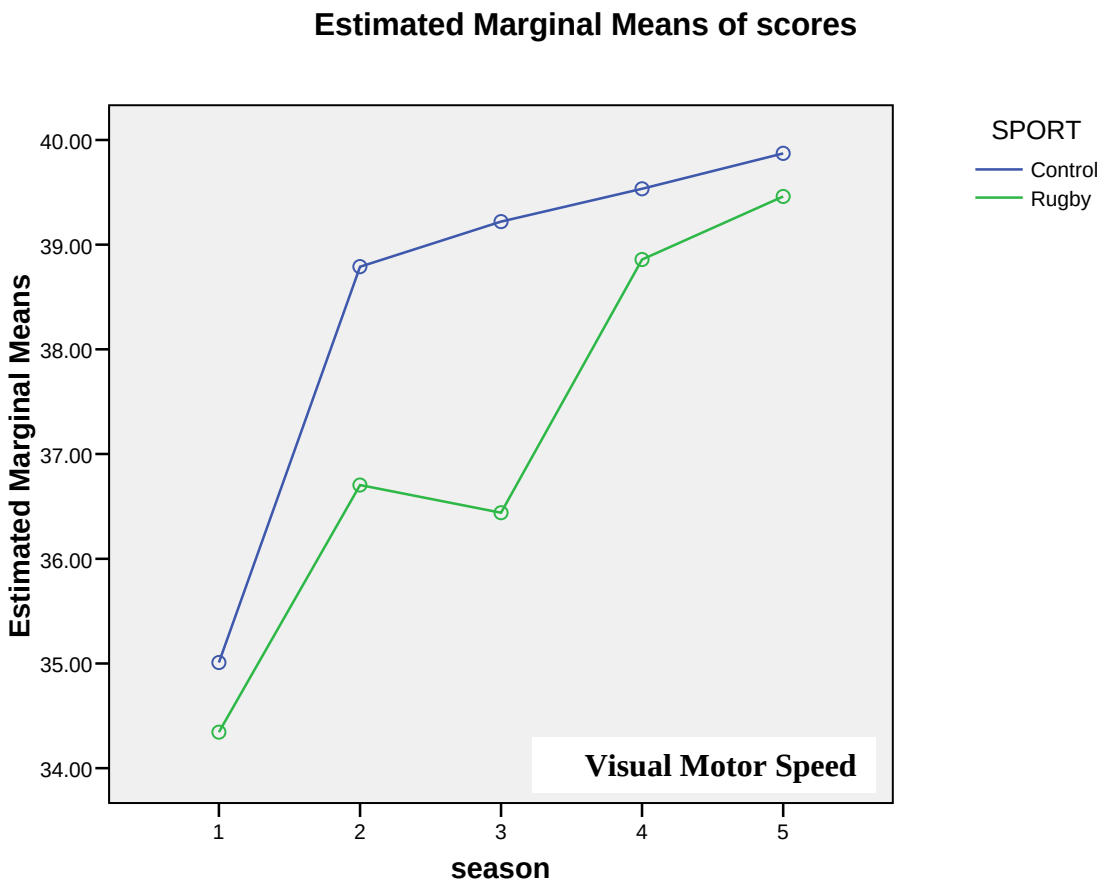


Figure 4. ImPACT Reaction Time scores at baseline versus four intervals for Concussed Rugby and Controls

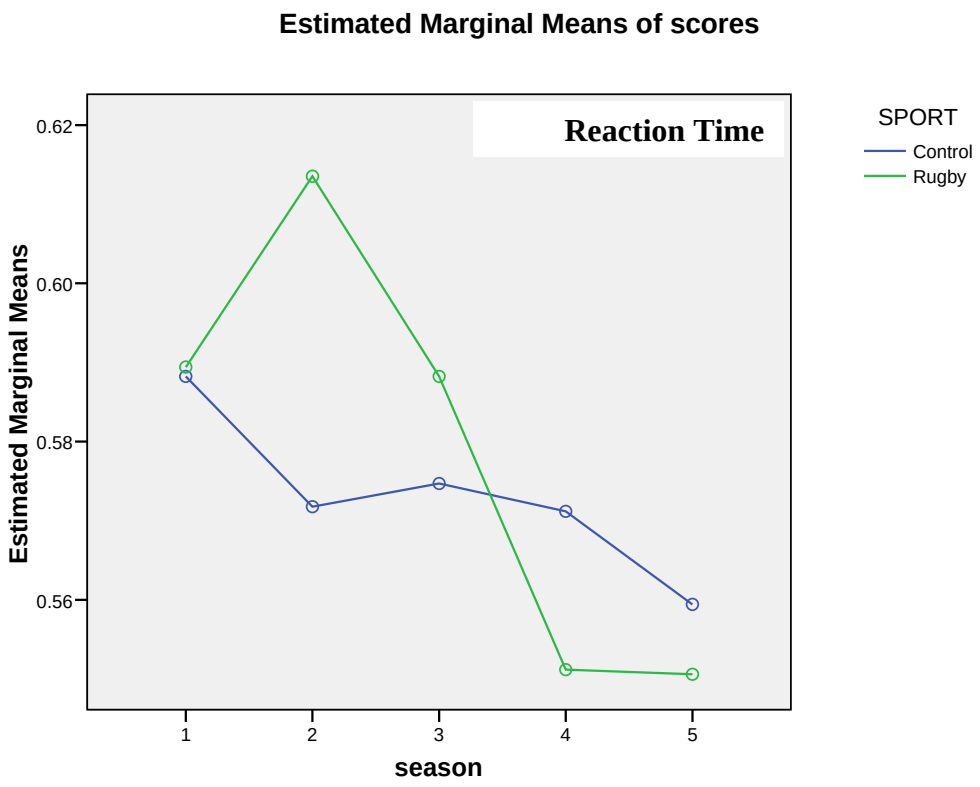


Figure 5. WMS-III scores at baseline versus first and fourth intervals for Concussed Rugby and Controls

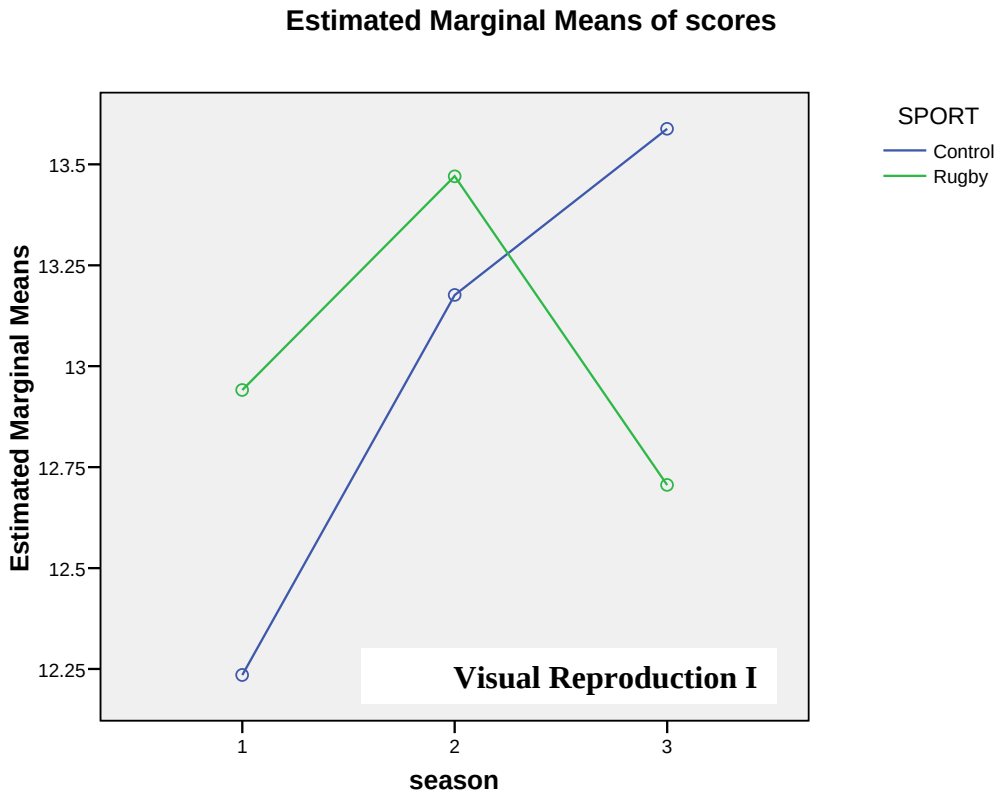


Figure 6. ImPACT Symptom scores at baseline versus four intervals for Concussed Rugby and Controls

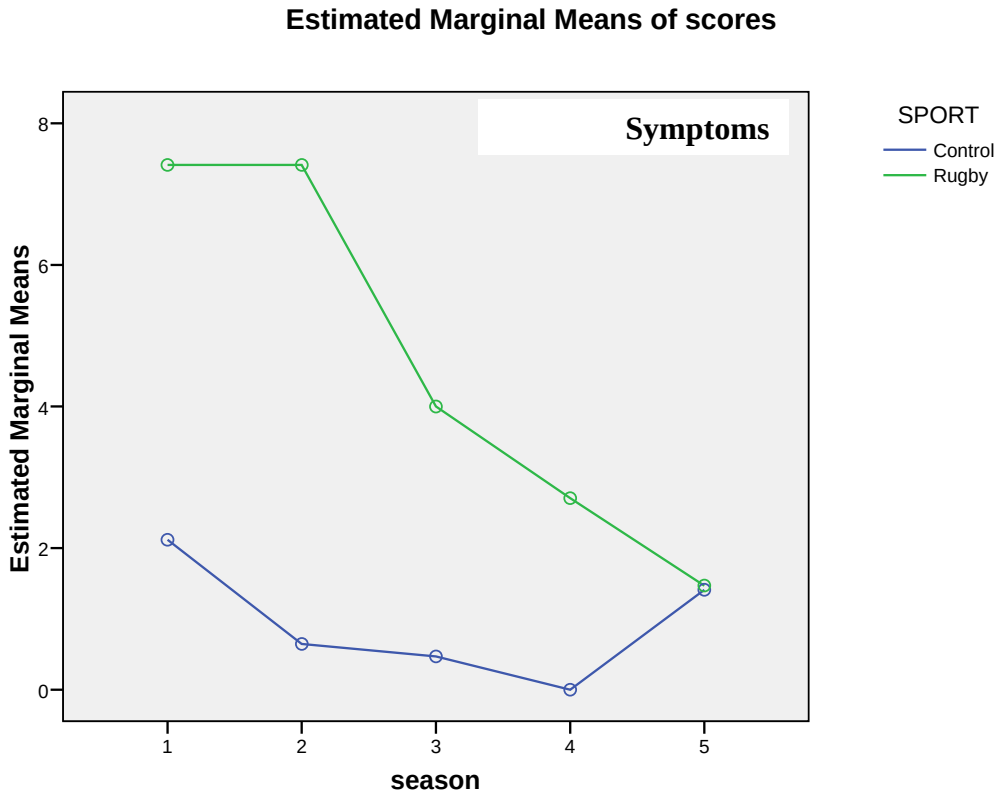
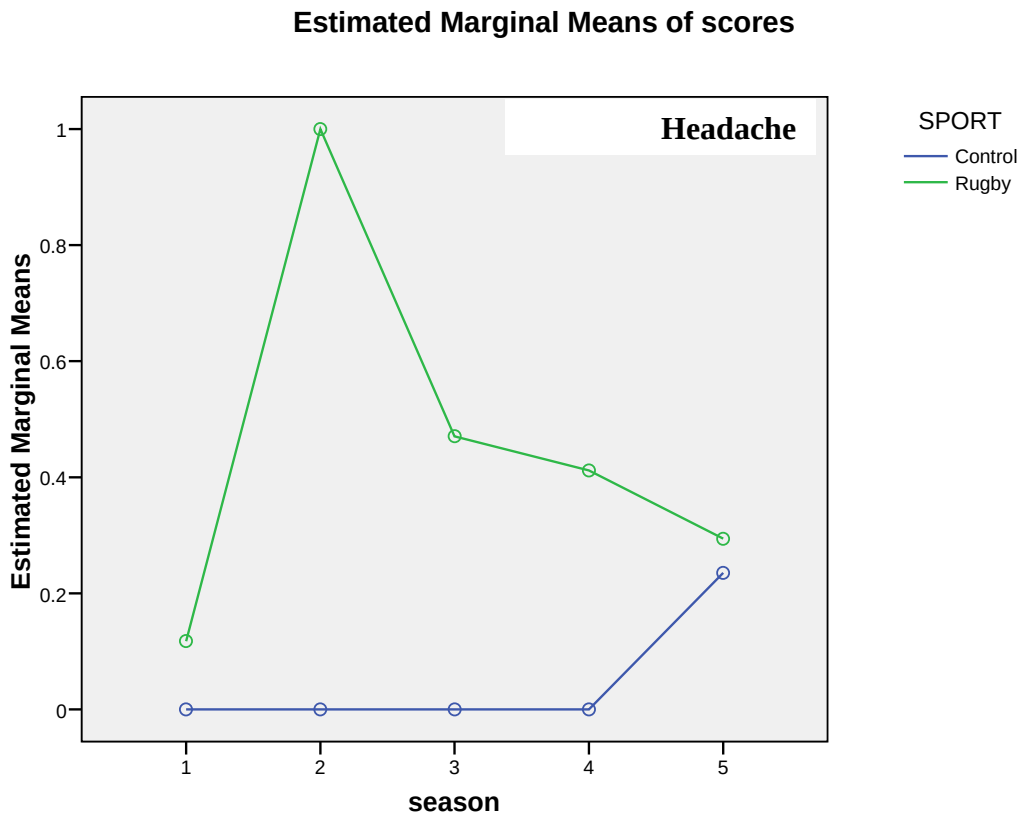


Figure 7. Headache symptom scores at baseline versus four intervals for Concussed Rugby and Controls



8.2.3 Synthesis of results for Concussed Rugby and Matched Control groups

Neurocognitive Measures

On the *independent* analyses between the Concussed Rugby group and Matched Control group no significant differences were revealed on the ImPACT and WMS-III neurocognitive measures at the baseline and first three follow-up intervals, however at the fourth follow-up interval a significant difference was revealed on one measure (WMS-III VPA I) in the direction of poorer performance for the Concussed Rugby group. Differences that were approaching significance were revealed for four measures at the baseline interval (ImPACT Impulse Control, and WMS-III VPA 1, VPA II, and VPA Recognition), for six measures at the first follow-up interval (ImPACT Reaction Time and Impulse Control, and WMS-III VPA II, VPA Recognition, VR II and VR Percent Retention) and for three measures at the fourth follow-up interval (WMS-III VPA First Recall, VPA II, and VR Recognition), in the direction of poorer performance for the Concussed Rugby group.

On the *dependent* analyses for the Concussed group and for the Matched Control group, the repeated measures ANOVAs revealed group by season interaction effects approaching significance for the WMS-III VR I and the ImPACT Reaction Time measures. A significant effect for season was also revealed for the ImPACT Reaction Time measures. For the Reaction Time measure, dependent analyses revealed that the Concussed Rugby group's slower performance at the first follow-up interval was approaching significance, whereas the Matched Control group's performance was faster at the first follow-up interval. However, the Concussed Rugby group's performance at the second follow-up interval was equivalent to their baseline performance, and a further significant improvement made at the third follow-up interval was maintained at the fourth follow-up interval, whereas the Matched Control group's Reaction Time at all four follow-up intervals was faster than that of their baseline performance. For the VR I measure, dependent analyses revealed that the Concussed Rugby group's performance improved at the first follow-up interval but was poorer than baseline performance at the fourth follow-up interval, whereas the Matched Control group's performance improved steadily at the first and fourth follow-up intervals. Significant effects for season revealed improvement by both groups at the follow-up intervals for seven further measures (ImPACT Visual Motor Speed and WMS-III VPA I, VPA First Recall, VPA II, VR II, VR Recognition and

VR Percent Retention), with dependent analyses revealing that the Concussed Rugby group, compared with the Matched Control group, appeared to make a less robust improvement for the ImPACT Visual Motor Speed and all four of the WMS-III VR measures. The dependent analyses of group means for the ImPACT neurocognitive measures at the four follow-up intervals and the WMS-III neurocognitive measures at the first and fourth follow-up intervals, versus baseline interval, revealed significant improvements for each group: The Concussed Rugby group improved on three measures at the first follow-up interval (ImPACT Visual Motor Speed and WMS-III VPA I and VPA First Recall), one measure at the second follow-up interval (ImPACT Visual Memory), three measures at the third follow-up interval (ImPACT Visual Memory, Visual Motor Speed and Reaction Time), and eight measures at the fourth follow-up interval (ImPACT Visual Motor Speed and Reaction Time, and WMS-III VPA I, VPA First Recall, VPA II, VR II, VR Recognition and VR Percent Retention). The Matched Control group improved on six measures at the first follow-up interval (ImPACT Visual Motor Speed and WMS-III VPA I, VPA First Recall, VR II, VR Recognition and VR Percent Retention), one measure at the second follow-up interval (ImPACT Visual Motor Speed), one measure at the third follow-up interval (ImPACT Visual Motor Speed), and nine measures at the fourth follow-up interval (ImPACT Visual Motor Speed and Reaction Time and WMS-III VPA I, VPA First Recall, VPA II, VR I, VR II, VR Recognition and VR Percent Retention). Overall trends were that both the Concussed Rugby and Matched Control groups' follow-up scores were all improvements on their baseline scores, apart from Reaction Time and VPA Recognition at the first follow-up interval and VR I at the fourth follow-up interval for the Concussed Rugby group, and Impulse Control at the second, third and fourth follow-up intervals for the Matched Control group.

In summary, taking into account significant results as well as trends, both the independent and dependent analyses for the ImPACT and WMS-III neurocognitive measures revealed a general (albeit not entirely consistent) tendency for the Concussed Rugby group to obtain comparatively poorer scores than the Matched Control group (who obtained no scores that were significantly weaker or approaching significance apart from ImPACT Impulse Control at the third follow-up) on the ImPACT and WMS-III neurocognitive measures at each test interval, and to make comparatively less pronounced improvement

between test intervals on the ImPACT and WMS-III neurocognitive measures compared with the Matched Control group.

Symptom Measures

On the *independent* analyses between the Concussed Rugby group and Matched Control group it was revealed that the Concussed Rugby group obtained higher Symptom Composite scores than the Matched Control group at each test interval on the ImPACT Symptom Scale, with the difference being significant at the baseline, first and second follow-up intervals, and approaching significance at the third follow-up interval. At the baseline interval the Concussed Rugby group, compared with the Matched Control group, evidenced higher scores for 19 of the 22 individual symptom measures, of which the difference was significant for 3 individual symptom measures (nervousness, difficulty concentrating and difficulty remembering) and approaching significance for one individual symptom measure (headache). At the first follow-up interval the Concussed Rugby group, compared with the Matched Control group, evidenced higher scores for 18 of the 22 individual symptom measures, with the difference being significant for 5 individual symptom measures (headache, fatigue, drowsiness, sensitivity to light and slowed down) and approaching significance for 3 individual symptom measures (nausea, sleeping more and numbness). At the second follow-up interval the Concussed Rugby group, compared with the Matched Control group, evidenced higher scores for 13 of the 22 individual symptom measures, with differences approaching significance for 5 individual symptom measures (headache, sensitivity to light, nervousness, mentally foggy and difficulty remembering). At the third follow-up interval the Concussed Rugby group, compared with the Matched Control group, evidenced higher scores for 9 of the 22 individual symptom measures (the Matched Control group reported no symptoms), with the difference being significant for one individual symptom measure (fatigue) and approaching significance for 2 individual symptom measures (headache and sensitivity to light). At the fourth follow-up interval no significant differences were revealed between the Concussed Rugby group and Matched Control group.

On the *dependent* analyses for the Concussed Rugby group and for the Matched Control group, the repeated measures ANOVAs for the ImPACT Symptom Scale revealed that the group by season interaction effect was significant for one individual symptom measure (headache), and approaching significance for the Symptom Composite measure.

A significant effect for season was revealed for the Symptom Composite measure, in that there was an overall decline in symptom reports for both the Concussed Rugby group and Matched Control group at the four follow-up intervals versus the baseline interval. The exception was for the first-follow-up interval, when the Concussed Rugby group's Symptom Composite score was equivalent to the baseline score. However, the Concussed Rugby group's overall symptom reports declined significantly at the third and fourth follow-up intervals. Both a significant group by season interaction effect and significant effect for season were revealed for the headache symptom measure, in that the Concussed Rugby group obtained a significantly higher mean score for this symptom at the first follow-up interval, whereas the Matched Control group did not report on the headache symptom measure at the baseline and first three follow-up intervals. A significant effect for season was revealed for the mentally foggy symptom measure, in that the Concussed Rugby group had higher scores than the Matched Control group on this symptom measure at the baseline and first two follow-up intervals. The effect for season was approaching significance for the slowed down symptom measure, in that the Concussed Rugby group obtained a significantly higher mean score on this symptom measure at the first follow-up interval.

The dependent analyses of group means for the ImPACT Symptom Scale at the four follow-up intervals versus the baseline interval for the Concussed Rugby group revealed significant findings or findings approaching significance for 2 significantly higher individual symptom scores (headache and slowed down), 3 significantly lower individual symptom scores (sadness, nervousness, and difficulty remembering), and 2 lower individual symptom scores approaching significance (trouble falling asleep and irritability) at the first follow-up interval. No significant findings but 4 lower individual symptom scores approaching significance were revealed (sleeping more, feeling more emotional, difficulty concentrating and difficulty remembering) at the second follow-up interval. Significantly lower scores for the Symptom Composite and 3 individual symptom scores (sadness, nervousness and difficulty remembering) and 4 lower individual symptom scores approaching significance were revealed (sleeping less, irritability, mentally foggy and difficulty concentrating) at the third follow-up interval. Significantly lower scores on the Symptom Composite and 6 individual symptom scores (fatigue, trouble falling asleep, sadness, nervousness, difficulty concentrating and difficulty remembering) and 3 lower individual symptom scores approaching significance

were revealed (sleeping more, sleeping less and irritability) at the fourth follow-up interval for the Concussed Rugby group. For the Matched Control group no significant findings were revealed for the Symptom Composite measure or for any specific individual symptom measures, but findings approaching significance were revealed on 5 lower individual symptom scores (fatigue, trouble falling asleep, sleeping less, irritability and difficulty remembering) at the first follow-up interval; on the lower Symptom Composite and 3 individual symptom scores (fatigue, irritability and difficulty remembering) at the second follow-up interval; on the lower Symptom Composite and 5 individual symptom scores (fatigue, trouble falling asleep, sleeping less, irritability and difficulty remembering) at the third follow-up interval; on 2 lower individual symptom scores (trouble falling asleep and sleeping less)at the fourth follow-up interval.

In summary, taking into account significant results as well as trends, both the independent and dependent analyses for the ImPACT symptom measures revealed that the Concussed Rugby group evidenced significantly higher symptom reports than the Matched Control group at the baseline and four follow-up intervals, and the overall symptom reports decreased at the four follow-up intervals versus the baseline interval for both groups, except for the Concussed Rugby group at the first follow-up interval.

CHAPTER NINE: DISCUSSION

As an introduction to the discussion, the broad aims of this study and the significance of the research questions will be presented. Following this, a discussion of the results will be presented in turn for the General Rugby and General Control groups, and for the Concussed Rugby and Matched Control groups. In all instances, the neurocognitive measures will be evaluated first followed by the symptom measures, in respect of the independent *t*-test comparisons of group means followed by the dependent *t*-test comparisons of group means. The final sections include an evaluation of the study, followed by a discussion on overall implications of the research outcomes.

9.1 BROAD OUTLINE OF THIS STUDY

Only a few known published sport-related MTBI studies investigating chronic or acute MTBI sequelae among adult athletes have used a combination of both computerised testing designed for sport-related MTBI and traditional pencil and paper neurocognitive tests, of which two studies relate specifically to rugby union (e.g., Bruce & Echemendia, 2009; Collie, Makdissi et al., 2006; Gardner et al., 2010; Makdissi et al., 2001; Shuttleworth-Edwards, Smith et al., 2008). However, only three of these studies reported on the symptoms, but did not use control groups of noncontact sport athletes for comparative purposes (Collie, Makdissi et al., 2006; Gardner et al., 2010; Makdissi et al., 2001). This provided the impetus for the present study to use a quasi-experimental non-equivalent design that incorporated noncontact sport controls, demographically matched on gender, age, language, race, IQ, education and years of sport participation. In addition this study used traditional and computerised measures for pretesting and posttesting purposes, to measure both neurocognitive and symptom complaints. Finally, only one prior rugby study included an investigation of the acute neurocognitive and symptom complaints among rugby union players, however, the sample of concussed rugby players was small, and preseason symptoms were not reported (Shuttleworth-Jordan et al., 1993). Therefore, in a non-equivalent quasi-experimental design, the present study investigated the neurocognitive and symptom profiles of *both* concussed and nonconcussed rugby union players in comparison with demographically equivalent controls over one season, utilising *both* the ImPACT computerised test and the WMS-III Verbal Paired Associates

and Visual Reproduction subtests. Broadly, the objective of this multi-faceted research design was to investigate the pattern of chronic and acute neurocognitive and symptomatic effects from concussive and subconcussive injuries at a provincial level of rugby, over one rugby season.

Specifically in order to achieve this objective the following was carried out. A sample of 97 provincial, national and academy male rugby players and 73 noncontact sportsmen participated in the study. The rugby players were divided into two groups. The General Rugby group not reporting any MTBI during the season, and the Concussed Rugby group diagnosed with MTBI during the season. There was a General Control group and a Matched Control group for comparison purposes with the General Rugby group and Concussed Rugby group, respectively. Following exclusion criteria (as described in Chapter 7, section 7.1.3), the final sample groups were General Rugby versus General Control (n = 54 versus n = 37), and Concussed Rugby versus Matched Control (n = 17 versus n = 17). Demographic variables on which the comparison rugby and control groups were matched, included age, education (years), estimated Full Scale IQ, Vocabulary scaled score, Matrix Reasoning scaled score, years of primary sport participation, language and race. There was a significant difference on the Matrix Reasoning subtest, in that the General Control group obtained a significantly higher score than the General Rugby group. However, this was an *isolated* finding in that there was equivalence on this parameter for the Concussed Rugby and Matched Control group. The number of prior reported MTBIs differed significantly between the rugby and control groups, with both rugby groups having obtained significantly more MTBIs than the control groups. *Importantly, this serves strongly to confirm the non-equivalence of the comparative groups, in terms of the key variable of exposure to MTBI.*

All participants were tested at baseline (preseason for rugby players). The General Rugby and General Control groups were tested again at the end of season. The Concussed Rugby and Matched Control groups were tested at four follow-up intervals following MTBI in the rugby players (averaging 4, 10, 16 and 24 days post MTBI). The neurocognitive test measures included the ImPACT Version 3.0 computerised test battery and the traditional pencil and paper Wechsler Memory Scales – Third Edition's (WMS-III) Verbal Paired Associates and Visual Reproduction subtests. All measures were administered at each test occasion, except the WMS-III VPA and VR measures at the second and third follow-up,

in an attempt to minimise practice effects. Independent *t*-test analyses were conducted on the group means of the General Rugby sample versus the General Control sample at the baseline and end of season test intervals, and of the Concussed Rugby sample versus the Matched Control sample at baseline and at the four postconcussion follow-up test intervals, to investigate differences in neurocognitive functioning and symptoms reported. Dependent *t*-test analyses were conducted on the group means of the General Rugby sample and the General Control sample at the baseline versus the end of season test intervals, and of the Concussed Rugby sample and the Matched Control sample at baseline versus the four post MTBI follow-up test intervals, to investigate prospective differences in neurocognitive functioning and symptoms reported. The Bonferroni Correction was implemented for the neurocognitive analyses to reduce the risk of Type I error, due to the use of multiple measures. Accordingly, at the 5% level of significance, $p = \leq .025$ for the neurocognitive measures, and $p = \leq .05$ for the symptom measures. For further interpretive purposes, results were considered to be approaching significance on all measures if *p* did not reach the 10% level ($p = < .01$). In keeping with APA journal format, specific *p* values delineated in the results section will not be provided in the discussion.

Broadly it was hypothesised that on both the cross-sectional independent analyses and the prospective postconcussion analyses there would be indications of neurocognitive vulnerability in the rugby players compared with noncontact sport controls, and higher symptom reporting amongst rugby players compared with noncontact sport controls. The detailed theoretical and statistical hypotheses appear in Chapter 6 (section 6.5.2) and Chapter 7 (section 7.3), respectively. Against the background of these hypotheses, the findings of the multi-level study will be discussed.

9.2 INTERPRETATION OF FINDINGS FOR THE GENERAL RUGBY AND GENERAL CONTROL GROUPS

This section discusses the results for the General Rugby and General Control groups. Baseline and end of the season findings across the neurocognitive measures are discussed first, followed by the baseline and end of season findings for the symptom measures. In each section, independent *t*-test comparisons between groups are discussed first, followed by the dependent t-test comparisons.

9.2.1 Interpretation of Findings on the Neurocognitive Measures

Independent *t*-test Comparisons

The independent analyses on the ImPACT and WMS-III neurocognitive test measures at the *baseline test interval* revealed no significant differences between the General Rugby and General Control groups. However, the General Rugby group exhibited poorer performance than the General Control group on three of the five ImPACT measures (apart from Verbal Memory and Reaction Time), with poorer performance on the Impulse Control measure approaching significance, and on five of the nine WMS-III measures with poorer performance on the VPA Percent Retention measure approaching significance. In contrast to these results, the General Rugby group's stronger performance than the General Control group on the ImPACT Reaction Time measure was approaching significance. Overall, however, the baseline results that approached significance and the general trend provide some tentative support for poorer performance for the General Rugby group relative to the General Control group.

The independent analyses on the ImPACT and WMS-III neurocognitive test measures at the *end of season test interval*, revealed significant differences for the ImPACT Impulse Control composite and the WMS-III VPA Percent Retention measures, in the direction of poorer performance for the General Rugby group, compared with the General Control group. Furthermore, the General Rugby group's poorer performance than the controls, on the ImPACT Visual Motor Speed and the WMS-III VPA I, VPA First Recall and VPA II measures, were approaching significance. The overall trend at the end of season interval revealed that the General Rugby group exhibited poorer performance than the controls on three of the five ImPACT measures and on eight of the nine WMS-III measures. Commensurate with the baseline outcome, these significant findings, findings approaching significance and the overall trend, provide support for generally poorer performance for the General Rugby group relative to the General Control group.

Specifically, in terms of the findings for the ImPACT test composites, the General Rugby group's stronger baseline performance than the controls for the ImPACT Reaction Time measure that was approaching significance, is an isolated finding contrary to the hypothesis that rugby would perform more poorly than controls, and contrary to what has been documented in other research in terms of the sensitivity of Reaction Time to sport-

related MTBI (e.g., Shuttleworth-Edwards, Border et al. 2004). Importantly, however, at the end of season interval there was equivalent performance for the General Rugby and General Control groups on the Reaction Time measure, such that this implicates cognitive vulnerability for the General Rugby group seen in other studies (e.g., Collie, Makdissi et al., 2006; Collins, Field et al., 2003; Iverson et al., 2004b; Makdissi et al., 2001; Shuttleworth-Edwards, Border et al. 2004; Sosnoff et al., 2007), in that they did not improve with practice over time as much as the General Control group (this aspect will be more fully elucidated upon in the dependent analyses discussion). The General Rugby group's poorer performance than the controls on the ImPACT Impulse Control measure that was approaching significance at the baseline interval and significant at the end of season interval, is suggestive of disinhibition, inattention and poor impulse control, typically described as MTBI sequelae (e.g., Lezak et al., 2004), further implicating cognitive vulnerability for the rugby players, particularly at the end of season interval, in comparison to the controls. Finally, the General Rugby group's poorer performance than the controls, on the ImPACT Visual Motor Speed measure, was approaching significance at the end of season. Several studies investigating the chronic effects of rugby-related MTBI have found deficits or a lack of practice effects for processing speed measures (e.g., 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). The previous rugby study using the ImPACT program, also found poorer end of season performance on measures of visuomotor speed, for university level rugby players in comparison to noncontact sport controls (e.g., Shuttleworth-Edwards, Smith et al., 2008).

In respect of the Wechsler Memory Scale tests, the General Rugby group's poorer performance than the controls on the WMS-III VPA Percent Retention measure, that was approaching significance at the baseline interval but was significant at the end of season interval, is suggestive of verbal memory difficulties. The significantly lower percentage of correctly learned and retained word pair associations is suggestive of neurocognitive compromise for the rugby players, in comparison to the controls, given that delayed recall tasks are sensitive to subtle MTBI neurocognitive deficits (Lezak et al., 2004). Furthermore, at the end of the season, the General Rugby group obtained lower scores than the controls on all the other VPA measures that, apart from VPA Recognition, were approaching significance. In view of there having beeen no significant differences

between the groups on the WAIS-III Vocabulary subtest scores (as an estimate of verbal IQ), suggests that these lower scores on the verbal memory measures are unlikely to be due to differences in verbal potential between the groups, and can more likely be attributed to the effects of rugby-related MTBI. Verbal memory difficulties for these rugby players, is in keeping with other MTBI related findings. Stuss et al. (1985), found significantly lowered scores for recovered MTBI patients on the WMS Paired Associates - delayed recall, distinguished them from the uninjured control participants (Lezak et al., 2004). Another study found, in comparison with the noncontact sport control groups, that a greater percentage of the national open rugby players obtained lower scores on the WMS Associate Pairs, the under-21 rugby players on the WMS Associate Pairs Hard-Immediate condition, and the school rugby players on the WMS Associate Pairs Delayed Recall condition (Shuttleworth-Edwards, Border et al., 2004). Furthermore, concussed female hockey players have shown a more pronounced decline on verbal than on non-verbal memory measures (Hatfield et al., 2004).

Overall therefore, results for the independent analyses, between the General Rugby and General Control groups, at both the baseline and end of season intervals viewed in conjunction with the relevant literature, are supportive of the first two hypotheses posed for this thesis (in Chapter 6, section 6.5.2 and Chapter 7, section 7.3), that the General Rugby group relative to the General Control group, would demonstrate poorer performance on the neurocognitive tests at both baseline and the end of season test intervals, due to long-term exposure to concussive and subconcussive events historically (baseline interval) together with an overlay of unreported concussive and subconcussive events sustained during the 2005 rugby season (end of season interval).

Dependent *t*-test Comparisons

Impact Measures

The repeated measures ANOVAs for the *end of season test interval versus the baseline test interval*, for the General Rugby and General Control groups, on the ImPACT neurocognitive measures, revealed a significant group by season interaction effect and a significant effect for season for the Reaction Time measure. A significant effect for season was also found for the Visual Motor Speed measure.

The dependent analyses for the ImPACT neurocognitive composites at the end of season interval versus baseline interval, found that both the General Rugby and General Control groups made significant improvements at the end of season on the ImPACT Reaction Time measure. However, the significant group by season effect implicated a substantially more pronounced improvement for the General Control group, for the reason that the General Rugby group did not sustain the superior performance evidenced at the baseline interval. As discussed in Chapter 6, slowed reaction time has proven a sensitive indicator of MTBI in several studies (e.g., Collie et al., 2006; Collins, Field et al., 2003; Cremona-Meteyard & Geffen, 1994; Downs & Abwender, 2002; Gaetz et al., 2000; Iverson et al., 2004b; Maddocks & Saling, 1996; Makdissi et al., 2001; Sosnoff et al., 2007). Specifically, both the General Rugby and General Control groups made significant improvements on the Visual Motor Speed composite at the end of season versus baseline intervals, with a trend of a greater improvement for the General Rugby group than the controls, although the group by season interaction effect failed to reach significance.

The absence of a reduced practice effect for the General Rugby group is in contrast to the Shuttleworth-Edwards, Smith et al. (2008) study, in which the university rugby group performed more poorly on the Visual Motor Speed composite and did not benefit from practice, and the control group made a very subtle improvement at the end of season versus baseline. The ImPACT program is designed for multiple repeat testing, and has multiple random forms to minimise practice effects (Shuttleworth-Edwards, Smith et al., 2008; Maroon et al., 2002). However, on repeat testing with an average retest interval of less than a week, a small practice effect was found for the Visual Motor Speed composite, which the researchers suggested might disappear over a longer retest interval (Iverson et al., 2003). It appears that a practice effect was apparent in this study, after a retest interval of 8.2 months, for both the General Rugby and General Control groups. Compared with the university cohort in the Shuttleworth-Edwards, Smith et al. (2008) study, for this elite sample of provincial level sportsmen who can be considered an exceptionally high functioning group. This factor, together with the fact that provincial players are exposed to significant input of hand-eye coordination practice over a season that would not apply to the same extent in respect of university players, may account for this contrasting finding in respect of the Visual Motor Speed composite. However, although practice effects did not serve to differentiate rugby from controls in the present study, it was not entirely spared when considering that for the independent *t*-tests, the rugby group's

poorer end of season performance on this measure was approaching significance (as discussed in the previous subsection).

Both the General Rugby group and the General Control group obtained poorer scores on the Impulse Control measure at the end of season interval versus the baseline interval. The General Rugby group's deterioration approached significance, whereas the General Control group's performance deteriorated only marginally, although the group by season interaction effect failed to reach significance. The poorer performance by the General Rugby group, at the end of season versus baseline, suggests increased neurocognitive disinhibition and impulsivity. This is suggestive of possible subconcussive effects, in that MTBI sequelae commonly include deficits in attention and concentration (Lezak et al., 2004). Furthermore, the General Control group's improved performance on the ImPACT Verbal Memory measure was approaching significance at the end of season interval versus the baseline interval. In contrast, the General Rugby group's performance on the Verbal Memory measure deteriorated marginally at the end of season, although the group by season interaction effect failed to reach significance. Lezak et al. (2004), consider that memory measures are more vulnerable to practice effects than other neurocognitive measures, despite the fact that Iverson et al. (2003) found that repeat testing with an average retest interval of less than a week, did not result in an increase in scores on the ImPACT Verbal and Visual Memory composites. In this instance, the control group's benefit and the General Rugby group's relative failure to benefit from practice on the ImPACT Verbal Memory composite, is suggestive of subtle memory difficulties for the rugby players.

WMS Measures

The repeated measures ANOVAs did not reveal any significant group by season interaction effects for any of the WMS-III VPA and VR measures, but significant effects for season were revealed for six measures, including: VPA I, VPA First Recall, VPA II, VR II, VR Recognition and VR Percent Retention. Specifically, the dependent analyses for the WMS-III measures at the end of season interval versus the baseline interval, found significant improvements by both groups on these same six WMS-III measures. The trend was for less robust improvement by the General Rugby group on five of these six measures, in comparison with the controls (VPA First Recall, VPA II, VR II, VR Recognition and VR Percent Retention). On the sixth measure (VPA I), both groups

attained an equivalent level of improvement. Furthermore, for the remaining three WMS-III measures (VPA Recognition, VPA Percent Retention and VR 1), improvement at the end of season interval by the General Rugby group was less robust than the improvement by the General Control group, although not reaching significance. The less robust improvement by the General Rugby group is possibly suggestive of subtle visual and verbal memory difficulties.

However, it appears that all nine WMS-III measures are subject to both ceiling and practice effects, even after an average interval of 8.2 months. It was anticipated that the control participants would make improvements at follow-up test intervals, based on the assumption of their being comparatively neurocognitively healthy and, therefore, able to benefit from practice. It was anticipated that the rugby players would reveal the absence or relative absence of practice effects, which would suggest neurocognitive impairment, shown as a decrement in learning (Knight, 1992). Practice effects occur for most memory tests (McCaffrey, Duff & Westervelt, 2000). A smaller practice effect with multiple repeat testing has previously been reported for the WMS-R Visual Reproduction-R in comparison with the remaining WMS-R subtests, and gains made on retesting were inconsequentially small in both the immediate and delayed recall conditions (e.g., Lezak, et al., 2004; Reite, Cullum, Stocker, Teale, & Kozora, 1993). In contrast, this study found all of the WMS-III VPA and VR subtest measures were subject to practice effects. However, this was more evident for the General Control group than for the General Rugby group, and taken together with similar indications for the ImPACT test Reaction Time, Verbal Memory and Impulse Control composites, provides further support for practice effects being more obvious for the controls than for the rugby players.

In summary, overall findings for the dependent analyses revealed a tendency of improved performance by both groups between the end of season versus the baseline intervals, in respect of both the ImPACT and the WMS measures. However, the general tendency was for a less pronounced practice effect for the General Rugby group than for the General Control group, implicating neurocognitive vulnerability in the rugby group, which is consistent with similar indications of cognitive vulnerability for the rugby group in the Shuttleworth-Edwards, Smith et al. (2008) study. There appears to have been only one other study, comparing football players prospectively over a season, being that of Miller, Adamson, Pink and Sweet (2007), which in contrast did not support cognitive

vulnerability however their research did not have a control group so they were not in a position to identify reduced practice effects. Therefore, taking the overall results of the dependent analyses, in conjunction with the only available comparative literature findings, provide support for this study's third and fourth hypotheses (in Chapter 6 section 6.5.2 and Chapter 7 section 7.3) viz., that, the General Rugby group (in contrast to the General Controls) would demonstrate poorer performance on the neurocognitive tests at the end of season test interval compared to the baseline test interval, in support of deleterious neurocognitive effects in the form of either poorer performance, or the absence of a practice effect when it is expected, due to long-term exposure to concussive and subconcussive events historically, and an overlay of unreported concussive and subconcussive events sustained during the 2005 rugby season.

Summary of Neurocognitive Findings for the Independent and Dependent Analyses

Overall findings for the General Rugby group and the General Control group, on the neurocognitive measures, were mostly in support of the hypotheses one to four (in Chapter 6 section 6.5.2 and Chapter 7 section 7.3), that broadly suggest that the General Rugby Group would reveal more cognitive vulnerability than the General Control Group due to long-term exposure to concussive and subconcussive events historically and during the 2005 season, either because of poorer performance at baseline and/or end of season testing (independent analyses), or the failure to benefit from practice to the same extent as the General Controls (dependent analyses). Albeit subtle in many instances, the highly consistent direction of the findings across both forms of statistical analysis (cross-sectional independent and prospective dependent) in the expected direction implicated from prior MTBI research, both generally as well as in the sports MTBI literature, adds potency to the findings in accordance with the hypotheses (as delineated in Chapter 6 section 6.5.2 and Chapter 7 section 7.3). Although it is understood that on the basis of the present research, it is not possible to differentiate between the extent to which current and past concussive and subconcussive events are contributing to this picture.

9.2.2 Interpretation of Findings on the Symptom Measures

Independent *t*-test Comparisons

The independent analyses between the General Rugby and General Control groups on the ImPACT Symptom Scale at the *baseline* test interval, revealed that the General Rugby

group obtained a significantly higher Symptom Composite score, having evidenced higher scores for 17 of the 22 individual symptoms, and the difference was significant for 5 symptoms (trouble falling, sleeping more, sadness, feeling more emotional and difficulty remembering), and approached significance for 2 symptoms (headache and mentally foggy). The independent analyses between the General Rugby and General Control groups on the ImPACT Symptom Scale at the *end of season interval* revealed that the General Rugby group obtained a significantly higher Symptom Composite score, having evidenced higher scores for 13 of the 22 individual symptoms, with the difference being significant for 2 symptoms (fatigue and trouble falling asleep), and approaching significance for a further 2 symptoms (numbness and difficulty concentrating).

Based on the evidence of two studies, it could be expected that the rugby group would report more symptoms than the control group. In comparison to control groups, Tysvaer (1992), found soccer players reported 30% more chronic symptoms and Shuttleworth-Edwards, Border et al. (2004), found rugby players reported 20 to 30% more neurobehavioural symptoms. Shuttleworth-Edwards, Border et al. (2004), found both adult rugby groups reported difficulty with sustained attention and memory, yet the open national rugby group reported less on the key physical and cognitive postconcussion symptoms (e.g., headache, limb weakness and visual disturbances), which was considered a tactic to avoid exclusion from sport participation. In this study the rugby group reported more typical postconcussion symptoms at baseline than the controls (feeling more emotional, difficulty remembering, headache and mentally foggy).

Overall findings for the baseline and end of season comparisons between the General Rugby and General Control groups, revealed significantly higher symptom reports for the General Rugby group than for the General Control group, with no results in the opposite direction. These findings show accordance with the literature and are suggestive of deleterious neuropsychological outcomes for the General Rugby group compared with the General Control group, and are in support of the fifth and sixth hypotheses (in Chapter 6, section 6.5.2 and Chapter 7, section 7.3), viz., that the General Rugby group relative to the General Control group, would demonstrate higher symptom reports on the Symptom Scale at the baseline and end of season test intervals, as a result of long-term exposure to subconcussive and concussive events historically, and such unreported events during the 2005 rugby season.

Dependent *t*-test Comparisons

The repeated measures ANOVAs for the *end of season test interval versus the baseline test interval*, for the General Rugby and the General Control groups, on the ImPACT Symptom Scale revealed a significant effect for season for the Symptom Composite measure, in that there was a significant overall decline in symptom reports at the end of season interval versus the baseline interval for both groups, and the group by season interaction effect on this measure was approaching significance, indicating a more robust decline in symptom reports for the General Rugby group than for the General Control group. More specifically, the dependent *t*-test analyses for the ImPACT Symptom Scale at the end of season versus baseline interval, found that the General Rugby group, revealed significantly lower scores for the Symptom Composite measure and for 6 individual symptoms (trouble falling asleep, sleeping more, sleeping less, irritability, mentally foggy and difficulty remembering), and lower scores approaching significance for a further 2 symptoms (headache and sensitivity to light). However, higher symptom scores for the General Rugby group were revealed on 5 symptoms at the end of season interval versus the baseline interval (fatigue, drowsiness, sensitivity to noise, numbness and difficulty concentrating).

In contrast to this, the control group did not reveal an increase in symptom scores for any of the symptoms, and in fact mainly reported zero symptoms (all except two symptoms, viz., trouble falling asleep and difficulty concentrating). Therefore, although the findings in respect of worsening symptom reports for the rugby players were not significant, they provide some reasonably compelling support for the presence of deleterious neuropsychological effects, given the absence of *any* increase in symptom scores for the control group, for whom only two symptoms were reported at the end of the season, and for which neither score was higher than the baseline score. These are findings therefore, that can be seen to provide support for the seventh and eighth hypotheses (in Chapter 6, section 6.5.2 and Chapter 7, section 7.3), viz., of increased symptoms in the General Rugby group that were not in evidence for the General Control group, due to long-term exposure to concussive and subconcussive events historically, and an overlay of unreported concussive and subconcussive events sustained during the 2005 rugby season.

Summary of Symptom Findings for the Independent and Dependent Analyses

Taking the independent and dependent analyses together, overall findings for the General Rugby group and the General Control group, on the symptom measures, were mostly in support of the hypotheses five to eight (in Chapter 6 section 6.5.2 and Chapter 7 section 7.3), that broadly suggest that the General Rugby Group would reveal more symptoms than the General Control Group due to long-term exposure to concussive and subconcussive events historically and during the 2005 season, either because of predominantly increased symptom reports at baseline and/or end of season testing (independent analyses), or the overriding increase in symptom reports between the two test intervals in comparison to the General Controls (dependent analyses).

9.3 INTERPRETATION OF FINDINGS FOR THE CONCUSSED RUGBY AND MATCHED CONTROL GROUPS

This section discusses the results for the Concussed Rugby and Matched Control groups. The first section discusses findings for the baseline and the four postconcussion follow-up test intervals, on the neurocognitive measures. The composite scores on the ImPACT computerised program are reported for the total five test intervals. The scores for the WMS-III Verbal Paired Associates (VPA) and Visual Reproduction (VR) traditional tests are reported for the baseline, first and fourth follow-up intervals. The second section discusses findings for the baseline and the four postconcussion follow-up test intervals, on the ImPACT symptom measures. In each section, the interpretation of results across the measures and in accordance with the hypotheses, will fall under the subheadings of the independent *t*-test comparisons of comparative group means and the dependent t-test comparisons for each group's means across the test occasions.

9.3.1 Interpretation of Findings on the Neurocognitive Measures

Independent *t*-test Comparisons

The independent analyses on the ImPACT and WMS-III neurocognitive test measures at the *baseline test interval*, revealed no significant differences between the Concussed Rugby and Matched Control groups. However, the Concussed Rugby group exhibited poorer performance than the Matched Control group on three of the five ImPACT measures (apart from Visual Memory, and an equivalent mean score on Reaction Time),

with poorer performance on the Impulse Control measure approaching significance and on seven of the nine WMS-III measures, with poorer performance on the VPA I, VPA II and VPA Recognition measures approaching significance.

The independent analyses on the ImPACT and WMS-III neurocognitive test measures at the first three *follow-up test intervals*, revealed no significant differences between the Concussed Rugby and Matched Control groups. However, at the fourth follow-up interval, a significant difference was revealed on the WMS-III VPA I, in the direction of poorer performance for the Concussed Rugby group compared with the Matched Control group. Differences that were approaching significance were revealed for six measures at the first follow-up interval (ImPACT Reaction Time and Impulse Control, and WMS-III VPA II, VPA Recognition, VR II and VR Percent Retention), and for three measures at the fourth follow-up interval (WMS-III VPA First Recall, VPA II, and VR Recognition), in the direction of poorer performance for the Concussed Rugby group.

Taken together, the significant result at the fourth follow-up interval, and for those approaching significance for the independent analyses at baseline and for the four follow-up test intervals (especially the baseline and fourth test interval), indicated vulnerability notably in the areas of the ImPACT Reaction Time and Impulse Control and several of the WMS-III verbal and visual memory measures. This provides support for the first and second hypotheses (in Chapter 6, section 6.5.2 and Chapter 7, section 7.3), viz., that the Concussed Rugby group relative to the Matched Control group, would demonstrate poorer performance on the neurocognitive tests due to long-term exposure to concussive and subconcussive events historically (baseline interval) together with an overlay of reported concussive events sustained during the 2005 rugby season (postconcussive intervals). Although the findings are subtle, they are nevertheless notable, considering that the first follow-up testing took place on average 4 days post MTBI (range 2-10 days). This is a longer period post MTBI than that of other studies that found significant results for the sensitive reaction time measure, within 48 hours and 72 hours of MTBI (e.g., Sosnoff et al., 2007; Makdissi et al., 2001). Moreover, significant effects that pertain to individuals, tend to be diluted in group analyses.

Dependent *t*-test Comparisons

The repeated measures ANOVAs for the four follow-up test intervals versus the baseline test interval, for the Concussed and Matched Control groups, revealed group by season interaction effects that were approaching significance for the ImPACT Reaction Time and WMS-III VR I measures. Significant effects for season were revealed for two ImPACT measures (Visual Motor Speed and Reaction Time), and for six WMS-III measures (VPA I, VPA First Recall, VPA II, VR II, VR Recognition, and VR Percent Retention. Specifically, for the dependent *t*-tests analyses, for the Reaction Time measure, dependent analyses revealed that the Concussed Rugby group's performance compared with the baseline interval was slower and approaching significance at the first follow-up interval, was equivalent to baseline performance at the second follow-up interval, and improved significantly at the third follow-up interval with this improvement maintained at the fourth follow-up interval. In contrast, for the Matched Control group, performance at all four follow-up intervals was faster on the Reaction Time measure than that of their baseline performance, suggestive of practice effects, with a significant improvement at the fourth follow-up interval. Several published studies have found reaction time measures sensitive to the effects of MTBI (e.g., Collie, Makdissi et al., 2006; Collins, Field et al., 2003; Cremona-Meteyard & Geffen, 1994; Iverson et al., 2004b; Maddocks & Saling, 1996; Majerske et al., 2008; Makdissi et al., 2001; Parker et al., 2007; Sosnoff et al., 2007). An implication for the present study is confirmation that the ImPACT Reaction Time measure is sensitive to the effects of concussion. Although the Concussed Rugby group's score at the first follow-up interval did not slow significantly from baseline level, it improved significantly at the third and fourth follow-up test intervals. This finding is similar to that of Lovell, Collins, Iverson et al. (2004), who found that high school athletes' reaction time slowed slightly but not significantly within 36 hours of MTBI, and improved significantly on baseline performance six days post MTBI. Furthermore, Parker et al. (2007) found that collegiate athletes' reaction times improved significantly day 5 post MTBI in comparison to their slower performance day 2 post MTBI. Therefore, had the Concussed Rugby group all been tested within two days of MTBI, in all probability their reaction times would have been significantly slower than their baseline performance.

Further, the dependent *t*-test analyses in respect of the ImPACT measures, revealed that the Concussed Rugby group's improved performance on the Visual Motor Speed measure in comparison to baseline was less robust than that of the Matched Control group,

implicating a less pronounced benefit from practice, particularly at the first two follow-ups. This is suggestive of the ImPACT Visual Motor Speed measure being sensitive to cognitive vulnerability following MTBI. Comparative visual motor speed difficulties for these rugby players, is in keeping with other MTBI related findings, in that several studies have found reduced speed or a lack of benefit from practice on processing speed measures at follow-up MTBI testing (e.g., Barth et al., 1989; Covassin et al., 2008; De Monte, Geffen, May et al., 2005; Echemendia et al., 2001; Hinton-Bayre et al., 1997, 1999; Hinton-Bayre & Geffen, 2002; Iverson et al., 2004b; Lovell, Collins, Iverson et al., 2004; Macciocchi et al., 1996; Makdissi et al., 2001, 2010; Mathias et al., 2004; McCrea et al., 2003; Mihalik et al., 2005; Parker et al., 2007; Peterson et al., 2003; Wilberger et al., 1991), and including one rugby-related MTBI study (Shuttleworth-Jordan, Puchert et al., 1993).

For the WMS-III measures, the Concussed Rugby group's improved performance on baseline scores was less robust than that of the Matched Control group, particularly on all four VR measures at the first and fourth follow-up intervals. The dependent *t*-test analyses revealed that the Concussed Rugby group's performance (for two WMS-III measures), lowered marginally on the VPA Recognition at the first follow-up interval, and on the VR I at the fourth follow-up interval (despite having improved marginally at the first follow-up interval), compared with baseline, although these findings were not significant. In contrast, the Matched Control group's performance reached the ceiling for VPA Recognition at the baseline and first and fourth follow-up intervals, and on the VR I their improvement at the first follow-up was reaching significance and was significant at the fourth follow-up interval, which again indicates practice effects being more obvious for the Matched Controls than for the Concussed Rugby group. These findings, suggest that the concussed rugby players experienced difficulty with their recognition of correct word-pairings and short-term visual memory for drawing designs. MTBI appeared to have a delayed effect on short-term visual memory, in that the fourth follow-up test interval averaged 24 days post MTBI.

In summary, overall findings for the dependent analyses revealed improvements by both the Concussed Rugby and Matched Control groups between the four follow-up intervals versus the baseline interval on most of the ImPACT and WMS measures. However, the Concussed Rugby group regularly revealed less pronounced practice effects than the

Matched Control group, that in conjunction with their poorer performance on the ImPACT Reaction Time and WMS VR I measures, and comparative findings from the literature, demonstrate support for the third and fourth hypotheses (in Chapter 6, section 6.5.2 and Chapter 7, section 7.3), viz., that, the Concussed Rugby group (in contrast to the Matched Controls) would demonstrate poorer performance on the neurocognitive tests at the postconcussion test intervals compared to the baseline test interval, in support of deleterious neurocognitive effects in the form of either poorer performance, or the absence of a practice effect when it is expected, due to long-term exposure to concussive and subconcussive events historically, and the overlay of acute effects of reported concussive events.

Summary of Neurocognitive Findings for the Independent and Dependent Analyses

Taking the independent and dependent analyses together, overall findings for the Concussed Rugby group and the Matched Control group, on the neurocognitive measures, were mostly in support of the hypotheses one to four (in Chapter 6 section 6.5.2 and Chapter 7 section 7.3), that broadly suggest that the Concussed Rugby Group would reveal more cognitive vulnerability than the Matched Control Group due to long-term exposure to concussive and subconcussive events historically and concussive events during the 2005 season, either because of poorer performance at baseline and/or follow-up (independent analyses), or the failure to benefit from practice to the same extent as the Matched Controls (dependent analyses). Albeit subtle in many instances, the highly consistent direction of the findings across both forms of statistical analysis (cross-sectional independent and prospective dependent) in the expected direction implicated from prior MTBI research, both generally as well as in the sports MTBI literature, adds potency to the findings.

9.3.2 Interpretation of Findings on the Symptom Measures

Independent *t*-test Comparisons

The independent analyses between the Concussed Rugby and Matched Control groups on the ImPACT Symptom Scale at the *baseline* interval, revealed that the General Rugby group obtained a significantly higher Symptom Composite score, having evidenced higher scores for 19 of the 22 individual symptoms, with the difference being significant for 3 of these symptoms (nervousness, difficulty concentrating and difficulty

remembering), and approaching significance for 1 symptom (headache). In contrast, the Matched Control group obtained a higher, but not significant, score on only one symptom (slowed down). The independent analyses between the Concussed Rugby and Matched Control groups on the ImPACT Symptom Scale at the *four follow-up intervals*, revealed that the Concussed Rugby group obtained significantly higher Symptom Composite scores than the Matched Control group at the first and second follow-up intervals, and a higher Symptom Composite score approaching significance at the third follow-up interval. Additionally, differences on the individual symptoms that were significant or approaching significance, were in the direction of greater symptom complaints by the Concussed Rugby group at the first, second and third follow-up intervals.

In terms of specific symptoms, the independent analyses between the Concussed Rugby and Matched Control groups on the ImPACT Symptom Scale at the *first follow-up* interval, revealed that the General Rugby group obtained a significantly higher Symptom Composite score, having evidenced higher scores for 18 of the 22 individual symptoms, with the difference being significant for 5 of these symptoms (headache, fatigue, drowsiness, sensitivity to light and slowed down), and approaching significance on 3 further symptoms (nausea, sleeping more and numbness). In contrast, the Matched Control group did not obtain any symptom scores that were higher than that of the Concussed Rugby group. These symptoms reported by the Concussed Rugby group, that were significant or approaching significance, are among those commonly reported soon after sustaining a MTBI, with headache being the most common symptom (e.g., Lovell et al., 2006). However, the Concussed Rugby group did not obtain significant findings, or findings approaching significance, for these other frequently reported symptoms: concentration difficulties, dizziness, confusion, balance problems, being mentally foggy, memory problems, and nervousness (e.g., Barbic et al., 2005; Collins et al., 2006; Erlanger, Kaushik et al., 2003; Gessel et al., 2007; Guskiewicz et al., 1997, 2000; Hinton-Bayre et al., 1999, 2004; Iverson et al., 2004b; Lovell et al., 2006; Macciocchi et al., 1996, 2001; Makdissi et al., 2001; McCrory & Johnston, 2002; Moser et al., 2007; Shuttleworth-Jordan et al., 1993).

Further, the independent analyses between the Concussed Rugby and Matched Control groups on the ImPACT Symptom Scale at the *second follow-up* interval, revealed that the General Rugby group obtained a significantly higher Symptom Composite score, having

evidenced higher scores for 13 of the 22 individual symptoms, with the difference approaching significance on 4 symptoms (headache, sensitivity to light, mentally foggy and difficulty remembering). In contrast, the Matched Control group obtained a higher score for only 1 symptom (sleeping less). The independent analyses between the Concussed Rugby and Matched Control groups on the ImPACT Symptom Scale at the *third follow-up* interval, revealed that the General Rugby group obtained a higher score approaching significance on the Symptom Composite, having evidenced higher scores for 9 of the 22 individual symptoms (the Matched Control group reported no symptoms), with the difference being significant for 1 symptom (fatigue), and approaching significance for 2 symptoms (headache and sensitivity to light). The independent analyses between the Concussed Rugby and Matched Control groups on the ImPACT Symptom Scale at the *fourth follow-up* interval revealed no significant differences or differences approaching significance between the Concussed Rugby and Matched Control groups. The Concussed Rugby group reported on 6 symptoms and the Matched Control group reported on 9 symptoms. The Concussed Rugby group obtained higher scores on 4 of the 22 symptoms, both groups obtained an equivalent score on 1 symptom, and the Matched Control group obtained higher scores on 4 symptoms. Some studies have found that concussed athletes in comparison to controls reported significantly higher symptom complaints up until and/or including day 5 post MTBI (e.g., Macciocchi et al., 1996; McCrea et al., 2003, 2010). Similarly, to this present study, Parker et al. (2007) found concussed adult athletes reported significantly higher symptom complaints than controls on the ImPACT Symptom Scale days 2, 5 and 14, post MTBI, and although their symptom reports remained higher than controls at day 28 post MTBI, the differences was not significant.

In summary as could be expected from the MTBI sport literature (e.g., Macciocchi et al., 1996; McCrea et al., 2003, 2010; Parker et al., 2007), the independent analyses of group means on the ImPACT Symptom scale at the baseline and four follow-up intervals found higher symptom reports for the Concussed Rugby group relative to the Matched Control group in support of the fifth and sixth hypotheses (in Chapter 6, section 6.5.2 and Chapter 7, section 7.3), viz., that the Concussed Rugby group would report more symptoms at baseline as a result of previous concussive and subconcussive incidents historically, and also following concussion, as an overlay of acute effects.

Dependent *t*-test Comparisons

The repeated measures ANOVAs for the four follow-up test intervals versus the baseline test interval, for the Concussed Rugby and the Matched Control groups, on the ImPACT Symptom Scale, revealed a significant group by season interaction effect for the headache symptom. For the Symptom Composite measure, the group by season interaction effect was approaching significance and the effect for season was significant. These effects revealed a general decline in overall symptom reports for both groups at the four follow-up intervals versus the baseline interval, apart from the first-follow-up interval when the Concussed Rugby group's Symptom Composite score was equivalent to their baseline score. The Symptom Composite score reduced significantly from the baseline score at the third and fourth follow-up intervals for the Concussed Rugby group, and this was taken to indicate complete symptom recovery. Furthermore and in comparison, the overall decline in symptom reports at the end of season interval versus the baseline interval was more robust for the General Rugby group.

Both a significant group by season interaction effect and significant effect for season for the headache symptom, revealed that the Concussed Rugby group obtained a significantly higher mean score for this symptom at the first follow-up versus baseline interval which is strongly suggestive of it being exacerbated by the MTBI, whereas the Matched Control group did not report on the headache symptom measure at the baseline and first three follow-up intervals. A significant effect for season was revealed for the mentally foggy symptom, in that the Concussed Rugby group revealed a higher score at the first follow-up versus baseline interval, although not approaching significance, while there was little difference in scores for this symptom between the first or fourth follow-up versus baseline interval for the Matched Control group. The effect for season was approaching significance for the slowed down symptom, in that the Concussed Rugby group obtained a significantly higher mean score on this symptom at the first follow-up versus baseline interval, whereas the Matched Control group reported on this symptom only at the baseline and fourth follow-up intervals.

Specifically, the dependent *t*-test analyses for the ImPACT Symptom Scale at the four follow-up intervals versus the baseline interval, found that the Concussed Rugby group revealed significant findings or findings approaching significance as follows. At the *first follow-up* interval only 2 symptoms were significantly higher than that reported at

baseline (headache and slowed down). In contrast, reports on the 3 symptoms were significantly lower than at baseline (sadness, nervousness, and difficulty remembering), and lowered reports on 2 symptoms were approaching significance (trouble falling asleep and irritability). At the *second follow-up* interval versus baseline, the overall symptom report lowered. In contrast to expectations, lowered reports on 4 symptoms were approaching significance (sleeping more, feeling more emotional, difficulty concentrating and difficulty remembering). At the *third follow-up* interval versus baseline, there was a significant lowering on the Symptom Composite, suggestive of complete recovery from the first-follow up (the score of which had equated the baseline report), and on 3 symptoms (sadness, nervousness and difficulty remembering). Lowered reports on 4 symptoms were approaching significance (sleeping less, irritability, mentally foggy and difficulty concentrating). At the *fourth follow-up* interval versus baseline, there was a significant lowering on the Symptom Composite and on 6 symptoms (fatigue, trouble falling asleep, sadness, nervousness, difficulty concentrating and difficulty remembering). Lowered reports on 3 symptoms were approaching significance (sleeping more, sleeping less and irritability). In contrast to the Concussed Rugby group, no significant findings were revealed on the symptom composite for the Matched Control group and their reports over all test intervals reflected a relatively flat profile for the consistency of symptom reports, as shown in other studies (e.g., McCrea et al., 2003; Parker et al., 2007).

Taken together, the main highlight of the results for the dependent analyses across the four follow-up intervals versus the baseline interval, are that headache and being slowed down are the most striking increased symptoms immediately postconcussion at the first follow up, and the other prominent finding is that the only significant reduction in overall total symptoms occurs at the third follow-up interval, neither of which occurred for the controls. The elevated and more persistent report on headache is in complete support with the literature (e.g., Collins, Iverson et al., 2003; Department of Veteran Affairs, 2009a; Evans, 2004; Guskiewicz, McCrea, Marshall et al., 2003; Guskiewicz et al., 2001; Legome & Alt, 2009; Lovell, Collins, & Bradley, 2004; Lovell et al., 2006, 2007; McCrory, Ariens, & Berkovic, 2000; Packard, 2008). Furthermore, the length of time for specific symptom resolution in comparison to the baseline performance (between approximately 5 to 10 days post MTBI), is similar to other studies in which symptom resolution occurs between days 4 to 7 days (e.g., Covassin et al., 2008; Lovell, Collins, Iverson et al., 2004; McCrea et al., 2003, 2010; Warden et al., 2001), and is in accordance

with the given ten days in which the neuropsychological effects of MTBI are commonly resolved (e.g., Bailes & Cantu, 2001; Barth et al., 1989; Belanger et al., 2005; Bleiberg et al., 2004; Collie, Makdissi et al., 2006; Collins, Grindel et al., 1999; Echemendia et al., 2001; Field et al., 2003; Hinton-Bayre et al., 1997; Hugenholtz & Richard, 1982; Iverson et al., 2004a; Macciocchi et al., 1996; McCrory et al., 2009; Shuttleworth-Edwards & Radloff, 2008; Shuttleworth-Edwards, Smith et al., 2008; Manko, 2003).

Generally, findings of the dependent analyses of changes in the expected direction for the Concussed Rugby group on the common post MTBI symptoms, in conjunction with comparative literature findings, and the relative lack of symptom changes for the Matched Control group provides reasonable support for the seventh and eighth hypotheses (in Chapter 6, section 6.5.2 and Chapter 7, section 7.3), viz., that the Concussed Rugby group (in contrast to the Matched Control group), would demonstrate increased symptom reports on the Symptom Scale at the postconcussion test intervals, and most notably at the first follow-up test interval, compared to the baseline test interval, in support of deleterious neuropsychological effects, due to long-term exposure to concussive and subconcussive events historically, and the overlay of acute effects of reported concussive events.

Summary of Symptom Findings for the Independent and Dependent Analyses

Taking the independent and dependent analyses together, overall findings for the Concussed Rugby group and the Matched Control group, on the symptom measures, were mostly in support of the hypotheses five to eight (in Chapter 6 section 6.5.2 and Chapter 7 section 7.3), that broadly suggest that the Concussed Rugby Group would reveal more symptoms than the Matched Control Group due to long-term exposure to concussive and subconcussive events historically and concussive events during the 2005 season, either because of increased symptom reports at baseline and/or follow-up testing (independent analyses), or the increase in symptom reports between the baseline and four follow-up test intervals in comparison to the Matched Controls (dependent analyses). Importantly, recovery took place longer than what is generally acknowledged, when using controls for comparisons. Again, as with the neurocognitive test findings, it is understood that on the basis of the present research, it is not possible to differentiate between the extent to which current and past concussive and subconcussive events are contributing to this picture.

9.4 SYNTHESIS OF FINDINGS FOR THE NEUROCOGNITIVE AND SYMPTOM MEASURES

9.4.1 Overall Synthesis of Findings For the Neurocognitive and Symptom Measures

Overall, the highly consistent direction of significant results and trends for the neurocognitive symptom measures across the General Rugby and General Control groups and Concussed Rugby and Matched Control groups, both cross sectional (independent) and prospective (dependent analyses) provides multifaceted cross-validation of outcome in support of all hypotheses (in Chapter 6, section 6.5.2 and Chapter 7, section 7.3). Generally, both rugby groups obtained poorer neurocognitive test results and higher symptom scores than their respective control groups. This provides fairly persuasive evidence that rugby players involved in contact sport incur adverse neuropsychological effects, as the result of exposure to concussive and subconcussive events, and that these adverse effects are exacerbated from one season of playing rugby. In contrast, sportsmen involved in noncontact sport do not incur adverse neuropsychological effects, and is explicable in that their exposure to concussive and subconcussive events is limited or unlikely. In particular, the additive effects from playing rugby over one season or sustaining MTBI during the season, cumulatively or synergistically, exacerbated the residual neurocognitive effects on ImPACT's measures of Impulse Control, Visual Motor Speed and Reaction Time, and the WMS-III measures of visual memory, but more notably verbal memory. Symptom measures on ImPACT were more pronounced on fatigue, difficulty concentrating, drowsiness, sensitivity to noise and numbness at the end of the season, and on headache and feeling slowed down post MTBI.

Specifically, with respect to the Concussed Rugby group, an important observation was that a significant reduction in overall symptom complaints in comparison to baseline and the first follow-up and in comparison to controls occurred at the third follow-up test (day 16), which is similar to findings by Parker et al. (2007) who at day 14 found the concussed athletes still reported a significantly higher number of symptoms than controls. Furthermore, following MTBI, despite symptom resolution, there was incomplete neurocognitive recovery still present at approximately 24 days post MTBI, in that the Concussed Rugby group obtained a significantly poorer score, than the Matched Control group, on the WMS-III VPA I measure. This implicates the presence of lingering

problems in the verbal learning area in the Concussed rugby group. While verbal memory problems were not identified by the ImPACT test at this late juncture, it is not an isolated finding in the rugby-related concussion research. The finding is similar to that of Shuttleworth-Jordan et al. (1993), who found the absence of symptoms in rugby players at three months post MTBI, while demonstrating incomplete neurocognitive recovery on verbal memory tasks, including Digit Span backwards, Digits Difference, and Digit Supraspan (Shuttleworth-Jordan et al., 1993). Accordingly, findings for the present study reveal that symptom recovery between approximately 5 to 16 days post MTBI (depending on whether comparisons are made on significantly elevated specific symptoms post MTBI and baseline performance, or between significantly higher symptom reports between rugby and controls, respectively), preceded neurocognitive recovery. This critical finding supports the importance of neurocognitive testing in that symptom reports appear unreliable among rugby players at this elite level. Moreover, in that the WMS-III Verbal Paired Associates was the superior indicator of lingering neurocognitive effects, it appears that the sensitivity of the ImPACT Verbal Memory composite could be enhanced via the inclusion of an associate learning task.

Finally, in reviewing the overall neurocognitive plus symptom report outcome for the Concussed Rugby group, several studies have found that slowed reaction time is associated with being symptomatic, following MTBI. Collie, Makdissi et al. (2006), found impaired reaction time more prevalent among symptomatic versus asymptomatic athletes, within 11 days of MTBI. Studies using ImPACT found that high school and collegiate athletes reporting a headache, migraine, fogginess, and/or increased symptoms, performed significantly worse on reaction time measures than those not reporting these symptoms, following MTBI (e.g., Collins, Field et al., 2003; Iverson et al., 2004b; Lau et al., 2009; Majerske et al., 2008; Mihalik et al., 2005). It might be expected, therefore, that there would have been a significant difference on the Reaction Time measure at the first follow-up, given that the Concussed Rugby group evidenced a significantly higher mean score versus baseline and between groups, on the headache symptom at the first follow-up. However, although there were significant group by season interaction and season effects for the Reaction Time measure, the Concussed Rugby group's score lowered, but not significantly, at the first follow-up versus baseline, and the between group difference on the Reaction Time was approaching significance. It may be that being a relatively high

functioning group, accounted for there being no significant reduction in reaction time at the first follow-up test interval.

Findings among various clinical samples in general neuropsychological research are often reported as being subtle (e.g., Demakis, 2006; Frencham et al., 2005; Reitan & Wolfson, 1999; Ruff, 2005; Woods et al., 2006). The findings in this study, of adverse neurocognitive and symptom effects for rugby players are also subtle. Nevertheless, these subtle findings gain in their persuasiveness in that pre-existing differences between the groups were reasonably well controlled for (e.g., gender, age, language, race, estimated IQ, education and years of sport participation). In terms of the Reserve theory, as the participants were cognitively high functioning, and on the basis that 35% of the final rugby sample reported no prior concussion at the outset of the study, significant effects are likely to have been diminished in the group analyses. Furthermore, the relative lack of significant findings might be Type II errors, as a result of the precautionary use of the Bonferroni Correction. Finally, ceiling effects, were a limitation of the WMS-III measures, and are highly likely to have provided a more limited impression of actual deficits for this high functioning group. Nevertheless, the subtle neurocognitive effects and residual symptoms found in this study, are generally consistent with subtle findings in numerous other contact sport-related MTBI studies, detailed in Chapter 6. An important qualifying observation from the present study, however, is that neurocognitive and symptom effects of MTBI take longer to recover than the period of ten days or less, that has typically been suggested in the available literature.

9.5 CRITICAL EVALUATION OF THE STUDY

Several studies on the neuropsychological effects of sport-related MTBI, suffer methodological limitations, which were discussed in Chapter 6, and the present study requires similar critical evaluation.

9.5.1 Strengths of the Study

Two previous studies that included a similar level of elite rugby players (national level), and controlled for gender, age, education and IQ between the rugby and control groups, investigated only the chronic neuropsychological effects at a preseason assessment, and

comprised relatively small samples (Shuttleworth-Edwards, Border et al., 2004; Shuttleworth-Edwards & Radloff, 2008). This study included noncontact sport control groups for comparison purposes. Gender, age, education, estimated IQ, WAIS-III Vocabulary subtest score, length of primary sport participation, language and race were all controlled for across all the comparison groups, with the only exception being that the General Control group demonstrated a significantly higher score on the WAIS-III Matrix Reasoning subtest than the General Rugby group. However, scores on the WAIS-III Vocabulary subtest and estimated Full Scale IQ (as calculated by the OPIE – 3 (2ST) formula) were not significantly different between the groups. In evaluating any possible confounding effects due to pre-existing differences on the Matrix Reasoning subtest, this might have been relevant had the WMS-III visual memory tasks been consistently impaired for the General Rugby group relative to the General Control group. However, this was not the case, in that it was the verbal memory aspect which consistently identified such vulnerability for the General Rugby group. Brandt (2007) criticises race or ethnicity traditionally being considered to account for a proportion of variance in cognitive test performance, however there was equivalence for racial distribution between the comparative groups. The sample size of the General Rugby and General Control groups was considered adequate. The sample size of the Concussed Rugby and Matched Control groups was inevitably relatively small given that only a proportion of players are concussed in a season, but was larger than the only other prospective rugby study carried out by Shuttleworth-Jordan et al. (1993). The proportion of concussed players identified for use in the study (i.e., 21 - 22%) was in keeping with the incidence reports in the available literature in respect of elite level rugby/football athletes (Shuttleworth-Edwards, Noakes et al., 2008).

It has been proposed that sport-related MTBI research should comprise the evaluation of both neurocognitive functioning and symptom reports, as measuring either aspect in isolation, is not adequately sensitive to the effects of MTBI (e.g., Aubrey et al., 2002; Guskiewicz, McCrea et al., 2003; McCrory, Johnston et al., 2005; Van Kampen et al., 2006). Therefore, a significant strength of the present study was that it evaluated both neurocognitive functioning and symptom reports. Furthermore, this study used both traditional and computerised testing, to enhance the sensitivity of the neurocognitive testing. The study by Shuttleworth-Edwards, Border et al. (2004), used similar subtests from a former version of the WMS, in addition to several other traditional tests, but did

not use computerised neuropsychological testing. This study used newer versions of the Verbal Paired Associates (VPA) and Visual Reproduction (VR) subtests, from the WMS-III edition. To this author's knowledge, no known MTBI research has used both the WMS-III VPA and VR neurocognitive subtests in combination with the ImPACT computerised neurocognitive program, to assess both the chronic and acute effects of sport-related MTBI, and in combined form this multi-faceted study allowed for a measure of cross-validation in the identification of acute and chronic postconcussion effects.

9.5.2 Limitations of the Study

A quasi-experimental cross-sectional design was used which involved the rugby group being divided into those players who were not knowingly concussed (General Rugby group), and those players who were concussed during the study (Concussed Rugby group), and forming the control participants into two control groups for comparison purposes. This cross-sectional design was implemented to evaluate the effects of MTBI as a result of exposure to a contact sport (rugby), as opposed to noncontact sports. Therefore, the control athletes formed non-equivalent comparison groups (i.e., they were non-equivalent on MTBI history), but were matched on demographic variables to ascertain demographic equivalence. A criticism of cross-sectional research is that findings from objective testing, between the comparative sample groups, could be the result of pre-existing differences i.e., other than exposure or non-exposure to concussive and subconcussive events. However, such is partly eliminated by this study: (i) implementing a baseline assessment against which end of season or postconcussive assessment results could be compared, for each individual, and (ii) the comparative sample groups were statistically matched on the variables of gender, age, estimated IQ, level of education, years of primary sport participation, language and race.

Intervals between Post Concussion Follow-up Testing

The time periods between baseline and end of season testing, for the General Rugby and General Control groups, were equivalent. The time periods between MTBI and postconcussion or equivalent follow-up testing, for the Concussed Rugby and Matched Control groups, were equivalent between groups. For the Concussed Rugby and the Matched Control participants, post concussion or equivalent testing took place at four follow-up intervals. The aim for this research, was to carry out the first follow-up post

concussion testing within 2-3 days of MTBI. However, due to constraints beyond the researcher's control, the interval between the MTBI and first follow-up testing averaged 4 days (ranging from 2 – 10 days postconcussion). Accordingly, a limitation of this study is that the effects of MTBI in the group analyses were in all likelihood diluted, in that testing did not take place within 2 to 3 days post MTBI in all cases, at which time deficits would in all probability have been more apparent. Thereafter, the aim was to carry out the second, third and fourth follow-up postconcussion testing at weekly intervals. However, the interval between the first and second postconcussion follow-up averaged 6 days (ranging from 2 – 9 days); the interval between the second and third postconcussion follow-up averaged 6 days (ranging from 2 – 8 days); and the interval between the third and fourth postconcussion follow-up averaged 8 days (ranging from 1 – 15 days). Despite the researcher's considerable attempt to maintain the planned time intervals between testing, this was not possible. Often players did not attend testing when scheduled. Frequently, re-testing was required prior to the scheduled time, to ascertain if the players were neuropsychologically recovered, in order to participate in important provincial rugby matches. Similarly, therefore, the uneven time intervals between MTBI follow-up testing would result in a less exact delineation of effects at very clearly demarcated time intervals. However, the subtle findings that were revealed for the Concussed Rugby group, appear more compelling for the first follow-up testing, given that most prominent neurocognitive impairments and symptom complaints would have diminished quite substantially at that time, therefore, diluting significant findings.

Comparative Groups

The pre-selected experimental group comprised contact sport rugby players who fell under the management of the provincial rugby administration, and the management volunteered their participation. The investigation of neuropsychological effects of concussive and subconcussive injuries in a contact sport group ideally requires a noncontact sport group for comparative purposes, in that noncontact athletes are less susceptible to concussive and subconcussive injuries (Shuttleworth-Edwards & Radloff, 2008). Noncontact sportsmen participated in the control groups, for this study. However, the control sample was not randomly selected but derived on an ad hoc voluntary basis, and effects could be due to a volunteer sampling bias. Furthermore, these sportsmen played a variety of sports, and unknown extraneous variables might be associated with the different noncontact sports.

Differences, for example, might exist between contact and noncontact sports. More aggressive or impulsive athletes might choose to play contact over noncontact sport, and test performance might be the consequence of these variables, rather than the effects of MTBI (e.g., Shuttleworth-Edwards & Whitefield, 2007a). The inherent differences between contact and noncontact groups, irrespective of MTBI histories, are covert. Therefore research outcomes based on comparisons beween these two groups in the final analysis remain somewhat speculative (e.g., Rutherford et al., 2003; Shuttleworth-Edwards & Radloff, 2008). To help negate such effects, attempts were made to match the comparative groups as closely as possible on extraneous demographic variables and, as indicated above, groups were statistically equivalent for gender, age, level of education, estimated IQ, years of sport participation, language and race. Comparative groups were also all well-matched for the Vocabulary subtest, i.e. a measure of Verbal IQ. A limitation in this regard was the significantly higher Matrix Reasoning score for the General Controls versus the General Rugby group, and an estimated IQ score that was approaching significance in the direction of General Controls scoring higher than the General Rugby group. However, these differences were not in evidence for the Concussed Rugby group and its matched Controls, and therefore cannot serve as a global explanation for the general trends of poorer outcome for the rugby groups identified across both aspects of the study. Moreover, as indicated above, significant lowering for the General Rugby group versus controls was largely in the verbal memory area rather than the non-verbal area, and this is also not readily explained by superior functioning on the non-verbal Matrix Reasoning task. However, the study would ideally have benefitted from improved control in this regard, if at all possible, for more certainty that this difference was not in any way influencing the results.

It is quite feasible that there could be differences in motivation between the rugby players, particularly those keen to return to play, and the control sportsmen. Therefore, it might be considered a limitation that a formal measure of 'effort' was not included as per that used in research involving group testing (e.g., Broglio et al., 2007), to assess if significant findings or a lack thereof may have been due to participants being fatigued or lacking in motivation. Each participant in this study was tested individually under the careful observation of the researcher throughout the test procedure, and therefore it seems unlikely that poor effort would have a confounding effect on results.

Shuttleworth-Edwards and Radloff (2008), note that sport-related MTBI studies have been subject to small sample sizes, with 20-30 participants in the comparative groups (e.g., Matser et al., 1999; Rutherford et al., 2005; Shuttleworth-Edwards, Border et al., 2004). In this study, the sample size was satisfactory for the General Rugby and General Control comparative groups (n = 54 versus n = 37). However, the sample size was small for the Concussed Rugby and Matched Control groups (n = 17 for both groups). Despite this, some significant and highly consistent trends in the hypothesised direction and in terms of the sport-related MTBI literature were still identified, which might have been strengthened were the sample numbers increased.

Overall the rugby and control participants in this study were predominantly in their early twenties, with estimated IQ scores falling in the upper average range. All of the participants were relatively well-educated and averaged more than thirteen years of formal education. Afrikaans or English was the first language for the majority of the participants, rather than an African language, and the distribution of race was skewed towards a white population. The sample consisted of sportsmen exclusively, therefore, findings from this study cannot be generalised to sportswomen. Overall, the sample is not representative of the South African population, and findings cannot be generalised to all South African cultural and socio-economic groups. The reasons for this are revealed by the following statistics. In 2007, the estimated South African population was 47.9 million, of which 38 million (79.6%) were black, 4.3 million (9.1%) white, 4.2 million (8.8% coloured), and 1.2 million (1.2%) Asian. African languages accounted for 78.5%, Afrikaans for 13.3% and English for 8.2% of the languages among the population. South African statistics for the average number of years of formal education were unavailable, but it was estimated that 86.4% of those over 15 years of age were literate. Furthermore, statistics were unavailable for the various sports played among the population (http:www.southafrican.info/about/people/population.htm). On the basis of the Reserve theory, the sample in this study is considered cognitively advantaged, in terms of being intellectually high functioning and well-educated. Therefore, results could differ in intellectually lower functioning or less educated samples and in terms of the tenets of Reserve theory (Satz, 1993, 2003; Satz et al., 2010; Stern, 2002, 2009; Stern et al., 2003), are likely to be substantially more pronounced in the direction of revealing more significant lowering in association with repetitive exposure to rugby-related MTBI.

Other Extraneous Variables

Rather than the cumulative effects of MTBI, the use of alcohol or cigarette-smoking could be considered extraneous variables accounting for neurocognitive impairment in contact sport groups (e.g., Macciocchi & Barth, 2004; Matser, Kessels et al., 2000; Shuttleworth-Edwards & Radloff, 2008). This study revealed that the percentage distributions of both weekly alcohol use and cigarette smoking were significantly less in the General Rugby group versus the General Control group. This implies a significantly higher consumption of each of these substances among the General Control group. No significant differences were revealed between the percentage distributions of the Concussed Rugby group versus the Matched Control group for the variables of weekly alcohol consumption and cigarettes smoked. The trends, however, were in the direction of higher consumption of each of these substances among the Matched Control group. Therefore, the use of these substances is not likely to contribute to a significant lowering in cognition or enhanced symptom reports for the rugby groups compared with the control groups, but could have done so for the control groups relative to the rugby groups. This factor may therefore have served to dilute significant effects among the rugby groups, in that there may have been pre-existing lowering of cognitive ability and enhanced symptom reports among some controls in association with alcohol use.

Jakoet and Noakes (1998), point out that the rugby player's on-field position (i.e., forward versus back), has an influence on the player's vulnerability for MTBI. The forwards, who are involved in the scrums, are more susceptible to MTBI than the backs. For the purposes of this study, the distinction between the forward and back positions was not made, and this could be considered a weakness. However, positional subgroups would have reduced the size of the sample inappropriately, especially for the Concussed Rugby group, and have created an over abundance of additional analyses causing the thesis to become less focused.

Research Measures

This study used only two traditional tests, the Verbal Paired Associates and Visual Reproduction subtests of the WMS-III. The majority of sport-related MTBI studies used several traditional tests (e.g., Collins, Grindel et al., 1999; Hatfield et al., 2004; Matser et al., 1998, 1999; Rutherford et al., 2005). This study could be criticised for not tapping several other neurocognitive functions, by using a greater number of traditional tests.

However, this provided a focused rather than a somewhat haphazard or indiscriminate approach to test selection in targeting specific neurocognitive functions. Importantly, the chances of incurring a Type I error decrease when fewer tests are used. When studies use a large number of tests, the results are considered 'exploratory', because this increases the likelihood of significant findings being found as a result of chance (Rutherford et al., 2003, 2005). Whereas some studies have used as many as 27 neuropsychological test comparisons (e.g., Matser et al., 1998, 1999), the present study only used 14 neurocognitive test comparisons. On the grounds of using relatively fewer tests, the present study can be considered less exploratory.

The WMS-III Verbal Paired Associates has been criticised for not being sensitive in detecting verbal learning and verbal association memory impairments and having a limited range of scores (e.g., Burton et al., 2003; Graf & Richter, 2002; Pearson Clinical Assessment, 2009; Riley & Zellinger, 2000; Uttl, et al., 2002). Therefore, this study could be criticised for having selected a test that is not sensitive enough to the subtle effects of MTBI. Despite the frequently cited limitations of the test, the findings suggestive of impairment on this measure are strengthened. Both the concussed and nonconcussed athletes revealed some level of compromised performance on the Verbal Paired Associates at the post-baseline test intervals. Specifically, there was a highly significant difference at the fourth follow-up interval in the direction of the Concussed Rugby group performing more poorly than controls. In addition, there were a number of findings approaching significance, in the direction of poorer performance by the General Rugby group at the end of season testing, that would have been significant had it not been for use of the Bonferroni Correction. It is of particular note that in contrast to these findings for the WMS-III Verbal Paired Associates subtest, at no point did the ImPACT Verbal Memory task discriminate significantly between the rugby and control groups. *This, therefore, is suggestive that the ImPACT Verbal Memory measure might be enhanced via inclusion of a verbal associate learning task.*

This study tapped MTBI-related symptom outcomes using the ImPACT Symptom scale. Measurement of both neurocognitive functions and postconcussive symptoms, provides a more holistic neuropsychological profile of rugby-related MTBI, and strengthens the study (e.g., Lovell, 2006; McCrory et al., 2009).

Bonferroni Correction

The Bonferroni Correction, discussed in Chapter 6, is the statistical adjustment made to compensate for multiple comparisons on the same data set, in order to reduce the risk of a Type I error i.e., rejecting the null hypothesis when it is true and finding a significant difference between the means, when such does not exist (e.g., Bland & Altman, 1995; Hsu, 1996; Perneger, 1998; Weisstein, 1996). It has been vehemently argued by some that use of the Bonferroni's Correction is likely to result in failing to discern statistical relevance in the interpretation of data, that would ordinarily provide a wealth of information (e.g., Brandt, 2007; Perneger, 1998). Brandt (2007) implies that this Correction is used in order to avoid criticism. Similarly a decision was taken for this study to use the Bonferroni Correction, in order to avoid criticism such as that levelled at previous MTBI studies (e.g., Matser et al., 1998, 1999, & 2001). On the other hand, this study might be criticised for using the Bonferroni Correction, and incurring the Type II error, by not finding significance when such does exist. For example, if the Bonferroni Correction had not been used, the poorer performance by the General Rugby group, in comparison with the General Rugby group, at the end of the season would have revealed significant findings on the WMS-III VPA I, VPA First Recall and VPA II measues in the direction of rugby being worse than controls. Furthermore, on the basis that 35% of the final rugby sample reported no prior concussion at the outset of the study, significant individual effects may be diminished in the analyses of group effects. This is a problem that has been strongly raised in the MTBI literature in general (Frencham et al., 2005; Shuttleworth-Edwards & Whitefield, 2007). Therefore, the study could fail to find significance by using this Correction. Notwithstanding this dilemma, the choice taken for the present study was towards stringency in order to reduce the risk of Type I error and therefore the significant findings as they have been identified, are unlikely to be due to chance.

However, the calculation of effect sizes (i.e. a form of power analysis) would have been a useful addition to the statistical analyses in the study and provided more certainty as to the clinical significance of the results.

9.6 IMPLICATIONS OF THE RESULTS

9.6.1 General Implications

The overall synthesis for the results of this study broadly supported the position of rugby players being neurocognitively and symptomatically vulnerable relative to noncontact sport controls. The general implication of this is that playing rugby at an elite provincial level may be neuropsychologically hazardous for a significant proportion of such relatively high functioning individuals. More specifically, a number of critical implications arise out of this study, as follows:

(i) Given that, MTBI symptom recovery occurred between 11 to 16 days, and incomplete neurocognitive recovery was revealed at an average of 24 days post MTBI (when comparing neurocognitive and symptom scores with controls), this recovery is far longer than the 7 to 10 days suggested in the literature. Accordingly for the adult athlete a quick recovery must not be assumed and should be cautiously investigated in every individual case.

(ii) Furthermore, in that symptom recovery preceded neurocognitive recovery in this study, alerts not only to the possibility of lingering effects in need of identification as per point (i), but also to the fact that elite athletes may under-report symptoms post MTBI to expedite return to play. Again, this carries with it the implication about being cautious in assuming quick recovery.

(iii) The two implications above (i.e. prolonged neurocognitive deficits and unreliable symptom reports) provide endorsement for the current recommendation of careful individualised assessment and management of concussive injury that ideally includes baseline and follow-up neurocognitive testing (Aubry et al., 2002; Echemendia et al., 2009; McCrory et al., 2009; Moser et al., 2007).

(iv) Specifically in this study, the ImPACT test demonstrated sensitivity more strongly for the Reaction Time, Visual Motor Speed and Impulse Control measures than for the memory measures. Therefore, in light of the WMS-III findings for the present study, it would appear that the sensitivity of the ImPACT Verbal Memory

component might be further enhanced by the inclusion of a verbal associate learning task. In the ongoing refinement of the ImPACT test this may be an interesting observation for the test developers to take into consideration, as the Verbal Memory composite does not include an associate learning aspect (see section 7.2.1).

(v) Finally, given the indications of adverse neuropsychological outcomes as a result of playing contact sport, steps need to be taken (as detailed in Chapter 4, section 4.3), to prevent MTBI occurring and to protect neuropsychological health in the event of MTBI. For example, prevention can occur through coaches teaching appropriate playing techniques, discouraging excessively competitive play, and for the IRB to review rules in the event that certain forms of play are found to increase players' risk for MTBI. Protecting neuropsychological health in the event of MTBI, relates to educating athletes, relatives, coaches and the general public to recognise and manage MTBI appropriately. In particular, this should include emphasis on the imperative need for individualised concussion follow-up, that includes neurocognitive evaluation as part of overall medical management.

9.6.2 Implications Concerning the Reserve Theory

Findings for this study provide support for the Reserve theory, that the cumulative effects from concussive and subconcussive events, over a lengthy and intense period of rugby participation depletes the individual's cognitive reserve. Rugby participation averaged 13.61 years for the General Rugby group and 14.76 years for the Concussed Rugby group. However, the overall findings of neurocognitive deficits were subtle for both rugby groups. A likely contributing reason for this is that the rugby groups were in the upper average range of intelligence and had more than 13 years formal education, which is suggestive of them having a higher cognitive reserve. Had the rugby sample consisted of rugby players with lower estimated IQ scores, less education, learning disabilities as well as current psychiatric or neurological difficulties, then the neurocognitive findings may have been significantly more evident. This would be due to such players having comparatively lower cognitive reserves compared with this study's relatively high functioning sample of rugby players. Therefore, with this sample of high functioning rugby players, it would appear that several of their scores improved across test intervals

because their reserves were substantial enough to benefit from practice effects. Consequently, deficits are likely to remain subclinical or undetected unless tasks are extremely challenging and of a high enough level of difficulty for their deficits to be revealed.

An important implication for neurocognitive baseline assessment is that this could help identify players who are neuropsychologically compromised and whose participation in contact sport should be reviewed, in order to preserve the player's lowered brain reserve capacity. This is particularly pertinent, because in years to come, rugby authorities might become liable for persistent and deleterious deficits incurred as a result of rugby as is happening in American NFL (Schwartz, 2009, 2010).

9.6.3 Implications for Future Research

Longitudinal neuropsychological studies of rugby players are required (using the same measures) to evaluate subtle neuropsychological effects over time. The strength of such type of study is that neurocognitive changes that might occur can be more reliably attributed to the effects of MTBI rather than being attributed to sampling effects. The IRB (2010a) has expressed the need for longitudinal studies and are, therefore, concerned about the long-term implications of MTBI.

Findings of this study revealed that rugby players evidenced neuropsychological deficits at the end of the season compared with the preseason baseline assessment. It would be pertinent for a longitudinal study to be carried out to see if rugby players achieve neuropsychological recovery following the period of abstinence from contact sport at year-end (i.e., second baseline comparison with previous year end results). It should be a cross-sectional study that compares those rugby players who had a longer break in comparison with those players who had a relatively short beak from rugby. The reason for the pertinence of such a study, is that the already long rugby season will be expanded for the Super-15 in 2011 and there is talk of the Springboks getting into an expanded Six Nations series (Gray, 2010; Long, 2010). A significant implication from such study might be that professional rugby players be given an adequate break between seasons to allow neurocognitive recovery and to preserve the players' neuropsychological integrity.

A study, such as the present one, that involves the analysis of group effects, dilutes individual effects. Importantly, *prospective* individual case-based MTBI rugby research is warranted, that allows for accurate medical diagnosis of concussion occurrence rather than reliance on self-reported MTBI history. This would provide a more reliable and more valid mode of measuring differential neuropsychological sequelae in association with the individual incidence of MTBI. Furthermore, it would be of value to conduct a study that investigates the fine detail of recovery duration between adult rugby players such as comparing recovery in those who rested and did no light exercise in comparison with those players who did not rest and did light exercise, or those returning to study and those who are removed from study (i.e., protected from cognitive exertion) until considered recovered.

In order to help prevent rugby-related MTBI, research should be carried out to ascertain which forms and positions of play result in either the most number of MTBIs or longer MTBI recovery. Such research is warranted in order to change rugby rules or to minimise dangerous forms of play, particularly for players whose on-field position incurs greater risk. Some dangerous forms of play are no longer tolerated, for example, spear tackles, tackling above shoulder height, and deliberate collapsing of the scrum (e.g., IRB, 2009b)

Findings from the current study were restricted to an elite high functioning male population and cannot be extrapolated confidently to wider populations. Therefore rugby studies should be carried out among culturally, economically, and educationally diverse populations, as well as among female rugby players and younger rugby players. Finally, future research is also required in order to develop sport-related norms for neuropsychological measures, that are based on players' estimated cognitive reserves or estimated IQ scores, and which takes practice effects into account.

9.7 FINAL WORD

The multi-faceted cross-sectional and prospective design of this study, using a focused battery of computerised and traditional test measures provides fairly persuasive evidence, albeit subtle, that provincial rugby players, in comparison with noncontact sportsmen, suffer adverse neuropsychological effects as a result of playing this collision sport, and that these effects are compounded during a rugby season as a result of subconcussive and

concussive events. The rugby sample was a high functioning group of sportsmen and therefore, in terms of Reserve theory, the evidence of deleterious neuropsychological consequences might appear less pronounced than in other less high functioning rugby populations. Furthermore, had the Concussed Rugby group not experienced the good follow-up medical care that was provided, in which they were withdrawn from rugby training and matches until asymptomatic and returned to baseline neurocognitive functioning, their recovery might have been impeded. Therefore, public awareness of the neuropsychological damage incurred as a result of concussive and subconcussive events needs to be promoted, in order to help ensure that compromised players are provided with sound medical and neuropsychological follow-up care.

REFERENCES

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

Ackerman, T. R. (2000). *Minor "dings"- major effects? A study into the cognitive effects of mild head injuries in high school rugby.* Rhodes University, Grahamstown, Republic of South Africa.

Agel, J., Dompier, T. P., Dick, R., & Marshall, S. W. (2007). Descriptive epidemiology of collegiate men's ice hockey injuries: National Collegiate Athletic Association injury surveillance system, 1988–1989 through 2003–2004. *Journal of Athletic Training, 42*, 241– 248.

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,* 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. Abstract retrieved February 8, 2010, from http://bjsm.bmj.com/content/43/Suppl_1/i3.abstract

Alvarez, J. A., & Emory, E. (2006). Executive function and the frontal lobes: A meta-analytic review. *Neuropsychology Review*, *16(1*), 17-42.

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 Congress of Rehabilitation Medicine. (1993). Definition of mild traumatic brain injury. *Journal of Head Trauma and Rehabilitation, 8*, 86-87

American Orthopaedic Society for Sports Medicine (AOSSM). (2006). Injury rate during preseason practice three times higher than during competition in college football. *The America's Intelligence Wire*. Retrieved March 9, 2010, from http://www.accessmylibrary.com/coms2/summary_0286-15184690_ITM

American Psychiatric Association (APA). (1994). *Diagnostic and statistical manual of mental disorders - Fourth edition (DSM-IV).* Washington, DC: American Psychiatric Association

American Psychiatric Association (APA). (2000). *Diagnosis and statistical manual of mental disorders (DSM-IV-TR)* 4th ed., Text Revision. Washington, DC: American Psychiatric Association. Retrieved September 01, 2010 from http://www.psychiatryonline.com/content.aspx?aID=5123&searchStr=post-concussion+syndrome

Andersson, E. E., Emanuelson, I., Olsson, M., Stalhammar, D., & Starmark, J. (2006). The new Swedish Post-Concussion Symptoms Questionnaire: A measure of symptoms after mild traumatic brain injury and its concurrent validity and inter-rater reliability. *Journal of Rehabilitation Medicine,* 38, 26-31. Retrieved October 19, 2010, from http://jrm.medicaljournals.se/files/pdf/38/1/26-31.pdf

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., &Murata, P. (2009). New VA/DoD guidelines for concussion and mild traumatic brain injury. *MedscapeCME Clinical Briefs*. Retrieved January 12, 2010, from http://www.medscape.com/viewarticle/714450

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

Anderson, T., Heitger, M., & Macleod, A. D. (2006). Concussion and mild head injury. *Practical Neurology,6,* 342–357. Retrieved August 16, 2010 from http://pn.bmj.com/content/6/6/342.full?ijkey=c41ed36819cfc7b4f1488218b0e825552c7ced43&keytype2=tf_ipsecsha

Anderson, V., & Catroppa, C. (2005). Recovery of executive skills following paediatric traumatic brain injury (TBI): A 2-year follow-up. *Brain Injury, 19,* 459-470.

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. Retrieved April 20, 2010, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2424119/

Arciniegas, D. B., Topkoff, J. L., Filley, C. M., Adler, L. E., Anderson, C. A., Ricketts, K. A. et al. (2003). Apolipoprotein-E4 in association with persistent neurophysiologic impairment after mild traumatic brain injury [Abstract]. *Journal of Neuropsychiatry and Clinical Neurosciences, 15(2)*, 276.

Ashtekar, J. L., & Patankar, T. U. (2009). Brain, MRI appearance of hemmorrhage. *eMedicine.* Retrieved April 4, 2009, from http://emedicine.medscape.com/article/344973

Asplund, C. A., McKeag, D. B., & Olsen, C. H. (2004). Sport-related concussion: Factors associated with prolonged return to play. *Clinical Journal of Sports Medicine, 14(6*), 339-343.

Aubry, M., Cantu, R. C., Dvorak, 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.

Australian Rugby Union Limited. (2009). *History of the game*. Retrieved November 10, 2009, from http://aru.rugby.com.au/community_rugby/what_is_rugby/history_of_the_game,24.html

Baddeley, A. (1992). Working memory. *Science,* 255(5044), 556-559. Retrieved August, 16, 2010, from http://www.ncbi.nlm.nih.gov/pubmed/1736359

Baddeley, A., Harris, J., Sunderland, A., Watts, K. P., & Wilson, B. A. (1987). Closed head injury and memory. In H. S. Levin, J. Grafman, & H. S. Eisenberg (Eds.), *Neurobehavioural recovery from head injury* (pp. 295-317). New York: Oxford University Press.

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. *DRC OhioLINK Electronic Thesis and Dissertation Center.* Retrieved November 2, 2009, from http://etd.ohiolink.edu/send-df.cgi/Baker%20Katherine%20Louise.pdf?acc_num=miami1209744334

Baker, R. J., & Patel, D. R. (2000). Sports Related Mild Head Injury in Adolescents. *Indian Journal of Pediatrics, 67(5)*, 317-321.

Bakhos, L. L., Lockhart, G. R., Myers, R., & Linakis, J. G. (2010). Emergency department visits for concussion in young child athletes. *Pediatrics.* Abstract retrieved September 27, 2010, from http://pediatrics.aappublications.org/cgi/content/abstract/peds.2009-3101v1

Barbic, D., Pater, J., & Brison, R, J. (2005). Comparison of mouth guard designs and concussion prevention in contact sports: A multicenter randomized controlled trial. *Clinical Journal of Sports Medicine, 15(5)*, 294-298.

Barclay, L. (2009). Mild traumatic brain injuries may cause trasnsient, persistent symptoms after injury. *Medscape Medical News*. Retrieved March 17, 2009, from http://cme.medscape.com/viewarticle/589259

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

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 April 27, 2010, from http://www.foresight.gov.uk/Mental%20Capital/SR-E4_MCW_v2.pdf

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. (2003). Neuropsychological testing of high school athletes. *Archives of Clinical Neuropsychology, 18(1),* 91-101

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.

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

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*, 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 N. R. Varney, & R. J. Roberts (Eds.), *The evaluation and treatment of mild traumatic brain injury*. Mahwah, NJ: Erlbaum.

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.

Bay, E., & McLean, S. A. (2007). Mild traumatic brain injury: An update of advanced practice for nurses. *Journal of Neuroscience in Nursing, 39(1)*, 43-51.

Bayir, H., Clark, R. S., & Kochanek, P. M. (2002). Promising strategies to minimize secondary brain injury after head trauma. *Critical Care Medicine, 31(1)*, 112-117.

Bazarian, J. J., McClung, J., Shah, M. N., Cheng, Y. T., Flesher, W., & Kraus, J. (2005). Mild traumatic brain injury in the United States, 1998-2000. *Brain Injury*, *19(2),* 85-91.

Begaz, T., Kyriacou, D. N., Segal, J., & Bazarian, J. J. (2006). Serum biochemical markers for post-concussion syndrome in patients with mild traumatic brain injury. *Journal of Neurotrauma, 23*, 1201–10.

Behan, L. A., Phillips, J., Thompson, C. J., & Agha, A. (2008). Neuroendocrine disorders after traumatic brain injury. *Journal of Neurology, Neurosurgery and Psychiatry, 79(7)*, 753-759.

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., & Vanderploeg, R. D. (2005). The neuropsychological impact of sports-related concussion: A meta-analysis. *Journal of the International Neuropsychological Society, 11*, 345-357.

Belasco, J. (undated). *How to learn the basics of hockey*. Retrieved March 24, 2010, from http://www.ehow.com/how-to_4845444_learn-basics-hockey.html

Bell, R. S., Neal, C. J., Lettieri, C. J., & Amonda, R. A. (2008). Severe traumatic brain injury: Evolution and current surgical management. *Medscape*. Retrieved June 26, 2008, from http://www.medscape.com/viewarticles/575753.

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 & Zeitlinger Publishers.

Benson, B. W., Hamilton, G. M., Meeuwisse, W. H., McCrory, P., & Dvorak, J. (2009). Is protective equipment useful in preventing concussion? A systematic review of the literature [Abstract]. *British Journal of Sports Medicine, 43(1)*, i56.

Benton, A. (undated). *Benton Visual Retention Test*. Retrieved October 26, 2010, from http://www.cps.nova.edu/~cpphelp/BVRT.html

Benton, A. (1974.). *Revised Visual Retention Test* (4th ed.). New York: Psychological Corporation.

Benton, A. L., Hamsher, K., & Sivan, A. B. (1989). *Multilingual aphasia examination.* Iowa City: AJA Association

Bernhardt, D. T. (2000). Football: A case-based approach to mild traumatic brain injury. *Pediatric Annals, 29(3)*, 172-176.

Bernhardt, D. T. (2009). Concussion. *Emedicine.* Retrieved September 7, 2009, from http:// *emedicine.medscape.com/article/92095-print.*

Bernstein, D. M. (2002). Information processing difficulty long after self-reported concussion. *Journal of the International Neuropsychological Society, 8*, 323-346. Retrieved August 24, 2010, from http://mykuc.biz/__shared/assets/Bernstein_20023960.pdf

Best, J., McIntosh, A., & Savage, T. (2005). The Rugby World Cup 2003 Injury Surveillance Project. *British Journal of Sports Medicine, 39*, 812-817.

Biasca, N., Wirth, S., & Tegner, Y. (2002). The avoidability of head and neck injuries in ice hockey: An historical review. *British Journal of Sports Medicine, 36*, 410-427.

Bigler, E. D. (1990). Neuropathology of traumatic brain injury. In E. D. Bigler (Ed.), *Traumatic brain injury.* Austin Texas: Pro-ed.

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. (2008). Neuropsychology and clinical neuroscience of persistent post-concussive syndrome. *Journal of the International Neuropsychological Society, 14(1)*, 1-22.

Bigler, E. D., & Orrison, Jr, W. W. (2004). Neuroimaging in sport-related brain injury. In M.R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 71-94). 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 implication. *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.

Biotech Week. (2009). *Sports concussions: Common and preventable*. Retrieved March 9, 2010, from http://www.accessmylibrary.com/coms2/summary_0286-37212399_ITM

Bird, Y. N., Waller, A. E., Marshall, S. W., Alsop, J. C., Chalmers, D. J., & Gerrard, D. F. (1998). The New Zealand injury and performance project: Epidemiology of a season of rugby injury. *British Journal of Sports Medicine, 32*, 319-325.

Bland, J. M., & Altman, D. G. (1995). Multiple significance tests: The Bonferroni method. *British Medical Journal, 310:* 170. Retrieved April 12, 2007, from http://www.bmj/cgi/content/full.

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., Halpern, E. L., Reeves, D., & Daniel, J. C. (1998). Future directions for the neuropsychological assessment of sports concussion. *Journal of Head Trauma Rehabilitation, 13(2)*, 36–44.

Bleiberg. J., Kane, R. I, Reeves, D. L., Garmoe, W. E., & Halpern, E. (2000). Factor analysis of computerized and traditional tests used in mild brain injury research. *Clinical Neuropsychology, 14,* 287-294.

Boake, C., McCauley, S. R., Levin, H. S., Pedroza, C., Contant, C. F., Song, J. X. et al. (2005). Diagnostic criteria for postconcussion syndrome after mild to moderate traumatic brain injury. *The Journal of Neuropsychiatry and Clinical Neurosciences, (17)*, 350-356. Retrieved September 1, 2010, from http://neuro.psychiatryonline.org/cgi/content/full/17/3/350

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.

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

Bohnen, N. I., Jolles, J., Twijnstra, A., Mellink., R., & Wijnen, G. (1995). Late neurobehavioural symptoms after mild head injury. *Brain Injury, 9*, 27-33.

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.

Borg, J., Holm, L., Peloso, P. M., Cassidy, J. D., Carroll, L. J., von Holst, H. et al. (2004). Non-surgical intervention and cost for mild traumatic brain injury: Results of the WHO collaborating centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine, 43,* 76-83.

Borgaro, S. R., Prigitano, 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.

Bottini, E., Poggi, E. J., Luzuriaga, F., & Secin, F. P. (2000). Incidence and nature of the most common rugby injuries sustained in Argentina (1991-1997). *British Journal of Sports Medicine, 34*, 94-97. Retrieved March 20, 2010, from http://bjsm.bmj.com/content/34/2/94.full

Bowen, A. P. (2003). Second Impact Syndrome: A rare, catastrophic, preventable complication of concussion in young athletes. *Journal of Emergency Nursing, 29,* 287-289.

Braham, R., Finch, C. F., McIntosh, A., & McCrory, P. (2004). Community level Australian Football: A profile of injuries. *Journal of Science and Medicine in Sport, 7(1)*, 96-105.

Brain Injury Association of Washington. (2009). *What is the Lystedt Law and why is the Brain Injury Association involved?* Retrieved April 12, 2010, from http://www.biawa.org/lystedt.htm

Brain Injury New Zealand. (2009). Concussion needs to be taken seriously. *Brainlink, 42*, 1-2. Retrieved March 26, 2010, from http://www.brain-injury.org.nz

Brandt, J. (2007). CE 2005 INS presidential addresses: Neuropsychological crimes and misdemeanors. *The Clinical Neuropsychologist, 21,* 553-568.

Briscoe, J. H. (1985). Sports injuries in adolescent boarding school boys. *British Journal of Sports Medicine, 19,* 67-70.

Broglio, S. P., Ferrara, M. S., Macciocchi, S. N., Baumgartner, T. A., & Elliott, R. (2007). Test-retest reliability of computerised concussion assessment programs. *Journal of Athletic Training, 42(4)*, 509-514.

Broglio, S. P., Ferrara, M. S., Piland, S. G., Anderson, R. B., & Collie, A. (2006). Concussion history is not a predictor of computerised neurocognitive performance. *British Journal of Sports Medicine, 40(1)*, 802-805.

Broglio, S. P., Macciocchi, S. N., & Ferrara, M. S. (2007a). Neurocognitive performance of concussed athletes when symptom free. *Journal of Athletic Training, 42(4)*, 504-508.

Broglio, S. P., Macciocchi, S. N., & Ferrara, M. S. (2007b). Sensitivity of the concussion assessment battery. *Neurosurgery, 60*, 1050-1058.

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. Retrieved March 26, 2010, from doi:10.1089/neu.2008.0766

Broglio, S. P., Vagnozzi, R., Sabin, M., Signoretti, S., Tavazzi, B., & Lazzarino, G. (2010). Concussion occurrence and knowledge in Italian football (soccer). *Journal of Sports Science and Medicine, 9,* 418-430. Retrieved October 3, 2010, from http://www.jssm.org/vol9/n3/10/v9n3-10pdf.pdf

Brooker, A. E. (1997). Performance on the Wechsler Memory Scale-Revised for patients with mild traumatic brain injury and mild dementia. *Journal of Clinical and Experimental Neuropsychology, 84*, 131-138.

Brooks, D. N. (1976). Wechsler Memory Scale performance and its relationship to brain damage after severe closed head injury. *Journal of Neurology, Neurosurgery and Neuropsychiatry, 39(6)*, 593-601.

Brooks, N. (1984). Cognitive deficits after head injury. In N. Brooks (Ed.), *Closed head injury: Psychological, social, and family consequences.* New York: Oxford University Press.

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. Abstract retrieved March 11, 2010, from http://journals.lww.com/jtrauma/toc/1999/01000

Brooks, J. H., Fuller, C. W., Kemp, S. P., & Reddin, D. B. (2005). Epidemiology of injuries in English professional rugby union: Part 1 match injuries. *British Journal of Sports Medicine, 39*, 757-766. Retrieved March 23, 2010, from doi: 10.1136/bjsm.2005.018135

Brooks, N., Kupshik, G., Wilson, L., Galbraith, S., & Ward, R. (1987). A neuropsychological study of active amateur boxers. *Journal of Neurology, Neurosurgery and Psychiatry, 50(8),* 997-1000. Retrieved March 11, 2010, from http://jnnp.bmj.com/content/50/8/997.full.pdf

Brooks, N., Symington, C., Beattie, A., & Campsie, L. (1989). Alcohol and other predictors of cognitive recovery after severe head injury. *Brain Injury, 3*, 235-246.

Broshek, D. K., Brazil, A. M., Freeman, J. R., & Barth, J. T. (2004). Equestrian sports. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports* (pp.149-168). The Netherlands: Swets & Zeitlinger Publishers.

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. Retrieved November 3, 2010 from http://www.impacttest.com/pdf/history_self_reported.pdf

Bruno, L. A., Gennarelli, T. A., & Torg, J. S. (1987). Management guidelines for head injuries in athletics. *Clinical Sports Medicine, 6*, 17-29.

Burgess, P., Sullivent, E. E., Sasser, S. M., Wald, M. M., Ossmann, E., & Kapil, V. (2010). Managing traumatic brain injury secondary to explosions. *Journal of Emergencies, Trauma, and Shock, 3(2)*, 164-172. Retrieved August 6, 2010, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2884448/

Burton, D. B., Ryan, J. J., Axelrod, B. N., Schellenberg, T., & Richards, H. M. (2003). A confirmatory factor analysis of the WMS-III in a clinical sample with cross validation in the standardization sample. *Archives of Clinical Neuropsychology, 18*, 629-641.

Butler, R. J., Forsythe, W. I., Beverly, D. W., & Adams, L. M. (1993). A prospective controlled investigation of the cognitive effects of amateur boxing. *Journal of Neurology, Neurosurgery and Psychiatry, 56(10)*, 1055-1061. Retrieved March 12, 2010 from, http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1015231/pdf/jnnpsyc00483-0013.pdf

Campbell, D. T. & Stanley, J. C. (1966). *Experimental and quasi-experimental designs for research*. Houghton Mifflin Company: Boston. Retrieved November 5, 2010, from http://www.uky.edu/~rford/CS_part1.pdf

Canadian Academy of Sport Medicine Concussion Committee. (2000). Guidelines for assessment and management of sport-related concussion. *Clinical Journal of Sport Medicine, 10*, 209-211.

Canadian Paediatric Society. (2006). Identification and management of children with sport-related concussion. *Paediatric Child Health, 11(7)*, 4420-428.

Cantu, R. C. (1986). Guidelines for return to contact sports after cerebral concussion. *Physician Sports Medicine, 14(10)*, 75-83.

Cantu, R. C. (1992). Cerebral concussion in sport. Management and prevention. *Sports Medicine, 14(1),* 64-74.

Cantu, R. C. (1995). Second impact syndrome: A risk in any contact sport. *Physician Sports Medicine, 23,* 27-34.

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. (1998a). Head injuries. In M. R. Safran, D. McKeag, & S. P. Van Camp (Eds.), *Manual of sports medicine.* Retrieved November 27, 2008, from http://books.google.com/books?id=7KzvoOXuEQ8C&pg=PA275&1pg=PA

Cantu, R. C. (1998b). Return to play guidelines after a head injury. *Clinics in Sport Medicine, 17(1),* 45-60.

Cantu, R. C. (1998c). Second-impact syndrome. *Clinical Sports Medicine, 17(1),* 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. *Clinical 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

Carroll, L. J., Cassidy, J. D., Peloso, P. M., Borg, J., von Holst, H., Holm, L. et al. (2004). Prognosis for mild traumatic brain injury: Results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitative Medicine, Suppl., 43*, 84-105.

Caselli, M. A., Gagne, A., & Kaplan, E. (2002). Ice hockey injuries: How to maximize treatment results. *Podiatry Today, 15(8).* Retrieved May 5, 2010, from http://www.podiatrytoday.com/article/666

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.

Casson, I. R., Sham, R., Campbell, E. A., Tarlau, M., & DiDomenico, A. (1982). Neurological and CT evaluation of knocked-out boxers. *Journal of Neurology, Neurosurgery and Psychiatry, 45,* 170-174. Retrieved March 9, 2010 from http://jnnp.bmj.com/content/45/2/170.full.pdf

Casson, I. R., Seigel, O., Sham, R., Campbell, E. A., Tarlau, M., & DiDomenico, A. (1984). Brain damage in modern boxers. *Journal of American Medical Association, 251*, 2663-2667. Retrieved March 9, 2010 from http://jama.ama-assn.org/cgi/reprint/251/20/2663

Catena, R. D., van Donkelaar, P., & Chou, L.S. (2009). Different gait tasks distinguish immediate vs long-term effects of concussion on balance control. *Journal of Neuroengineering and Rehabilitation 2009 July 7; 6:25.* Retrieved 8 September 2009, from *http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2713249.*

Catroppa, C. , & Anderson, V. (2009). Neurodevelopmental outcomes of pediatric traumatic brain injury. *Future Neurology, 4(6),* 811-821. Retrieved January 7, 2010, from http://www.medscape.com/viewarticle/713315

Cekic, M., & Stein, D. G. (2010). Progesterone treatment for brain injury: An update. *Future Neurology, 5(1),* 37-46. Retrieved February 4, 2010, from http://www.medscape.com/viewarticle/715373

Centers for Disease Control and Prevention (CDC). (2003). *Glasgow Coma Scale.* Retrieved July 16, 2008 from http://www.cdc.gov/ncipc/pub-res/tbi_toolkit/physicians/gcs.pdf

Centers for Disease Control and Prevention (CDC). (2004). *Traumatic brain injury facts*. Retrieved July 16, 2008 from www.cdc.gov/ncipc/factsheets/tbi.htm

Centers for Disease Control and Prevention. (2010). *Concussion and mild TBI: Concussion in sports*. Retrieved May 26, 2010 from www.cdc.gov/concussion/sports/index.html

Cernich, A., Reeves, D., Sun, W., & Bleiberg, J. (2007). Automated Neuropsychological Assessment Metrics sports medicine battery. *Archives of Clinical Neuropsychology, 22(1)*, 101-114.

Cervilla, J., Prince, M., & Mann, A. (2000). Smoking, drinking, and incident cognitive impairment: A cohort community based study included in the Gospel Oak project. *Journal of Neurology, Neurosurgery and Psychiatry, 68*, 622-626.

Chamelian, L., &Feinstein, A. (2004). Outcome after mild to moderate traumatic brain injury: The role of dizziness. *Archives of Physical Medicine and Rehabilitation,* 85 (10), 1662-1666.

Chamelian, L., Reis, M., & Feinstein, A. (2004). Six month recovery from mild to moderate traumatic brain injury: The role of APOE-epsilon4 allele. *Brain, 127*, 2621-2628.

Chan, R. C., Shum, D., Toulopoulou, T., & Chen, E. Y. (2008). Assessment of executive functions: Review of instruments and identification of critical issues. *Archives of Clinical Neuropsychology, 23*, 201-206. Retrieved May 26, 2010 from http://www.sciencedirect.com/ doi:10.1016/j.acn.2007.08.010

Chou, L. S., Kaufman, K. R., Walker-Rabatin, A. E., Brey, R. H., & Basford, J. R. (2004). Dynamic instability during obstacle crossing following traumatic brain injury. *Gait Posture, 20*, 245-254. (PubMed)

Christensen, H., Anstey, K. J., Parslow, R. A., Maller, J., Mackinnon, A., & Sachdev, P. (2007). The Brain Reserve hypothesis, brain atrophy and aging. *Gerontology, 53*, 82-95.

Claassen, N. C. W., Krynauw, A. H., Paterson, H., & Mathe, M. (2001). *A standardization of the WAIS-III for English-speaking South Africans.* Pretoria: Human Sciences Research Council.

Clausen, H., McCrory, P., & Anderson, V. (2005). The risk of chronic traumatic brain injury in professional boxing: Change in exposure variables over the past century. B*ritish Journal of Sports Medicine, 39*, 661–664. Retrieved March 16, 2010, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1725298/pdf/v039p00661.pdf

Clinical Psychology Associates. (2008). *Classification of coma, concussion and traumatic brain injury: Concussion, head injury, traumatic brain injury and coma classification systems*. Retrieved November 21, 2008, from http://cpancf.com/headinjuryclassification.asp

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 18, 2010 from http://www.cogstate.com/go/sport

Cohen, M. (1997). *Children's Memory Scale*. San. Antonio: Texas: The psychological Corporation.

Collie, A., Darby, D., & Maruff, P. (2001). Computerized cognitive assessment of athletes with sports related head injury. *British Journal of Sports Medicine, 35(5)*, 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. Retrieved August 13, 2008 from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2077582/

Collie, A., & Maruff, P. (2003). Computerised neuropsychological testing. *British Journal of Sports Medicine, 37 (1)*, 2-3.

Collie, A., Maruff, P., Darby, D. G., & McStephen. (2003). The effects of practice on the cognitive test performance of neurologically normal individuals assessed at brief test-retest intervals. *Journal of the International Neuropsychological Society, 9*, 419-428.

Collie, A., Maruff, P., Makadissi, M., McCrory, P., McStephen, M., & Darby, D.G. (2004). CogSport: Reliability and correlation with conventional cognitive tests used in postconcussion medical evaluations. *Clinical Journal of Sports Medicine, 13,* 28-32.

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 (6)*, 556-559.

Collie, A., McCrory, P., & Makadissi, M. D. (2006). Does history of concussion affect current cognitive status? *British Journal of Sports Medicine, 40 (6)*, 550-551.

Collins, C. L., Micheli, L. J., & Yard, E. E. (2008). Injuries sustained by high school rugby players in the United States, 2005-2006. *Archives of Pediatrics and Adolescent Medicine, 1621)*, 49-54.

Collins, M. (2003, February). Recovery and return to play following sports-related concussion in high school athletes [Abstract]. In K. Podell (Chair), *Sports-related concussion: Focus on high school athletes*. Symposium conducted at The Thirty-First Annual International Neuropsychological Society Meeting, Honolulu, Hawaii. *Journal of the International Neuropsychological Society, 9(2)*, 155.

Collins, M. W., Echemendia, R. J., & Lovell, M. R. (2004a). Collegiate and high school sports. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 111-127). The Netherlands: Swets & Zeitlinger Publishers.

Collins, M. W., Field, M., Lovell, M. R, Iverson, G. L., Johnston, K. M., Maroon, J., & Fu, F. H. (2003). Relationship between post-concussion headache and neuropsychological test performance in high school athletes. *American Journal of Sports Medicine, 31(2)*, 168-173.

Collins, M. W., Grindel, S. H., Lovell, M. R., Dede, D. E., Moser, D. J., Phalin, B. R. 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., Iverson, G. L., Lovell, M. R., McKeag, D. B., Norwig, J., & Maroon, J. (2003). On-field predictors of neuropsychological and symptom deficits 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., Iverson, G. L., Ide, T., & Maroon, J. (2006). Examining concussion rates and return to play in high school football players wearing newer helmet technology: A three-year prospective cohort study. *Neurosurgery, 58(2),* 275-286.

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.

Colvin, A. C., Mullen, J., Lovell, M. R., West, R. V., Collins, M. W., & Groh, M. (2008, July). *The role of concussion history and gender in recovery from soccer-related concussion.* Paper presented at the 34th Annual meeting of the AOSSM, Florida.

Comerford, V. E., Geffen, G. M., May, C., Medland, S. E., & Geffen, L. B. (2002). A rapid screen of the severity of mild traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology, 24(4)*, 409-419.

Committee on Head Injury, Nomenclature of the Congress of Neurological Surgeons (1966). Glossary of head injury, including some definitions of injury to the cervical spine. *Clinical Neurosurgery, 12*, 386-394.

Congress of Neurological Surgeons. Committee on Head Injury Nomenclature: Glossary of head injury. (1991). *Clinical Neurosurgery, 12*, 386-394.

Corrigan, J. D., Selassie, A. W., & Orman, J. A. (2010). The epidemiology of traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 25(2)*, 72-80. Abstract retrieved April 06, 2010, from doi:10.1097/HTR.0b013e3181ccc8b4

Covassin, T., Elbin, R., Kontos, A., & Larson, E. (2010). Investigating baseline neurocognitive performance between male and female athletes with a history of multiple concussions. *Journal of Neurology, Neurosurgery, and Psychiatry, 81(6)*, 597-601. Abstract retrieved June, 17, 2010, from http://www.ncbi.nlm.nih.gov/pubmed/20522868

Covassin, T., Schatz, P., & Swanik, C. B. (2007). Sex differences in neuropsychological function and post-concussion symptoms of concussed collegiate athletes. *Neurosurgery, 61(2)*, 345-350. Abstract retrieved March 26, 2010, from http://www.ncbi.nlm.nih.gov/pubmed/17762747

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., Swanik, C. B., & Sachs, M. L. (2003). Sex differences and the incidence of concussions among collegiate athletes. *Journal of Athletic training, 38*, 238-244. Retrieved March 25, 2010, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC233178/

Covassin, T., Swanik, C. B., Sachs, M. L., Kendrick, Z., Schatz, P., Zillmer, E. et al. (2006). Sex differences in baseline neuropsychological function and concussion symptoms of collegiate athletes. *British Journal of Sports Medicine, 40(11)*, 923-927. Retrieved September 6, 2010, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2465022/

Covassin, T., Weiss, L., Powell, J., & Womack, C. (2007). A maximal exercise test can affect the results of a neurocognitive test (ImPACT test) – therefore, a neuropsychological test battery should not be administered immediately after a practice or a game session. *British Journal of Sports Medicine, 41,* 370-374.

Craft, S., Murphy, C., & Wenstrom, J. (1994). Glucose effects on complex memory and nonmemory tasks: The influence of age, sex and glucoregulatory response. *Physiobiology*, 22, 95-105.

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

Crippen, D. W. (2008). *Head trauma*. Retrieved November 25, 2008, from http://www.emedicine.com/med/TOPIC2820.HTM.

Crippen, D. W. (2009). *Head trauma*. Retrieved August 20, 2009, from http://www.emedicine.com/article/433855-print

Cuddon, C. (2004). The history of rugby union and the rules of rugby union (two sections). *Microsoft Encarta 2005 (Premium Suite)*. Seattle, WA, USA: Microsoft Corporation.

Cullum, C. M., & Thompson, L. L. (1997). Neuropsychological diagnosis and outcome in mild traumatic brain injury. *Applied Neuropsychology, 4 (1),* 6-15.

Cunningham, M. (2007). *Effects of pre-participation sports concussion education upon the anticipated reporting of potential concussion symptoms in high school football players*. Unpublished masters thesis, Pacific University Library. Abstract. Retrieved March 26, 2010, from http://commons.pacificu.edu/pa/24/

Currie Cup. (2010, January 4). In Wikipedia, The Free Encyclopaedia. Retrieved January, 25, 2010, from http://en.wikipedia.org/wiki/Currie_Cup

Daniel, J. C., Olesniewicz, M. H., Reeves, D. L., Tam, D., Bleiberg, J., Thatcher, R. et al. (1999). Repeated measures of cognitive processing efficiency in adolescent athletes: Implications for monitoring recovery from concussion. *Neuropsychiatry, Neuropsychology and Behavioural Neurology, 12(3),* 167-169.

Davies, J. E., & Gibson, T. (1978). Injuries in rugby union football. *British Medical Journal, (2), 23-30.*

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. Abstract retrieved February 8, 2010 from http://bjsm.bmj.com/content/43/Suppl_1/i36.abstract

Dawodu, S. T. (2009). Traumatic Brain Injury (TBI) - Definition, Epidemiology, Pathophysiology. *eMedicine*. Retrieved July, 28, 2010, from http://emedicine.medscape.com/article/326510-overview

Dawson, K. S., Batchelor, J., Meares, S., Chapman, J., & Marosszeky, J. E. (2007). Applicability of neural reserve theory in mild traumatic brain injury. *Brain Injury 21(9)*, 943-949. Abstract retrieved March 17, 2010, from http://www.informaworld.com/smpp/content-db=all-content=a781502999

Deary, I. J., Pattie, A., Taylor, M. D., Whiteman, M., Starr, J., & Whalley, L. (2003) Smoking and cognitive change from age 11 to age 80. *Journal of Neurology, Neurosurgery and Psychiatry,* 74, 1003-1007.

De Beaumont, L., Brisson, B., Lassonde, M., & Jolicoeur, P. (2007). Long-term electrophysiological changes in athletes with a history of multiple concussions. *Brain Injury 21(6)*, 631-644. Abstract retrieved March 17, 2010, from http://www.informaworld.com/smpp/content-db=all-content=a779661952

De Beaumont, L., Lassonde, M., Leclerc, S., & Théoret, H. (2007). Long-term and cumulative effects of sports concussion on motor cortex inhibition. *Neurosurgery, 61(2)*, 329-337. Abstract retrieved March 17, 2010, from http://journals.lww.com/neurosurgery/Abstract/2007/08000/Long_Term_and_Cumulative_Effects_of_Sports.13.aspx

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.

de Kruijk, J. R., Leffers, P., Meerhoff, S., Rutten, J., & Twijnstra, A. (2002). Effectiveness of bed rest after mild traumatic brain injury: A randomised trial of no versus six days of bed rest. *Journal of Neurology, Neurosurgery, and Psychiatry*. Retrieved March 9, 2010, from http://www.accessmylibrary.com/coms2/summary_0286-25795189_ITM

de Kruijk, J. R., Leffers, P., Menheere, P.P., Meerhoff, S., Rutten, J., & Twijnstra, A. (2002). Prediction of post-traumatic complaints after mild traumatic brain injury: Early symptoms and biochemical markers. *Journal of Neurology, Neurosurgery, and Psychiatry*. Retrieved March 9, 2010, from http://www.accessmylibrary.com/article-1G1-95501231/prediction-post-traumatic-complaints.html

de Boussard, C. N., Lundin, A., Karlstedt D., Edman, G., Bartfai, A., & Borg, J. (2005). S100 and cognitive impairment after mild traumatic brain injury. *Journal of Rehabilitative Medicine, 37*, 53–57.

Delaney, J. (2005). Concussion risk factors and return-to-play variables. *Physician and Sports Medicine, 33(9)*, 6.

Delaney, J. S., Abuzeyad, F., Correa, J. A., & Foxford, R. (2005). Recognition and characteristics of concussions in the emergency department population. *The Journal of Emergency Medicine, 29(2)*, 189-197.

Delaney, J. S., Al-Kashmiri, A., Drummond, R., & Correa, J. A. (2008). The effect of protective headgear on head injuries and concussion in adolescent football (soccer) players. *British Journal of Sports Medicine, 42(2),* 110-115.

Delaney, J. S., Lacroix, V. J., Gagne, C., & Antoniou, J. (2001). Concussions among university football and soccer players: A pilot study. *Clinical Journal of Sports Medicine, 11*, 234-240.

Delaney, J. S., Lacroix, V. J., Leclerc, S., & Johnston, K. M. (2000). Concussion during the 1997 Canadian football league season. *Clinical Journal of Sport Official: Official Journal of the Canadian Academy of Sport, 10*, 9-14.

Delaney, J. S., Lacroix, V. J., Leclerc, S., & Johnston, K. M. (2002). Concussion among university football and soccer players. *Clinical Journal of Sports Medicine, 12*, 331-338.

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 and practice effects of rapid screening of mild traumatic brain injury. *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 mild traumatic brain injury. *Brain Injury, 20 (13-14)*, 1345-1354.

De Monte, V. E., Geffen, G. M., May, C., & McFarland, K. A. (2004). Double cross-validation and improved sensitivity of the rapid screen of mild traumatic brain injury. J*ournal of Clinical and Experimental Neuropsychology, 26(4)*, 628-644. [Abstract].

De Monte, V. E., Geffen, G. M., May, C. R., McFarland, K., Heath, P., & Neralic, M. (2005). The acute effects of mild traumatic brain injury on finger tapping with and without word repetition. J*ournal of Clinical and Experimental Neuropsychology, 27(2)*, 224-239.

den Heijer, T., Geerlings, M. I., Hoebeek, F. E., Hofman, A., Koudstall, P. J., & Breteler, M. M. (2006). Use of hippocampal and amygdalar volumes on magnetic resonance imaging to predict dementia on cognitively intact elderly people. *Archives of General Psychiatry, 63(1)*, 57-62. Retrieved September 14, 2010, from http://archpsyc.ama-assn.org/cgi/content/full/63/1/57

Dennis, M., Spiegler, B. J., & Hetherington, R. (2000). New survivors for the new millennium: Cognitive risk and reserve in adults with childhood brain insults. *Brain and Cognition,* 42, 102-105.

Department of Sport and Recreation (2010). *Dimensions for rugby league*. Retrieved October 27, 2010, from http://www.dsr.wa.gov.au/rugbyleaguedimensions

Department of Veteran Affairs. (2009a). *VA/DoD Clinical practice guideline for management of concussion/mild traumatic brain injury*. Retrieved February 24, 2010, from http://www.healthquality.va.gov/mtbi/concussion_mtbi_full_1_0.pdf

Department of Veteran Affairs. (2009b). *VA/DoD Clinical practice guideline summary for management of concussion/mild traumatic brain injury*. Retrieved February 24, 2010, from http://www.healthquality.va.gov/mtbi/concussion_mtbi_sum_1_0.pdf

Dick, R. W. (2003). *National Collegiate Athletic Association (NCAA) Injury Surveillance System 2002-2003*. Indianapolis, Indiana: National Collegiate Athletic Association.

Dick, R. W. (2009). Is there a gender difference in concussion incidence and outcomes? *British Journal of Sports Medicine, 43, (Suppl 1), i46-i50.*

Dicker, G., & Maddocks, D. (1988). An objective measure of recovery from concussion in Australian Rules footballers. *The Australian Journal of Science and Medicine in Sport, December, 17.*

Dik, M. G., Deeg, D. J., Visser, M., & Jonker, C. (2003). Early life activity and cognition at old age. *Journal of Clinical and Experimental Neuropsychology, 25*, 643-653.

Dikmen, S. S., Heaton, R. K., Grant, I., & Temkin, N. R. (1999). Test-retest reliability and practice effects of expanded Halstead-Reitan neuropsychological test battery. *Journal of the International Neuropsychological Society, 5*, 346-356.

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.

Dikmen, S., Machamer, J., & Temkin, N. (2001). Mild head injury: Facts and artefacts. *Journal of Clinical and Experimental Neuropsychology, 23(6), 729-738.*

Dimitrov, D. M., & Rumrill, P. D. (2003). Pretest-posttest designs and measurement of change. *Work, 20*, 159–165.

Di Sclafani, V., Clark, H. W., Tolou-Shams, M., Bloomer, C. W., Salas, G. A., Norman, D. et al. (1998). Premorbid brain size is a determinant of functional reserve in abstinent crack-cocaine-alcohol-dependent adults. *Journal of International Neuropsychological Society, 4(6),* 559-565.

Dixon, T., & Malinoski, D. J. (2009). Devastating brain injuries: Assessment and management part 1: Overview of brain death. *Western Journal of Emergency Medicine.* Retrieved April 23, 2009, from http://www.medscape.com/viewarticle/587273

Downs, D. S., & Abwender, D. (2002). Neuropsychological impairment in soccer athletes. The *Journal of Sports Medicine and Physical Fitness, 42,* 103-107.

Dupuis, F., Johnston, K. M., Lavoie, M., Lepore, F., & Lassonde, M. (2000). Concussions in athletes produce brain dysfunction as revealed by event-related potentials. *Clinical Neuroscience ans Neuropathology, 11(18)*, 4087-4092.

Durazzo, T. C., Gazdzinski, S., & Meyerhoff, D. J. (2007). The neurobiological and neurocognitive consequences of chronic cigarette smoking in alcohol use disorders. *Alcohol and Alcoholism, 42*, 174-185. Retrieved October, 20, 2010, from doi: 10.1093/alcalc/agm020

Durkin, T. E. (1977). A survey of injuries in a 1st class rugby union football club from 1972 – 1976. *British Journal of Sports Medicine, 11 (7), 7-11.*

Duthie, G., Pyne, D., & Hooper, S. (2003). Applied Physiology and game analysis of Rugby Union. *Sports Medicine, 33(13),* 973-991.

Dvorak, J., McCrory, P., Aubry, M., Molloy, M., & Engebretsen, L. (2009). Concussion sans frontieres. *British Journal of Sports Medicine, 43(1).* Abstract retrieved February 8, 2010 from http://bjsm.bmj.com/content/43/Suppl_1/i1.abstract

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)*, 48-55.

Echemendia, R. J., & Cantu, R. C. (2004). Return to play following cerebral brain injury. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 479-498). The Netherlands: Swets & Zeitlinger Publishers.

Echemendia, R. J., Herring, S., & Bailes, J. (2009). Who should conduct and interpret the neuropsychological assessment in sports-related concussion? Abstract retrieved February 8, 2010, from http://bjsm.bmj.com/content/43/Suppl_1/i32.abstract

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 Sport Medicine, 11*, 23-31.

Echemendia, R. J., Putukian, M., & Phillips, T. G. (1997). Neuropsychological Baseline Testing in the Management of Head Injured college Athletes: The Penn State Concussion Program. *Clinical Journal of Sport Medicine, 7(4)*, 319.

Eckner, J. T., & Kutcher, J. S. (2010). Concussion symptom scales and sideline assessment tools: A critical literature update. *Current Sports Medical Reports, 9(1)*, 8-15.

Egger, J., De Mey, H., & Janssen G. (2007). Assessment of executive functioning in psychiatric disorders: Functional diagnosis as the overture of treatment. *Clinical Neuropsychiatry 4(3)*, 111-116. Retrieved October 11, 2010, from http://www.clinicalneuropsychiatry.org/pdf/egger.pdf

Ellemberg, D., Henry, L. C., Macciocchi, S. N., Guskiewicz, K. M., & Broglio, S. P. (2009). Advances in sport concussion assessment: From behavioral to brain imaging measures. *Journal of Neutotrauma (26)*, 2365-2382. Retrieved October 12, 2010, from http://www.liebertonline.com/doi/pdfplus/10.1089/neu.2009.0906

Ellemberg, D., Leclerc, S., Couture, S., & Daigle, C. (2007). Prolonged neuropsychological impairments following a first concussion in female university soccer athletes. *Clinical Journal of Sport Medicine, 17*, 369–374.

Elwood, P. C., Gallacher, J. E., Hopkinson, C. A., Pickering, J., Rabbitt, P., Stollery, B. et al. (1999). Smoking, drinking, and other life style factors and cognitive function in men in the Caerphilly cohort. *Journal of Epidemiology and Community Health, 53(1)*, 9-14.

Elwood, R. W. (1997). Episodic and semantic memory components of verbal paired-associate learning. *Assessment, 4(1)*, 73-77. (Abstract).

Erlanger, D., Feldman, D. J., & Barth, J. T. (2001). Statistical techniques for interpreting post-concussion neuropsychological tests. *British Journal of Sports Medicine, 35(5)*, 370-371.

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., Kaushik, T., Cantu, R., Barth, J. T., Broshek, D. K., Freeman, J. R. et al. (2003). Symptom-based assessment of the severity of a concussion. *Journal of Neurosurgery, 98(3)*, 477-484.

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.

Ewing, R., McCarthy, D., Gronwall, D., & Wrightson. P. (1980). Persisting effects of minor head injury observable during hypoxic stress. *Journal of Clinical Neuropsychology, 2(2),* 147-155.

Eyres, S., Carey, A., Gilworth, G., Neumann, V., & Tennant, A. (2005). Construct validity and reliability of the Rivermead Post-Concussion Symptoms Questionnaire. *Clinical Rehabilitation, 19*, 878–887. Abstract retrieved October 14, 2010 from http://cre.sagepub.com/content/19/8/878.abstract

Fairnpour, R., Miller, E. N., Satz, P., Selnes, O. A., Cohen, B. A., Becker, J. T. et al. (2003). Psychosocial risk factors of HIV morbidity and mortality: Findings from the Multicenter AIDS cohort study. *Journal of Clinical and Experimental Neuropsychology, 25*, 654-670.

Farace, E., Barth, J. T., Brosherk, D. K., Hollier, J. A., DeAngelo, K. B., & Shaffrey, M. E. (2000). Neurosurgical management of concussions in rugby football. *Neurosurgery, 47(2)*, 513-514. (Abstract).

Farace, E., Ferree, R. M., Hollier, J. A., Barth, J. T., & Shaffrey, M. E. (2003, February). Trails A: Neurocognitive effect of previous concussions in a women's rugby sample [Abstract]. In K. Podell (Chair), *Sports-related concussion: Focus on high school athletes*. Symposium conducted at The Thirty-First Annual International Neuropsychological Society Meeting, Honolulu, Hawaii. *Journal of the International Neuropsychological Society, 9(2),* 207.

Faul, M., Xu, L., Wald, M. M., & Coronado, V. G. (2010). *Traumatic Brain Injury in the United States: Emergency Department Visits, Hospitalizations and Deaths 2002–2006*. Retrieved May 26.2010, from http://www.cdc.gov/traumaticbraininjury/tbi_ed.html

Fazio, V. C., Lovell, M., Pardini, J. E., & Collins, M. W. (2007). The relation between post concussion symptoms and neurocognitive performance in concussed athletes. *NeuroRehabilitation, 22(3),* 207-216.

Feise, R. J. (2002). Do multiple outcome measures require p-value adjustment? *BMC Medical Research Methodology, 2*, 8. Retrieved November 11, 2010, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC117123/

Ferguson, R. J., Mittenberg, W., Barone, D. F., & Schneider, B. (1999). Postconcussion syndrome following sports-related head injury: Expectation as etiology. *Neuropsychology, 13(4),* 582-589.

Fick, D. S. (1995). Management of concussion in collision sports; guidelines for the sidelines. *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 Paediatrics, 142(5),* 546-553.

FIFA. (2007). FIFA Big Count 2006: 270 million people active in football. Retrieved June 8, 2010, from http://www.fifa.com/aboutfifa/media/newsid=529882.html

Finch, C., Best, J., McIntosh, A., Chalmers, D., & Eime, R. (2002). *Research report: Preventing rugby union injuries*, Department of Epidemiology and Preventive Medicine, Monash University.

Fischer, J. M., & Vaca, F .E. (2004). Sport-related concussion in the emergency department. *Topics in Emergency Medicine, 26(3)*, 260-266.

Flanagan, S. (1999). Psychiatric management of mild traumatic brain injury. *The Mount Sina Journal of Medicine, 66(3),* 152-159.

Ford, M. R., & Khalil, M. (1996a). Evoked potential findings in mild traumatic brain injury 1: Middle latency component attenuation. *Journal of Head Trauma Rehabilitation, 11(3)*, 1-15.

Ford, M. R., & Khalil, M. (1996b). Evoked potential findings in mild traumatic brain injury 2: Scoring system and individual discrimination. *Journal of Head Trauma Rehabilitation, 11(3)*, 16-21.

Franzen, M. D., Frerichs, R. J., & Iverson, G. L. (2004). Reliability, validity and the measurement of change in serial assessments of athletes. In M. R. Lovell, R.J. Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 299-321). The Netherlands: Swets & Zeitlinger Publishers.

Frencham, K. A., 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.

Friedman, D. (2003). Cognition and aging: A highly selective overview of event-related potential (ERP) data. *Journal of Clinical and Experimental Neuropsychology, 25(5)*, 702-720.

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.

Fryatt, K., Rose, J., & Slessor, J. (2001). Alberta Occupational Medicine Newsletter: Summer 2001. Retrieved September 3, 2007, from http://hdl.handle.net/1880/43243

Gabbe, B, Finch, C., Wajsweiner, H., & Bennell, K. (2002). Australian football: Injury profile at the community level. *Journal of Science and Medicine in Sport, 5(2)*, 149-160.

Gabbett, T. J. (2003) Incidences of injury in semi-professional rugby league players. *British Journal of Sports Medicine, 37*, 36-44.

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.

Gandey, A. (2010). New brain death guidelines issued. *Medscape Medical News.* Retrieved June, 12, 2010, from http://www.medscape.com:80/viewarticle/723342

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., Hutton, S. J., Russell, E. B., & Macleod, D. A. (2000). Impact of professionalism on injuries in rugby union. *British Journal of Sports Medicine, 34*, 348-351.

Gentilini, M., Nichelli, P., & Schoenhuber, R. (1989). Assessment of attention in mild head injury. In H. Levin, M. Eisenberg & A.L. Benton (Eds.), *Mild head injury*. New York: Oxford University Press.

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

Georgetown EEG/ERP Laboratory (2009). *Electroencephalography/Event Related Potentials*. (EEG/ERP) Laboratory. Retrieved October 20, 2009, from http:// brainlang.georgetown.edu / erplab.htm.

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

Gessel, L. M., Fields, S. K., Collins, C. L., Dick, R. W., & Comstock, R. D. (2007). Concussion among United States High School and collegiate athletes. *Journal of Athletic Training*, *42(4)*, 495–503.

Gianotti, S., & Hume, P. A. (2007). Concussion sideline management intervention for rugby union leads to reduced concussion claims. *NeuroRehabilitation, 22(3)*, 181-189. Abstract retrieved March 26, 2010, from http://www.ncbi.nim.nih.gov/pubmed/17917168

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.

Gioia, G. A., Schneider, J. C., Vaughan, C. G., & Isquith, P. K. (2009). Which symptom assessments and approaches are uniquely appropriate for paediatric concussion? *British Journal of Sport Medicine, 43(1)*,i13. Abstract retrieved February 8, 2010 from http://bjsm.bmj.com/content/43/Suppl_1/i13.abstract

Giza, C. C., & Hovda, D. A. (2004). The pathophysiology of traumatic brain injury. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 45-69). The Netherlands: Swets & Zeitlinger Publishers.

Golden, C. A. (1978). *Stroop color and word test manual*. Chicago: Stoelting Company.

Gonder-Frederick, L., Hall, J. L., Vogt, J., Cox, D. J., Green, J., & Gold, P. E. (1987). Memory enhancement in elderly humans: Effects of glucose ingestion. *Physiology and Behaviour*, 41, 503-504.

Gosselin, N., Theriault, M., Leclerc, S., Montplaisir, J., & Lassonde, M. (2006). Neurophysiological anomalies in symptomatic and asymptomatic concussed athletes. *Neurosurgery, 58(6)*, 1151-1161.

Gouvier, W. D., Cubic, B., Jones, G., Brantley, P., & Cutlip, Q. (1992). Postconcussion symptoms and daily stress in normal and head-injured college populations. *Archives of Clinical Neuropsychology, 7(3)*, 193-211.

Gray, W. (2010). Rugby season too trying for staunchest of fans, *New Zealand Herald.* Retrieved November 27, 2010, from http://www.nzherald.co.nz/opinion/news/article.cfm?c_id=466&objectid=10689672

Green, G., & Jordan, S. (1996). Chronic head and neck injuries. In W. Garret, D. Kirkendall, & S. Contiguglia, S. (Eds.), *The US football sports medicine book* (pp. 191-204). Baltimore: Williams & Wilkins.

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. M. (1977). Paced Auditory Serial-Addition task: A measure of recovery from concussion. *Perceptual Motor Skills, 44*, 367-373.

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 function after minor head injury. *The 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.

Grouvier, W. D., Uddo-Crane, M., & Brown, L .M. (1998). Base rates of post-concussive symptoms. *Archives of Clinical Neuropsychology, 8*, 273-278.

Grubenhoff, J. A., Kirkwood, M., Gao, D., Deakyne, S., & Wathen, J. (2010). Evaluation of the standardized assessment of concussion in a pediatric emergency department. *Pediatrics, 126(4)*, 688-695. Abstract retrieved October 19, 2010 from http://www.ncbi.nlm.nih.gov/pubmed/20819901

Guilmette, T. J., & Rasile, D. (1995). Sensitivity, specificity, and diagnostic accuracy in three verbal memory measures in the assessment of mild brain injury. *Neuropsychology, 9*, 338-344.

Guskiewicz, K. M. (2000). Postural stability assessment following concussion: One piece of the puzzle. *Clinical Journal of Sports Medicine, 11*, 182-189.

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-life cognitive impairment in retired professional football players. *Neurosurgery, 57(4)*, 719-726.

Guskiewicz, K. M., Marshall, S. W., Bailes, J., McCrea, M., Harding, H. P., Jr., Matthews, A. et al. (2007). Recurrent concussion and risk of depression in retired professional football players. *Medical Science and Sports Exercise, 39(6)*, 903-909.

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. *The Journal of the American Medical Association, 290(19),* 2549-2555.

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. Abstract retrieved September 11, 2009, from PubMed database.

Guskiewicz, K. M., Riemann, B. L., Perrin, D., & Nashner, L. M. (1997). Alternative approaches to the assessment of mild head injury in athletes. *Official Journal of the American College of Sports Medicine, 29(7)*, S213-S221.

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,* 263-273.

Guskiewicz, K. M., Weaver, N. L., Padua, D. A., & Garrett, W. E. (2000). Epidemiology of concussion in collegiate and high school football players. *American Journal of Sports Medicine, 28(5),* 643-650.

Halstead, M. E., & Walter, K. D. (2010). Sport-related concussion in children and adolescents. *Pediatrics, 126(3)*, 597-615. Retrieved September 6, 2010, from http://pediatrics.aappublications.org/cgi/reprint/126/3/597.pdf

Halterman, C. I., Langan, J., Drew, A., Rodriguez, E., Osternig, L. R., Chou, L. et al. (2005). Tracking the recovery of visuospatial attention deficits in mild traumatic brain injury. *Brain, 129*, 747-753. Retrieved August 18, 2010 from http://www.uoregon.edu/~paulvd/lab/pvd-brain-05.pdf

Hannay, J. H., & Levin, H. S. (1985). Selective reminding test: An examination of the equivalence of four forms. *Journal of Clinical and Experimental Neuropsychology, 7*, 251-263.

Hartlage, L. C., Durant-Wilson, D., & Patch, P. C. (2001). Persistent neurobehavioural problems following mild traumatic brain injury. *Archives of Clinical Neuropsychology, 16(6)*, 561-570.

Hatfield, R., Bieliauskas, L., Begloff, P., Steinberg, B., & Kauszler, M. (2004). Youth hockey. In M. R. Lovell, R. J. Echemendia, J.T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 129-147). The Netherlands: Swets & Zeitlinger Publishers.

Haydel, M. J., Preston, C. A., Mills, T. J., Luber, S., Blaudeau, E., & DeBlieux, P. M. C. (2000). Indications for computed tomography in patients with minor head injury. *The New England Journal of Medicine, 343(2)*, 100-105.

Hayman-Abello, S. E., Rourke, B. P., & Fuerst, D. R. (2003). Psychosocial status after pediatric traumatic brain injury: A subtype analysis using the child behaviour checklist. *Journal of the International Neuropsychological Society, 9*, 887-898.

Heilbronner, R. L., Buck, P., & Adams, R. L. (1989). Factor analysis of verbal and nonverbal clinical memory tests. *Archives of Clinical Neuropsychology, 4(4)*, 299-309.

Heilbronner, R. L., Bush, S. S., Ravdin, L. D., Barth, J. T., Iverson, G. L., Ruff, R. M. et al. (2009). Neuropsychological consequences of boxing and recommendations to improve safety: A national academy of neuropsychology education paper. *Archives of Clinical Neuropsychology 24*, 11 – 19. Retrieved March 12, 2010, from http://nanonline.org/NAN/Files/Research%20and%20Publications/NP%20Consequences%20of%20Boxing%20-%20NAN%20paper.pdf

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

Heilbronner, R. L., & Ravdin, L. D. (2004). Boxing. In M. R. Lovell, R. L. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic Brain Injury in Sports* (pp. 231-254). The Netherlands: Swets & Zeitlinger Publishers.

Hemmer, M. (2000). Outcome prediction as a guide to withdrawing or withholding of therapy. *European Journal of Anesthesiology, 17*, 27-29. Abstract retrieved July, 28, 2010, from http://journals.lww.com/ejanaesthesiology/Fulltext/2000/00001/Outcome_prediction_as_a_guide_to_withdrawing_or.16.aspx#

Hermann, N., Rapoport, M. J., Rajaram, R. D., Chan, F., Kiss, A., Ma, A. K. et al. (2009). Factor analysis of the Rivermead Post-Concussion Symptoms Questionnaire in mild-to-moderate traumatic brain injury patients. *Journal of Neuropsychiatry and Clinical Neuroscience, 21*, 181-188. Retrieved October 14, 2010, from http://neuro.psychiatryonline.org/cgi/content/full/21/2/181

Hibbard, M. R., Uysal, S., Kepler, K., Bogdany, J., & Silver, J. (1998). Axis I psychopathology in individuals with traumatic brain injury. *Journal of Head Trauma Rehabilitation, 13(4), 24-39.*

Hickey, J. (2004). Concussion. *Canadian Family Physician.* Retrieved November 7, 2007, from http://www..cfpc/2004/Feb/vol50-feb-clinical-2asp?stype=advanced&

Hillis, W. S., Mc McIntyre, P. D., Maclean, J., Goodwin, J. F., & McKenna, W. J. (1994). ABC of sports medicine: Sudden death in sport. *British Medical Journal, 309*, 657-660.

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* (pp. 169–192). 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.

History of Rugby Union. (2010, January 16). *History of rugby union*. In Wikipedia, the free encyclopaedia. Retrieved January, 22, 2010, from http://en.wikipedia.org/wiki/History_of_rugby_union

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. et al. (2009). Texture analysis of corpus callosum in mild traumatic brain injury patients. Abstract retrieved February 3, 2010 from http://www.springerlink.com/content/g6m194086246r674/

Hollis, S. J., Stevenson, M. R., McIntosh, A. S., Shores, E. A., Collins, M. W., & Taylor, C. B. (2009). Incidence, risk, and protective factors of mild traumatic brain injury in a cohort of Australian nonprofessional male rugby players. *American Journal of Sport Medicine, 37*, 2328-2333. Abstract retrieved March 30, 2010 from http://ajs.sagepub.com/content/37/12/2328.abstract

Hootman, J. M., Dick, R., & Agel, J. (2007). Epidemiology of collegiate injuries for 15 sports: Summary and recommendations for injury prevention initiatives. *Journal of Athletic Training, 42(2)*, 311-319.

Hoskins, W. T., Chiro, B., & Pollard, H. (2003). Australian Rules football injuries in children and adolescents. *ACO, 11(2), 49-56.* Retrieved September 29, 2010, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2051317/pdf/aco112-049b.pdf

Hovda, D. A., Prins, M., Becker, D. P., Lee, S., Bergsneider, M., & Martin, N. A. (1999). Neurobiology of concussion. In J. E. Bailes., M. R. Lovell, & J. C. Maroon (Eds.), *Sports-related concussion* (pp. 327-332). St. Louis: Quality Medical Publishing.

Hsiang, J. N., Yeung, T., Yu, A. L., & Poon, W. S. (1997). High-risk mild head injury. *Journal of Neurosurgery, 87,* 234-238.

Hsu, J. C. (1996). *Multiple comparisons theory and methods*. London: Chapman & Hall.

Hugenholtz, H., & Richard, M. T. (1982). Return to athletic competition following concussion. *Canadian Medical Associate Journal, 127*, 827-829.

Hugenholtz, H., Stuss, D. T., Stethem, L. L., & Richard, M. T. (1988). How long does it take to recovery from mild concussion? *Neurosurgery, 22(5),* 833-858.

Huitt, W., Hummel, J., & Kaeck, D. (1999). Internal and external validity: General issues. Retrieve November 5, 2010, from http://www.edpsycinteractive.org/topics/intro/valdgn.html

ImPACT. (2004). *Clinical Interpretation Manual for ImPACT 3.0*. Pittsburgh, USA: ImPACT.

ImPACT. (2007a). *Graphic display of data*. Retrieved September 18, 2007, from http://www.impacttest.com/graphicdisplay.php.

ImPACT. (2007b). *ImPACT Background*. Retrieved September 18, 2007, from http://www.impacttest.com/impactbackground.php.

ImPACT. (2007c). *Test section 3: Neuropsychological Tests (Baseline and Post-Concussion)*. Retrieved September 18, 2007, from http://www.impacttest.com/testmodules.php

ImPACT. (2010). *Overview and Features of the ImPACT Test*. Retrieved October 23, 2010, from http://impacttest.com/about/background

Ingebrigsten, 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, 48(4)*, 760-765.

Ingebrigsten, T., Waterloo, K., Marup-Jensen, S., Attner, E., & Romner, B. (1998). Quantification of post-concussion symptoms 3 months after minor head injury in 100 consecutive patients. *Journal of Neurology, 245(9)*, 609-612.

International Rugby Board [IRB]. (2009a). *A beginner's guide to rugby union.* Retrieved November 20, 2009, from http://www.irb.com/mm/document/training/0/beginners20guide20en_7391.pdf

International Rugby Board [IRB]. (2009b). *Law amendments explained*. Retrieved November 20, 2010, from http://www.irb.com/mm/document/lawsregs/0/090430sglaexplanatory_7684.pdf

International Rugby Board [IRB]. (2010a). *Education top priority for player welfare (5 November 2010)*. Retrieved November 10, 2010, from, http://www.irb.com/newsmedia/mediazone/pressrelease/newsid=2040676.html#education+priority+player+welfare

International Rugby Board [IRB]. (2010b). *IRB organisation*. Retrieved November 10, 2010, from, http://www.irb.com/aboutirb/organisation/index.html

International Rugby Board [IRB]. (2010c). *Regulation 10: Medical*. Retrieved April 12, 2010, from http://www.irb.com/mm/document/lawsregs/0/regulation10090603%5f8285.pdf

International Touch Rugby. (2008). *What is touch rugby?* Retrieved January, 29, 2010, from http://touchrugby.com/what_is_touch

Iverson, G. (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., Brooks, B. L., Collins, M. W., & Lovell, M. R. (2006). Tracking neuropsychological recovery following concussion in sport. *Brain Injury, 20*, 245-252.

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., Franzen, M. D., Lovell, M. R., & Collins, M. W. (2003). Construct validity of ImPACT in athletes with concussion. *Presented at the National Academy of Neuropsychologist's Annual Meeting.* Retrieved April 6, 2010, from http://www.impacttest.com/ArticlesPage_images/Articles_Docs/4ConstructValidityNAN2004.pdf

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. (2004b). Relation between subjective fogginess and neuropsychological testing following concussion. *Journal of the International Neuropsychological Society, 10*, 904-906.

Iverson, G. L., & Lange, R. T. (2003). Examination of 'postconcussion-like' symptoms in a healthy sample. *Applied Neuropsychology, 10(3)*, 137-144.

Iverson, G. L., Lange, R. T., & Franzen, M. D. (2005). Effects of mild traumatic brain injury cannot be differentiated from substance abuse. *Brain Injury, 19(1)*, 11-18.

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.*

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(6),* 683-689.

Iverson, G. L., Lovell, M. R., & Collins, M. W. (2010). *Normative data: For Foxpro (Original Platform) version of ImPACT 2003 (for reference only)*. Retrieved October 23, 2010 from http://impacttest.com/publications/baseline_data/normative

Iverson, G. L., Lovell, M. R., Collins, M. W., & Norwig, J. (2002). *Tracking recovery from concussion using ImPACT: Applying Reliable Change methodology*. Paper presented at the National Academy of Neuropsychology Annual Conference. Miami, Florida.

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 Neuropsycholoy, 15 (7), 643-648.*

Iverson, G., & McCracken, L. M. (1997). "Post-concussive" symptoms in persons with chronic pain. *Brain Injury, 11*, 783-790.

Iverson, G. L., Zasler, N. D., & Lange, R. T. (2007). Post-concussive disorder. In N. D. Zasler, D. I. Katz AND R. D. Zafonte (Eds.), *Brain Injury Medicine: Principles and Practice.* New York: Demos Medical Publishing LLC.

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.

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.

Jagaroo, V. (1999). Methodological commentary: Towards an analytic framework for the visuospatial domain: Spatial reference frames, cognitive operations, and neural systems. *Journal of Neuroradiology, 25, 738-745.*

Jagaroo, V. (2009). *Neuroinformatics for neuropsychologists*. New York: Springer Science Medicine.

Jantzen, K. J., Anderson, B., Steinberg, F L., & Kelso, J. A. (2004). A prospective functional MR imaging study of mild traumatic brain injury in college football players. *American Journal of Clinical and Experimental Neuropsychology, 21(1)*, 134-146.

Jeanty, L., & Della Porta, G. Z. (2009). Collisions and particle physics. *Harvard University Summer School in Physics, August 2009.* Poster retrieved April 7, 2010, from http://www.ippp.dur.ac.uk/export/sites/IPPP/Workshops/09/SUSSP65/Posters/Rugby.pdf

Jeffrey, S. (2010). Reduced Melatonin Associated With Sleep Disturbance in Traumatic Brain Injury. *Medscape Medical News* Retrieved May 29, 2010, from http://www.medscape.com/viewarticle/722485?src=mpnews&spon=26&uac=119539SX

Jellinger, K. A. (2004). Head injury and dementia*. Current Opinion in Neurology, 17*, 719-723.

Jennett, B., & Bond, M. (1975). Assessment of outcome after severe brain damage: A practical scale. *The Lancet, 305(795)*, 480-484.

Jennett, B., & Frankowski, R. F. (1990). Epidemiology of head injury. In R. Brinkman (Ed.), *Handbook of clinical neurology* (pp. 1-16). New York: Elsevier.

Jeret, J. S., Mandell, M., Anziska, B., Lipitz, M., Vilceus, A. P., Ware, J .A. et al. (1993). Clinical predictors of abnormality disclosed by computer tomography after mild brain trauma. *Neurosurgery, 32(1)*, 15-16.

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. (2007). Letters to the editor. *Clinical Journal of Sport Medicine, 17(4)*, 330.

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.

Johnston, K. M., Ptito, A., Chankowsky, J., & Chen, J. K. (2001). New frontiers in diagnostic imaging in concussive head injury. *Clinical Journal of Sports Medicine, 11(3)*, 166-175.

Jones, M. K. (1974). Imagery as a mnemonic aid after left temporal lobectomy: Contrast between material-specific and generalized memory disorders. *Neuropsychologia 12*, 12–30.

Jones, J. H., Violen, S. L., Laban, M. M., Schynoll, W. G., & Krome, R. L. (1992). The incidence of post minor traumatic brain injury syndrome. A retrospective survey of treating physicians. *Archives of Physical Medicine and Rehabilitation, 73*, 145-146.

Jones, W. P., Loe, S. A., Krach, S. K., Rager, R. Y., & Jones, H. M. (2008). Automated Neuropsychological Assessment Metrics (ANAM) and Woodcock-Johnson III tests of cognitive ability: A concurrent validity study. *The Clinical Neuropsychologist, 22*, 305–320, Retrieved October 13, 2010, from http://www.iapsych.com/articles/jones2008.pdf

Jordan, A. B. (1997). *The Shuttle Effect : The development of a model for the prediction of variability in cognitive test performance across the adult life span.* Unpublished doctoral dissertation, Rhodes University, South Africa.

Jordan, B. D. (1987). Neurologic aspects of boxing. *Archives of Neurology, 44(4)*, 453-459.

Jordan, B. D. (2004). Genetic aspects of traumatic brain injury. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 358-374). The Netherlands: Swets & Zeitlinger Publishers.

Jordan, B. D., Matser, E., Zimmerman, R., & Zazula (1996). Sparring and cognitive function of professional boxers. *Physician and Sports Medicine, 24(5),* 87-98.

Jordan, B. D., Relkin, N. R., Ravdin, L. D., Jacobs, A. R., Bennett, A., & Gandy, S. (1997). Apolipolprotein E e4 associated with chronic traumatic brain injury in boxing. *Journal of the American Medical Association, 278(2),* 136-140. Retrieved March 10, 2010, from http://jama.ama-assn.org/cgi/reprint/278/2/136.pdf

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

Joy, S., Kaplan, E., & Fein, D. (2004). Speed and memory in the WAIS-III Digit Symbol—Coding subtest across the adult lifespan. *Archives of Clinical Neuropsychology, 19*, 759–767. Retrieved October 11, 2010, from http://www.pearsonassessments.com/hai/images/dotcom/sciencedirect/WAIS-III_DS.pdf

Junge, A., Cheung, K., Edwards, T., & Dvorak, J. (2004). Injuries in youth amateur soccer and rugby players - comparison of incidence and characteristics. *British Journal of Sports Medicine, 38*, 168-172.

Kalmijn, S., van Boxtel, M. P., Verschuren, M. W., Jolles, & Launer, L. J. (2002). Cigarette smoking and alcohol consumption in relation to cognitive performance in middle age. *American Journal of Epidemiology,156*, 936-944.

Kashluba, S., Casey, J. E., & Paniak, C. (2006). Evaluating the utility of ICD-10 diagnostic criteria for postconcussion syndrome following mild traumatic brain injury. *Journal of the International Neuropsychological Society, 12(1),* 111-118.

Kashluba, S., Paniak, C., Blake, T., Reynolds, S., Toller-Lobe, G., & Nagy, J. (2004). A longitudinal, controlled study of patient complaints following treated mild traumatic brain injury. *Archives of Clinical Neuropsychology, 19,* 805-816.

Kaufman, A. S. (2003). Practice effects. *Speech and Language Forum.* Retrieved March 15, 2008, from http://www.speechand language.com/café/13.asp

Kaste, M., Vikki, J., Sainio, K., Kuurne, T., Katevuo, K., & Meurala, H. (1982). Is chronic brain damage in boxers a hazard of the past? *Lancet, 2,* 1186-1188.

Katzman, R., Terry, R., De Teresa, R., Brown, T., Davies, P., Fuld, P. et al. (1988). Clinical, pathological, and neurochemical changes in dementia: A subgroup with preserved mental status and numerous neurocortical plaques. *Annals of Neurology, 23*, 138-144.

Kaut, K. P., DePompei, R., Kerr, J., & Congeni, J. (2003). Reports of head injury and symptom knowledge among college athletes: Implications for assessment and educational intervention. *Clinical Journal of Sport Medicine, 13*, 213-221.

Kelly, J. C. (2010). Traumatic brain injuries rising 3 times faster than population rate. *Medscape Medical News.* Retrieved March 27, 2010, from http://www.medscape.com.viewarticle/719184

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(3)*, 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., & Fuller, C. W. (2008). The epidemiology of head injuries in English professional rugby union. *Clinical Journal of Sport Medicine, 18(3)*, 227-234. Abstract retrieved March 19, 2010, from doi: 10.1097/JSM.0b013e31816a1c9a

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. Abstract retrieved September 30, 2010, from http://bjsm.bmj.com/content/42/7/595.abstract

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(3)*, 153-162. Retrieved April 27, 2010, from http://www.informaworld.com/smpp/content~content=a783683471&db=all

Kibby, M. Y., & Long, C. J. (1996). Minor head injury: Attempts at clarifying the confusion. *Brain Injury, 10(3),* 159-186.

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. A (2010). Injuries in rugby league: Incidence, influences, tackles and return to play decision. Abstract retrieved October 27, 2010, from http://hdl.handle.net/10292/1007

King, D. A., Hume, P. A., Milburn, P., & Gianotti, S. (2009). Rugby league injuries in New Zealand: A review of 8 years of Accident Compensation Corporation injury entitlement claims and costs. *British Journal of Sports Medicine,* 43, 595-602.

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

King, N. S. (2003). Post- concussion syndrome: Clarity amid the controversy? *British Journal of Psychiatry, 183*, 276-278. Retrieved September 22, 2010 from http://bjp.rcpsych.org/cgi/reprint/183/4/276

King, N. S., Crawford, S., Wenden, F. J., Caldwell, F. E., & Wade, D. T. (1999). Early prediction of persisting post-concussion symptoms following mild and moderate head injuries. *British Journal of Clinical Psychology*, 38 (1), 15-25.

King, N. S., Crawford, S., Wenden, F. J., Moss, N. E., & Wade, D. T. (1995). The Rivermead Post Concussion Symptoms Questionnaire: A measure of symptoms commonly experienced after head injury and its reliability. *Journal of Neuroogy, 242(9)*, 587–592. Retrieved October 19, 2010, from http://www.springerlink.com/content/r937732618q72765/fulltext.pdf

Kirkendall, D. T., & Garrett, W. E. (2001). Heading in soccer: Integral skill or grounds for cognitive dysfunction? *Journal of Athletics Training, 36(3)*, 328-333.

Kirkendall, D. T., Jordan, S. E., & Garrett, W. E. (2001). Heading and head injuries in soccer. *Sports Medicine, 31*, 369-386.

Klein, M., Houx, P. J., & Jolles, J. (1996). Long-term persisting cognitive sequelae of traumatic brain injury and the effect of age. *The Journal of Nervous and Mental Disease, 184(8)*, 459-467.

Knight, R. G. (1992). *The neuropsychology of degenerative brain diseases.* Hillsdale, NJ: Lawrence Erlbaum Associates Inc.

Knight, R. G., & Titov, N. (2009). Use of virtual reality tasks to assess prospective memory: Applicability and evidence. *Australian Academic Press, 10*, 3-13. Retrieved June 10, 2010 from http://www.atypon-link.com/AAP/doi/pdf

Knowles, S. B., Marshall, S. W., & Guskiewicz, K. M. (2006). Issues in estimating risks and rates in sports injury research. *Journal of Athletic Training, 41 (2),* 207-215. Retrieved September 11, 2009, from PubMed database.

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. (2003). Concussion in rugby – an update. *Sports Medicine*, 16-20. Retrieved March 16, 2010, from http://ajol.info/index.php/sasma/article/viewFile/31879/23614

Kohler, R. M. (2004). Concussion in sport: Practical management guidelines for medical practitioners. *Continuing Medical Education, 22*, 122-125.

Kontos, A. P., Collins, M., & Russo, S. A. (2004). An introduction to sports concussion for the sport psychology consultant. *Journal of Applied Sport Psychology,* 16, 220-235.

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.* Advanced access to abstract retrieved October 6, 2010, from http://www.ncbi.nlm.nih.gov/pubmed/20861034

Kozora, E., & Gerber, D. (2004). Special considerations and implications of neuropsychological testing in professional athletes. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 358-374). The Netherlands: Swets & Zeitlinger Publishers.

Kozorovitskiy, Y., & Gould, E. (2003) Adult neurogenesis: A mechanism for brain repair. *Journal of Clinical and Experimental Neuropsychology, 25(5)*,721-732.

Kujala, U. M., Taimela, S., Antti-Poika, I., Orava, S., Tuominen, R., & Myllynen, P. (1995). Acute injuries in soccer, ice hockey, volleyball, basketball, judo and karate: Analysis of national registry data. *British Journal of Sport Medicine, 311*, 1465-1468.

Kurca, E., Sivak, S., & Kucera, P. (2006). Impaired cognitive functions in mild traumatic brain injury patients with normal and pathologic magetric resonance imaging. *Neuroradiology, 48(9)*, 661-669.

Kushner, D. S. (2001). Concussion in sports: Minimizing the risk for complications. *American Family Physician, 64(6)*, 1007-2001.

Kutner, K. C., Erlanger, D. M., Tsai, J., Jordan, B., & Relkin, N. R. (2000). Lower cognitive performance of older football players possessing apolipoprotein E ϵ4. *Neurosurgery, 47(3),* 651-657.

Kupperman, N., Holmes, J. F., Dayan, P. S., Hoyle, J. D., Atabaki, S. M., Holubkov, R. et al. (2009). Identification of children at very low risk of clinically-important brain injuries after head trauma: A prospective cohort study. *Lancet (374)*, 1160-170.

LaBotz, M., Martin, M. R., Kimura, I. F., Hetzler, R. K., & Nichols, A. W. (2005). A comparison of preparticipation evaluation history form and a symptom-based concussion survey in the identification of previous head injury in collegiate athletes. *Clinical Journal of Sport Medicine, 15*, 73-78.

Lampert, P. W., & Hardman, J. M. (1984). Morphological changes in the brains of boxers. *Journal of the American Medical Association, 251(20),* 2676-2679.

Landre, N., Poppe, C. J., Davis, N., Schmaus, B., & Hobbs, S. E. (2006). Cognitive functioning and postconcussive symptoms in trauma patients with and without mild TBI. *Archives of Clinical Neuropsychology, 21(4)*, 255-273.

Langburt, W., Cohen, B., Akhthar, N., O'Neil, K., & Lee, J. C. (2001). Incidence of concussion in high school football players in Ohio and Pennsylvania. *Journal of Child Neurology, 16*, 83-85. Abstract retrieved September 29, 2010 from http://jcn.sagepub.com/content/16/2/83.abstract

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*, 375-378.

Lau, B., Lovell, M., Collins, M., & Pardini, J. (2009). Neurocogntive and symptom predictors of recovery in high school athletes. *Clinical Journal of Sport Medicine, 19(3)*, 216-221.

Launer, L. J., Feskens, E. J., Kalmijn, S., & Kromhout, D. (1996). Smoking, drinking and thinking: The Zutphen elderly study. *American Journal of Epidemiology, 143(3)*, 219-227.

Lavoie, M. E., Dupuis, F., Johnston, K. M., Leclerc, S., & Lassonde, M. (2004). Visual P300 effects beyond symptoms in concussed athletes. *Journal of Clinical and Experimental Neuropsychology, 26(1),* 55-73.

Leclerc, S., Lassonde, M., Delaney, S., Lacroix, V., & Johnston, K. (2001). Recommendations for grading concussions in athletes. *Sports Medicine, 31(8)*, 629-636.

Leddy, J. J., Kozlowski, K., Donnelly, J. P., Pendergast, D. R., Epstein, L. H., & Willer, B. (2010). A preliminary study of subsymptom threshold exercise training for refractory concussion syndrome. *Clinical Journal of Sport Medicine, 20(1*), 21-27. Abstract retrieved May 27, 2010, from doi: 10.1097/JSM.0b013e3181c6c22c

Lees. A. J. (1997). Mise au point: Trauma and Parkinson's disease. *Rev Neorol (Paris), 153(10)*, 541-546.

Lees-Haley, P. R., & Brown, R. S. (1993). Neuropsychological-complaint base rates of 170 personal-injury claimants. *Archives of Clinical Neuropsychology, 8(3)*, 203-209.

Legome, E., & Alt, R. (2009). Postconcussive syndrome. *eMedicine*. Retrieved April 04, 2010 from http://emedicine.medscape.com/article/828904-overview.

Leininger, B. E., Gramling, S. E., Farrell, A. D., Kreutzer, J. S., & Peck, E. A. (1990). Neuropsychological deficits in symptomatic minor head injury patients after concussion and mild concussion. *Journal of Neurology, Neurosurgery, and Psychiatry, 53*, 293-296. Retrieved February, 9, 2010, from http://jnnp.bmj.com/content/53/4/293.full.pdf

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., Goldstein, F. C., High W. M., Jr., & Eisenberg, H. M. (1988). Disproportionately severe memory deficit in relation to normal intellectual functioning after closed head injury. *Journal of Neurology, Neurosurgery, & Psychiatry, 51*, 1294-1301.

Levin, H. S., Lippold, S. C., Goldman, A., Handel, S., High, W. M., Eisenberg, H. N. et al. (1987). Neurobehavioural functioning and magnetic resonance imaging findings in young boxers. *Journal of Neurosurgery, 67(5)*, 657-667.

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. S., Papanicolaou, A., & Eisenberg, H. M. (1984). Observations on amnesia after nonmissile head injury. In L. R. Squire & N. Butters (Eds.), *Neuropsychology of memory* (pp. 247-257). New York: Guilford Press.

Levin, H. S., Williams, D., Crofford, M. J., High, W. M., Jr., Eisenberg, H. M., Amparo, E. G. et al. (1988). Relationship of depth of brain lesions to consciousness and outcome after closed head injury. *Journal of Neurosurgery, 69*, 861-866.

Levine, Z. (2010). Mild traumatic brain injury part 2: Concussion management. *Canadian Family Physician, 56*, 658-662. Retrieved July 27, 2010 from http://www.cfp.ca/cgi/content/full/56/7/658

Lewandowski, L., Rieger, B., Smyth, J., Perry, L., & Gathje, R. (2009). Measuring post-concussion symptoms in adolescents: Feasibility of ecological momentary assessment. *Archives of Clinical Neuropsychology, 24(8)*, 791-796.

Lezak, M. D. (1979). Recovery of memory and learning functions following traumatic brain injury. *Cortex*, 15, 63-72.

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

Lezak, M. D., Howieson, D. B., & Loring, D. W. (2004). *Neuropsychological assessment.* (4th ed.). New York: Oxford University Press.

Lichtman, S. W., Seliger, G., Tycko, B., & Marder, K. (2000). Apolipoprotein E and functional recovery from brain injury following post acute rehabilitation, *Neurology, 55,* 1536-1539.

Liebeskind, D. S. (2009). Epidural hematoma. *eMedicine.* Retrieved April 4, 2009, from http://emedicine.medscape.com/article/1137065

Lima, D. P., Simao Filho, C., Abib, S. C., & Poli de Figueiredo, L. F. (2008). Quality of life and neuropsychological changes in mild head trauma: Late analysis and correlation with S100B protein and cranial CT scan performed at hospital admission. *Injury, 39*, 604–611.

Lindsay, K. W., McLatchie, G., & Jennett, B. (1980). Serious head injury in sport. *British Medical Journal, 281*, 789-791.

Lingard, D. A., Sharrock, N E., & Salmond, C. E. (1976). Risk factors of sports injuries in winter. *The New Zealand Medical Journal, 557 (83),* 70-73.

Lishman, W.A. (1987). *Organic psychiatry* (2nd Ed.). Oxford: Blackwell.

Lishman, W. A. (1999). *Organic psychiatry: The psychological consequences of cerebral disorder* (3rd ed.). Oxford: Blackwell Science.

Long, D. (2010). *South Africa rugby bosses need to get real*. Retrieved November 27, 2010, from http://www.stuff.co.nz/sport/rugby/opinion/4031511/South-Africa-rugby-bosses-need-to-get-real

Loosemore, M., Knowles, C. H., & Whyte, P. (2007). Amateur boxing and risk of chronic traumatic brain injury. *British Medical Journal, 335*, c809. Retrieved March, 19, 2010, from http://www.bmj.com/cgi/content/full/335/7624/809

Lovell, M. R. (2004). *ImPACT Version 2.0 Clinical User's Manual.* Accessed 3.12.2004 at http://www.impacttest.com/clients.htm

Lovell, M. R. (2006). Letters to the editor. *Journal of Athletic Training, 41 (2)*, 137-138.

Lovell, M., Bradley, J., Collins, M., & Burke, C. (2003). Amnesia, confusion may signal concussion. *American Journal of Sports Medicine*, *31.*

Lovell, M. R., & Collins, M. W. (1998). Neuropsychological assessment of the college football player. *Journal of Head Trauma and Rehabilitation, 13(2)*, 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., Iverson, G. L., Field, M., Maroon, J. C., Cantu, R. et al. (2003). Recovery from mild concussion in high school athletes. *Journal of Neurosurgery, 98(2),* 296-301.

Lovell, M. R., Collins, M. W., Iverson, G. L., Johnston, K. M., & Bradley, J. P. (2004). 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., Collins, M. W., Podell, K., Powell, J., & Maroon, J. (2000). *ImPACT: Immediate post-concussion assessment and cognitive testing.* Pittsburgh, PA: NeuroHealth Systems, LLC.

Lovell, M., Collins, M., & Fu, F. H. (2003). New technology and sports-related concussion. *Orthopedic Technology Review, 5 (1)*. Retrieved February 18, 2003, from http://www.ortho[edictechreview.com/issues/janfeb03/pg35.htm

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., Echemendia, R. J., & Burke, C. J. (2004). Professional ice hockey. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 221-229). The Netherlands: Swets & Zeitlinger Publishers.

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,* 193-199.

Lovell M. R., Iverson, G. L., Collins, M. W., Podell, K., Johnston, K. M., Pardini, D. et al. (2006). Measurement of symptoms following sports-related concussion: Reliability and normative data for the post-concussion scale. *Applied Neuropsychology, 13(3)*, 166-174.

Lovell, M. R., Pardini, J. E., Welling, J., Collins, M., Bakal, J., Lazar, N. et al. (2007). Functional brain abnormalities are related to clinical recovery and time to return to play in athletes. *Neurosurgery, 61(2)*, 359-360.

Lowry, F. (2010a). Brain training programs do not increase cognitive function. *Medscape Medical News*. Retrieved April, 26, 2010, from http://www.medscape.com/viewarticle/720577?src=mpnews&spon=26&uac=119539SX

Lowry, F. (2010b). New decision rule identifies kids with minor head trauma who need CT. *Medscape Medical News*. Retrieved February 20, 2010, from http://www.medscape.com/viewarticle/716931

Lubit, R. H. (2008). Postconcussive Syndrome. *eMedicine.* Retrieved September, 1, 2010, from http://emedicine.medscape.com/article/292326-overview

Lynch, S., & Yarnell, P. R. (1973). Retrograde amnesia: Delayed forgetting after concussion. *American Journal of Concussion, 86(3)*, 643-645.

Macciocchi, S. N., & Barth, J. T. (2004). Methodological concerns in traumatic brain injury. In M. R. 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(3),* 510-514.

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. (1993). Risks and injuries in rugby football. In G. R. McLatchie and C. M. Lennox (Eds.), *The soft tissues: Trauma and sport injuries* (pp. 371-381). London: Butterworth-Heinemann Ltd.

MacQueen, A. E., & Dexter, W. W. (2010). Injury trends and prevention in rugby union football. *Current Sports Medicine Reports, 9(3)*, 139-143. Abstract retrieved September 30, 2010, from http://journals.lww.com/acsm-csmr/Fulltext/2010/05000/Injury_Trends_and_Prevention_in_Rugby_Union.8.aspx

Maddocks, D. L., & Dicker, G. D. (1988). An objective measure of recovery from concussion in Australian Rules footballers. *The Australian Journal of Science and Medicine in Sport, December, 17*, 6-7.

Maddocks, D. L., & Saling, M. M. (1991). Neuropsychological sequelae following concussion in Australian Rules Footballers [Abstract]. *Journal of Clinical and Experimental Neuropsychology*, 13,439.

Maddocks, D. L., & Saling, M. M. (1996). Neuropsychological deficits following concussion. *Brain Injury, 10(2)*, 99-103.

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

Mahoney, D. (2009). Mild brain injury can have long-term effects. *Family Practice News.* Retrieved March 9, 2010, from http://www.accessmylibrary.com/article-1G1-198544428/mild-brain-injury-can.html

Majerske, C. W., Mihalik, J. P., Ren, D., Collins, M. W., Reddy, C. C., Lovell, M. R. et al. (2008). Concussion in sports: Postconcussive activity levels, symptoms, and neurocognitive performance. *Journal of Athletic Training, 43(3)*, 265-274. Retrieved September 6, 2010 from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2386420/

Makdissi, M., Collie, A., Maruff, P., Darby, D. G., Bush, A., McCrory, P. et al. (2001). Computerised cognitive assessment of concussed Australian Rules footballers. *British Journal of Sports Medicine, 35,* 354-360.

Makdissi, M., Darby, D., Maruff, P., Ugoni, A., Brukner, P., & McCrory, P. (2010). Natural history of concussion in sport: Markers of severity and implications for management. *American Journal of Sports Medicine, 38,* 464-471.

Mandel, S. (1989). Minor head injury may not be 'minor'. *Postgraduate Medicine, 85*, 213-225.

Manko, S. (2003). Amnesia, confusion may signal concussion. *UPMC News Bureau.* Retrieved March 1, 2010, from http://www.impacttest.com/pdf/Amnesia_Confusion.pdf

Manly, J. J., Toradji, P., Tang, M., & Stern, Y. (2003). Literacy and memory decline among ethnically diverse elders. *Journal of Clinical Experimental Neuropsychology, 25 (5)*, 680-690.

Maroon, J. C., Field, M., Lovell, M., Collins, M., & Bost, 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.

Marshall, L. F., Marshall, S. B., & Klauber, M. R. (1991). A new classification of head injury based on computerised tomography. *Journal of Neurosurgery, 75*, S14- S20.

Marshall, S. W., & Spencer, R. T. (2001). Concussion in rugby: The hidden epidemic. *Journal of Athletic Training, 36(3)*, 334-338.

Martelli, M. F., Grayson, R. L., & Zasler, N. D. (1999). Posttraumatic headache: Neuropsychological and psychological effects and treatment implications. *Journal of Head Trauma Rehabilitation, 14(1)*, 49-69.

Martineau, C., Kingma, J. J., Bank, L., & Valovich-McLeod, T. C. (2007). Guidelines for treatment of sport-related concussions. *JAAPA, 20(5)*, 22-28. Retrieved Aril 02, 2010 from http://media.haymarketmedia.com/documents/8/concussion0507_1921.pdf

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*, 971-973.

Matser, J. T., Kessels, A. G., Lezak, M. D., Troost, J., & Jordan, B. D. (2000). Acute traumatic injury in amateur boxing. *Physician and Sports Medicine, 28*, 87-92.

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 M.R. Lovell, R.L. Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic brain injury in sports.* (pp. 193-208). The Netherlands: Swets & Zeitlinger Publishers.

Maxwell, J. P., & Visek, A. J. (2009). Unsanctioned aggression in rugby union: Relationships among aggressiveness, anger, athletic identity, and professionalization. *Aggressive Behavior, 35(3)*, 237-243. Abstract retrieved April 7, 2010, from http://www3.interscience.wiley.com/journal/122270754/abstract

Mayeux, R., Ottman, R., Maestre, G., Ngai, C., Tang, M. X., Ginsberg, H. et al. (1995). Synergistic effects of traumatic head injury and apolipoprotein epsilon 4 in patients with Alzheimer's disease. *Neurology, 45(3),* 555-557.

McAllister, T. W., & Arciniegas, D. (2002). Evaluation and treatment of postconcussive symptoms. *NeuroRehabilitation, 17(4)*, 265-283.

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.

McAllister, T. W., Sparling, M. B., Flashman, L. A., Guerin, S. J., Mamourian, A. C., & Saykin, A.J. (2001). Differential working memory load effects after mild traumatic brain injury. *NeuroImage*, 14, 1004-1012.

McCaffrey, R. J., Duff, K., & Westervelt, H. J. (2000). *Practitioner's guide to evaluating change with neuropsychological assessment instruments.* New York: Kluwer Academic/Plenum Press.

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.

McCauley, S. R., Boake, C., Levin, H. S., Contant, C. F., & Song, J. X. (2001). Postconcussional disorder following mild to moderate brain injury: Anxiety, depression, and social support as risk factors and comorbidities. *Journal of Clinical and Experimental Neuropsychology, 23 (6)*, 792-808.

McCauley, S. R., Boake, C., Pedroza, C., Brown, S. A., Levin, H. S., Goodman, H. S. et al. (2008). Correlates of persistent Postconcussional disorder: DSM-IV criteria versus ICD-10 *Journal of Clinical and Experimental Neuropsychology, 30(3)*, 360 - 379

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. (2001). Standardized mental status assessment of sports concussion. *Clinical Journal of Sports Medicine*, *11*, 176-181.

McCrea, M. (2008). *Mild traumatic brain injury and postconcussion syndrome: The new evidence* (pp. 1-4). Oxford University Press: New York. Read online April 7, 2010, from http://books.google.com/books?id=-WFl91VSWyEC&pg=PA4&lpg=PA4&dq=TBI+a+continuum&source

McCrea, M., Barr, W. B., Guskiewicz, K., Randolph, C., Marshall, S. W., Cantu, R. et al. (2005). Standard regression-based methods of 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)*, 2604-2605. Retrieved June 21, 2010 from http://jama.ama-assn.org/cgi/content/full/290/19/2556

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 Sport Medicine, 14(1)*, 13-17.

McCrea, M., Kelly, J. P., Kluge, J., Ackley, B., & Randolph, C. (1997). Standardized assessment of concussion in football players. *Neurology, 48(3)*, 586-588.

McCrea, M., Kelly, J. P., Randolph, C., Cisler, R., & Berger, L. (2002). Immediate neurocognitive effects of concussion. *Neurosurgery, 50*, 1032-1041.

McCrea, M., Kelly, J. P., Randolph, C., Kluge, J., Bartolie, E., Finn, G. et al. (1998). Standardized assessment of concussion (SAC). On site mental status evaluation of the athlete. *Journal of Head Trauma and 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 study. *Journal of Head Trauma and Rehabilitation, 25(4)*, 283-292. Abstract retrieved September 23, 2010 from http://www.ncbi.nlm.nih.gov/pubmed/20611046

McCrory, P. R. (1999a). The eighth wonder of the world: The mythology of concussion management. *British Journal of Sports Medicine, 33,* 136-137.

McCrory, P. R. (1999b). 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. (2002a). What advice should we give to athletes postconcussion? *British Journal of Sports Medicine, 36*, 316-318.

McCrory, P. (2002b). Treatment of recurrent concussion? *Current Sports Medicine Reports, 1*, 28-32.

McCrory, P., Heywood, J., & Coffey, C. (2005). Prevalence of headache in Australian footballers [Abstract]. *British Journal of Sports Medicine, 39(2)*, e10. Retrieved 14 August, 2009 from http://bjsm.bmj.com/cgi/content/abstract/39/2/e10

McCrory, P., & Shrier, I. (2002, October). *Recovery of clinical and neuropsychological function following acute concussion using survival analysis* [Abstract]. Sports Medicine and Science at the Extremes. Paper presented at the Australian Conference of Science and Medicine in Sport 2002, Australia. *Sports Medicine Australia.* Retrieved February 20, 2010, from http://fulltext.ausport.gov.au/fulltext/2002/acsms/Papers/McCrory1.asp

McCrory, P. R. (2001). When to retire after concussion? *British Journal of Sports Medicine, 35*, 380-382.

McCrory, P. R., Ariens, M., & Berkovic, S. F. (2000). The nature and duration of acute concussive symptoms in Australian football. *Clinical Journal of Sports Medicine, 10*, 235-238.

McCrory, P. R., & Berkovic, S. F. (1998). Second impact syndrome. *Neurology, 50*, 677-683.

McCrory, P. R., Berkovic, S. F., & Cordner, S. M. (2000). Deaths due to brain injury among footballers in Victoria, 1968-1999. *Medical Journal of Australia, 175(5)*, 217-219.

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. (2002). Acute clinical symptoms of concussion: Assessing prognostic significance. *The Physician and Sports Medicine, 30 (8)*, 43-47.

McCrory, P. R., Johnston, K., Meeuwisse, W., Aubry, M., Cantu, R., Dvorak, J. et al. (2005). Summary and agreement statement of the 2nd International Conference on Concussion in Sport: The 3rd International Conference on Concussion in Sport, Prague 2004. *Clinical Journal of Sports Medicine, 15(2),* 48-55.

McCrory, P. R., Makdissi, M., Davis, G., & Collie, A. (2005). Value of neuropsychological testing after head injuries in football. *British Journal of Sports Medicine, 39(Suppl 1)*, i58-i63.

McCrory, P. R., Meeuwisse, W., Johnston, K., Dvorak, J., Aubry, M., Molloy, M. et al. (2009). Consensus statement on concussion in sport, held in Zurich, November 2008. *British Journal of Sports Medicine*, *43 (1)*, i76-i84. Retrieved May 19, 2009 from doi:10.1136/bjsm.2009.058248.

McDaniel, L. W. (2010). Neurocognitive Performance: Returning to Competition. *International Education Studies, 3(3)*, 137-140. Retrieved October 13, 2010 from http://www.ccsenet.org/journal/index.php/ies/article/viewFile/6788/5319

McGuine, T. (2006). Sports injures in high school athletes: A review of injury-risk and injury-prevention research. *Clinical Journal of Sport Medicine, 16(6)*, 488-499. Abstract retrieved March 25, 2010, from http://www.ncbi.nim.nih.gov/pubmed/17119362

McIntosh, A. (2005). Rugby Injuries. In N. Maffulli & D. J. Caine DJ (Eds.), Epidemiology *of Pediatric Sports Injuries: Team Sports, Medical Sport Science, 49* (pp. 120-139). Basel: Karger. (DOI: 10.1159/000085394)

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., Best, J. P., Chalmers, D. J., & Wolfe, R. (2009). Does padded headgear prevent head injury in rugby union football? *Medicine & Science in Sports & Exercise,* 307-313. Retrieved September 30, 2010, from http://www.udel.edu/PT/PT%20Clinical%20Services/journalclub/sojc/08-09/Mar09/Padded%20head%20gear%20in%20rugby,%20Mcintosh.pdf

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. Retrieved October 28, 2010 from http://bjsm.bmj.com/content/44/3/188.full

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. Abstract Retrieved September 6, 2010, from http://www.ncbi.nlm.nih.gov/pubmed/19535999

McKee, A. C., Gavett, B. E., Stern, R. A., Nowinski, C. J., Cantu, R. C., Kowall, N. W. et al. (2010). TDP-43 proteinopathy and motor neuron disease in chronic traumatic encephalopathy. *Journal of Neuropathology and Experimental Neurology*, ePub ahead of print Retrieved September 6, 2010, from http://journals.lww.com/jneuropath/Abstract/publishahead/TDP_43_Proteinopathy_and_Motor_Neuron_Disease_in.99709.aspx

McKeever, C. K., & Schatz, P. (2003). Current issues in the identification, assessment, and management of concussions in sports-related injuries. *Applied Neuropsychology, 10(1)*, 4-11.

McKinley, J. (2007). New challenges in assessing and managing concussion in sports [Electronic version]. *American Family Physician, 76(7)*. Retrieved November 7, 2007, from http://www.aafp.org/afp/20071001/editorials.html

McLatchie G., Brooks, N., Galbraith, S., Hutchinson, J. S., Wilson, L., Melville, I. et al. (1987). Clinical neurological examination, neuropsychology, electroencephalography and computed tomographic head scanning in active amateur boxers. *Journal of Neurological Psychiatry, 50*, 96-99. Retrieved March 11, 2010, from http://jnnp.bmj.com/content/50/1/96.full.pdf

McLatchie G., & Jennett, B. (1994). ABC of sports medicine: Head injury in sport. *British Journal of Sports Medicine, 308*, 1620-1624.

McMahon, K. A., Nolan, T., Bennett, T., & Carlin, J. B. (1993). Australian rules football injuries in children and adolescents. *Medical Journal; Australia, 159(5)*, 301-306

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.

McMillan, T. M. (1997). Minor head injury. *Current opinion in Neurology, 10,* 479-483.

Meares, S., Shores, E. A., Taylor, A. J., Batchelor, J., Bryant, R. A., Baguley, I. J. et al. (2008). Mild traumatic brain injury does not predict acute postconcussion syndrome. *Journal of Neurology, Neurosurgery and Psychiatry, 79*, 300-306. Abstract retrieved March 26, 2010, from http://jnnp.bmj.com/content/79/3/300

Meehan, W. P., & Bachur, R. G. (2009). Review article: Sport-related concussion. *Pediatrics, 123(1),* 114-123.

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. Retrieved October 20, 2009, from http://www.mirecc.va.gov/docs/visn6/windows-2005-sportsTBI.pdf.

Meyers, J. E., & Meyers, K. R. (2008). *RCFT - Rey Complex Figure Test and recognition trial*. Retrieved October 26, 2010 from http://www.hogrefe.co.uk/?/test/show/130/

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. et al. (2007). Effectiveness of mouthguards in reducing neurocognitive deficits following sports-related concussion. *Dental Traumatology, 23(1),* 14-20. Abstract retrieved September 11, 2009, from PubMed database.

Mihalik, J. P., Stump, J. E., Collins, M. W., Lovell, M. R., Field, M., & Maroon, J. C. (2005). Posttraumatic migraine characteristics in athletes following sports-related concussion. *Journal of Neurosurgery, 102*, 850-855.

Milberg, W. P., Hebben, N., & Kaplan, E. (1996). The Boston process approach to neuropsychological assessment. In I. Grant, & K. M. Adams (Eds.), *Neuropsychological Assessment of Neuropsychiatric Disorders* (2nd ed.). New York: Oxford University Press.

Miller, J. D. (1986). Minor, moderate and severe head injury. *Neurosurgical Review, 9(1-2),* 135-139.

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.

Mitchener, A., Wyper, D. J., Patterson, J., Hadley, D. M., Wilson, J. T., Scott, L. C. et al. (1997). SPECT, CT and MRI in head injury: Acute abnormalities followed up at six months. *Journal of Neurology, Neurosurgery and Psychiatry, 62(6),* 633-666.

Mittl, R. L., Grossman, R. I., Hiehle, J. F., Hurst, R. W., Kauder, D. R., Gennarelli, T. A. et al. (1994). Prevalence of MR evidence of diffuse axonal injury in patients with mild head injury and normal CT findings. *Journal of Neuroradiology, 15(8),* 1583-1589.

Molsa, J., Kujala, U., Nasman, O., Lehtipuu, T-P., & Airaksinen, O. (2000). Injury profile in ice hockey from the 1970s through the 1990s in Finland. *American Journal of Sports Medicine, 28*, 322-327.

Moriarity, J., Collie, A., Olson, D., Buchanan, J., Leary, P., McStephen, M. et al. (2004). A prospective controlled study of cognitive function during an amateur boxing tournament. *Amercian Academy of Neurology, 62(9),* 1497-1502.

Mortimer, J. A. (1997). Brain reserve and the clinical expression of Alzheimer's disease. *Geriatrics, 52 (Suppl. 2),* S50-S53.

Mortimer, J. A., & Graves, A. B. (1993) Education and other socioeconomic determinants of dementia and Alzheimer's disease. *Neurology, 43(4)*, S39- S44.

Mortimer, J. A., Snowdon, D. A., & Markesbery, W. R. (2003). Head circumference, education and risk of dementia: Findings from the nun study. *Journal of Clinical and Experimental Neuropsychology, 25 (5), 671-679.*

Mortimer, J. A., van Duijn, C. M., Chandra, V., Fratiglioni, L., Graves, A. B., Heyman, A. et al. (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),* S28-S35.

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). Neuropsychological evaluation in the diagnosis and management of sports-related concussion. *Archives of Clinical Neuropsychology, 22, 909-916.*

Mower, W. R. (2010). What rules should guide imaging decisions in injured children? *Medscape Emergency Medicine.* Retrieved March 4, 2010, from http://www.medscape.com/viewarticle/717110

Mrazik, M., Ferrara, M. S., Peterson C. L., Courson, R. W., Elliott, R. E., & Hynd, G. W. (Personal Communication Chris Paniak, September 21, 2007). Detecting individual change: A model for classifying mild head injuries in sports.

Mrazik, M., Ferrara, M. S., Peterson, 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.

Myers, P. T. (1980). Injuries presenting from rugby union football. *The Medical Journal of Australia, 2,* 17-20.

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

National Center for Injury Prevention and Control. (2003) *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. Retrieved August 11, 2010, from http://www.cdc.gov/ncipc/pub-res/mtbi/mtbireport.pdf

NCAA (2002). Concussion and second-impact syndrome. *NCAA Guideline 2o*.

Necajauskaite, O., Endziniene, M., & Jureniene, K. (2005). The prevalence, course and clinical features of post-concussion syndrome in children. *Medicina (Kaunsas), 41(16)*, 457-464. Abstract retrieved August 13, 2009 from http://www.ncbi.nlm.nih.gov/pubmed/15998982

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.

Neppe, V. M., & Godwin, G. T. (1999). The neuropsychiatric evaluation of the closed head injury of transient type (CHIT) (pp. 149-208). In N. Varney & R. Roberts (Eds.), *Evaluation and Treatment of Mild Traumatic Brain Injury.* Mahweh, NJ:Erlbaum and Associates.

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., & Coleman, P. (1996). Letters: Acute sports injuries. *British Journal of Sports Medicine, 312*, 844.

Nicholl, J., & LaFrance, W. C. (2009). Neuropsychiatric sequelae of traumatic brain injury. *Seminars in Neurology, 29(3)*, 247-255. Retrieved April 04, 2010, from http://www.medscape.com.viewarticle/706300

Nicol, A., Pollock, A., Kirkwood, G., Parekh, N., & Robson, J. (2010). Rugby union injuries in Scottish schools. *Journal of Public Health*, 1-6 |. Retrieved September 30, 2010, from http://jpubhealth.oxfordjournals.org/content/early/2010/06/25/pubmed.fdq047.full.pdf+html

Noakes, T., & Jakoet, I. (1995). Spinal cord injuries in rugby union. *British Medical Journal, 310,* 1345-1346.

Notebaert, A. J., & Guskiewicz, K. M. (2005). Current trends in athletic training for concussion assessment and management. *Journal of Athletic Training, 40(4)*, 320-325. Retrieved September 11, 2009, from PubMed database.

Oliaro, S., Anderson, S., & Hooker, D. (2001). Management of cerebral concussion in sports: The athletic trainer's perspective. *Journal of Athletic Training, 36(2)*, 257-262.

Oliaro, S. M., Guskiewicz, K. M., & Prentice, W. E. (1998). Establishment of normative data on cognitive tests for comparison with athletes sustaining mild head injury. *Journal of Athletic Training, 33(1)*, 36-40. Retrieved October 8, 2010, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1320373/pdf/jathtrain00009-0038.pdf

Ombac Rugby. (2010, January 26). Retrieved January, 26, 2010, from http://www.ombac.org/ombac_rugby/rulesofrugby.htm

O'Meara, E. S., Kukull, W. A., Sheppard, L., Bowen, J. D., McCormick, W. C., Teri. L. et al. (1997). Head Injury and Risk of Alzheimer's disease by Apolipoprotein E Genotype. *American Journal of Epidemiology, 146(5)*, 373.384. Retrieved September 15, 2010, from http://aje.oxfordjournals.org/content/146/5/373.short

Orchard, J., & Seward, H. (2002). Epidemiology of injuries in the Australian football league seasons 1997-2000. *British Journal of Sports Medicine, 36,* 39-45.

Orchard, J., & Seward, H. (2009). Injury Report 2008: Australian Football League. Retrieved September 29, 2010, from http://www.afl.com.au/portals/0/afl_docs/2008_injury_survey.pdf

Orchard, J., Wood, T., Seward, H., & Broad, A. (1998). Comparison of injuries in elite senior and junior Australian football. *Journal of Science and Medicine in Sport, 1(2)*, 82-88.

Oriard, M. (undated). Gridiron football. *Britannica concise encyclopeda*. Retrieved September 29, 2010, from http://www.britannica.com/EBchecked/topic/212839/gridiron-football

Osterrieth, P. A. (1944). Le test de copie d'une figure complexe. *Archives de Psychologie, 30*, 206-356 (trans. J. Corwin and F.W. Bylsma (1993), *The Clinical Neuropsychologist, 7,* 9-15.

Ostrosky-Solis, F. (2004). Can literacy change brain anatomy? *International Journal of Psychology, 39(1)*, 1-4.

Oxford University Women's Rugby football Club. (undated). The difference between rugby union and rugby league. http://users.ox.ac.uk/~ouwrugby/TheDifferenceBetweenRugbyUnionandRugbyLeague.shtml

Packard, R. C. (2008). Chronic post-traumatic headache: Associations with mild traumatic brain injury, concussion, and post-concussive disorder. *CurrentPain and Headache Reports, 12(1)*, 67-73. Retrieved March 17, 2010, from http://www.springerlink.com/content/37h12q2147552176/

Paniak, C., MacDonald, J., Toller-Lobe, G., Durand, A., & Nagy, J. (1998). A preliminary normative profile of mild traumatic brain injury diagnostic criteria. *Journal of Clinical and Experimental Neuropsychology, 20(6),* 852-855.

Paniak, C., Reynolds, S., Phillips, K., Toller-Lobe, G., Melnyk, A., & Nagy, J. (2002). Patient complaints within 1 month of mild traumatic brain injury: A controlled study. *Archives of Clinical Neuropsychology, 17),* 319-334.

Paniak, C., Toller-Lobe, G., Reynolds, S., Melnyk, A., & Nagy, J. (2000). A randomized trial of two treatments for mild traumatic brain injury: 1 year follow-up. *Brain Injury, 14(3)*, 219-226. Abstract retrieved June 15, 2010, from http://www.informaworld.com/smpp/content~db=all~content=a713802706

Parker, R. S. (2001). *Concussive brain trauma. Neurobehavioural impairment and maladaption.* Boca Raton, FL: CRC Press.

Parker, T. M., Osternig, L. R., van Donkelaar, P., & Chou, L. S. (2006). Gait stability following concussion. *Medicine and Science in Sports and Exercise*, 1032-1949.

Parker, T. M., Osternig, L. R., van Donkelaar, P., & Chou, L. (2007). Recovery of cognitive and dynamic motor function following concussion. *British Journal of Sports Medicine, 41,* 868-873.

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. Retrieved October 20, 2010 from http://projects.ict.usc.edu/vrcpat/PDF/Parsons(IJN)_RCI_Neuro.pdf

Parsons, M. W., & Gold, P.E. (1992). Glucose enhancement in memory in elderly humans: An inverted-U dose-response curve. *Neurobiology of Aging, 13*, 410-404.

Pearson Clinical Assessment (2009). *WMS–III to WMS–IV: Rationale for Change.* Retrieved August 19, 2010, from http://www.pearsonassessments.com/hai/images/Products/WMS-IV/WMS-RationaleforChange.pdf

Pelletier, J. C. (2006). Sports related concussion and spinal injuries: The need for changing spearing rules at the National Capital Amateur Football Association (NCAFA). *The Journal of the Canadian Chiropractic Association, 50(3)*, 195-208. Retrieved March 24, 2010, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1839959

Pellman, E. J., Powell, J. W., Viano, D. C., Casson, I. R., Tucker, A. M., Feuer, H. et al. (2004). Concussion in Professional Football: Epidemiological features of game injuries and review of literature – Part 3. *Neurosurgery, 54,* 81-96.

Pellman, E. J., Viano, D. C., Casson, I. R., Tucker, A. M., Waeckerle, J. F., Powell, J. W. et al. (2004). Concussion in Professional Football: Repeat injuries –Part 4. *Neurosurgery, 55(4*), 860-876.

Pellman, E. J., Viano, D. C., Casson, I. R., Arfken, C., & Powell, J. W. (2004). Concussion in Professional Football: Injuries involving 7 or more days out –Part 5. *Neurosurgery, 55(5*), 1100-1119.

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*), 263-274.

Peloso, P. M., Carroll, L. J., Cassidy, D., Borg, J., von Holst, H., Holm, L. et al. (2004). Critical Evaluation of the Existing Guidelines on Mild Traumatic Brian Injury. *Journal of Rehabilitation Medicine, Suppl. 43*, 106-112.

Peloso, P. M., von Holst, H., & Borg, J. (2004). Mild traumatic brain injuries presenting to hospitals in Sweden during the years 1987-2000. *Journal of Rehabilitation Medicine, Suppl. 43*, 22-27.

Perneger, T. V. (1998). What's wrong with Bonferroni adjustments. *British Medical Journal, 316,* 81236-1238.

Petchprapai, N., & Winkelman, C. (2008). Mild traumatic brain injury: Determinants and subsequent quality of life. A review of the literature [Electronic version]. *The Journal of Neuroscience Nursing.* July 15, 2008, from http://www.medscape.com/viewarticle/567651.

Peterson, C. L., Ferrara, M. S., Mrazik, M., Piland, S., & Elliot, R. (2003). Evaluation of neuropsychological domain scores and postural stability following cerebral concussion in sports. *Clinical Journal of Sports Medicine, 13(4)*, 230-237.

Pettersen, J. A., & Skelton, R. W. (2000). Glucose enhances long-term declarative memory in mild head-injured varsity rugby players. *Psychobiology, 28,* 81-89.

Philips, S. M. (2003). Concussion: A review and update. *Athletic Therapy Today, 8(5)*, 42-44.

Pieter, W., & Zemper, E. D. (1998). Incidence of reported cerebral concussion in adult taekwondo athletes. *Journal of the Royal Society for the Promotion of Health, 118(5),* 272-279.

Piland, S. G., Ferrara, M. S., Macciocchi, S. N., Broglio, S. P., & Gould, T. E. (2010). Investigation of baseline self-report concussion symptom scores. *Journal of Athletic Training, 45(3)*, 273-278.

Piland, S. G., Motl, R. W., Ferrara, M. S., & Peterson, C. L. (2003). Evidence for the factorial and construct validity of a self-report concussion symptoms scale. *Journal of Athletic Training, 38(2)*, 104-112. Retrieved October 14, 2010, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC164898/

Piland, S. G., Motl, R. W., Guskiewicz, K. M., McCrea, M., & Ferrara, M. S. (2006). Structural validity of a self-report concussion-related symptom scale. *Medicine and Science in Sports and Exercise, 38*, 27-32. Retrieved October 14, 2010 from http://csmfoundation.homestead.com/piland_-_structural_validity_of_symtpm_scale_-_MSSE_2006.pdf

Podell, K. (2004). Computerized assessment of sports-related brain injury. In M. R. Lovell, R. L. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 375-393). The Netherlands: Swets & Zeitlinger Publishers.

Poirier, M. P. (2003). Concussions: Assessment, management, and recommendations for return to activity. *Clinical Pediatric Emergency Medicine, 4 (3),* 179-185.

Poirier, M. P., & Wadsworth, M. R. (2000). CME review article: Sports-related concussions. *Pediatric Emergency Care, 16(4), 2*78-283.

Ponsford, J., Sloan, S., & Snow, P. (1995). *Traumatic brain injury: Rehabilitation for everyday adaptive living.* Hove, UK: Lawrence Erlbaum Associates Ltd.

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. Retrieved July, 27, 2010 from Science Direct database. http://kch.illinois.edu/Research/Labs/neurocognitive-kinesiology/files/Articles/Pontifex_2009_TheAssociationBetweenMild.pdf

Porter, M. D. (2003). A 9-year controlled prospective neuropsychologic assessment of amateur boxing. *Clinical Journal of Sports Medicine, 13(6),* 339-352.

Porter, M. D., & Fricker, P. A. (1996). Controlled perspective neuropsychological assessment of active experienced boxers. *Clinical Journal of Sports Medicine, 6*, 90-96.

Potter, S., Leigh, E., Wade, D., & Fleminger, S. (2006). The Rivermead Post Concussion Symptoms Questionnaire: A confirmatory factor analysis. *Journal of Neurology, 253(12)*, 1603-1614. Retrieved October 19, 2010 from http://www.ncbi.nlm.nih.gov/pubmed/17063314

Powell, J. W. (1999). Injury patterns in selected high school sports. In J. E. Bailes, M .R. Lovell, & J. C. Maroon (Eds.), *Sports-related concussion* (pp. 75-90). St Louis: Quality Medical Publishing.

Powell, J. W. (2001). Cerebral concussion: Causes, effects, and risks in sports. *Journal of Athletic Training, 36(3)*, 307-311.

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 & Zeitlinger Publishers.

Powell, J. W., & Barber-Foss, K. D. (1999). Traumatic brain injury in high school athletes. *Journal of the American Medical Association, 282(10)*, 958-963.

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.

Preiss-Farzanegan, S. J., Chapman, B., Wong, T. M., Wu, J., & Bazarian, J. (2009). The relationship between gender and postconcussion symptoms after sport-related mild traumatic brain injury. *Physical Medicine and Rehabilitation, 1(3)*, 245-253. Abstract retrieved February, 03, 2010, from Science Direct database.

Pretz, L. C. (2007). *Assessment of risk factors in sports-related concussion: Incidence rate and recovery patterns*. Retrieved June 26, 2009, from Miami University Oxford Ohio, Department for Speech Pathology and Audiology Web site: http://www.ohiolink.edu/etd/send-pdf.cgi/Pretz%20Laura%20Christine.pdf?miami11770863c08

Provvidenza, C. F., & Johnston, K. M. (2009). Knowledge transfer principles as applied to sport concussion education. *British Journal of Sports Medicine, 43(September)*, i68. Abstract retrieved February 8, 2010 from http://bjsm.bmj.com/content/43/Suppl_1/i68.abstract

Purcell, L. (2009). What are the most appropriate return-to-play guidelines for concussed child athletes? Abstract retrieved February 8, 2010 from http://bjsm.bmj.com/content/43/Suppl_1/i51.abstract

Putukian, M., Aubry, M., & McCrory, P. (2009). Return to play after sports concussion in elite and non-elite athletes? *British Journal of Sports Medicine, 43(September)*, i28. Abstract retrieved February 8, 2010 from http://bjsm.bmj.com/content/43/Suppl_1/i28.abstract

Putukian, M., & Echemendia, R. J. (1996). Managing successive minor head injuries: Which tests guide return to play. *The Physician and Sports Medicine, 24(11)*, 25-38.

Putukian, M., Echemendia, R. J., & Mackin, S. (2000). The acute neuropsychological effects of heading in soccer: A pilot study. *Clinical Journal of Sports Medicine, 10,* 104-109.

Quality Standards Subcommittee of the American Academy of Neurology. (1997). Practice Parameter: The management of concussion in sports (summary statement). *Neurology, 48,* 581-585.

Quarrie, K. L., Cantu, R. C., & Chalmers, D. J. (2002). Rugby union injuries to the cervical spine and spinal cord, *Sports Medicine, 32*, 633-653.

Quarrie, K. L., Gianotti, S. M., Hopkins, W. G., & Hume, P. A. (2007). Effect of nationwide injury prevention programme on serious spinal injuries in New Zealand rugby union: Ecological study. *British Medical Journal, 334*, 1150. Retrieved March 20, 2010, from http://www.bmj.com/cgi/content/full/334/7604/1150

Rabadi, M. H., & Jordan, B. D. (2001). The cumulative effect of repetitive concussion in sports. *Clinical Journal of Sport Medicine, 11(3),* 194-198.

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., Barr, W. B., & McCrea, M. (2006). Letters to the editor. *Journal of Athletic Training, 41 (2)*, 138-140.

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.

Randolph, C., Millis, S., Barr, W. B., McCrea, M., Guskiewicz, K. M., Hammeke, T. A. et al. (2009). Concussion symptom inventory: An empirically derived scale for monitoring resolution of symptoms following sport-related concussion. *Archives of Clinical Neuropsychology, 24(3)*, 219-229. Retrieved June, 22, 2010, from http://www.smf.org/articles/hic/Concussion_Symptom_Inventory_rem.pdf

Rangel-Castilla, L., Gasco, J., Hanbali, F., & Salinas, P. (2008). Closed head trauma. Retrieved July 15, 2008, from http://emedicine.medscape.com/article/251834-overview

Rao, V., & Rollings, P. (2002). Sleep disturbances following traumatic brain injury. *Current Treatment Options in Neurology, 4*, 77-87.

Raskin, S. A., & Rearick, E. (1996). Verbal fluency in individuals with mild traumatic brain injury. *Neuropsychology, 10,* 416-422.

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., Zuacha, K., McArthur, D. L. et al. (2006). Functional outcome in TBI I: Neuropsychological, emotional, and behavioral 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., Thorne, R., Winter, S., & Hegge, F. (1989). *The United Tri-Service Cognitive Performance Assessment Battery (UTC-PAB). Report 89-1.* San Diego, CA: US Naval Aerospace Medical Research Laboratory and Walter Reed Army Institute of Research.

Register-Mihalik, J. K., Mihalik, J. P., & Guskiewicz, K. M. (2008). Balance deficits after sports-related concussion in individuals reporting posttraumatic headache. *Neurosurgery, 63(1)*, 76-80.

Reid, D. B., & Kelly. M. P. (1993). Wechsler Memory Scale-Revised in closed head injury. *Journal of Clinical Psychology, 49(2)*, 245-254.

Reitan, R. M. (1955). The relation of the Trail Making Test to organic brain damage. *Journal of Consulting Psychology, 19*, 393-394.

Reitan, R. M. (1958). Validity of the Trail Making Test as an indicator of organic brain damage. *Perceptual & Motor Skills, 8*, 271-276.

Reitan, R. M., & Wolfson, D. (1985). *The Halsted-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.

Reite, M., Cullum, C. M., Stocker, J., Teale, P., & Kozora, E. (1993). Neuropsychological test performance and MEG-based brain lateralisation: Sex differences. *Brain Research Bulletin, 32*, 325-328.

Rey, A. (1941). L'examen psychologique dans les cas d'encephalopathie traumatique. *Archives de Psychologie, 28,* 286-340 (see Corwin, & Bylsma, 1993b for translation).

RFU. (2010). *Rugby Tens*. Retrieved January, 29, 2010, from http://www.rfu.com/TakingPart/Play/Leisure/TypeOfLeisureRugby

Richards, M., & Deary, I. (2005). A Life Course Approach to Cognitive Reserve: A Model for Cognitive Aging and Development? *Annual Neurologist, 58*, 617-622. Retrieved April 26, 2010, from http://www.psy.ed.ac.uk/people/iand/Richards%20(2005)%20Arch%20Neurol%20cognitive%20reserve.pdf

Richards, M., & Sacker, A. (2003). Lifetime antecedents of cognitive reserve. *Journal of Clinical and Experimental Neuropsychology, 25*, 614-624.

Richardson, J. T. (1990). *Clinical and Neuropsychological aspects of closed head injury*. Great Britain: Burgess Science Press.

Riley, R., & Zellinger, M. (2000). The WMS-III verbal paired associates recognition task: Exploration of an alternative approach [Abstract]. *Archives of Clinical Neuropsychology,* 15, 679.

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.

Robinson, R. (2010). Is diffusion tensor imaging a potential biomarker for mild traumatic brain injury? *Neurology Today, 10(6)*, 8-9.

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. Retrieved August 13, 2009, from http://www.beyondthesideline.com/Documents/Microsoft%20Word%20-%20ATR%20490%20Concussions%20Paper.pdf

Roe, C, Sveen, U., Alvsaker, K., & Bautz-Holter, E. (2009). Post-concussion symptoms after mild traumatic brain injury: Influence of demographic factors and injury severity in a 1-year cohort study. *Disability and Rehabilitation, 31(15)*, 1235-1243. Abstract.

Ropacki, M. T., & Elias, J. W. (1999). Further validation of the Oklahoma premorbid intelligence estimate with closed head injury and mixed neurologic patients. Abstracts from the 19th annual meeting. *Archives of Clinical Neuropsychology, 14*, 788. Abstract retrieved February 22, 2010 from http://scienceDirect-Archvesofclinical neropsychology:preliminary examination

Ropacki, M. T., & Elias, J. W. (2003). Preliminary examination of cognitive reserve theory in closed head injury. *Archives of Clinical Neuropsychology, 18(6)*, 643-654.

Ropper, A. H., & Gorson, K. C. (2007). Concussion. *The New England Journal of Medicine, 356(2)*, 166-172.

Rosnow, R. L., & Rosenthal, R. (1996). *Beginning behavioural research.* New Jersey: Prentice Hall.

Ross, R. J., Casson, I. R., Siegel, O., & Cole, M. (1987). Boxing Injuries, Radiologic, and Neuropsychologic Evaluation. *Clinics in Sports Medicine, 6(1),* 41-51.

Rotem, T., James, S. L., Wilson, S. F., Engel, S., Rutkowski, S. B., & Aisbett, C. W. (1998). Severe cervical spinal cord injuries related to rugby union and league football in New South Wales. *Medical Journal of Australia, 168,* 379-381.

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

Roy, S. P. (1974). The nature and frequency of rugby injuries. *S.A. Medical Journal, 2321-2327.*

Royal College of Surgeons of England. (2007). *Good surgical practice*. Retrieved January 14 2007, from http://jalt.org/test/bro_27.htm

Ruchinskas, R. A., Francis, J. P., & Barth, J. T. (1997). Mild head injury in sports. *Applied Neuropsychology, 4(1)*, 43-49.

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., & Grant, I. (1999.) Postconcussional Disorder: Background to DSM-IV and future considerations. In N. R., Varney & R. J. Roberts (Eds.), *The evaluation and treatment of mild traumatic brain injury* (pp. 315-325). Mahwah, N. J.: Erlbaum. Retrieved September 1, 2010 from http://www.hnrc.ucsd.edu/publications_pdf/297art1999.pdf

Ruff, R. M., & Jurica, P. (1999). In search of a unified definition for mild traumatic brain injury. *Brain Injury, 13(12)*, 943-952.

Ruff, R. M., Levin, H. S., Mattis, S., High, W. M., Marshall, L. F., Eisenberg, H. M. et al. (1989). Recovery of memory after mild head injury: A three-center study. In H. S. Levin and H. M. Eisenberg & A. L. Benton (Eds.), *Mild Head Injury* (pp.176-188). New York: Oxford University Press. Viewed March 5, 2010 at http://www.google.com/search?client=safari&rls=en&q=Mattis-Kovner+Verbal+Learning+and+Memory+Test&ie=UTF-8&oe=UTF-8

Rugby Football History. (undated). Retrieved November 10, 2009, from http://www.rugbyfootballhistory.com

Rugby School. (2009, November 1). In Wikipedia, The Free Encyclopedia. Retrieved November, 11, 2009, from http://en.wikipedia.org/wiki/Rugby_School

Rugby Tens. (2010, January 1). In Wikipedia, The Free Encyclopedia. Retrieved January, 29, 2010, from http://en.wikipedia.org/wiki/ Rugby_ tens

Rugby Union Rules (undated). Retrieved November 10, 2009, from http://www.rugbyunionrules.com

Rugby World Cup. (2010, January 28). In Wikipedia, The Free Encyclopedia. Retrieved January, 28, 2010, from http://en.wikipedia.org/wiki/Rugby_World_Cup

Rugby World Cup Sevens. (2010, January 9). In Wikipedia, The Free Encyclopedia. Retrieved January, 28, 2010, from http://en.wikipedia.org/wiki/ Rugby_ World_Cup_Sevens

Russell, W. R., & Smith, A. (1961). Post-traumatic amnesia in closed head injury. *Archives of Neurology, 5*, 16-29.

Rutgers, D. R., Toulgoat, F., Cazejust, J., Fillard, P., Lasjaunias, P., & Ducreux, D. (2008). White matter abnormalities in mild traumatic brain injury: A diffusion tensor imaging study. *Amrican Journal of Neuroradiology, 29*, 514-519. Retrieved March 26, 2010, from http://www.ajnr.org/cgi/content/full/29/3/514

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.

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

Ryan, J. P., Atkinson, T. M., & Dunham, K. T. (2004). Sports-related and gender differences on neuropsychological measures of frontal lobe functioning. *Clinical Journal of Sports Medicine, 14(1),* 18-24.

Ryan, L. M., & Warden, D. L. (2003). Post concussion syndrome. *International Review of Psychiatry, 15*, 310-316.

Salinas, C. M., Webbe, F. M., & Devore, F. T. (2009). The epidemiology of soccer heading in competitive youth. *Journal of Clinical Sports Psychology, 3*, 14-33.

Sallis, R. J., Sunshine, S., & Simon, L. (2001). Comparing sports injuries in men and women. *International Journal of Sports Medicine*, *22*, 420-423.

Sánchez, J. L., Rodríguez, M., & Carro, J. (2002). Influence of cognitive reserve on neuropsychologic functioning in Alzheimer's disease type sporadic in subjects of Spanish nationality. *Neuropsychiatry, Neuropsychology & Behavioral Neurology, 15(2)*, 113-122.

Santalucia, P., & Feldmann, E. (2000). Concussion and head injury. *Medicine and Health/Rhode Island, 83(6)*, 173-177.

SANZAR. (2009, December 27). In Wikipedia, The Free Encyclopedia. Retrieved February, 1, 2010, from http://en.wikipedia.org/wiki/SANZAR

SARugby. (2009, October, 25). Currie Cup history. Retrieved November 10, 2009, from http://www.sarugby.com/news/News/article/sid=8000.html

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., 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.

Scarmeas, N., & Stern, Y. (2003). Cognitive reserve and lifestyle. *Journal of Clinical and Experimental Neuropsychology, 25(5), 625-633.*

Scarmeas, N., Zarahn, E., Anderson, K. E., Hilton, J., Flynn. J., Van Heertum, R. L. et al. (2003). Cognitive reserve modulates functional brain responses during memory tasks: A PET study in healthy young and elderly subjects. Retrieved February 22, 2010, from http://cumc.columbia.edu/dept/sergievsky/cnd/pdfs/CogRes4.pdf

Schatz, P. (2010). Long-term test-retest reliability of baseline cognitive assessments using ImPACT. *American Journal of Sports Medicine, 38(1)*, 47-53. Retrieved October 14, 2010 from http://schatz.sju.edu/downloads/research/AJSM_Schatz_PreView.pdf

Schatz, P., & Browndyke, J. (2002). Applications of computer-based neuropsychological assessment. *Journal of Head Trauma Rehabilitation, 17(5)*, 395-410.

Schatz, P., Neidzwski, K., Moser, R. S., & Karpf, R. (2010). Relationship between subjective test feedback provided by high-school athletes during computer-based assessment of baseline cognitive functioning and self-reported symptoms. *Archives of Clinical Neuropsychology, 25(4)*, 285-292.

Schatz, P., Pardini, J.E., Lovell, M.R., & Collins, M.W., & Podell, K. (2006). Sensitivity and Specificity of the ImPACT Test Battery for concussion in athletes. *Archives of Clinical Neuropsychology, 21(1)*, 91-99.

Schatz, P., & Putz, B. O. (2006). Cross-validation of measures used for computer-based assessment of concussion. *Applied Neuropsychology, 13(3)*, 151-159.

Schatz, P., & Zillmer, E. (2003). Computer-based assessment of sports-related concussion. *Applied Neuropsychology, 10(1)*, 42-47.

Scher, A. T. (1977). Rugby injuries of the cervical spine and spinal cord – has the situation improved? *Sports Medicine, 5(2),*9-14.

Scher, A. T. (1987). Rugby injuries of the spine and spinal cord. *Clinics in Sports Medicine, 6(1),* 87-99.

Schick, D. M., Molloy, M. G., & Wiley, J. P. (2008). Injuries during the 2006 Women's Rugby World Cup. *British Journal of Sports Medicine, 42*, 447-451.

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). N.F.L. Acknowledges long-term concussion effects. *New York Times, December 20, 2009.* Retrieved September 6, 2010, from http://www.nytimes.com/2009/12/21/sports/football/21concussions.html?ref=boston_university

Schwartz, A. (2010). N.F.L. Asserts greater risks of head injury. *New York Times, July 26, 2010.* Retrieved September 6, 2010, from

Scott, J. G., Krull, K. R., Williamson, D. J., Adams, R. L., & Iverson, G. L. (1997). Oklahoma premorbid intelligence estimation (OPIE): Utilization in clinical samples. *The Clinical Neuropsychologist, 11*, 146-154.

Scrum (rugby). (2010, January 12). In Wikipedia, The Free Encyclopedia. Retrieved February, 2, 2010, from http://en.wikipedia.org/wiki/ Scrum_ (rugby)

Scrum (rugby union). (2010, January 4). In Wikipedia, The Free Encyclopedia. Retrieved February, 2, 2010, from http://en.wikipedia.org/wiki/ Scrum_ (rugby_union)

Sebastianeli, W. J. (2006). *Injury rate during preseason practice three times higher than during competition in college football*. Presented at the Sports Medicine and Football Conference, Received September 28, 2010 from http://www.sportsmed.org/tabs/newsroom/AOSSMPressReleaseDetails.aspx?DID=285

Segalowitz, S. J., Bernstein, D. M., & Lawson, S. (2001). P300 event related potential decrements in well-functioning university students with mild head injury. *Brain and Cognition, 45*, 342-356.

Segatore, M., & Way, C. (1992). The Glasgow Coma Scale: Time for a change. *Heart and Lung, 21*(6), 548-557.

Seliger, G., Lichtman, S. W., & Polsky, T. (1997) The effect of apolipoprotein E on short-term recovery from head injury. *Lancet, 350*, 1069-1071.

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.

Shah, M., Bazarian, J., Mattingly, A., Davis, E., & Schneider, S. (2004). Patients with head injuries refusing emergency medical services transport. *Brain Injury, 18(8)*, 765-773.

Shallice, T., & Burgess, P. W. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain, 114*, 727-741.

Shapiro, A. M., Benedict, R. H., Schretlen, D., & Brandt, J. (1999). Construct and current validity of the Hopkins Verbal Learning Test-revised. *Clinical Neuropsychology, 13*, 348-358.

Sharp, J. C., Murray, G. D., & Macleod, D. A. (2001). A unique insight into the incidence of rugby injuries using referee replacement reports. *British Journal of Sports Medicine, 35(1)*, 34-37.

Shaw, N. A. (2002). The neurophysiology of concussion. *Progress in Neurobiology, 67(4)*, 281-344.

Shawdon, A., & Brukner, P. (1994). Injury profile of amateur Australian rules footballers. *Australian Journal of Science and Medicine in Sport, 26(3-4),* 59-61.

Sheldon, T. (2003). Boxing should be banned unless rules are tightened, advises Dutch body. *British Medical Journal, (327*), 1186. Retrieved Marc 20, 2010 from doi:10.1136/bmj.327.7425.1186-b.

Sheridan, L. K., Fitzgerald, H. E., Adams, K. M., Nigg, J. T., Martel, M. M., Puttler, L. I. et al. (2006). Normative Symbol Digit Modalities Test performance in a community-based sample. *Archives of Clinical Neuropsychology, 21(1)*, 23-28. Retrieved October 26, 2010 from http.www.science direct

Shores, E. A., Lammel, A., Hullick, C., Sheedy, J., Flynn, M., Levick, W. et al. (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.* Retrieved February 09, 2008, from http://www.jnnp.bmj.com/cgi/reprint/79/10/1100.

Shuttleworth-Edwards, A. B. (2002). Fine tuning of the Digit Symbol Paired Associate Recall Test for practitioner purposes in clinical and research settings. *The Clinical Neuropsychologist, 16(3)*, 232-241.

Shuttleworth-Edwards, A. (2004). Never say never: Taking account of additive and variability effects in sports concussion research. In M. Lovell (Chair), *Mild traumatic brain injury in sports: An international neuropsychological perspective.* Symposium conducted at the Thirty-Second Meeting for International Neuropsychological Society, Baltimore, Maryland, USA.

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. A. (2002). Computer based screening in concussion management: Use versus abuse. *British Journal of Sports Medicine, 36*, 473.

Shuttleworth-Edwards, A., Border, M., Reid, I., & Radloff, S. (2004). South African Rugby Union. In M. R. Lovell, R. J. Echemendia, J. T. Barth, & M. W. Collins (Eds.), *Traumatic brain injury in sports* (pp. 149-168). The Netherlands: Swets & Zeitlinger Publishers.

Shuttleworth-Edwards, A. B., Gaylard, E. K., & Radloff, S.E. (in press). WAIS-III test performance in the South African context: Refinement of an existing cross-cultural normative database. In: K. Cockcroft & S. Laher (Eds.), *Psychological Assessment in South Africa: Research and Applications*. Johannesburg: Wits Press.

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., Radloff, S. E., Whitefield, V. J., Clark, S. B., Roberts, C. O. et al. (2008). The comparative incidence of reported concussions presenting for follow-up management in South African Rugby Union. *Clinical Journal of Sport Medicine, 18(5),* 403-409.

Shuttleworth-Edwards, A. B., & Radloff, S. E. (2008). Compromised visuomotor processing speed in players of Rugby Union from school through to the national adult level. *Archives of Clinical Neuropsychology, 23(5)*, 511-520.

Shuttleworth-Edwards, A. B., Smith, I., & Radloff, S. (2008). Neurocognitive vulnerability amongst university rugby players versus noncontact sport controls. *Journal of Clinical and Experimental Neuropsychology, 30(8)*, 870-884.

Shuttleworth-Edwards, A. B., Van der Merwe, A. S., van Tonder, P. & Radloff, S. E. (in press). WISC-1V test performance in the South African context: A collation of cross-cultural norms. In: K. Cockcroft & S. Laher (Eds.), *Psychological Assessment in South Africa: Research and Applications*. Johannesburg: Wits Press.

Shuttleworth-Edwards, A. B., & Whitefield, V. J. (2007a). 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, V. J. (2007b). Optimal application of neurocognitive assessment in concussion management: A professional dilemma. *South African Journal of Sports Medicine, 19(2),* 101-104.

Shuttleworth-Edwards, A. B., Whitefield-Alexander, V. J., & Radloff, S. E. (Submitted). 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*.

Shuttleworth-Edwards, A. B., Whitefield-Alexander, V. J., Radloff, S. E., Taylor, A. M., & Lovell, M. R. (2009). Computerized neuropsychological profiles of South African versus US athletes: A basis for commentary on cross-cultural norming issues in the sports concussion arena. *Physical Sportsmedicine, 37(4)*, 45-52.

Shuttleworth-Jordan, A. B. (1996). On not reinventing the wheel: A clinical perspective on culturally relevant test usage in South Africa. *South African of Psychology, 26(2)*, 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. J. Anderson (Eds.), *Proceedings of the 5th national neuropsychology conference* (pp 39-68). Durban. South Africa: University of Natal. South African Clinical Neuropsychological Association (SACNA).

Siedlecki, K. L, Stern, Y, Reuben, A., Sacco, R. L., Elkind, M. S., & Wright, C. B. (2009). Construct validity of cognitive reserve in a multiethnic cohort: The Northern Manhattan Study. *Journal of the International Neuropsychological Society, 15*, 558-269.

Sigurdardottir, S., Andelic, N., Roe, N., Jerstad, T., & Schanke, A. (2009). Post-concussion symptoms after traumatic brain injury at 3 and 12 months post-injury: A prospective study. *Brain Injury, 23(6)*, 489-497. Abstract retrieved October 14, 2010, from http://www.ncbi.nlm.nih.gov/pubmed/19484622

Silver, J. M., McAllister, T. W., & Arciniegas, D. B. (2009). Depression and cognitive complaints following mild traumatic brain injury. *The American Journal of Psychiatry, 166*, 653-661. Retrieved June 15, 2010, from http://ajp.psychiatryonline.org/cgi/content/full/166/6/653

Silver, J.M., McAllister, T.W., & Yudofsky, S.C. (2005). *Textbook of Traumatic Brain Injury.* Washington, DC, American Psychiatric Publishing, Inc.

Silver, J. M., Yudofsky, S. C., & Anderson, K. E. (2005). Aggressive Disorders. In J. M. Silver, T. W. McAllister, & S. C. Yudofsky (Eds.). *Textbook of Traumatic Brain Injury*. Washington DC: American Psychiatric Publishing, Inc.

Silver, J. R. (1984). Injuries of the spine sustained in rugby. *British Medical Journal, 288*, 37- 43.

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.

Skinner, J. H. (2008). *A pilot project to investigate a novel computerized concussion assessment tool for use in the emergency department and other outpatient settings.* Unpublished masters thesis, Queens University, Kingston, Canada. Retrieved March 26, 2010, from https://qspace.library.queensu.ca/bitstream/1974/1469/1/Skinner_Jennifer_HC_20080 9_MSc.pdf

Slack, M. K., & Draugalis, J. R. (2001). Establishing the internal and external validity of experimental studies. *American Journal of Health-System Pharmacy, 58(22).* Retrieved November 5, 2010 from http://www.medscape.com/viewarticle/414875

Slobounov, S., Slobounov, S., Sebastianelli, W., Cao, C., & Newell, K. (2007). Differential rate of recovery in athletes after first and second concussion episodes. *Neurosurgery, 61(2)*, 338-344.

Smith, A. (1973). *Symbol Digit Modalities Test Manual.* Los Angeles: Western Psychological Services

Smith, A. (1991). *A Symbol Digit Modalities Test.* Los Angeles: Western Psychological Services

Smith, S. (1999). *The union game: A rugby history*. London: BBC Worldwide Ltd.

Snow, N. (2010). *MIAA coaches required to take concussion education course*. Retrieved October 31, 2010 from http://concord.patch.com/articles/miaa-coaches-required-to-take-concussion-education-course-7

Soccer Training Guide. (undated). *Basic soccer rules - why you need to follow them?* Retrieved June 8, 2010, from http://www.soccer-training-guide.com/soccer-rules.html

Solomon, G.S., Johnston, K.M., & Lovell, M.R. (2006). *The heads-up on sport concussion*. United States: Human Kinetics.

Sortland, O., & Tysvaer, A. T. (1989). Brain damage in former association football players. *Neuroradiology, 31(1),* 44-48.

South African Government online. (undated). Retrieved May 28, 2006, from http://www.gov.za/

South African Wechsler Adult Intelligence Scale manual. (1969). Johannesburg: National Institute for Personnel Research.

Sosnoff, J. J., Broglio, S. P., Hillman, C. H., & Ferrara, M. S. (2007). Concussion does not impact intraindividual response time variability. *Neuropsychology, 21(6)*, 796 – 802. Retrieved October 20, 2010, from http://kch.illinois.edu/Research/Labs/neurocognitive-kinesiology/files/Articles/Sosnoff_2007_ConcussionDoesNotImpact.pdf

Sparks, J. P. (1981). Half a million hours of rugby football. *British Journal of Sports Medicine, 15(1),* 30-32.

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

Sportex. (2009). *Concussion in sport*. Retrieved August 13, 2009, from http://www.averonleisurecentre.co.uk.

Sports Dentistry Online (2009). *Concussion prevention and athletic mouthguards*. Retrieved September 7, 2009, from http://www.sportsdentistry.com/concussion.html

Spreen, O., & Straus, E. (1998). *A compendium of neuropsychological tests* (2nd ed.). New York: Oxford University Press.

Stalnacke, B. (2009). Relationship between symptoms and psychological factors five years after whiplash injury. *Journal of Rehabilitative Medicine, 41*, 353-359.

Stalnacke, B., Tegner, Y., & Sojka, P. (2003). Playing ice hockey and basketball increases serum levels of S-100B in elite players: A pilot study. *Clinical Journal of Sport Medicine, 13*, 292–302.

Stalnacke, B., Tegner, Y., & Sojka, P. (2004). Playing soccer increases serum concentrations of the biochemical markers of brain damage S-100B and neuron-specific enolase in elite players: A pilot study. *Brain, 18*, 899–909.

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.

Stephens, R., Rutherford, A., Potter, D., & Fernie, G. (2010). Neuropsychological consequence of soccer play in adolescent U.K. school team soccer players. *The Journal of Neuropsychiatry and Clinical Neurosciences, 22*, 295-303. Retrieved October 12, 2010 from http://neuro.psychiatryonline.org/cgi/content/full/22/3/295

Stern, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society, 8,* 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. (2009). Cognitive Reserve. *Neuropsychologia,* 10, 2015-2028. Retrieved April 23, 2010, from http://cpmcnet.columbia.edu/dept/sergievsky/cnd/pdfs/sdarticle-1.pdf

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.

Stevenson, M., Finch, C., Hamer, P., & Elliott, B. (2003). The Western Australia sports injury study. *British Journal of Sports Medicine, 37(5)*, 380–381. *Retrieved March, 20, 2010, from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1751370/pdf/v037p00380.pdf*

Stiver, S. I., & Manley, G. T. (2008). Prehospital management of traumatic brain injury. *Journal of Neurosurgical Focus, 25(4)*, E5. Retrieved February 19, 2009, from http://www.medscape.com/viewarticle/585165

Stoddard, E. (2007). *Measuring learning modalities with neuropsychological memory measures in a college population.* Doctoral thesis submitted to DrexelUniversity. Retrieved August 10, 2010, from http://idea.library.drexel.edu/bitstream/1860/1797/1/Stoddard_Eve.pdf

Straume-Naesheim, T. M., Andersen, T. E., Dvorak, J., & Bahr, R. (2005). Effects of heading exposure and previous concussions on neuropsychological performance among Norwegian elite footballers. *British Journal of Sports Medicine, 39 (1),* i70-i77.

Strauss, E., Sherman, E. M.S., & Spreen, O. (2006). *A compendium of neuropsychological tests.* Oxford: Oxford University Press.

Stuart, J. (n. d.). *The history and origins of rugby union*. Hubpages Incorporated. Retrieved November 1, 2009, from http://aru.rugby.com.au/community_rugby/what_is_rugby/history_of_the_game,24.html

Sturmi, J.E., Smith, C., & Lombardo, J. A. (1998). Mild brain trauma in sports. *Sports Medicine, 25(6)*, 352-358.

Stuss, D. T., & Alexander, M. P. (2000). Executive functions and the frontal lobes: A conceptual view. *Psychological Research*, 63, 289-298.

Stuss, D. T., Binns, M. A., Carruth, F. G., Levine, B., Brandys, C. E., Moulton, R. J. et al. (1999). The acute period of recovery from traumatic brain injury: Posttraumatic amnesia or posttraumatic confusional state? *Journal of Neurosurgery,* 90, 635-643.

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

Sullivan, S. J., Bourne, L., Choie, S., Eastwood, B., Isbister, S., McCrory, P. et al. (2009). Understanding of sport concussion by the parents of young rugby players: A pilot study. *Clinical Journal of Sport Medicine, 19(3)*, 228-230.

Super 14. (2010, January 21). In Wikipedia, The Free Encyclopedia. Retrieved January, 25, 2010, from http://en.wikipedia.org/wiki/Super_14

Swann, I. J., & Teasdale, G. M. (1999). Current concepts in the management of patients with so-called 'minor' or 'mild' head injury. *Trauma, 1*, 143-155.

Sye, G., Sullivan, S. J., & McCrory, P. (2006). High school rugby players' understanding of concussion and return to play guidelines. *British Journal of Sports Medicine, 40*, 1003-1005. Retrieved March 26, 2010, from doi;10.1136/bjsm.2005.020511

Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: A practical scale. *Lancet, 13, 2(7872)*, 81-84.

Teasdale, G. M., Murray, G. D., & Nicoll, J. A. (2005). The association between APOE 4, age and outcome after head injury: A prospective cohort study. *Brain, 128(11)*, 2556-2561. Retrieved March 16, 2010, from http://brain.oxfordjournals.org/cgi/reprint/128/11/2556

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, T. W., & Engberg, A. (1997). Duration of cognitive dysfunction after concussion, and cognitive dysfunction as a risk factor: A population study of young men. *British Medical Journal, 315,* 569-572.

Tegeler, C., Kim, J., Collins, G., Steelman, D., Westwood, K., Reynolds, P. et al. (work in progress). Transcranial Doppler ultrasound for concussion in amateur athletes. Retrieved November 2, 2009, from http://www.newhealthsciences.com/NewsItems/2-Concussion%20Poster.pdf

Tegner, Y., & Back, B. (2000). Injury pattern in Swedish elite ice hockey. *Hygiae, 199*, 204.

Tegner, Y., & Lorentzon, R. (1996). Concussion among Swedish elite ice hockey players. *British Journal of Sports Medicine, 30*, 251-255.

Tellier, A., Malva, L. C., Cwinn, A., Grahovac, S., Morrish, W., & Brennan-Barnes, M. (1999). Mild head injury: A misnomer. *Brain Injury, 13 (7),* 463-475.

Terrell, T. R. (2004). Concussion in athletes. *Sports Medicine, Southern Medical Journal, 97(9)*, 837-842. Retrieved March 9, 2010, from http://www.accessmylibrary.com/article-1G1-123332690/concussion-athletes-featured-cme.html

The Fife Headway Group. *Understanding TBI (Traumatic Brain Injury).* Retrieved August, 17, 2009, from http://www.fifeheadway.org.uk/index.asp?MainID=9117.

Theisen, M. (1997). *The effects of practice in repeated administration of the Wechsler Memory Scale-Revised and the California Verbal Learning Test in normal adults.* Michgan: Wayne State University.

Thomassen, A., Juul-Jensen, P., Olivarius, B. D, Briemer, J., & Christensen, A-L. (1979). Neurological, electroencephalographic and neuropsychological examination of 53 former amateur boxers. *Acta Neurologica Scandanavica, 60(6)*, 352-362.

Thompson, J. W. (2006). EEG changes and balance deficits following concussion: One piece of the puzzle. In S. Slobounov & W. Sebastianelli (Eds.), *Foundations of sport-related brain injuries.* USA: Springer. Abstract retrieved October 20, 2009, from http://www.springerlink.com

Thompson, J., Sebastianelli, W., & Slobounov, S. (2005). EEG and postural correlates of mild traumatic brain injury in athletes. *Neuroscience Letters, 377*, 158-163. Abstract retrieved March 11, 2010, from ScienceDirect http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6T0G-4F320NH-6

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., & Guerrero, J. (1997). Trends in hospitalization associated with traumatic brain injury. *Journal of the American Medical Association, 282 (10)*, 754-757.

Tinker, J. R. (2010). *Reported visual disturbance and post-concussion cognitive function in collegiate athletes: The relationship between symptom report and neurocognitive outcome*. Thesis submitted to Drexel University. Retrieved July 27, 2010 from http://idea.library.drexel.edu/bitstream/1860/3264/1/Tinker_Jennifer.pdf

Titolo, T. R. (2009). *Brain and spine law blog: Mild traumatic brain injury*. Retrieved August, 13, 2009, from http://www.brainandspine.titololawoffoce.com/2009/08/articles/articles-1/mild-traumatic-brain-injry/print.html.

Tombaugh, T. N., Rees, L., Stromer, P., Harrison, A. G., & Smith, A. (2007). The effects of mild and severe traumatic brain injury on speed of information processing as measured by the computerized tests of information processing (CTIP). *Archives of Clinical Neuropsychology, 22(1)*, 25-36. Abstract retrieved March 26, 2010, from http://can.oxfordjournals.org/cgi/content/abstract/22/1/25

Tommasone, B.A., & Valovich McLeod, T.C. (2006). Contact sport concussion incidence. *Journal of Athletic Training 41(4)*, 470-472.

Townend, W., & Ingebrigtsen, T. (2006). Head injury outcome prediction: A role for protein S-100B?" *Injury 37(12)*, 1098-1108.

Tri Nations (rugby union). (2010, January 16). In Wikipedia, The Free Encyclopedia. Retrieved February, 1, 2010, from http://en.wikipedia.org/wiki/Tri_Nations_(rugby_union)

Tromp, E., & Mulder, T. (1991). Slowness of information processing after traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology, 13(6)*, 821-830.

Tsacopoulos, M., & Magistretti, P. J. (1996). Metabolic coupling between glia and neurons. *Journal of Neuroscience, 16(3)*, 877-885.

Tucker, A. (1997). Common soccer injuries. *Sports Medicine, 23(1)*, 21-32.

Tulsky, D. S. (2003). Troubleshooting concerns raised about the WMS-III. [Abstract]. In D.S. Tulsky (Chair), Thirty-First Annual International Neuropsychological Society Meeting Abstracts. *Journal of the International Neuropsychological Society, 9(2), 197.*

Tulsky, D., Zhu, J., & Ledbetter, M. F. (2002). *WAIS-III/WMS-III technical manual*. USA: The Psychological Corporation.

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

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.

Ullman, M. T. (2004). Contributions of memory circuits to language: The declarative/procedural model. *Cognition, 92*, 231–70.

University of South Africa (UNISA). (1996). *Psychological Research: Tutorial Letter 109 for PSY314-E 1996*. Pretoria: Muckleneuk.

Uttl, B., Graf, P., & Richter, L. K. (2002). Verbal Paired Associates tests limits on validity and reliability. *Archives of Clinical Neuropsychology, 17(6),* 567-681.

Uzzell, B. P. (1999). Mild head injury: Much ado about something. In R. Varney, & R. J. Roberts (Eds.), *The Evaluation and Treatment of Mild Traumatic Brain Injury* (pp. 1-13). New Jersey: Lawrence Erlbaum Associates, Publishers.

Valenzuela, M. J. (2008). Brain reserve and the prevention of dementia. *Current Opinion in Psychiatry, 21(3)*, 296-302. Retrieved April 26, 2010, from http://www.medscape.com/viewarticle/572545

Valenzuela., M. J., Breakspear, M., & Sachdev, P. (2007). Complex mental activity and the aging brain: Molecular, cellular and cortical network mechanisms. *Brain Research Reviews*, *56*, 198-213.

Valenzuela, M. J., & Sachdev, P. (2006). Brain reserve and dementia: A systematic review. *Psychological Medicine, 36*, 441-454. Retrieved April 26, 2010, from http://journals.cambridge.org/action/displayAbstract?fromPage=online&aid=414382

Valovich McLeod, T. C. (2009). The value of various assessment techniques in detecting the effects of concussion on cognition, symptoms and postural control. *Journal of Athletic Training, 44 (6),* 663-665.

Valovich McLeod, T. C., Heil, J., McVeigh S. D., & Bay, R. C. (2006). Identification of sport and recreational activity concussion history through the pre-participation screening and symptom survey in young athletes. *Journal of Athletic Training, 41*, S-91. (Abstract).

Valovich McLeod, T., Schwartz, C., & Bay, R. C. (2007) Sport-related concussion misunderstandings among youth coaches. *Clinical Journal of Sports Medicine, 17*, 140-142.

Van der Elst, W., Van Boxtel, M. P., Van Breukelen, G. J., & Jolles, J. (2006). The Stroop Color-Word Test influence of age, sex, and education: and normative data for a large sample across the adult age range. *Assessment, 13 (1)*, 62-79. Retrieved October 26, 2010, from http://arno.unimaas.nl/show.cgi?fid=4598 DOI:10.1177/1073191105283427

Van der Naalt, J. (2001). Prediction of outcome in mild to moderate Head Injury: A Review. *Journal of Clinical and Experimental Neuropsychology, 23(6)*, 837-851.

van der Naalt, J., van Zomeren, A. H., Sluiter, W. J., & Minderhoud, J. M. (1999). One year outcome in mild to moderate head injury: The predictive value of acute injury characteristics related to complaints and return to work. *Journal of Neurology, Neurosurgery and Psychiatry, 66*, 207-213.

Vanderploeg, R. D., Belanger, 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. (Abstract). Retrieved October 20, 2009, from http://www.springerlink.com

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.

Van Dyke, S. A., Axelrod, B. N., & Schutte, C. (2010). The utility of the post-concussive symptom questionnaire. *Archives of Clinical Neuropsychology, 25(7)*, 634-639. Abstract retrieved November 8, 2010, from http://www.ncbi.nlm.nih.gov/pubmed/20710017

Van Kampen, D. A., Lovell, M. R., Pardini, J. E., Collins, M. W., & Fu, F. H. (2006). The "value added" of neurocognitive testing following sport-related concussion. *American Journal of Sports Medicine, 34(10)*, 1630-1635.

Varney, N. R., & Menefee, L. M. (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., Bir, C. A., Zhang, L., Sherman, D. C. et al. (2005). Concussion in Professional Football: Comparison with Boxing Head Impacts-Part 10. *Neurosurgery, 57(6)*, 1154-1172. Retrieved March 8, 2010, from doi: 10.1227/01.NEU.0000187541.87937.D9

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.

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

Wallack, M. (2006). *Cognitive reserve: Evolution and adaptation of an explanatory concept*. Prepared for presentation at the Ninth Annual International Symposium on the Treatment of Alzheimer Disease, October 19-21, 2006, Wolfville, Nova Scotia. Retrieved April 23, 2010, from http://www.ucs.mun.ca/~mwallack/cr.pdf

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

Wechsler, D. (1981). *WAIS-R manual.* New York: The Psychological Corporation.

Wechsler, D. (1997). *Wechsler Adult Intelligence Scale – III*. San Antonio: The Psychological Corporation.

Wechsler, D. (2002). *WAIS-III/WMS-III technical manual.* San Antonio: The Psychological Corporation.

Wechsler, D., Wycherley, R. J., Benjamin, L., Callanan, M., Lavender, T., Crawford, J. et al. (1998a). *WAIS-III^{UK} administration and scoring manual.* Harcourt Place, London: The Psychological Corporation Limited.

Wechsler, D., Wycherley, R. J., Benjamin, L., Callanan, M., Lavender, T., Crawford, J. et al. (1998b). *WMS-III^{UK} Administration and Scoring Manual.* Harcourt Place, London: The Psychological Corporation Limited.

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

Weisstein, E. W. (undated). Bonferroni Correction. *MathWorld - A Wolfram Web Resource.* Retrieved July 27, 2007, from http://mathworld.wolfram.com/BonferroniCorrection.html

Wekesa, M., Asembo, J. M., & Njororai, W. W. (1996). Injury surveillance in a rugby tournament. *British Journal of Sports Medicine, 30*, 61-63.

West, R., Murphy, K. J., Armilio, M., Craik, F. I., & Stuss, D. T. (2002). Lapses of intention and performance variability reveal age-related increases in fluctuations of executive control. *Brain and Cognition, 49*, 402–419.

Whitefield, V. (2007). *Complete handbook for concussion management and interpretation*. South Africa: Cape Town.

Wilberger, J. E. (1993). Minor head injuries in American football: Prevention of long term sequelae. *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.

Willer, B., & Leddy, J. J. (2006). Management of concussion and post-concussion syndrome. *Current Treatment Options in Neurology, 8*, 415-426.

Williams, D. H., Levin, H. S., & Eisenberg, H. M. (1990). Mild head injury classification. *Neurosurgery, 27*, 422-428.

Wills, S. M., & Leathem, M. (2001). An investigation of brain injury incurred in New Zealand club-grade rugby. *Journal of International Neuropsychological Society, 7*, 405.

Wilner, A. (2010). Mild traumatic brain injury/concussions – new Department of Defense guidelines. *Neuro Notes.* Retrieved February 4, 2010, from http://boards.medscape.com/forums

Wilson, J. T., Wiedmann, K. D., Hadley, D. M., Condon, B., Teasdale, G., & Brooks, D. N.(1988). Early and late magnetic resonance imaging and neuropsychological outcome after head injury. *Journal of Neurology, Neurosurgery and Psychiatry, 51(3),* 391-396.

Witol, A. D., & Webbe, F. M. (2003). Soccer heading frequency predicts neuropsychological deficits. *Archives of Clinical Neuropsychology, 18*, 397-417.

Wong, J. L., & Gilpin, A. R. (1993). Verbal vs. visual categories on the Wechsler Memory Scale-Revised: How meaningful a distinction. *Journal of Clinical Psychology, 49*, 847-854.

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., Parsons, 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. et al. (1999). Concussion in sports. *The American Journal of Sports Medicine, 27(5),* 676-687.

World Health Organization. (1993). *The ICD-10 classification of mental and behavioural disorders: Diagnostic criteria for research*. Geneva: World Health Organization. Retrieved September 1, 2010, from http://www.who.int/classifications/icd/en/GRNBOOK.pdf

Wrightson, P., & Gronwall, D. (1981). Time off work and symptoms after minor head injury. *Injury, 12*, 445-454.

Wrightson, P., & Gronwall, D. (1998). Mild head injury in New Zealand: Incidence of injury and persisting symptoms. *New Zealand Medical Journal, 111*, 99-101.

Wrightson, P., & Gronwall, D. (1999). *Mild head injury.* Oxford: Oxford University Press.

Yang, C. C., Tu, Y. K., Hua, M. S., & Huang, S. J. (2007). The association between the postconcussion symptoms and clinical outcomes for patients with mild traumatic brain injury. *Journal of Trauma, 62(3*), 657-663.

Yarnell, P.R., & Lynch, S. (1973). The "ding": amnestic states in football trauma. Retrograde memory immediately after concussion. *Neurology, 23*, 196-197.

Yu, C., & Ohlund, B. (2010). *Threats to validity of research design*. Retrieved November 5, 2010 from http://www.creative-wisdom.com/teaching/WBI/threat.shtml

Zetterberg, H., Jonsson, M., Rasulzada, A., Popa, C., Styrud, E., Hietala, M. A. et al. (2007). No neurochemical evidence for brain injury caused by heading in soccer. *British Journal of Sports Medicine, 41,* 574–577.

Zheng, Y., Myerson, J., & Hale, S. (2000). Age and individual differences in visuospatial processing speed: Testing the magnification hypothesis. *Psychonomic Bulletin and Review 7(1),* 113-120.

Appendix A.

GENERAL INFORMATION FOR PARTICIPANTS' CONSENT

I, Sue Clark, am conducting research on the neuropsychological effects of concussion, as a requirement for a PhD in Psychology degree at Rhodes University.

What I am doing here is getting some assessment results that will help the doctor when managing the after effects of a head injury, should any of you get concussed while playing sport or for 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. US and Australia), and is already in place for the All Black rugby teams. To my knowledge you are going to be the first adults in South Africa to have this system in place, using the computer-based test ImPACT and tests from the Wechsler Memory Scale-111.

I am going to ask some questions, and collect some scores on a number of small tasks. Today the tasks will be verbal, on paper, and on computer. The assessment should take about an hour and a quarter. After the 2005 rugby season you will be assessed again on some of these tasks, which should take less than an hour.

Should you receive a concussion then you will be reassessed using some of the tests, and the results will be compared with those obtained in this first assessment we are doing now (before the rugby season begins). From this comparison it will be possible to gauge your recovery process, and the doctor will be able to make a more informed decision about how serious your concussion is, and when you are ready to go back to exercising and playing sport. The researcher only acts in a research capacity, and does not take responsibility for the decision about return to play.

Follow-up assessment after a concussion will be done at the Discovery SharkSmart medical offices at ABSA Stadium if you are in Durban or on a computer elsewhere if you are away from Durban. You will be given all that information if you do get concussed.

Whenever a rugby player is concussed, I will contact a matching person (e.g., same age) from the noncontact sport group who, not being concussed, will be reassessed along with the

person who is concussed. This will provide information about what happens when you repeat the tests when you are concussed compared with when you are not concussed.

The information collected is confidential and will only be looked at individually should you have a head injury. The identity of individual participants will not be made known, except to the research collaborators and the doctor treating the concussion. The information obtained will be used for research purposes (publication of a thesis and/or publication of research articles in medical/psychology journals) where readers will be unable to identify individuals. At a later date, this test data may be released for collaborative national or international research, but only if anonymity of the participants is guaranteed.

It is very important to do your best and to be as accurate and honest as possible when you answer the questions. If for any reason the information you give me is inaccurate or you cannot do your best on the tasks, this might cause the seriousness of your head injury to be underestimated, which would not benefit you medically. So, if at the end of the session you believe you have not been able to do your best for any reason, please tell me. Reasons might be because you have a headache, are worrying about something else, or felt distracted because there is a noise, and so on.

Your participation in this research is greatly appreciated as it will help increase future knowledge about concussion management. It is hoped that you will be committed to participating throughout the research period. Participation, however, is voluntary and you are free to withdraw from the study at any stage if some unusual circumstances occur or you have concerns about participating. You are invited to discuss your concerns with Sue Clark. If you do decide against participating, then it is important that you contact Sue Clark (Tel 031 561 1183 or 083 253 6751) to advise her.

Is there anything you don't understand, or are uncertain about? Are there any further questions? If you are happy to go ahead, please read and sign the consent form that will be given to you.

Appendix B.

RHODES UNIVERSITY
DEPARTMENT OF PSYCHOLOGY
AGREEMENT
BETWEEN STUDENT RESEARCHER AND RESEARCH PARTICIPANT

I _____ player of _____

have read and understood the information sheet about the research on the effects of concussion in rugby that will be conducted by Rhodes University student, Sue Clark.

I understand that:

1) The researcher is a student conducting research as a requirement for a Ph.D. in Psychology degree at Rhodes University.
2) The researcher is interested in the neuropsychological effects of concussion e.g., how attention, memory and reaction times are affected in the few weeks after concussion. Of interest is whether concussion has a long-term permanent effect on the brain. Also of interest is how effective the computerised test is compared to the non-computerised tests.
3) The research will involve rugby players and a control group of sportsmen involved in noncontact sport. The participants will be assessed using a computer-based neuropsychological assessment as well as traditional pencil and paper/ verbal tests. Testing will take place before and after the 2005 rugby season, at times convenient to players. The first pre-season assessment will take approximately one and a quarter hours. Subsequent assessments will take approximately 25-50 minutes. Follow-up assessments of concussed players will take place within 3 days of injury and then again three times at weekly intervals, or until symptoms resolve. Concussion follow-ups will usually take place at the Discovery Shark Smart medical offices at ABSA Stadium.
4) I will be asked to answer questions of a personal nature but I can choose not to answer any personal question that I am unwilling to disclose. The biographic questionnaire with medical information relevant to the research will be kept confidential.

5) The 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 a medical practitioner, and should only return to exercise or contact sport on the advice of the medical practitioner.
6) Participation in the research is voluntary, however I commit myself to full participation. I have the right to withdraw if some unusual circumstances occur or I am concerned about participating. The researcher invites participants to voice any concerns that they may have. I agree to contact Sue Clark (Tel 031 561 1183 or 083 253 6751) if I do not wish to participate in the study.
7) The test information collected on me will be strictly confidential and will only be available to the research collaborator or medical practitioner if I sustain a concussion. This information may form part of the management decision in individual cases. The researcher or research collaborators, however, will not be held accountable for medical decisions made by coaches, medical practitioners or players on the basis of test information.
8) Data arising out of this project will be used for thesis and publication purposes where individual identities will remain anonymous. At a later date, this test data may be released for collaborative national or international research, but only if anonymity of participants is guaranteed.

I agree to the above conditions and agree to participate in the research project of Sue Clark on concussion in rugby, and to be assessed by the researcher or a research assistant.

SIGNED **ON** **(DATE)**

_____

Appendix C.

INVITATION

You are invited to participate in an interesting sport research program.
The research is about CONCUSSION and how it affects Verbal and Visual Memory, Processing Speed, Reaction Time, and how long it takes to recover from concussion. The research subjects are the Sharks and Wildebeest Rugby players. It is hoped that you will volunteer to be a control participant – that is you are paired with a rugby player and should he become concussed and need post concussion assessments, you too would have these assessments to ensure that any changes in his functioning are due to concussion and not other reasons.
This research is being conducted by Sue Clark (Psychologist) as a requirement for a PhD in Psychology degree at Rhodes University.
The trial will involve at least two visits to Sue Clark – the first one to baseline your functioning should take 1 ¼ hours, and the second at the end of the rugby season to assess whether you are still performing to your baseline level, should take ¾ hour. Should your rugby partner become concussed then you would be required for four follow-up assessments that range from ½ to ¾ hour. The All Black rugby team uses ImPACT which is the computer test that you would be required to do.

The benefit to you would be that should you become concussed during the research you would also be monitored and this would help your medical practitioner decide when it is safe for you to return to sport. Most important is you would be benefiting science as a whole in understanding and treating concussion.
Should you be interested please fill in your details and hand to or fax to Sue Clark (031) 561 1183 or phone Sue on 083 253 6751.

Name: _____
Age: _____
Address: _____
_____
Phone: _____ Cell: _____
Email: _____

Appendix D.

OKLAHOMA PREMORBID INTELLIGENCE ESTIMATE: OPIE – 3 (2ST) ESTIMATION FORMULA USING ONLY THE WAIS-111 VOCABULARY AND MATIX REASONING SUBTESTS. (Schoenberg, Scott, Duff & Adams, 2002)

SE est = 6.63

Formula:

FSIQ = 45.997 + .652 (Vocabulary raw score) + 1.287 (Matrix Reasoning raw score) + .157 (age in years) + 1.034 (education) + .652 (ethnicity) – 1.015 (gender)

Coding Variables:

Age: In years

Ethnicity: 1 = African 2 = Hispanic 3 = Other 4 = Caucasian

Education: 1 (0 - 8 years) 2 (9 - 11 years) 3 (12 years) 4 (13 – 15 years) 5 (16 + years)

Gender: 1 = male 2 = female