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**"Glory is temporary, brain injury may be forever"  
A neuropsychological study on the cumulative effects of sports-related  
concussive brain injury amongst Grade 12 school boy athletes.**

**VOLUME ONE  
LITERATURE REVIEW**

**BY**

**VICTORIA JANE WHITEFIELD**

**Thesis presented for the Degree of  
DOCTOR OF SCIENCE  
in the Department of Science  
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## ABSTRACT

The study investigated the long-term neuropsychological effects of repetitive mild traumatic brain injury (MTBI) due to participation in a contact sport amongst South African final year male high school athletes (N=189). The sample was divided by sports affiliation (Contact n = 115; Non-Contact n = 74) and concussion history (2+ Concussion n = 43; 0 Concussion n = 108). Comparative subgroups were statistically equivalent for age, education and estimated IQ ( $p > 0.05$ ), with the Contact sport groups having markedly higher incidences of concussion than controls ( $p < 0.000$ ). Measures included the ImPACT Verbal and Visual Memory, Visuomotor Speed and Reaction Time Composites, Digit Symbol Substitution and Digit Symbol Incidental Recall (immediate and delayed), the ImPACT Symptom Scale and a Postconcussion Symptom (PCS) questionnaire. Independent t-tests on cognitive measures at pre-and post-season revealed a predominant trend of Contact and 2+ Concussion groups performing worse, although only ImPACT Reaction Time at pre-season reached significance ( $p = 0.014$ ). PCS comparisons revealed an overwhelming tendency of enhanced symptoms for Contact and 2+ Concussion groups with total scores being significantly different in most instances at pre-and post-season. Fatigue and aggression were the symptoms most pervasively high for the Contact and 2+ Concussion groups. Dependent t-test analyses at pre- versus post-season, revealed significant practice effects for the Contact group, not in evidence for controls on ImPACT Visual Motor Speed and Digit Symbol Incidental Recall–Delayed. Overall the results imply the possible presence of lingering neurocognitive and symptomatic concussion sequelae amongst South African final year high school participants of a contact sport. The indications gain potency when understood against the background of (i) Brain Reserve Capacity threshold theory, and (ii) the known risk of Type II error in group MTBI research, that might result in under-emphasis of subtle effects and miscalculation of cost-benefit risks. Clinical implications, and the need for prospective case-based research to ratify the results of this predominantly cross-sectional study, are discussed.

## CHAPTER ONE

### INTRODUCTION TO THE STUDY

There is a general attitude amongst high impact sports players of putting success, admiration and stardom over pain, the extent of which is exemplified by a slogan that appeared on the top team rugby jersey of a leading South African rugby playing school at an inter-schools jubilee: "*Pain is temporary and glory lasts forever*". Accordingly, the broad question underlying the motivation for this thesis is to enquire as to whether deleterious consequences specifically in relation to the brain are worth the cost of participation in a contact sport, and whether the level of damage here warrants the strongly prevailing mind-set exemplified by this rugby jersey template. More appropriately such a slogan might be turned around to read "*Glory is temporary and brain damage is forever*".

Specifically, in light of such a challenge, the central concern of the present study is with the long-term neuropsychological effects of repetitive mild traumatic brain injury sustained as a consequence of participation in a contact sport amongst South African final year high school learners from a relatively advantaged educational institution in the Western Cape. A contact sport is a sport associated with a high degree of contact between the participants involved (Wikipedia, n.d.). Examples of contact sports available in South Africa include Rugby Union, soccer, boxing, and the martial arts such as Karate, Kung Fu, and Jeet Kune Do.

In order to contextualize the field of mild traumatic brain injury in sport, a broad sketch of issues relating to traumatic brain injury in general will be provided as a frame of reference within which to locate the current study. This will be followed by a discussion of the measures of severity used in the treatment and diagnosis of traumatic brain injury, which includes the various grading scales used in the acute management of concussion. This paves the way for introducing the central role of neuropsychological testing in the evaluation and management of sports concussion. Finally there is an explication of the broad aims of this study.

#### 1.1 BROAD CATEGORIES OF TRAUMATIC BRAIN INJURY

Approximately two million people world-wide suffer from traumatic brain injury (TBI) each year, making TBI the leading cause of brain damage (National Institute of Neurological

Disorders and Stroke-NINDS, 1989). Traumatic brain injury refers to any injury involving the brain, although in most cases the term is used synonymously with head injury (Lezak, Howieson, & Loring, 2004). Due to its greater specificity, the term 'TBI' has currently gained favour over the term 'head injury' such that leading contemporary textbooks and periodicals in both the child and adult literature make use of the term TBI rather than head injury (e.g. Anderson, Northam, Hendy & Wrennall, 2001; Lovell, Echemendia, Barth, & Collins, 2004; Ruff, 2005; Rees, 2003; Silver, McAllister, & Yudofsky, 2005). It will therefore be the term of choice for the purposes of the present thesis, even when literature is being cited that has used the term head injury.

In broad terms, TBI can be classified as either open (penetrating) or closed. The closed TBI differs from the penetrating TBI not only in terms of the external causes but also in the neurological and neuropsychological sequelae that tend to occur (Levin, Benton, & Grossman, 1982). Typically the penetrating TBI is a focal lesion which penetrates the skull and dura mater. The most common causes are gunshot wounds or fragments from exploding shells which cause lacerations to the scalp, perforation or fracture of the skull and laceration of brain tissue in the path of the foreign body (Richardson, 1990). Because of the nature of the penetration, the morphological changes produced are restricted to the path of the projectile or the area directly under the depressed fracture (Adams, Parsons, Culbertson, & Nixon, 1996). The depth of dural penetration and loss of brain tissue represent measures of injury severity (Levin et al., 1982). Coma is uncommon after a penetrating TBI. This is supported by the fact that diffuse axonal injury is far less common after a penetrating TBI than a closed TBI (Adams et al., 1996). Posttraumatic amnesia (the absence of continuous memory), in terms of neuropsychological sequelae, is also infrequent after penetrating TBI.

In contrast to the penetrating TBI, most TBIs are closed where the skull remains intact and the contents of the brain are not exposed (Richardson, 1990), and for the purposes of this thesis, when the term TBI is used, it will be specifically with reference to the closed brain injury. Typically, a closed TBI involves blunt trauma to the head, resulting either from an impact by a moving object upon a stationary or slower moving head (acceleration), or when the head and body are decelerated by a stationary or slower moving object (Gualtierri, 1995). The type and extent of injury will be directly related to the speed and direction of the head movements (linear/translational acceleration, or rotational acceleration) and the time and distance of

deceleration (Adams et al., 1996). Newton's law illustrates this: force equals mass times acceleration or force divided by mass equals acceleration (Cantu, 1992). The movement of the head and neck on impact produces angular acceleration which involves a combination of both translational (linear) and rotational acceleration (Richardson, 1990).

In static injuries in which a relatively still individual receives a blow to the head, the site at which the impact occurs is called *coup* and directly across the skull from the point of contact is called *contrecoup* (Courville, 1942). Coup and contrecoup lesions account for specific and localized behavioural changes that follow closed head injuries (Richardson, 1990), and a blow to the head which is sufficient to cause even the slightest alteration in consciousness, may produce detectable structural damage (Jennett and Galbraith, 1983).

Acceleration-deceleration trauma that occurs as a consequence of a collision in sport or in a motor vehicle accident is usually accompanied by the brain twisting and rotating within the skull (rotational-acceleration) which puts strains on the delicate nerve fibres and blood vessels that can stretch to the point of shearing (Pang, 1989; Strich, 1961; Teasdale & Mendelow, 1984). Microscopic shearing occurs simultaneously within a rapidly rotating brain resulting in myriad axonal and neuronal disruptions within the white matter of both hemispheres (Pang, 1989). The rotational velocity also plays a role in producing a loss of consciousness (Adams, Graham, & Gennarelli, 1985; Ommaya & Gennarelli, 1974; Parker, 2001). The effects of these immediate disturbances in neurological functions caused by the rapid acceleration-deceleration forces, is commonly referred to as *concussion* (Lezak et al., 2004). The concussion syndrome covers a range of symptoms and severity depending on the amount of diffuse injury incurred, with the spectrum of severity ranging from mild to moderate to severe (Levin et al., 1982). However, in the sports literature the term concussion is commonly used synonymously with the term mild traumatic brain injury (MTBI) probably due to the fact that most concussions within the sports context do not involve a loss of consciousness (Lovell, Iverson, Collins, McKeag & Maroon, 1999) and have a tendency to fall within the mild rather than the moderate or severe categories. Given that the focus of this research is on sports related brain injury, for the purpose of this study the terms concussion and mild traumatic brain injury will be used interchangeably. Researchers also refer to sub-concussive head trauma or microtrauma (Green & Jordan, 1998; Erlanger, Kutner, Barth, & Barnes, 1999) in the sports literature which occur as a result of forces which are

not great enough to result in a concussive injury but which operate below the concussion threshold level. The effects of sub-concussive head injury in sport are considered to be cumulative and thus, chronic (Killam, Cautin, & Santucci, 2005; Rutherford, Stephens, Potter, & Fernie, 2005). *Accordingly, the basic assumption underlying the rationale for the present study is that participation in a contact sport places athletes at risk for repeated incidence of both unreported concussive and sub-concussive brain trauma.*

## **1.2 TRAUMATIC BRAIN INJURY CLASSIFICATION OF SEVERITY**

The severity of TBI can be measured in terms of the duration of its initial features, the incidence of complications and long-term consequences (Jennett, 1976). The range of trauma severity varies from mild bumps or bruises, causing no ill effects on the one end of the severity continuum, to prolonged coma or vegetative states on the other (Lezak et al., 2004). Severity ratings are generally more directed towards patients with moderate-to-severe injuries (Schatz & Chute, 1995) although in the milder cases these parameters are still used in order to assist with the management of the patient and hence are of relevance to the present study. The most commonly used measures of severity include a rating on the Glasgow Coma Scale, the length of posttraumatic amnesia and the duration of loss of consciousness, as delineated below.

### **1.2.1 Glasgow Coma Scale (GCS)**

The development of the Glasgow Coma Scale (GCS) (see Table 1.2.1) by Teasdale and Jennett in 1974 marked a milestone in the evaluation, prognosis and clinical research of head injury (Segatore & Way, 1992). The GCS is used as a means of assessing the depth and duration of impaired consciousness and coma and for gauging deterioration and improvement at the acute stages of brain damage as well as predicting the final outcome (Teasdale & Jennett, 1974). By observing the patient's motor responsiveness, verbal performance and eye opening, the GCS is able to define levels in the patient's comatose state more accurately than by measuring the length of consciousness (Schatz & Chute, 1995). Patients are scored according to their eye opening on a scale of 4 to 1, motor response on a scale of 6 to 1 and verbal response on a scale of 5 to 1 with the sum of the three categories totalling 15, which equals to the total GCS score (Teasdale & Jennett, 1974). Patients with a GCS of 8 or less are considered to have a severe head injury and

are classified as “in coma”; scores of 9 to 12 are classified as a moderate head injury and 13 to 15 classified as mild or no TBI.

Psychometric testing illustrates that the GCS has excellent interrater reliability ( $r = .95$ ) and test-retest reliability (Spearman’s  $\rho = .85$ ,  $p < .001$ ), with good construct validity (Pearson’s  $r = .68$ ,  $p < .001$ ), especially when applied by medical, nursing or paramedical personnel in relation to more severe levels of TBI (Rowley & Fielding, 1991). However it reveals a less desirable predictive validity of outcome (Pearson’s  $r = .56$ ) for patients with middle to mild range GCS scores (Segatore & Way, 1992). Some trauma patients on arrival have little or no loss of consciousness but deteriorate thereafter and are routinely misclassified (Young, Gleave, Schmidek, & Gregory, 1984). The presence of alcohol or drugs in a patient may produce unreliable scores with impaired consciousness attributed inappropriately to head trauma severity in some cases, to alcoholic stupor in others (Segatore & Way, 1992). Additional criticism of the GCS is that one or more of the modalities may not be measurable during the first few days of observation: eye opening may be closed due to facial swelling, verbal responses may be impeded by endotracheal tube and immobilization or paralysis precludes limb movement (Lezak et al., 2004). Its use may also be inappropriate with younger children or patients who speak a different language other than that of the observer (Sorenson & Kraus 1991). Using the GCS as a research tool in classifying patients, particularly at the lower end of severity, is also highly questionable.

**Table 1.2.1**  
**Glasgow Coma Scale**

<b>Eye Opening</b>	<b>E</b>	<b>Best Motor Response</b>	<b>M</b>	<b>Best Verbal Response</b>	<b>V</b>
Spontaneous	<b>4</b>	To verbal commands	<b>6</b>	Oriented and converses	<b>5</b>
To speech	<b>3</b>	To painful Stimulus: localizes pain	<b>5</b>	Disoriented and converses	<b>4</b>
To Pain	<b>2</b>	Flexion-withdrawal	<b>4</b>	Inappropriate words	<b>3</b>
No response	<b>1</b>	Flexion-abnormal	<b>3</b>	Incomprehensible sounds	<b>2</b>
		Extension	<b>2</b>	No response	<b>1</b>
		No response	<b>1</b>		

(After Teasdale & Jennett, 1974, as cited in Lezak et al., 2004)

**E+M+V = 3 to 15**

- **13 - 15 = Mild or No TBI**
- **9 - 12 = Moderate TBI**
- **3 - 8 = Severe TBI**

### 1.2.2 Posttraumatic amnesia (PTA)

Posttraumatic amnesia is the length of time from the moment of injury to the time of resumption of normal continuous memory (Lishman, 1987) (See Table 1.2.2). It represents the time after the TBI in which the patient is confused, disoriented and has difficulty acquiring and retrieving new information (Baddeley, Sunderland, Watts, & Wilson, 1987). Some regard it as a state of anterograde amnesia (an inability to remember the continuous flow of new information) (Levin et al., 1982).

Posttraumatic amnesia is assessed through direct questioning and can only be determined once an individual has recovered from a coma sufficiently so that he or she can speak (Lezak, 1995). It therefore not only includes a period of anterograde amnesia but also extends beyond the length of coma (Levin et al., 1982). Although the duration of PTA is highly related to that of coma, the relationship between the two is quite variable, measuring different but overlapping neurological phenomena (Levin et al., 1982). From a clinical view point, the length of time over which remembering is impaired is broadly proportional to the length of coma and is relevant to both diagnosis and prognosis (Richardson, 1990). According to Brooks, Symington, Beattie, & Campsie (1989), PTA lasts about four times the length of coma. Hence, clinicians usually regard the duration of PTA as an excellent indicator of the degree of severity in head injury. Researchers Katz and Alexander (1994) found that PTA was the strongest predictor of outcome at 12 months in patients with diffuse axonal injury, whilst amongst the more general traumatic brain injury population; the duration of PTA has been related to aspects of neurocognitive recovery six months and two to five years after injury (Levin, Papanicolaou, & Eisenberg, 1984).

The period of PTA also tends to be directly related to the duration of retrograde amnesia (an impairment of memory for events experienced before a traumatic brain injury) although it is postulated that the former is typically much longer than the latter (Russell, 1932; Russell & Nathan, 1946; Russell & Smith, 1961). It is thought that a loss of consciousness inevitably gives rise to retrograde amnesia, however, the latter may certainly occur without the former (Richardson, 1990). Posttraumatic amnesia has proved to be more valid and useful than retrograde amnesia in assessing severity since in general a great majority of retrograde amnesias are brief and are less valuable as a guide to severity (Lezak, 1995). PTA has also proved to be more valid than a measure of the duration of unconsciousness or confusion as the latter depends

more closely on secondary complications (Lezak, 1995). The duration of PTA correlates well with GCS ratings except for some finer scaling at the extremes where PTA has been found to better predict outcome (Evans, 1975; Levin et al., 1982).

Difficulties in defining and therefore determining the duration of posttraumatic amnesia, however, have made its usefulness as a measure of severity questionable (Lezak, 1995). While it is in agreement that PTA does not end when the patient first begins to register experience but when the registration is continuous, deciding when continuous memory returns is difficult especially with confused or aphasic patients. In addition, many patients with mild traumatic brain injury (MTBI) are discharged while still in PTA leaving the clinician to estimate the duration of PTA based on reports by the parents or family (Lezak, 1995). According to researchers, whilst PTA is generally accurate at predicting cognitive functioning two years post-injury (Brooks, McKinley Symington, Beattie, & Campsie, 1987) it does not discriminate well between the different levels of severity and might not be sensitive enough for classifying subjects for research (Brooks & McKinley, 1983; Brooks, McKinley, et al., 1987).

### **1.2.3 Loss of consciousness (LOC)**

The word 'conscious' or 'consciousness' is the state of being aware of oneself and of one's surroundings and must not be confused with 'attention'. The level of consciousness ranges over a continuum from full alertness, to drowsiness, to stupor and then coma (Lezak et al., 2004). In addition to the length of PTA, the diagnosis and classification of TBI is also based on the length of loss of consciousness (LOC). Disturbances in consciousness usually reflect pathological conditions in the brain (Lishman, 1987) and both the duration of PTA and LOC have been seen to correlate with the severity and outcome of TBI (Levin et al., 1988; Newcombe, Rabbitt, & Briggs, 1994).

The International Classification of Diseases (ICD) was developed by the World Health Organization for the classification and comparison of morbidity and mortality data (Richardson, 1990). The duration of unconsciousness, as a measure of the degree of severity (see Table 1.2.3), could therefore be classified together with the type of head injury. For example, concussion

coded as 850 with a brief loss of consciousness would therefore be classified as 850.2 using the ICD-9 classification system (International Classification of Diseases, 2006).

Taken together, the above severity indications differentiate MTBI from moderate or severe brain injury in terms of a GCS of 13 to 15, PTA < 60 minutes and LOC < 30 to 60 minutes. It is this category of TBI that is most prevalent in the contact sports populations and provided the basis for the development of concussion grading scales in sport. The role of grading scales in sport is to offer useful prognostic information to aid with the management of the athlete and to assist with return-to-play decisions, and will be delineated more fully in a subsequent section. Although grading scales continue to be employed in the acute assessment and management of concussion throughout the sporting world, the need for more empirically based assessment measures led to an interest in neuropsychology. Neuropsychological assessment provides objective cognitive testing which can be used to evaluate the cognitive functioning of the athlete, thereby providing a more accurate means of assisting with return-to-play decisions.

**Table 1.2.3**  
**ICD-9 Sub-classification of levels of consciousness**

Level	Duration of consciousness
0	Unspecified state of consciousness
1	With no loss of consciousness
2	With brief (less than one hour) loss of consciousness
3	With moderate ( 1 to 24 hours) loss of consciousness
4	With prolonged (more than 24 hours) loss of consciousness and return to pre-existing conscious level
5	With prolonged (more than 24 hours) loss of consciousness, without return to pre-existing conscious level
6	With loss of consciousness of unspecified duration
9	With concussion, unspecified

(International Classification of Diseases, 2006)

## **1.3 THE DEVELOPMENT OF THE ROLE OF NEUROPSYCHOLOGICAL ASSESSMENT IN THE MANAGEMENT OF MTBI IN SPORT**

### **1.3.1 Origins of neuropsychological assessment in MTBI in sport**

Prior to the 1980's, investigations into the effects of traumatic brain injury focused predominantly on moderate to severe brain injury. Mild traumatic brain injury, on the other hand,

was not viewed as problematic since its features were associated with minimal or no underlying neuropathology and the period of recovery was considered to be brief (Barth et al., 1989).

Clinical investigations into MTBI began in the late 1970's and continued into the early 1980's and 1990's. Work by researchers such as Gronwall and Wrightson (1974, 1975, 1980) found organic brain damage manifested by the slow processing of information, impaired attention or concentration and delayed return to work amongst some MTBI patients (Gronwall & Wrightson, 1974). Similarly, Rimel, Giordani, Barth, Boll, & Jane (1981) also documented the presence of long-term sequelae in the form of persistent headaches and memory problems, preventing a large number of MTBI patients from returning to work. Whilst researchers Barth et al. (1983), Dikmen, McLean & Temkin (1986), Levin, Lippold, et al. (1987), and Ruff et al. (1989) all revealed neurocognitive deficits and postconcussive symptoms in their investigations of MTBI patients.

Although methodological considerations, such as the absence of adequate controls and poor selection criteria, were just some of the many problems confronting researchers in the late 1980's (Bender, Barth & Irby, 2004), these studies paved the way for a new found interest in the neuropathological effects following MTBI (Barth et al., 1989). Mild traumatic brain injury thus became known as the 'silent epidemic' which could be associated with significant morbidity and the potential for devastating cognitive, emotional and economic consequences (Barth et al., 1989).

Two schools of thought have been evident in the MTBI literature to explain the persistence of neurocognitive deficits and postconcussive symptoms following MTBI. Some researchers have postulated that symptoms develop as a consequence of psychological vulnerability, psychiatric disturbance, or secondary gain (malingering) (Miller, 1961). Others found evidence rooted in neurogenic origins where postmortem microscopic lesions in the brains of individuals thought to have sustained a MTBI, were discovered (Oppenheimer, 1968). Research on animals by Gennarelli et al. (1982) also supported the neurogenic basis by providing objective evidence that MTBI produces microscopic damage in the form of diffuse axonal injury. These two schools of thought continue today (Barth, Diamond, & Errico, 1996; Sweet, Peck, Abramowitz, & Etzeiler, 2003) and paved the way for litigation, where in the 1990's MTBI patients entered litigation at

an alarming rate with neuropsychological assessment gaining increasing recognition and popularity for being the most sensitive method for capturing subtle neuropsychological sequelae following MTBI (Ruff, 2005).

Investigations into the effects of MTBI in sports began as far back as the 1970's where researchers such as Yarnell and Lynch (1970) found that concussion resulted in temporary disturbances in memory functioning amongst college football players. Subsequently, an historic, large multi-site research project was undertaken by Barth and his colleagues in the late 1980's, to investigate the effects of concussion amongst college football players at the University of Virginia, the Ivy League schools and the University of Pittsburgh (Barth et al., 1989). Barth et al. (1989) addressed some of the methodological limitations facing researchers at that time by using a test-retest design to evaluate athletes before and after sustaining a MTBI (concussion). They undertook pre-injury testing (baseline evaluation) on 2350 athletes, with more than 200 concussed players evaluated at intervals of 24 hours, 5 days and 10 days. Their findings revealed mild cognitive decline at five days post-injury with most athletes returning to baseline performance within 10 days. Barth et al.'s (1989) study thus provided a model for evaluating change in neuropsychological test performance from pre-injury to post-injury (Lovell, 2002).

Although Barth et al.'s (1989) study confirmed the importance of neuropsychological evaluation in the management of concussion, it did not result in the widespread use of neuropsychological testing amongst athletes at the clinical level (Barth et al., 1989). From the 1990's onwards there was an increased focus on defining, analyzing and categorizing concussion in sports for medical appropriate management (Bailes & Cantu, 2001). Numerous grading scales for the acute management of concussion were developed based on the clinical experience and expert opinions of medical sports specialists with only one rating scale, Cantu's Evidence-Based Rating Scale (2001) reported to be based on empirical research (Solomon, Johnston, & Lovell, 2006).

During the years 1997 to 1998 a large multi-site college football study was undertaken involving 395 football players from Michigan State University, the University of Florida, the University of Pittsburgh and the University of Utah (Collins, Lovell & McKeag, 1999). When researchers compared the concussed athletes relative to the controls, deficits were noted up to seven days post-injury especially in terms of memory and speed of information processing (Collins, Grindel

et al., 1999). The resolution of postconcussive symptoms however, took place between three to five days which mirrored the underreporting of symptoms in athletes wanting to return-to-play, whilst still exhibiting the effects of concussion on formal testing (Collins, Echemendia, & Lovell, 2004). This study thus had important implications for high profile players wanting to return to play whilst still symptomatic and resulted in the shift of neuropsychological testing to the clinical arena where large-scale studies implementing the use of baseline neuropsychological testing took place (Lovell, 1999; Lovell & Burke, 2000).

The sports arena thus became an ideal setting in which to study the effects of concussion, with sports such as boxing (e.g. Casson et al., 1984; Jordan, Ravdin, Jacobs, Bennett, & Gandy, 1997), ice hockey (e.g. Hatfield, Bieliauskas, Bergloff, Steinberg, & Kauszler, 2004; Gaetz, Goodman, & Weinberg, 2000; Killam et al., 2005), soccer (e.g. Matsler, Kessels, Lezak, Jordan, & Troost, 1999; Rutherford et al., 2005), American football (e.g. Barth et al., 1989; Pellman, Lovell, Viano, Casson, & Tucker, 2004), Australian Football League and Rugby League (e.g. Collie, Makdissi, Maruff, Bennell, & McCrory, 2006; Hinton-Bayre, Geffen, & Friis, 2004; Hinton-Bayre, Geffen, & McFarland, 1997) and Rugby Union (e.g. Shuttleworth-Edwards, Border, Reid & Radloff, 2004; Shuttleworth-Jordan, Puchert, & Balarin, 1993) becoming targets of increased attention and interest as they represented highly predictable environments for the occurrence of single and repetitive MTBI. Whilst most athletes who sustain concussions make a rapid and complete recovery, some athletes continue to be symptomatic with effects persisting over time. Hence, the consequence of concussion involves both immediate (acute) and long-term (chronic) effects (Grindel, Lovell, & Collins, 2001). In the acute condition, cognitive and symptom sequelae usually resolve within three months post-injury, whereas in the chronic condition the effects persist for longer than three months post-injury (Barth et al., 1989 Frencham, Fox, & Maybery, 2005; Schretlen & Shapiro, 2003). In both conditions, cognitive deficits, identified on objective neurocognitive testing, usually include impairments in attention and memory, processing speed, reaction time, planning and cognitive flexibility which occur with or without postconcussive symptoms such as headache, nausea, irritability, drowsiness, balance problems etc.

### **1.3.2 Computer-based advancements in neuropsychological assessment of MTBI in sport**

Paralleling the development and interest in MTBI in sport was the sudden advancement in neuropsychological assessment. Although Barth and his colleagues' (1989) multi-site research study demonstrated that neuropsychological tests could be adapted to assess large groups of athletes, the conventional paper-and-pencil tests used in the initial assessment of athletes, although effective in the diagnosis and management of concussion, were not very efficient and proved to be very time-consuming and costly. Another potential drawback was that athletes had to be re-tested over short test intervals and the standard tools for assessment proved to have significant practice effects when used in serial testing (Spreeen & Straus, 1998).

In spite of strong evidence to support conventional paper-and-pencil measures as being sensitive to the acute effects of concussion (Echemendia, Petukian, Mackin, Julian, & Shoss, 2001; Field, Collins, Lovell, & Maroon, 2003; Hinton-Bayre et al., 1997; McCrea et al., 2003), computerized and web-based neurocognitive test batteries were subsequently introduced in order to address some of the potential difficulties with paper-and pencil measures. Apart from being efficient and cost-effective, with easy access to the data received and stored, computerized neurocognitive tests provided more accurate measurement of processes such as reaction time and processing speed in milliseconds and allowed for the randomisation of test stimuli thereby minimizing practice effects (Lovell & Collins, 2002).

Currently, there are a number of computer-based neurocognitive tests available these include ImPACT (Immediate Postconcussion Assessment and Cognitive Testing) (Maroon et al., 2000), CogSport (Collie, Darby, & Maruff, 2001), Headminder or Concussion Resolution Index (Erlanger et al., 2001) and ANAM (Automated Neuropsychological Assessment Metric) (Reeves, Throne, Winter, & Hegge, 1989). Most of these programmes were modelled on traditional neuropsychological tests and are designed to evaluate measures of attention, reaction time, working speed, speed of information processing and memory (Podell, 2004).

The advancement of neuropsychological testing also developed alongside the progression of neuroradiological interventions. Traditional techniques such as computed tomography (CT) scanning and magnetic resonance imaging (MRI) were seen to be largely invisible to the

pathophysiology of concussion (Bigler & Orrison, 2004) and with the development of functional magnetic resonance imaging (fMRI), functional abnormalities associated with concussion could be detected. Functional magnetic resonance imaging (fMRI) also served to compliment neuropsychological testing by providing task-specific information about neural function. It was therefore able to provide data regarding the sensitivity and specificity of neuropsychological testing in detecting subtle changes in brain function post-injury (Lovell & Collins, 2002).

The growing interest and concern for the safety and health of athletes who are exposed to the effects of MTBI initiated the first ever International Symposium on Concussion in Sport hosted in Vienna in 2001 (Aubry et al., 2002). The same group met again in 2004 to host the second symposium which took place in Prague (McCrory, Johnston, et al., 2005). The Prague agreement statement was designed to build on the principles outlined in the Vienna document as well as to develop further conceptual understandings of the problem of concussion. The general consensus in both symposiums was that neuropsychological evaluation should play an integral part in the overall management and treatment of athletes who have sustained concussions (Aubry et al., 2002; McCrory et al., 2005). These recommendations occurred in parallel with evidence of major advancements in methods of neuropsychological evaluation through the development of designated computerized programmes specifically for this purpose.

### **1.3.3 Management of MTBI in sport with specific reference to the younger athlete**

In the original Vienna statement the recommendations outlined were for concussion management amongst the adult population. Although the Prague agreement statement identified the recommendations put forward at the Vienna conference as relevant and applicable to children between the ages of 5 to 18 years of age, which involves the thorough resolution of symptoms and the return of cognitive function back to baseline levels (McCrory, Johnston, et al., 2005), there was consensus that different recommendations for children need to be considered and that more research in the area was needed. The necessity to develop cognitive assessment tools that evaluate younger athletes, was also encouraged in order to better clarify the differences between the adult and child groups younger than the age of 15 years. Whilst research amongst young children is limited, research investigating the effects of concussion amongst adolescents has

increased substantially in the last three years owing to the high incidence of concussion reported for this age group (Mihalik et al., 2005; Schatz et al., 2006).

Children's ability to endure the biomechanical forces involved in producing a concussive injury differs from that in adults. This is due to a number of factors relating to the geometry of the head and skull, physiological response to stress etc., where a greater impact or force is required in children to produce the same clinical symptoms as those found in adults (McCrory, Collie, Anderson, & Davis, 2004; Ponsford, 1999). For children between the ages of 5 to 15 years rapid cognitive maturation is taking place, the brain is therefore more vulnerable to the effects of concussion owing to the interruptions in the neuronal maturation caused by the trauma (Anderson, 2001). Cognitive evaluation is also difficult over this period as the brain is cognitively maturing, therefore any baseline or post-injury testing needs to consider the normal maturation process that is occurring over this period. This is important because maturational development may cause improvements in test scores between baseline and post-injury testing and may offset injury related cognitive impairment in concussed children (McCrory et al., 2004).

Participation in sport is strongly advocated in all societies but the potential for head injury particularly amongst the younger athlete is largely overlooked. Boys and girls are encouraged at an early age to take part in team sport but the risk for concussion amongst the more popular contact sports is very high and can seriously compromise cognitive functioning. Cognitive sequelae following concussion in adults are much the same as those in children, namely reduced information processing, poor attention and impaired executive functioning (Macciocchi, Barth, Alves, Rimel, & Jane, 1996; McCrory et al., 2004). These functions are important for successful achievement at school and a concussion may impact negatively upon learning as children may have difficulty acquiring new knowledge and attending to schoolwork. It has also been suggested that adolescents between the ages of 14 to 18 years might experience prolonged cognitive recovery when compared with young adults between the ages of 18 to 25 years (Field et al., 2003; Pellman, Lovell, Viano, & Casson, 2006). This may be due to the degree of impact of the biomechanical forces involved with children who sustain concussions. Also, amongst children and teenagers rare but well recognised post-injury sequelae can involve diffuse cerebral swelling that usually results in brainstem herniation and death (Theye & Mueller, 2004). Multiple concussions have been suggested as a possible cause and risk factor to this syndrome referred to

as second-impact syndrome (Bailes & Cantu, 2001), although some authors have disputed this (McCrory, 2001).

Researchers do not know to what extent the brain may be compromised during the scholastic years as a result of exposure to multiple concussive trauma in a contact sport. The concept of cognitive reserve (Stern, 2003; 2006), alternatively designated Brain Reserve Capacity Theory-BRC (Satz, 1993) was adopted as an interpretive framework for the present study. The concept has been widely applied in brain injury research, being a powerful mode for interpreting individual variability in the mediation of cognitive processes in the brain and is useful when conceptualising the risks associated with multiple concussions. Specifically, the theory postulates that a threshold factor or what Randolph (2001) terms as a 'functional reserve', exists in each individual prior to the manifestation of symptoms resulting from dysfunction in the central nervous system. Each subsequent concussive injury, however minor, results in the further depletion of this reserve capacity thereby limiting the rate and perhaps the degree to which functional recovery can occur (Moser, Schatz, & Jordan, 2005; Randolph, 2001). Moreover, vulnerability factors such as prior head injuries, low IQ, low level of education, genetic factors, neurological conditions and learning disorders constitute vulnerability factors, either alone or in additive form, that predispose individuals to the early onset of symptoms. Thus, amongst learners whose functional reserve is already comprised in terms of such vulnerability factors either in isolation or in combined form, the additive effect of receiving further insult to the brain could push the individual over the threshold and produce deleterious permanent effects (Satz, 1993). It is important that the inter-individual variability in terms of such vulnerability factors is taken into account when designing studies on cumulative MTBI in the school contact sport arena. This involves controlling for IQ and educational levels, and isolating individuals with prior LD or a history of neurological conditions for separate analysis or exclusion from the study.

Most research investigating the effects of MTBI with respect to neurocognitive and postconcussive sequelae have mainly focussed on professional or elite athletes and amateur, collegiate or university athletes (e.g. Farace et al., 2003; Hinton-Bayre et al., 2004; Jordan et al., 1997; Kutner, Erlanger, Tsai, Jordan, & Relkin, 2000; Macciocchi et al., 1996; Matser et al., 1999; McCrory, Ariens, & Berkovic, 2000). To the author's knowledge there are no studies investigating these effects amongst junior athletes (< 12 years), with the exception of one study

investigating the effects of concussion amongst junior ice hockey players (Hatfield et al., 2004). Recently, there has been an increase in the number of studies investigating the effects of concussion amongst high school athletes (13 to 18 years), particularly within the last three years (e.g. Barr, 2003; Collins, Field, et al., 2003; Field et al., 2003; Lovell et al., 2003; Lovell, Collins, Iverson et al., 2004; Iverson, Gaetz, Lovell, & Collins, 2004a, 2004b; Iverson, Brooks, Lovell, Collins, 2006; McClincy, Lovell, Pardini, Collins, & Spore 2006; Mihalik et al., 2005; Moser et al., 2005; Pellman et al., 2006; Ramirez, 1998; Stephens, Rutherford, Potter, & Fernie, 2005; Webbe & Ochs, 2003; Witol & Webbe, 2003). To the author's knowledge, there are no studies investigating the effects of concussion amongst high school athletes in respect of the contact sports of Australian Football League and Rugby League. Few studies have investigated these effects amongst high school athletes in the contact sports of boxing, ice hockey, soccer and Rugby Union, where only one study has been reported in boxing (Ramirez, 1998) and one study in ice hockey (Ferguson, Mittenberg, Barone, & Brown, 1999), three in soccer (Stephen et al., 2005; Webbe & Ochs, 2003; Witol & Webbe) and one in Rugby Union (Shuttleworth-Edwards et al., 2004). The majority of studies, (n=15) investigating the effects of concussion amongst high school athletes have employed American football high school athletes (Barr, 2003; Collins, Iverson, et al., 2003; Field et al., 2003; Guskiewicz, Weaver, Padua, & Garrett, 2000; Iverson, Lovell, & Collins, 2002a, Iverson et al., 2004a, 2004b; Iverson et al., 2006; Lovell et al., 2003; Lovell, Collins, Iverson, et al., 2004; McClincy et al., 2006; Mihalik et al., 2005; Moser et al., 2005; Pellman et al., 2006; Wilberger, Haag, & Maroon, 1991), although eight of these studies have also included collegiate or university and professional athletes into their samples (Field et al., 2003; Guskiewicz et al., 2000; Iverson et al., 2002a; Iverson et al., 2004a; Iverson et al., 2006; McClincy et al., 2006; Mihalik et al., 2005; Pellman et al., 2006). Moreover, in the one ice hockey study reported on above (Ferguson et al., 1999), both high school and collegiate athletes were investigated together in one sample. This was also the case with two of the three soccer studies where high school athletes were incorporated together with collegiate athletes in one study (Webbe & Ochs, 2003) and high school athletes incorporated with amateur and professional athletes in another (Witol & Webbe, 2003). Although the Rugby Union study investigated the effects of concussion at three different levels of play (high school, university and professional), all these analyses were conducted separately for each level of play (Shuttleworth-Edwards et al., 2004).

Of the studies employing high school athletes, five studies investigated the chronic effects only consisting of one ice hockey study (Ferguson et al., 1999), one soccer study (Stephens et al., 2005), two American football studies (Moser et al., 2005; Iverson et al., 2006), and one Rugby Union study (Shuttleworth-Edwards et al., 2004). Eight studies investigated the acute effects only, consisting of one boxing study (Ramirez, 1998), one soccer study (Webbe & Ochs, 2003), and six American football studies (Collins, Iverson, et al., 2003; Field et al., 2003; Guskiewicz et al., 2000, Iverson et al., 2004a, Mihalik et al., 2005; Pellman et al., 2006). Seven studies investigated both the acute and chronic effects (Barr, 2003; Iverson et al., 2002a; Iverson et al., 2004a; Lovell et al., 2003; Lovell, Collins, Iverson, et al., 2004; McClincy et al., 2006; Wilberger et al., 1991) all involving American football athletes. In total there are only seven studies which have investigated the chronic effects of concussion employing high school athletes only, these include one soccer study (Stephens et al., 2005), five American football studies (Barr, 2003; Lovell et al., 2003; Lovell, Collins, Iverson, et al., 2004; Moser et al., 2005; Wilberger et al., 1991) and one Rugby Union study (Shuttleworth-Edwards et al., 2004). One American football study (Barr, 2003), investigated the neurocognitive sequelae following concussion only and employed paper-and-pencil measures. Two studies, both involving American football athletes (Lovell et al., 2003; Lovell, Collins, Iverson, et al., 2004), employed a computerized neurocognitive battery, ImPACT and three studies employed paper-and-pencil measures to investigate both the neurocognitive and postconcussion sequelae following concussion (Moser et al., 2005; Shuttleworth-Edwards et al., 2004; Wilberger et al., 1991). Only one soccer study employed a combination of both paper-and-pencil and computerized neurocognitive tests, although no postconcussion inventory was used (Stephens et al., 2005). None of the studies used a combination of both paper-and-pencil and computerized neurocognitive and postconcussion symptom inventories in their studies investigating high school athletes.

Thus in summary, few studies up to now have focussed on the long-term neuropsychological effects of concussion amongst high school athletes, comprising only seven and (as reported above) none of these studies have used a combination of both paper-and-pencil and computerized neurocognitive and postconcussion symptom inventories in their investigations of concussive and sub-concussive effects in this age group.

#### 1.4 RATIONALE AND HYPOTHESIS FOR THE PRESENT STUDY

In light of this background it was decided to conduct an extensive investigation into the long-term consequences of male high school participation in a contact sport. In South Africa Rugby Union has traditionally been the most popular contact sport played amongst the white population attending relatively advantaged English medium schools in urban areas. These schools can be distinguished from the relatively disadvantaged black township or rural schools that until recently had minimal resources for organized sports of any description. Although Rugby Union continues to be the predominant contact sport across all school population groups in South Africa today, since the end of the Apartheid era other contact sports such as soccer, boxing and the martial arts have gained in popularity. For this particular study, the main contact sport played within the school targeted for the research was Rugby Union, although a small proportion of the boys were involved in other contact sports such as soccer and the martial arts.

Further, the current study followed modern developments in concussion assessment, by utilizing an internationally recognised and widely used computerized test for this purpose, viz. the ImPACT programme. This test appears to be the most comprehensive and extensively researched computerized neuropsychological tool used in the assessment of MTBI in sport (Podell, 2004). For comparative purposes in terms of test sensitivity to concussion sequelae, the ImPACT test was used in conjunction with a prototypically sensitive paper-and-pencil measure to the non-specific effects of diffuse brain injury that occur in association with MTBI, the Digit Symbol Substitution Test. The Digit Symbol Substitution Test taps visuo-perceptual processing at speed, and has been frequently used to identify neurocognitive deficits in association with concussion across most sports studies, either in isolation (e.g. Maddocks, Saling, & Dicker, 1995) or as part of a wider test battery (e.g. Abreau, Templer, Schuyler, & Hutchinson, 1990; Matser, Kessels, Jordan, Lezak, & Troost, 1998; Matser et al., 1999; Shuttleworth-Edwards et al., 2004). It has the added advantage of providing access to an adjunctive Digit Symbol Incidental Recall task which was incorporated for the purposes of this study in both immediate and delayed form. The Digit Symbol Paired Associate Incidental Recall task is also considered to be sensitive to mild presentations of diffuse brain injury (Shuttleworth-Edwards, 2002), thereby further enhancing the discriminating ability of the standard administration of the Digit Symbol Substitution Test.

In addition to the above cognitive measures, two postconcussion symptom measures were employed in the research for the purpose of evaluating the postconcussive symptoms experienced by the high school athletes. One formed part of the ImPACT programme and the other, a paper-and pencil questionnaire, based on a questionnaire used in prior research in South Africa on Rugby Union athletes (Shuttleworth-Edwards et al., 2004) contained four additional symptoms over the ImPACT scale. Two of the additional symptoms (aggression and speech problems) were shown to be sensitive to the effects of diffuse closed MTBI (Shuttleworth-Edwards et al., 2004). Specifically the aims of the research were as follows:

1. The first objective of this study was to establish whether there are indications of permanent (residual) neuropsychological sequelae relative to non-contact sport controls in respect of (i) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and pencil tests), and (ii) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories, in sports playing final high school male students, due to postulated exposure to repetitive concussion (MTBI), including both concussive and sub-concussive events, during high school participation in a contact as opposed to a non-contact sport.
2. The second objective was to establish whether there are indications of a deterioration in neuropsychological functioning relative to non-contact sport controls in respect of (i) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and pencil tests), and (ii) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories, in sports playing final high school male students, due to the effects of unreported concussive and sub-concussive events, sustained during participation in a contact sport over a six month winter sports season.
3. The third objective of this study was to establish whether there are indications of permanent (residual) neuropsychological sequelae for sports playing high school boys with two or more concussions versus no concussions in respect of (i) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and

pencil tests), and (ii) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories.

4. A fourth objective in respect of isolating residual concussive and subconcussive effects of participation in a contact sport, was to examine (i) the relative sensitivity of the cognitive composite scores of the ImPACT computer-based test especially designed for the assessment of sports concussion injuries compared with a prototypically sensitive paper and pencil measure of visuo-perceptual processing at speed and its adjunctive memory component (Digit Symbol Substitution test and Digit Symbol Incidental Recall-Immediate and Delayed), and (ii) the discriminating ability of four additional symptoms not present on the ImPACT Symptom Scale.

## **1.5 STRUCTURE OF THE THESIS**

The thesis is divided into two volumes. Volume One consists of the literature review which is divided into six chapters, including the introductory chapter. Volume Two consists of the research study which includes three chapters, chapters seven to nine. This is followed by the references and appendixes. For ease of reference all sections and tables are prefixed with the number of the chapter to which they belong. Thus, for example, Table 2.1 is the first table to appear in chapter two. Tables appear at the end of the first appropriate paragraph or page break following their initial mention in the text.

Volume One:

Chapter one consists of the introduction to the present study.

Chapter two discusses the field of mild traumatic brain injury in general. In this section terms and concepts which relate to the severity indicators, Glasgow Coma Scale, posttraumatic amnesia and loss of consciousness, are used to define the category of MTBI in general. The delineation of the severity indicators within this category is taken a step further with the development of the different grading scales used in the treatment and management of MTBI within the sports arena and which are addressed in this section.

Chapter three examines the epidemiology, neurophysiology, and neuroimaging techniques in respect of mild traumatic brain injury (MTBI) in general. This is followed by a discussion on outcome following MTBI, which includes identified risk factors which may serve to influence the presentation of this outcome. The Brain Reserve Capacity Theory concludes this chapter and provides an explanation for the variability in outcome following MTBI. This theory is useful when conceptualising the risks associated with multiple concussions and provides the theoretical backdrop for the present study.

Chapter four focuses on the evaluation of objective neurocognitive test sequelae following MTBI. Sensitive paper-and-pencil measures used in both MTBI research in general as well as in the sports arena are reviewed first followed by a review of the cognitive evaluation of MTBI in sport. The different levels of neuropsychological evaluation in sports will be discussed first followed by a discussion on the various computerized neurocognitive test batteries made available for the investigation of MTBI in sport.

Chapter five reports specifically on the evaluation of self-reported postconcussive symptoms (PCS) following MTBI as identified on postconcussion symptom inventories and scales. This chapter begins by reviewing the etiology of PCS which includes the Diagnostic and Statistical Manual for Mental Disorders - 4<sup>th</sup> Edition's (DSM-IV) diagnosis of postconcussional disorder, followed by an investigation into postconcussion in sport.

Chapter six focuses specifically on mild traumatic brain injury (concussion) in contact sport. It begins by reporting on the incidence of concussion in contact sports in general and then focuses specifically on the sports of boxing, the martial arts, ice hockey, soccer, American football, Australian Rules Football, Rugby League, and Rugby Union. This section is followed by a review of existing research regarding the presence and potential for both acute and chronic consequences of concussion in contact sport, in respect of both neurocognitive and postconcussive symptom sequelae. Methodological concerns relating to studies investigating MTBI in sport are next discussed. Finally, the rationale and hypothesis for the present study are delineated, which concludes the chapter.

Volume Two:

Chapter seven describes the research design and methodology; chapter eight presents the results, and chapter nine concludes the dissertation with a discussion. This is followed by the references and appendixes.

## CHAPTER TWO

### DEFINITIONS AND GRADING SYSTEMS IN MILD TRAUMATIC BRAIN INJURY

The central concern of this chapter is to define terms and concepts, and the various grading systems utilized in respect of mild traumatic brain injury (MTBI), first generally and then more specifically in the sports arena.

#### 2.1 DEFINITION OF TERMS AND CONCEPTS

Within the MTBI literature there has been much confusion and debate over the use of the different terms and concepts used to define MTBI. As with the term traumatic brain injury, MTBI seems to be the preferred term of choice in leading contemporary textbooks and periodicals (e.g. Anderson et al., 2001; Lezak et al., 2004; Lovell, Collins, & Bradley, 2004; Vanderploeg, Curtiss, & Belanger, 2005), although in many cases the term is still used interchangeably with the terms 'mild head injury', 'minor head injury' and 'concussion' (e.g. Barth Varney, Ruchinskias, & Francis, 1999; Van der Naalt, 2001). Whilst many researchers use the terms 'minor head injury' and 'mild head injury' interchangeably in the literature, others suggest differences, where minor head injury includes a number of patients with a GCS score of 15, transient loss of consciousness and PTA, whilst mild head injury normally encompasses more patients with a GCS score of between 13-14, clouded consciousness and variable PTA (Van der Naalt, 2001). Alternatively, some researchers group both mild and moderate TBI patients into one group, which they describe as a minor head injury group (Kibby and Long, 1996).

Although generally the term 'MTBI' has been used interchangeably with the term 'concussion' especially in the sports literature, some researchers attribute different meanings to these terms thereby limiting the generalizability of their use in MTBI research (Bender et al., 2004). For example, some researchers prefer to use the term 'MTBI' rather than 'concussion' because they regard the latter term as describing the mechanism of injury rather than the injury itself (Nelson, Jane and Gieck, 1984). Moreover, other researchers refer to a 'concussion' as a characteristic injury of MTBI or a particular type of MTBI unique to contact sport caused by rapid acceleration

or deceleration of the brain inside the skull (Lezak et al., 2004). For the purpose of this study, however, 'MTBI' will be the term of choice and will be used interchangeably with the term 'concussion', even when literature is cited that has used the terms mild or minor head injury.

## **2.2 MTBI CLASSIFICATION OF SEVERITY**

In addition to the inconsistencies in the use of terms in MTBI literature, there is also much discrepancy over the severity indicators used in the management and diagnosis of MTBI. Hence, although the severity indicators discussed in the introduction differentiate MTBI from moderate or severe TBI in terms of a Glasgow Coma Scale (GCS) score of 13 to 15, posttraumatic amnesia (PTA) < 60 minutes and loss of consciousness (LOC) < 60 minutes, this classification and measurement of severity is somewhat inconsistent amongst researchers investigating this population. Whilst some researchers have defined MTBI as a GCS score ranging between 13 to 15, a loss of consciousness of less than or equal to 20 minutes (or no loss of consciousness), less than 48 hours of hospitalisation (no physical complications) and normal scanning (Levin, Eisenberg, & Benton, 1989; Rimel et al., 1981), the use of PTA as a measure of severity has been excluded from their diagnosis. This is also supported by other definitions of MTBI (e.g. Gentilini et al., 1985; Hsiang, Yeung, Yu, & Poon, 1997). Russell and Smith (1961) proposed that a MTBI is defined as a concussion with transient disturbance of consciousness and duration of PTA less than one hour; however neither a GCS score, nor a measure of LOC was included in this definition. Minderhoud, Boelens, Huizenga & Saan (1980) also did not include LOC nor PTA as a criterion for MTBI in their definition. The reason for the exclusion of these criteria by some researchers in their definitions could be because they are viewed as potential sources of measurement error. They are therefore seen to be unreliable in that in many cases the measures LOC and PTA rely on untrained observers at the scene or on self-reporting (Dikmen & Levin, 1993), and moreover many MTBI patients are diagnosed hours, days and even months after the injury so a GCS score would not be useful (Ruff, 2005).

The lack of generalizability and consensus around the criteria used to define MTBI has been further compounded by the fact that some researchers include additional criteria in their definitions, such as the presence of lesions on imaging (Levin, Mattis, et al., 1987), duration of hospital stay (Rimel et al., 1981) and focal neurological signs (Gentilini et al, 1985). In an

attempt to modify the current definition of MTBI, some researchers divided all TBI patients with a GCS score ranging from 13 to 15 into MTBI and high-risk MTBI groups (Hsiang et al., 1997). The MTBI group was defined as having a GCS score of 15, with no acute radiological abnormalities whereas the high-risk MTBI group was defined as a GCS score of 13 or 14, or a GCS score of 15 with radiological abnormalities. This more precise definition was meant to help avoid confusion caused by current classification systems and to assist with the treatment process but it rather served to promote further confusion. By dividing individuals with a GCS score of 15 and normal imaging (MTBI group) from a so-called 'high risk' MTBI group, does not necessarily mean that the MTBI group is without injury or risk. Individuals with MTBI rarely show abnormalities on scanning (Bigler & Orrison, 1996), moreover some neuropsychological deficits associated with MTBI have been documented two years post-injury (Alexander, 1992).

Other researchers attempted to classify MTBI patients by also providing sub-classifications based on neuroimaging. In a study by Levin, Lippold, et al. (1987), MTBI patients with positive CT or MRI findings were classified as complicated MTBI and patients with negative CT or MRI findings were referred to as uncomplicated MTBI. Similarly, Williams, Levin & Eisenberg (1990) studied 155 MTBI patients with a GCS score ranging from 13 to 15 and divided their group based on their CT findings. Seventy-seven patients were classified as complicated based on the presence of intracranial abnormalities and 78 were classified as uncomplicated having normal scans. Williams and his colleagues (1990) found that those complicated MTBI patients exhibited neuropsychological impairment at discharge relative to patients with identical GCS scores who had normal CT scans. Borgaro, Prigatano, Kwasnica and Rexer (2003) recently expanded on this by defining complicated MTBI patients as those with positive neuroimaging, with space occupying lesions, LOC and the presence of cognitive symptoms over emotional ones. Uncomplicated patients included those MTBI patients without space-occupying lesions, brief or no LOC and primarily emotive symptoms (Borgaro et al., 2003).

In light of the above it becomes clear that many different classification approaches exist to define MTBI in the acute phase. While some studies only use the GCS (Minderhoud et al., 1980), others use the GCS and LOC, without PTA as a criterion (Rimel et al., 1981), and others still use PTA, and not LOC or the GCS (Russel & Smith, 1961). Moreover, studies range from a PTA of one hour or less (Russel & Smith, 1961), to 24 hours (Kibby & Long, 1998), to less than 48 hours

(Miller, 1986) and LOC of less than 20 minutes (Levin et al., 1989; Miller, 1986) to 30 minutes (American Congress of Rehabilitation Medicine, 1993). In addition researchers in their definitions include GCS scores ranging from 13 to 15 (Hsiang et al., 1997; Levin et al., 1989) or from 13 to 14 (Van der Naalt, 2001). Other researchers also rely on the use of neuroimaging, the presence of space occupying lesions and cognitive symptoms to classify the level of severity amongst MTBI patients (Borgaro et al., 2003; Levin, Mattis et al., 1987; Williams et al., 1990).

Generally the lack of empirical evidence to support the delineation of the different severity levels within this MTBI category would account for the discrepancies amongst researchers. Moreover, the transient and variable nature of the sequelae following MTBI could also contribute to the differences in classification (Johnston, Ptito, Chankowsky, & Chen, 2001; Satz et al., 1997). In an attempt to provide a unified definition of MTBI, the mild traumatic brain injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (ACRM) proposed the following definition of MTBI in 1993, principally based on the duration of LOC and PTA which could be applied retrospectively (Ruff & Jurica, 1999). The Committee defined MTBI to be traumatically induced physiological disruption of brain function manifested by one or more of the following symptoms:

1. Any period of LOC;
2. Retrograde or PTA;
3. Any alteration of mental state (e.g. feeling dazed, disorientated or confused); and
4. focal neurological deficit(s) that may or may not be transient (e.g. double vision, loss of balance, taste or smell).

Where the injury severity does not exceed the following:

1. LOC less than or equal to 30 minutes
  2. An initial GCS score of 13-15
  3. PTA equal to or not greater than 24 hours
- (Kibby & Long, 1996)

It is of note that the above ACRM definition does not require LOC as a necessary component for a diagnosis for MTBI, and that in terms of the committee's parameters, the presence of PTA or neurological symptoms are sufficient to support a diagnosis (Ruff, 2005).

This definition is similar to the one proposed recently by the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury (Carroll, Cassidy, Peloso, et al., 2004) who, in response to the weak literature on MTBI diagnosis, attempted to try and operationalize a definition of MTBI. Mild traumatic brain injury was thus defined as an acute injury resulting from mechanical energy to the head from external physical forces. Operational criteria for clinical identification include a GCS score of 13 to 15, thirty minutes post-injury (or later upon presentation for healthcare), with one or more of the following: confusion or disorientation, LOC for 30 minutes or less, PTA for less than 24 hours or other transient neurological abnormalities such as focal signs, seizures and intracranial lesions not requiring surgery. These manifestations must not be due to drugs, alcohol, or medications taken as a result of other injuries (Carroll, Cassidy, Peloso, et al., 2004).

Although these definitions are a step forward towards operationalizing a definition of MTBI by covering a broader range of injury severity and encouraging the investigation of patients without hospital admission, they are not without criticism. Earlier studies have defined MTBI as having PTA less than one hour and moderate TBI as having PTA ranging from one to 24 hours (Bigler, 1990). This overlap in definition of the mild and moderate TBI populations in both the ACRM and WHO definitions makes it difficult to draw comparisons with future research. Moreover, the presence of neurological abnormalities is suggestive of a more severe TBI (Richardson, 1990).

In addition to the mild and moderate TBI inclusion, the ACRM and WHO's definitions cover a vast range of severity, for example some would argue that an individual who sustained a MTBI and who experienced LOC or PTA for a few seconds would have milder ramifications than one whose LOC is of 30 minutes duration with a PTA of 24 hours (Kibby & Long, 1996, Rutherford, Merrett & McDonald, 1979). Satz et al. (1997) argue that the usage of arbitrary and a priori cut-off points which are not empirically validated in order to designate grades of severity is another weakness.

One month after the ACRM definition was published, the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV) (1994) included criteria for postconcussive disorders (see Chapter Four) that occur as a consequence of closed TBI (Ruff, 2005). They included no specific criteria for diagnosing a concussion other than proposing a period of LOC of more than five minutes, a PTA of more than 12 hours, and the development of seizures or worsening of pre-existing

seizures (Ruff, 2005). Although both the ACRM and DSM-IV definitions have the advantage of being administered retrospectively to all MTBI patients, their criteria for both LOC and PTA are also very inconsistent. The ACRM diagnosis includes a period of LOC to be less than or equal to 30 minutes, whereas the DSM-IV requires a LOC of greater than five minutes. For PTA, the ACRM require any loss of memory before or after the injury but not greater than 24 hours, whereas the DSM-IV includes a period of more than 12 hours (Ruff & Jurica, 1999). Ruff and Jurica's (1999) study illustrates this discrepancy between the two definitions, where 76 patients were diagnosed as having sustained a concussion according to ACRM criteria yet only 34% of these patients were classified as having sustained a concussion according to the DSM-IV criteria. This discrepancy further supports the need for injury severity classification to be dependent on empirically based outcomes rather than on arbitrary definitions.

### **2.3 DIAGNOSTIC CLASSIFICATION OF MTBI VIA GRADING SCALES**

In order to address the issue around the heterogeneity of the MTBI population and to further attempt a more uniform definition of MTBI, researchers divided the diagnostic criteria of MTBI into grades or severity levels, i.e. Type I, Type II and Type III, being from least to greatest degree of severity, which are aligned with definitions like that of the ACRM and the DSM-IV (Ruff & Jurica, 1999) (see Table 2.3.1). These different levels resemble the current grading systems used to assist with the diagnosis and overall management of MTBI in sport. In the gradation categories Type I corresponds with the ACRM definition, with Type III associated with the DSM-IV definition and Type II connecting the two (Ruff, 2005). Ruff (2005) strongly recommends that the range of severity within the MTBI population be captured by sub-classifications which in turn will assist with the refined treatment of MTBI and should therefore be included in future DSM editions. Further research is indicated in order to validate these gradations clinically.

Whilst the different severity levels used to classify MTBI are not empirically based but are rather seen to be estimations based on clinical experience, it also appears that the above severity measures themselves have little meaning when applied to the milder categories. For example, most injured patients cannot be diagnosed using GCS criteria since it cannot be administered retrospectively and has to be administered as soon as possible, as most of the symptoms are

captured in the first hours following the brain trauma (Ruff & Jurica, 1999). A GCS score of 10 could therefore improve to 13 from the time it takes to transport the patient to the emergency department (Ruff & Jurica, 1999). Furthermore a GCS score of 15, three hours post-injury does not mean that no brain injury has taken place as it does not take into account the presence or absence of focal neurological injury (Rees, 2003). The effects of alcohol and drugs are other confounding variables amongst MTBI patients, where a GCS score may have otherwise been 15 (Dikmen & Levin, 1993).

**Table 2.3.1**  
**Gradation of severity for mild traumatic brain injury**

Criteria	Type I	Type II	Type III
Loss of consciousness	Altered state or transient LOC	Definite LOC with time unknown or less than 5 min	LOC 5 to 30 min
Posttraumatic amnesia	1 to 60 seconds	60 seconds to 12 hours	> 12 hours to 24 hours
Neurological symptoms	One or more	One or more	One or more

(Ruff & Jurica, 1999)

Although clinical outcome of MTBI patients correlates better with PTA than with GCS, as noted previously, subjective estimates of length of PTA have met with criticisms and therefore make the estimate of severity questionable and unreliable (Dikmen & Levin, 1993). Traditionally LOC has been a pre-requisite for a diagnosis of MTBI but this too has been challenged by researchers where it is seen to be a poor predictor of outcome (McMillan, 1997). Some authors believe that neither LOC nor PTA is necessary for a diagnosis of MTBI (Cicerone, 1996a; Stablum, Mogentale & Umilta, 1996). Moreover, the use of these measures appears somewhat obsolete when applied to the assessment of MTBI in the chronic condition.

Despite these misgivings, over the past three decades many grading scales for the acute management of MTBI (concussion) in sport have been published (Johnston, McCrory, Mohtadi, & Meeuwisse, 2001). Guidelines have been developed in order to assist both practitioner and patient with the necessary management procedures to predict length of disability and to facilitate return-to-play (Solomon et al., 2006). The lack of conclusive scientific data is the main reason for the differences between the various guidelines that are based on current literature and clinical

experience (Cantu, 1997b; McCrory, 1999a). It is estimated that at least 25 different grading systems (Johnston, McCrory, et al., 2001) and 14 different return-to-play grading scales (Collins, Lovell et al., 1999) have been used for concussion management over the last few decades.

Peloso, Carroll, et al. (2004) appraised 41 existing MTBI guidelines of which 18 were related to sport and developed a set of methodological criteria that allowed them to assess the scientific quality of those guidelines. After appraising the guidelines against a validated index only three of the 41 guidelines could be categorized as evidence based. Two focused on paediatric patients and one on adult patients. Although, in general, criteria relating to guideline development and format were frequently met, they concluded that owing to the limited methodological quality in the current guidelines, conflicting recommendations were evident (Peloso, Carroll, et al., 2004). As with the criteria used to define the severity levels within the MTBI category in general, amongst the existing sports guidelines the researchers reported great variability with none of those assessed being evidence based. They found that the current sports guidelines do not suggest a standard method of grading concussion severity during competition and are variable in their markers of severity. Whilst some guidelines focus on the importance of LOC (Hugenholtz & Richard, 1982), others recommended PTA as a measure of severity (Bruno, Gennarelli, & Torg, 1987). Many guidelines also suggest that the athlete's symptoms are important (Aubry et al, 2002; Johnston, Lassonde & Ptito, 2001; Kelly & Rosenberg, 1997) and most recommend the athlete to be asymptomatic before returning to play (Aubry et al., 2002; McCrory, Johnston, et al., 2005).

In 1963 the Congress of Neurological Surgeons established a committee to study TBI with an emphasis on sports related concussion (Maroon, Field, Lovell, Collins, & Post, 2002). Based on their definition of concussion, they divided concussion into three separate levels (mild, moderate and severe) with management recommendations attached to each level (see Table 2.3.2). Similarly, Maroon, Steele, and Berlin (1980) initially divided concussion into three grades based on the duration of unconsciousness: (i) mild (no loss of consciousness); (ii) moderate (loss of consciousness with retrograde amnesia) and (iii) severe (unconsciousness for greater than five minutes). It was thus assumed that injuries resulting in a LOC required a greater degree of force to the head than those blows that resulted in confusion or PTA.

**Table 2.3.2**  
**Congress of Neurological Surgeons Guidelines for cerebral concussion**

Level	Definition	Management recommendations
<b>I</b>	Mild, no loss of consciousness, transient neurological disturbance	Athlete removed from contest but may return to play after completely normal neurologically
<b>II</b>	Moderate, loss of consciousness with complete recovery occurring in < 5 minutes	Athlete removed from contest and not permitted to resume contact during that game: may return in 1 week contingent upon neurological and neuropsychological findings
<b>III</b>	Severe, unconsciousness lasting > 5 minutes	Managed as a severe head injury, with possible hospitalization and appropriate diagnostic testing

(Maroon et al., 2002)

The focus on LOC as a severity measure developed out of research by Denny-Brown and Russell (1941) and later by Ommaya and Gennarelli (1974) using animal models. In their animal study, Ommaya and Gennarelli (1974) showed that of their six grades of concussion, three did not involve LOC. They postulated that cortical and subcortical structures could be affected to produce amnesia and confusion but unless the shearing forces reach the reticular activating system no LOC would occur (Ommaya & Gennarelli, 1974). As pointed out by Lovell et al. (1999), these initial studies were able to show the importance of LOC in predicting outcome for more severe injuries but not for milder injuries, moreover the studies were conducted exclusively on animal models.

Most sports guidelines, excluding Cantu's (2001) revised grading system, use LOC as an indicator of severity more than confusion or amnesia (Collins, Lovell, et al., 1999). Recent evidence however, has questioned the validity of emphasizing LOC as a significant marker in concussion severity (Maroon et al., 2002). For example in a study by Lovell et al. (1999) there was no difference between those patients who experienced LOC and those who did not on neuropsychological testing, which supports other research investigating MTBI in patient populations where LOC was also seen to be a poor predictor of outcome (Dikmen & Levin, 1993; McMillan, 1997). McCrea and colleagues (2002) contradicted those findings however, by reporting significantly lower scores on their neurocognitive, brief screening test in athletes with LOC compared to those without, although all groups showed no significant difference on testing compared to baseline 48 hours post-injury. Whilst neither study looked at the length of period of

LOC, researchers report prolonged LOC to be a relatively rare phenomenon amongst sports concussion (Collins, Iverson, et al., 2003). In the Prague agreement statement, mentioned previously, which was developed out of the second International Symposium on Concussion in Sport, hosted in Prague in 2004 (McCrory, Johnston, et al., 2005), the Concussion in Sport Group agreed that LOC, as a symptom, no matter the length of time, does not necessarily imply severity nor does its presence classify the concussion as complex (involving persistent symptoms, specific sequelae or prolonged cognitive impairment).

The three most widely recognised concussion management guidelines are those devised by Robert Cantu (1986), the Colorado Medical Society (Kelly et al., 1991) and the American Academy of Neurology (1997) (see Table 2.3.3). Cantu (1986) modified the above definitions by incorporating PTA into his grading scale. In 1991 Kelly and his colleagues through the sports medicine committee of the Colorado Medical Society, developed the Colorado guidelines. The difference between their guidelines and Cantu's guidelines was that LOC was not required as a criterion for their Grade II concussion but rather confusion and amnesia for a period of 15 minutes (Maroon et al., 2002). The Quality Standards Subcommittee of the American Academy of Neurology (AAN) took an official position concerning sports concussion in 1997 and developed the next set of guidelines which emphasized the importance of the alteration in mental status without LOC and symptoms of confusion and amnesia as the hallmarks of concussion.

Recently Hinton-Bayre & Geffen (2002) used the three above-mentioned management guidelines depicted in Table 2.3.3, to grade concussion severity amongst a group of 21 concussed athletes from two national rugby clubs. It was hypothesized that the more severe grades of concussion would be associated with greater prevalence and longer duration of impairment (Hinton-Bayre & Geffen, 2002). Although the number of athletes showing deterioration of performances two days post-injury was similar across all grading systems at 10 days post-injury, more athletes with AAN and Cantu Grade I concussions were impaired compared to those with Grade II concussions. Similarly, under the Colorado guidelines a comparable number of athletes with Grade I and II concussions were impaired at 10 days. Interestingly, no athletes with Grade III concussions (LOC) under both AAN and Colorado systems recorded impairment at 10 days post-

**Table 2.3.3**  
**Concussion Grading Scales**

<b>CANTU (1986)</b>	<b>Grade 1 (mild)</b>	<b>Grade 2 (moderate)</b>	<b>Grade 3 (severe)</b>
<b>Signs and Symptoms</b>	No LOC; PTA < 30 min	LOC < 5min or PTA > 30min but < 24hrs	LOC > 5min or PTA > 24 hrs
<b>1<sup>st</sup> Concussion</b>	May return to play (RTP) is asymptomatic for 1 week, terminate season if CT/MRI scans reveal abnormalities	RTP is asymptomatic for 2 weeks terminate season if CT/MRI scans reveal abnormalities	Withdraw athlete for minimum of 1 month; may then return to play if asymptomatic for 1 week
<b>2<sup>nd</sup> Concussion</b>	RTP in 2 weeks if asymptomatic at the time for 1 week	Withdraw athlete for one month; may RTP if asymptomatic for 1 week consider withdrawing athlete from play for the rest of the season	Withdraw athlete from play for the rest of the season; may return to play next season if asymptomatic
<b>3<sup>rd</sup> Concussion</b>	Withdraw athlete from play for the rest of the season; may RTP next season if asymptomatic	Withdraw athlete from play for the rest of the season; may return to play next season if asymptomatic	Consider no further contact sports
<b>COLORADO MEDICAL SOCIETY (1991)</b>	<b>Grade 1 (mild)</b>	<b>Grade 2 (moderate)</b>	<b>Grade 3 (severe)</b>
<b>Signs and Symptoms</b>	Confusion without amnesia, no LOC	Confusion with amnesia; no LOC	LOC
<b>1<sup>st</sup> Concussion</b>	May RTP if without symptoms for at least 20min	Terminate contest; may RTP if without symptoms for at least 1 week	Terminate Athlete's contest or practice and transport to hospital; may RTP in 1 month, after 2 consecutive weeks without symptoms
<b>2<sup>nd</sup> Concussion</b>	Terminate contest or practice; may return to play if without symptoms for at least 1 week	Consider withdrawing athlete from play for the rest of the season; may return to play in 1 month if without symptoms	Withdraw athlete from play for the rest of the season; may RTP next season if without symptoms
<b>3<sup>rd</sup> Concussion</b>	Withdraw athlete from play for the rest of the season, or may return to play in 3 months if without symptoms	Withdraw athlete from play for the rest of the season, may return to play next season if without symptoms	Withdraw athlete from play for the rest of the season; strongly discourage return to contact or collision sport
<b>AMERICAN ACADEMY OF NEUROLOGY (1997)</b>	<b>Grade 1 (mild)</b>	<b>Grade 2 (moderate)</b>	<b>Grade 3 (severe)</b>
<b>Signs and Symptoms</b>	Transient confusion; no LOC; symptoms or abnormalities resolve < 15 min	Transient confusion; no LOC; symptoms or abnormalities last > 15 min	Any LOC either brief or prolonged
<b>1<sup>st</sup> Concussion</b>	Remove from contest may RTP if abnormalities or symptoms clear within 15 min	Terminate athlete's contest; may RTP after 1 full asymptomatic week at rest and with exertion	Terminate contest; transport to hospital if unconscious or if there is evidence of neurological abnormalities. If concussion is brief may RTP in 1 week if no symptoms at rest and with exertion; if concussion prolonged; RTP in 2 weeks if no symptoms
<b>2<sup>nd</sup> Concussion</b>	Terminate athlete's contest; may RTP after 1 week without symptoms at rest and with exercise	Terminate athlete's contest; may RTP after at least 2 asymptomatic week at rest and with exertion; withdraw athlete from play for the rest of the season if CT/MRI scan abnormalities	Terminate athlete's contest; may RTP after min of 1 month asymptomatic; withdraw athlete from play for the rest of the season if any CT/MRI scan abnormalities
<b>3<sup>rd</sup> Concussion</b>			

(Bailes and Cantu, 2001)

injury (Hinton-Bayre & Geffen, 2002). The results of this study suggest that no relationship between concussion severity grade and cognitive impairment was observed between the systems. Posttraumatic amnesia was the only reliable predictor of impairment at two days but neither its presence nor duration was related to duration of impairment. Athletes with LOC also did not appear more likely to show impairment (Hinton-Bayre & Geffen, 2002) which is interesting since both the AAN and Colorado systems put great emphasis on LOC as an indicator of a severe concussion. Athletes who sustained Grade I concussions were still cognitively impaired at 10 days which is concerning because the present guidelines would permit these impaired athletes to return to play. The authors propose reasons for this could be that those athletes had a history of multiple concussions and would take longer to recover from a seemingly minor concussion, as well as those with Grade I concussions may have been returned to play prematurely thereby exposing themselves to further sub-concussive trauma (Hinton-Bayre & Geffen, 2002).

Similarly, Lovell and colleagues (2004) studied 43 high school athletes who sustained Grade I concussions and whose symptoms resolved within 15 minutes. Within 36 hours post-injury, however the concussed athletes had an increase in postconcussive symptoms and demonstrated a decline in memory functioning as evident on neurocognitive testing. Again in this study, according to current concussion guidelines these concussed athletes would have been allowed to return to play (Lovell, Collins, et al., 2004). Hence, while the grading systems may assist in the emergent management of concussion, they are not useful in predicting return to play nor in predicting outcome (Hinton-Bayre & Geffen, 2002).

Cantu (2001) recently revised his guidelines (see Table 2.3.4), which were a significant departure from those of the AAN, to rely more on symptom duration and PTA than LOC (Echemendia & Cantu, 2004). For a Grade II concussion, there is a LOC for less than a minute or PTA or post-concussive symptoms and signs for greater than 30 minutes (Cantu, 2001). Cantu (2001) also associates a PTA lasting 24 hours or more, or postconcussive signs and symptoms greater than seven days as indicative of a severe or a Grade III concussion. In contrast the AAN regards any concussion with LOC a Grade III or severe concussion, and for a Grade II concussion transient confusion, no LOC and symptoms lasting less than 15 minutes (Maroon et al., 2002).

Further support for these guidelines is illustrated in a study investigating the relationship between on-field markers of concussion severity and post-injury neurocognitive and symptom presentation amongst high school and college athletes (Collins, Iverson, et al., 2003). Researchers found that PTA and not LOC appeared to be predictive of symptom and neurocognitive deficits following concussion. Although recent research has suggested that neither these factors individually or together are good predictors of injury severity (Guskiewicz, et al., 2004). For example, in a study evaluating 1003 concussions sustained by high school and college football players, only 9% of their players presented with LOC and only 27% suffered from PTA, whereas signs and symptoms such as headache, dizziness, confusion and others, were more common (Guskiewicz et al., 2000).

**Table 2.3.4**  
**Evidence based revised concussion grading guidelines**

Grade	Definition	PTA/Post concussion signs and symptoms
1	Mild, no LOC	<30 minutes
2	Moderate, LOC < 1 minute	>30 minutes
3	Severe, LOC	PTA = 24 hours/ PCS > 7 days

(Cantu, 2001)

From the above review it can be seen that in essence, there is currently no consensus amongst medical professionals concerning concussion grading scales, return to play criteria and standardized methods of concussion management (Collins, Lovell, et al., 1999). McCrea et al. (2003) therefore recommend that less emphasis be placed on grading concussion and more emphasis should be placed on a standardized approach to measuring recovery. Moreover, the grading scales are almost entirely dependant on the symptom reporting of the athlete, who is often motivated to underreport their symptoms, or on second hand information though direct observation by significant others, for example, coaches, parents and first aid personnel (Echemendia & Cantu, 2003). The current grading systems also do not take into account the pre-existing cognitive abilities of athletes (Maroon et al., 2002), nor sex or any other individual differences (Broshek, Kaushik, Erlanger, Webbe, & Barth, 2005). Since females comprise a large percentage of the athlete population and research indicates a poorer outcome for females after concussion, individual differences such as sex and age need to be taken into account (Broshek et al., 2005).

At both the Prague and Vienna International Conferences on Concussion in Sport, the Concussion in Sport Group recommended that injury-grading scales be abandoned in favour of combined measures of recovery (e.g. medical evaluation, neuropsychological assessment, objective balance assessment, neuroimaging etc.) to determine injury severity and therefore individually guide the return to play decision-making process (Aubry et al., 2002; McCrory, Johnston, et al., 2005). The Prague group, whilst supporting this approach, emphasized the need to categorize concussion for management purposes as either simple or complex. With a simple concussion an athlete suffers an injury that progressively resolves without complication over seven to ten days. Complex concussions involve cases with persistent symptoms, specific sequelae (e.g. prolonged LOC and seizures) or prolonged cognitive impairment. Since there is limited published evidence that concussion injury severity correlates with the number and duration of acute concussion signs and symptoms or the degree of neurocognitive impairment, it was suggested that concussion severity could only be determined retrospectively after all concussion symptoms and neurological signs have cleared and cognitive functioning has returned to baseline (McCrory, Johnston, et al., 2005).

Whilst this process does stress a more individualized approach to concussion management, its use may result in further confusion amongst clinicians as the distinction between the different classifications of simple versus complex concussion appears somewhat loose. Although the classification of severity takes place retrospectively, it is difficult to say at what point a clinician classifies the concussion as simple or as complex. Many simple concussions could well develop into complex ones as evidence has shown the delayed effects of Grade I concussions in the latent development of cognitive deficits and postconcussive symptoms (Hinton-Bayre & Geffen, 2002; Lovell, Collins, et al., 2004). Moreover, a serious problem that arises out of the Prague International Conference recommendations is that only concussions categorized as complex concussions warrant neuropsychological assessment. Since it is well-known that athletes underreport symptoms, cognitive symptoms as a result of concussion may not be identified in the first place if neuropsychological assessment had not taken place. This means that any individual with a simple concussion may well have had a complex concussion had they been tested neuropsychologically. The development of validated, empirically based severity grading scales will continue to be a work in progress (McCrory, Johnston et al., 2005).

Finally, from the above review it is evident that the definition of MTBI is full of difficulties and contradictions, and that this problem has become most exacerbated in the sports context due to the close relationship between definition of severity of MTBI and its subsequent management. Thus, whilst multiple grading systems have been developed within the sports arena which lack empirical validation and are in the process of being abandoned, new attempts at validating scales are currently in progress.

## CHAPTER THREE

### GENERAL ISSUES IN MILD TRAUMATIC BRAIN INJURY

The central concern of this chapter is to examine the epidemiology, neurophysiology, and neuroimaging techniques in respect of mild traumatic brain injury (MTBI) in general (as distinct from specifically with reference to sport which is the subject matter of Chapter six). This will be followed by a discussion on outcome following MTBI, which includes identified risk factors which may serve to influence the presentation of this outcome. A discussion of the Brain Reserve Capacity Theory concludes this chapter and provides an explanation for the variability in outcome following MTBI.

#### 3.1 EPIDEMIOLOGY OF MTBI IN GENERAL

In general most closed TBIs are classified as mild, with global estimates ranging from 55% (Rimel et al., 1981), to 85% (Bennett & Raymond, 1997; Wrightson and Gronwall, 1999) to between 70 to 90% (Cassidy et al., 2004; Kraus & Nourjah, 1988). The WHO Collaborating task force on MTBI reviewed 169 studies, after screening 38,806 abstracts on the epidemiology, diagnosis, treatment and prognosis of MTBI (Cassidy et al., 2004). The reviewed articles showed that between 70 to 90% of all treated TBIs were mild and that the incidence of hospital treated patients with MTBI was 100 to 300 per 100,000 population but is probably above 600 per 100,000 since most MTBIs are not treated in hospitals. The incidence of MTBI is therefore difficult to ascertain particularly from hospital data since many do not seek treatment or are seen only in the outpatient environment.

The Centers for Disease Control and Prevention (1997), recently reported that MTBI has reached epidemic proportions in the United States. In 1991 the National Health Interview Survey analysed the incidence of MTBI in the United States and estimated that 1.5 million non-institutionalized civilians suffer from MTBIs each year at a rate of 618 per 100,000 annually (Sosin, Sniezek, & Thurman, 1996). Estimates of the number of cases hospitalised in the USA were as high as 327 000 in the mid 1990's which excludes those not seeking hospitalisation (Binder, 1997). Recently, in a study by Bazarian et al. (2005), the incidence of United States

emergency department attended MTBI was 503.1 per 100,000, whilst in North America the incidence rates vary from between 51 to 618 per 100,000 (Cassidy et al., 2004). In Great Britain of the 250-300 per 100,000 hospital admissions, at least 75% are classified as mild (Jennett & MacMillan, 1981; Kraus & Nourjah, 1988). Similarly, in a prospective study by Thornhill and colleagues (2000) documenting disability amongst young people and adults following head injury, 90% of the sample reported to have MTBIs, which relates to an incidence of 154 per 100,000. The incidence rate in Spain is 80 per 100,000 (Vásquez-Barquero et al., 1992) and in France (Tiret et al., 1990) and Italy (Servadei et al., 1988) two hundred and seven and 297 per 100,000 respectively. In Auckland, New Zealand the incidence of MTBI ranges from 782 per 100,000 (Wrightson and Gronwall, 1998) and in New South Wales, Australia 64 per 100,000 (Tate, McDonald, & Lulham, 1991). To the author's knowledge, no epidemiological data for MTBI are currently reported in the scientific literature in respect of South Africa.

As with TBI most MTBIs are due to falls and traffic related accidents, with recreational activities and sports accounting for the rest. Bailes and Cantu (2001) differentiate between recreational activities and organized, sanctioned sports. The former are classified as recreational or non-organized sports where there is no formal structure, organization or rules whereas the latter requires structured training, specialized equipment, physicians, athletic trainers and enforced rules. It is estimated that 750,000 Americans sustain injuries in recreational activities each year and of that 82,000 sustain some form of TBI (Bailes & Cantu, 2001). Bicycles and sports accounted for 26.4% of all MTBIs attended at a United States emergency department (Bazarian et al., 2005) and in a civilian survey performed in the United States, sports and recreational activities accounted for 20% of all MTBIs (Sosin et al., 1996). Falls accounted for 50 to 60% of all MTBI admitted to Swedish hospitals during the period from 1987 to 2000, followed by traffic related injuries which accounted for 25% (Peloso, von Holst, & Borg, 2004). Similarly, Sosin et al. (1996) found that of their sample of 46,671 civilians in the United States, 28% of the sample who sustained a MTBI were involved in motor-vehicle accidents. In a French study 59.7% of MTBI cases were caused by motor-vehicle accidents and 32.5% by falls (Tiret et al., 1990).

Teenagers, young adults and males tend to be the highest risk group for sustaining MTBIs (Bazarian et al., 2005; Cassidy et al., 2004; Parkinson, Stephensen, & Phillips, 1985; Meerhoff de Kruijk, Rutten, Letters, & Twijnstra, 2000; Sosin et al., 1996). More than 5% of high school

athletes in the United States suffer from MTBIs each year whilst playing sport (Powell & Barber-Foss, 1999). In general, school-going males are more than twice as likely as females of the same age to suffer a TBI (Kraus, 1995). A Canadian study conducted on high school students reported an incidence of 37% amongst males in comparison to 23% for girls (Segalowitz & Brown, 1991). North American studies African Americans are at greater risk than Caucasians for MTBI (Jager, Weiss, Coben, & Pepe, 2000; Whitman, Coonley, & Desai, 1984), although in another study white American females were at greater risk than African-American females (McCarroll & Gunderson, 1990). Individuals aged between 18 to 23 years are 3.8 times at risk for traffic related MTBI than individuals 50 years or older (Cassidy et al., 2004).

Although there is limited information on paediatric MTBI, the estimated incidence rate of TBI amongst children per year is 180 per 100, 000, of which 85% are classified as mild (Kraus, 1995; Kraus, Fife, Cox, Ramstein, & Conroy, 1986; Durkin, Olsen, Barlow, Virella, & Connolly, 1998; Engberg & Teasdale, 1998; Goh & Poon, 1995). In a study examining children up to the age of 15 years admitted to hospitals in San Diego, 88% of those admitted had a MTBI (Kraus, Fife, & Conroy, 1987). The lack of information on the epidemiology of MTBI amongst children represents a growing need for more research in this area.

Although these studies support the fact that incidence rates of MTBI are on the increase it is difficult to draw consistent conclusions from these findings. The incidence rates vary between studies owing to the heterogeneity of the populations studied with regards to the different inception periods, diverse source populations, differing inclusion and exclusion criteria and definitions used in the diagnosis and treatment of MTBI (as seen with the inconsistencies in the different classifications and grading scales), which lends itself to information bias and misclassification of cases (Cassidy et al., 2004).

## **3.2 NEUROPHYSIOLOGY OF MTBI**

### **3.2.1 Mechanisms of injury**

In the past MTBI was not considered to involve any gross structural damage (Stritch, 1961). This was largely due to the fact that the changes taking place are microscopic and diffuse in nature in that the injury is scattered throughout the brain, and that the changes to the brain cells do not

result in sufficient alteration to the tissue density to be detected on conventional scanning such as computed tomography (CT) scanning and magnetic resonance imaging (MRI) (Gaetz, 2004). Today however, MTBI is thought to cause both structural (stretching, compressing and tearing of axons) and functional damage (disruptions in cognitive functioning such as processing speed, memory, reaction time and the presence of postconcussive symptoms) (Bigler, 2003; Gaetz, 2004). MTBI typically involves the acceleration and deceleration from a direct or indirect impact which in turn creates linear or rotational forces on the brain (Barth et al., 1999). Factors such as mass, weight, velocity, hardness and surface area of the impacting object are all important elements that need to be considered in predicting the extent of neuronal damage (Bender et al., 2004). Therefore, the severity of the injury is a function of both the magnitude and direction of these forces separately and in combination with each other.

However, whilst it makes sense that the force at the time of contact would be the determining factor for injury severity, there are cases, as Cantu (1996) points out, where the forces applied seem to be high enough to result in injury and no injury occurs, whilst on the other hand there are cases where injury has occurred from a seemingly low force. What researchers have now demonstrated that MTBI can occur in any activity, regardless of its nature and when it does occur the outcome can have serious, long-term effects and consequences (Baker & Patel, 2000; Gaetz, 2004; King, 1997; Macciocchi, Barth, & Littlefield, 1998; Sterr, Herron, Hayward, & Montaldi, 2006), which is contrary to earlier beliefs that such injuries result in reversible and non-structural changes (Pang, 1985). Research on primates has shown that LOC is induced via angular (rotational) forces to the brain and that whilst linear acceleration forces appear to be relatively well tolerated in the brain angular forces are not (Gennarelli et al., 1982). Other researchers, however, caution that one cannot attribute the forces to the brain as being uniquely angular or linear in nature and that both types of head acceleration are involved in virtually all injury events (Elson & Ward, 1994). These angular forces are commonly seen in injuries that rotate the head on impact such as are often seen in motor vehicle accidents and sports injuries (Ponsford, Sloan, & Snow, 1995). There are three types of stresses generated as a result of the applied force: compressive, tensile (the opposite of compressive) and shearing (Gaetz, 2004). Whilst compressive forces are well tolerated due to the nature of the cerebrospinal fluid (CSF), which acts as a shock absorber converting external stresses to compressive stress, shearing stresses are not well tolerated. Despite the nature of the CSF, if rotational forces are applied to

the head, shearing forces will occur at those sites where rotational gliding is hindered (Anderson et al., 2001).

More specifically, structural and functional damage can be seen to occur as a consequence of brain trauma in a number of different ways. The first way relates to the anatomical housing of the brain within the skull. The brain is supported and protected by three membranes, the outer dura mater, the middle arachnoid layer and a tough pia mater, and cerebrospinal fluid, which fill the subarachnoid space and ventricles. The brain has a limited amount of movement within the skull and as a result its inertia when mechanical forces accelerate the head and its momentum when the moving head is accelerated may cause the brain within its protective membranes to contact the inner surface of the skull (Elson & Ward, 1994), which results in shearing and contusion of the cortical surface (Lezak et al., 2004). In the case of a physical blow to the head, injury to the brain near the site of impact is not implicated due to the nature of the cranium which is sufficiently robust to protect the brain (Lezak et al., 2004). The frontotemporal regions proximal to the bony irregularities forming rough corrugated surfaces at the anterior and middle cranial fossae are most likely to be affected (Lezak et al., 2004). This is seen in the case of a contrecoup injury which occurs when an individual makes contact with an object whilst in motion causing a maximum injury directly across the skull from the point of contact (Solomon et al., 2006). In contrast, a coup injury occurs beneath the point of cranial impact usually as a result of an individual at rest receiving a blow beneath the point of cranial impact.

The second way is related to the interference within the white fibre tracts that connect the cortex to the midbrain and brainstem (Rutherford, Stephens, & Potter, 2003). High acceleration may lead to the formation of microscopic lesions, undetected by traditional imaging methods, which occur as a result to the tearing of the radiating axons which retract and expel balls of axoplasm (Lezak et al., 2004). With high acceleration, diffuse axonal injury is likely to result in prolonged coma, a 33% mortality rate and extensive neurological damage amongst survivors (Rutherford et al., 2003). These forces have a direct influence on the reticular system in the dorsal midbrain leading to loss of consciousness and may affect the corpus callosum and the diencephalic structures proximal to the third ventricle (Kirkendall, Jordan, & Garrett, 2001). According to Kirkendall et al. (2001), damage to the latter structures may be responsible for longer-term memory deficits associated with MTBI. Recently researchers have proposed to replace the term

diffuse axonal injury with the term traumatic axonal injury, as damage is believed to occur primarily at the axons and since trauma can occur to individual axons among many non-injured cells, damaged axons are not truly considered to be diffuse because they tend to group in various areas of the brain (Gaetz, 2004). The term diffuse axonal injury, however, still continues to be the preferred term of choice by researchers in leading contemporary books and periodicals (e.g. Bigler, 2001; Hammoud & Wasserman, 2002; Lezak et al., 2004; Lovell, Echemendia, & Burke, 2004; Solomon et al., 2006).

The third way in which trauma can cause damage to the brain is through a series of neurochemical changes that begin within the first hour after the trauma and continue for up to 10 days (Rutherford et al., 2003). The cascade of neurochemical changes places the nerve cells under metabolic stress which increases their vulnerability to further and fatal damage (Hovda et al., 1999). An increase in the demand for glucose and a reduction in cerebral blood flow are indicative of this vulnerable state that may predispose the individual to further injury. This period of nerve cell vulnerability may also have consequences for second impact syndrome (to be described in more detail below), although some researchers are uncertain whether this period of diminished cerebral metabolism is protective rather than representing as a second potential period of vulnerability (Giza & Hovda, 2004).

Following head injury there is a marked elevation of extracellular potassium ( $K^+$ ) which activates the ATP dependent sodium-potassium ( $Na^+-K^+$ ) pumps (Wojtys et al., 1999). This increase in glucose demand is triggered in response to cell activating sodium-potassium pumps resulting in metabolic stress (Gaetz, 2004). In addition to the potassium efflux, there is an influx in the concentration of Calcium ( $Ca^{2+}$ ) which occurs in response to a reduction in cerebral blood flow following MTBI occurring near the site of injury (Wojtys, et al., 1999). Increases in  $Ca^{2+}$  can lead to metabolic dysfunction and even energy failure but mostly following mild to moderate traumatic brain injury,  $Ca^{2+}$  accumulation peaks in 48 hours and then resolves without significant cell loss (Giza & Hovda, 2004). The increase in glucose metabolism during the first minutes of injury can last for days after the injury and is an important factor in delayed injury or increased vulnerability to further injury (Giza & Hovda, 2004). The duration of decreased cerebral blood flow is also an important factor in determining outcome as brief interruptions in cerebral blood flow can lead to ischemia and neurocognitive deficits (Bender et al., 2004).

Animal research has identified acute metabolic dysfunction following MTBI. Although this has yet to be confirmed in the human model, this might explain the increased neuronal vulnerability that can exist for several days following a MTBI (Echemendia & Cantu, 2003; Guskiewicz et al., 2003). Neurons that have been injured remain alive but in a vulnerable state and are susceptible to minor changes in cerebral blood flow, intracranial pressure and anoxia (Echemendia & Cantu, 2003). This disrupted cellular metabolism leaves the cells more vulnerable to further injury (Giza and Hovda, 2000). Decreased cerebral blood flow has been reported to last up to 10 days following MTBIs in animal models and is consistent with researchers Guskiewicz et al's (2003) findings where collegiate athletes sustained repeat concussions within seven and ten days of the first injury, suggesting a period of increased susceptibility to recurrent injury. These neurochemical and structural changes have parallels in neurocognitive functioning, where several studies have shown that neurocognitive changes can exist for up to ten days following a MTBI, consistent with most sport studies investigating the acute recovery of athletes (Barth et al., 1989; Bleiberg et al., 2004; Collins, Grindel, et al., 1999; Guskiewicz, Riemann, Perrin, & Nashner, 1997; Lovell, Collins, et al., 2004; Macciocchi et al., 1996).

### **3.2.2 Neurophysiological outcome following MTBI**

The mechanisms and severity of injury affect the magnitude and duration of postconcussive metabolic dysfunction which can lead to long-term cognitive dysfunction even in the absence of obvious structural damage (Giza & Hovda, 2004). Children and adults respond differently to trauma, and whilst some researchers suggest that generally the young brain may recover more quickly than in adults (Giza & Hovda, 2004), others have suggested that children may experience prolonged recovery and present to be more vulnerable to cerebral swelling and subdural hematomas post-injury (Field et al., 2003; Pellman et al., 2006). Furthermore, researchers postulate that the developing brain may be up to 60 times more sensitive to glutamate, a neurotransmitter involved in neurochemical changes following MTBI thereby increasing the likelihood of permanent or severe neurological deficit (Lovell, Collins, et al., 2004). This may provide further evidence to support the occurrence of second impact syndrome amongst young individuals (Cantu & Voy, 1995).

Haematomas and haemorrhages are uncommon in MTBI but can result from serious injuries. The leading cause of death from serious injuries is intracranial haemorrhage of which there are four

types: (i) extradural haematoma which is associated with a fracture of the temporal bone and results from a tear in one of the arteries covering the brain; (ii) subdural haematoma occurs between the brain surface and the dura and often results from a torn vein running from the surface of the brain to the dura; (iii) an intracerebral haematoma results in bleeding into the brain substance itself, usually from a torn artery and (iv) subarachnoid haemorrhage which is confined to the surface of the brain as a result of the rupturing of the tiny surface brain vessels (Anderson et al., 2001; Gaetz, 2004).

Acute complications associated with MTBI involve raised intracranial pressure as a result of either an intracranial haematoma or brain swelling. This can be seen in cases of second impact syndrome and malignant brain edema associated with diffuse swelling, which are both similar in pathology and more common in individuals younger than 21 years of age (Cantu, 1992). Although second impact syndrome was first described by neurosurgeon Schneider in 1973, Drs Saunders and Harbaugh (1984) coined the term second impact syndrome. This condition occurs when an individual sustains a head trauma whilst still symptomatic from a previous head injury. Although the second injury may be minor, the consequences could be fatal (Bailes & Cantu, 2001). Second impact syndrome involves a loss of autoregulation in the brain's blood supply, edema and uncontrolled intracranial hypertension (Bailes & Cantu, 2001, Kelly et al., 1991). The loss of autoregulation, which involves the loss of an organ's ability to maintain constant blood flow despite changes in arterial pressure (Solomon et al., 2006), leads to vascular engorgement which results in increased intracranial pressure, followed by uncal herniation, cerebellar herniation or both (Junger et al., 1997). The time from second impact to brain stem failure is between two to five minutes and once this and brain herniation occur ocular involvement and respiratory failure follow (Bailes & Cantu, 2001).

Results from animal studies suggest a 'critical period' in which recovery from MTBI takes place which is related to the cerebral maturation of the brain rather than chronological age (Anderson et al., 2001). This explains why the condition occurs more frequently in individuals less than 21 years of age and why children who engage in activities such as contact sports, are at a greater risk of developing this condition (Heilbronner & Ravdin, 2004). Although children, adolescents and young adults are prone to this type of pathological vascular congestion, and more cases have been reported amongst boys between the ages of 16 to 24 years (McCrory & Berkovic, 1998a),

the precise population at risk is unknown and so is the true incidence of second impact syndrome (Bailes & Cantu, 2001). These findings, however, are of particular importance to the current study since the sample group all fell within this age range (i.e. 16 to 18 years).

Whilst it is clear that second impact syndrome is a rare phenomenon requiring further investigation, as researchers have questioned the validity of some cases (McCrory and Berkovic, 1998a), of critical concern is that it is characterized by extreme morbidity and that currently there are no published studies indicating that the condition does not exist (Erlanger, Kutner, Barth, & Barnes, 1999). Moreover, the effects of repeated MTBI need to be considered as a possible risk factor for the development of second impact syndrome, although researchers have also questioned this association (McCrory, 2001). The role of the neuropsychologist used in the evaluation of second impact syndrome is of great importance since second impact syndrome occurs whilst the athlete is still symptomatic and neuropsychological tests are designed to be sensitive enough to detect even the most subtle effects of cognitive decline.

### **3.3 NEUROIMAGING IN MTBI**

Neuroimaging investigations can be classified as either structural or functional. Structural measures include traditional imaging methods such as computed tomography (CT) scanning and magnetic resonance imaging (MRI) and are usually normal following MTBI (Borg et al., 2004; Levin, Mattis, et al., 1987). Functional measures discussed here include functional magnetic resonance imaging (fMRI), single photon emission computed tomography (SPECT) and positron emission tomography (PET), and are usually abnormal following MTBI as they measure the functions underlying the brain structures (Bigler & Orrison, 2004; Jacobs, Put, Ingels, & Bossuyt, 1996; Johnston, Ptito, et al., 2001). Other functional measures include electroencephalograms (EEG), evoked potentials and transcranial Doppler Ultrasound.

#### **3.3.1 Computed tomography (CT) scanning, and magnetic resonance imaging (MRI)**

Computed Tomography (CT) scanning is usually performed in clinical settings in the acute stages of TBI, primarily to rule out the possibility of intracranial bleeding but also because they are able to detect practically all of the surgically significant lesions (Adams et al., 1996; Lezak et al., 2004). Although CT scanning is both cost-effective and beneficial and has proved to be of

significant benefit with severe TBI (Johnston, Ptito, et al., 2001), in milder cases it does not provide conclusive evidence (Bigler & Orrison, 2004). As already stated, MTBI patients rarely have abnormal CT scans which is due to the pathophysiology of MTBI where changes taking place are usually diffuse and microscopic (Gaetz, 2004; Baker and Patel, 2000) rendering it largely invisible to traditional radiological techniques (Echemendia & Cantu, 2003). For example researchers Borg et al. (2004) found CT scan abnormalities in 5% of patients presenting to hospitals with a GCS score of 15 and 30% or more of these patients with a GCS score of 13. Yet only 1% of the MTBI patients required neurosurgery (Borg et al., 2004). This is comparable to other findings where 95% of severe TBI patients showed abnormalities on CT scanning, in comparison with 25% of individuals with moderate injuries and only 5% of individuals with MTBI (Levin, Amparo, et al., 1987). Lack of evidence on CT scanning does not suggest that no obvious brain damage has taken place, however, since evidence of both behavioural and cognitive deficits on post-injury performances have been documented in the neuropsychological assessment of MTBI, in the absence of a positive CT scan (Lord-Maes & Obrzut, 1996).

Whilst Magnetic Resonance Imaging (MRI) has become the preferred technique in the evaluation of MTBI, and provides evidence of diffuse axonal injury (Anderson et al., 2001), findings have been inconsistent where in some studies they have been demonstrated (Mittl et al., 1994) and in others there have been none (Johnston, Ptito, et al., 2001). This lack of evidence is probably due to the fact that both CT and MRI scanning do not specifically demonstrate the areas in the brain in which structural impairment is likely to occur, with factors such as edema and swelling also playing a role (Kreiter et al., 2002; Lezak et al., 2004). Whilst MRI is more expensive than CT scanning, it is to some extent more superior in defining the number, location and depth of lesions across all levels of injury severity, especially non-haemorrhagic diffuse axonal injuries, some subdural collections and brainstem lesions that are known to predict poorer global and neuropsychological outcome (Adams et al., 1996; Levin, Amparo, et al., 1987; Lezak et al., 2004). For example, researchers Levin, Amparo, et al. (1987) and Levin et al. (1994, 1997), have shown correlations between MRI abnormalities and deficits on neuropsychological tests of frontal lobe function. Although MRI is used in the evaluation of acute TBI, it is a better prognostic measure at the chronic stage (more than three months post-injury) (Bigler, 2001; Guskiewicz et al., 2004; Wilson, 1988). For example amongst the sports arena MRI changes

have been documented in repeated MTBI amongst professional boxers (Casson et al., 1984) and in elite soccer players (Jordan, Matser, Zimmerman, & Zazula, 1996).

The clinician's decision to scan MTBI patients is a difficult one since evidence suggests that neither LOC nor mild altered mental status is a reliable indicator on which to select patients for scanning, although a liberal policy of scanning is warranted for paediatric patients with a high risk of injury despite normal neurological status (Simon, Letourneau, Vitorino, & McCall, 2001). Furthermore a normal neurological examination and no LOC do not exclude significant rates of intracranial injury (Simon et al., 2001). Researchers also report a weak correlation between neuroimaging findings and neurocognitive outcome (Bigler, 2001; Hofman et al., 2001). The adoption of a multiple neuroimaging approach has been suggested in order to increase the sensitivity of detecting residual injury (Bigler & Orrison, 2004). Here researchers have found that by just using CT imaging alone an 8% abnormality rate in concussed patients was found compared with a 77% abnormality rate when using both MRI and single photon emission computed tomography (SPECT) (Hofman et al., 2001).

### **3.3.2 Functional magnetic resonance imaging (fMRI)**

Functional magnetic resonance imaging on the other hand provides task-specific information about neural function and is well suited to detect functional abnormalities associated with MTBI (Jantzen, Anderson, Steinberg, & Kelso, 2004). Functional magnetic resonance imaging technology is based on the measurement of specific correlates of brain activation such as cerebral blood flow and oxygenation which is promoted through the in-scanner implementation of specific neurocognitive tasks (Bigler & Orrison, 2004; Lovell & Collins, 2002). In this way pre-injury baseline measures of neural function can be obtained in anticipation of any future deleterious effects which may occur such as brain injury. FMRI has become a widely used method for investigating traditional psychological processes in both acute and chronic conditions across the different levels of severity. It is therefore able to provide data regarding the sensitivity and specificity of neuropsychological testing in detecting subtle changes in brain function post-injury (Lovell & Collins, 2002). Jantzen et al. (2004) were amongst the first to utilize fMRI in a prospective study of MTBI. Their results showed MTBI-induced differences in neural

functioning, which suggest that fMRI may increase sensitivity to MTBI compared with neuropsychological evaluation alone (Jantzen et al., 2004).

Predictable and reliable changes in brain activity can be seen during the scanning process of fMRI whilst individuals simultaneously perform neurocognitive tests (Lovell & Collins, 2002). In studies on TBI patients whilst performing cognitive tasks, the fMRI showed larger areas of activation in the brains of the patients relative to the controls (McAllister et al., 1999). Axonal injury after MTBI is thought to reduce processing efficiency, thereby imposing additional demands on neural networks involved in task performance. Clinicians are thus able to make measurable and predictable outcomes by systematically tracking the recovery process (Lovell & Collins, 2002). Functional magnetic resonance imaging has the added advantage over other imaging techniques such as PET and SPECT in that it involves no exposure to radiation and multiple sessions can be performed on a subject at one time (Jantzen et al., 2004). Due to considerable expense, however, fMRI is used in only a few clinical settings and is yet to be implemented on a wider basis. It does, however, validate neuropsychological testing as a sensitive and reliable method to detect subtle changes in brain functioning (Lovell & Collins, 2002).

### **3.3.3 Single photon emission computed tomography (SPECT) and positron emission tomography (PET)**

Positron emission tomography (PET) has increased in popularity in the last decade and is useful in investigating metabolic processes in the brain such as glucose utilization and neurotransmitter concentrations (Johnston, Ptito, et al., 2001). Single photon emission computed tomography (SPECT) is also involved in the investigation of glucose utilization and other metabolic processes and has also been found to correlate with persisting postconcussive symptoms (Jacobs, Put, Ingels, & Bossuyt, 1994) and cognitive deficits (Bigler & Orrison, 2004). Although the American Academy of Neurology (AAN) does not support the use of SPECT in the routine evaluation of patients with closed head injury or postconcussion syndrome, probably because it involves the intravascular injection of a radiopharmaceutical (Bigler & Orrison, 2004), it is still used in research (Rees, 2003). Jacobs et al. (1996) performed SPECT imaging on 136 MTBI patients four weeks post-injury, where postconcussive symptoms (PCS) were also documented.

Abnormalities were reported in 73 (54%) of these patients and of these, 32 patients reported PCS. At 12 months, nine patients (7%) showed both abnormal SPECT images and PCS, whereas three patients (2%) showed abnormal SPECT images only, whilst none showed just PCS (Jacobs et al., 1996). The authors concluded that based on their findings SPECT is a good predictor of outcome in the acute phase post-injury although it is more commonly used in assessing functioning in the chronic phase (Hofman et al., 2001).

The issue of whether an abnormal SPECT image is indicative of organic lesions rather than of a psychological disorder is an important consideration (McMillan, 1997). An abnormal SPECT however, does not always relate to the injury and can be caused by vascular headaches, altered mood and other variables (Rees, 2003). In a study by Mitchener et al. (1997) SPECT abnormalities were found to correlate poorly with focal CT and MRI findings as well as with clinical outcome. What SPECT and PET do is improve the ability to examine other brain regions that may not have observable structural defects (Bigler & Orrison, 2004). Although less expensive than PET, SPECT still involves similar limitations and is rarely used in younger children due to the fact that the procedure involves the injection of a small amount of radioactive material (Lovell & Collins, 2002).

### **3.3.4 Electroencephalograms (EEG) and Evoked Potentials**

Electroencephalography (EEG) may provide an index of abnormality in severe TBI, although with MTBI, it is usually associated with normal or non-specific EEG findings (Johnston, Pfito, et al., 2001). There is some evidence that EEG used together with CT scanning is predictive of both neurological outcome and educational success following mild to moderate TBIs in paediatric samples (Anderson et al., 2001; Ruijs, Gabreels, & Thijssen, 1994). Electroencephalography has also been shown to detect neurological abnormalities in patients with PCS (Mandel, 1989), whilst in studies on populations with persistent and cumulative MTBI (e.g. boxers), significant abnormalities on EEG testing when compared to controls have also been shown (Tysvaer, Storli, & Bachen, 1989). This evidence, however, does not improve the diagnostic utility of EEG in MTBI populations as a whole (King, 1997) and EEG measurements are still inconsistent in identifying long-term impairments post-injury (Haglund & Persson, 1990). The incidence of posttraumatic epilepsy in MTBI patients is very low, although MTBI can reduce the seizure

threshold in individuals with epilepsy (Mandel, 1989). According to Evans (1992), the sensitivity and specificity of EEG is limited with factors such as the use of pharmacological agents, interference from other electronic equipment and the behaviour of the patient who can be agitated and uncooperative, reducing its overall effectiveness as a diagnostic instrument in MTBI.

Event related potentials (ERP) represent useful tools for clinical diagnosis in detecting anomalies following TBI, in that the procedure is relatively inexpensive and non-evasive (Lovell & Collins, 2002). The ERP represent the averaged EEG signal time-locked to the stimulus and consist of different components labelled by their amplitude polarity (e.g. P = positive) and temporal range in milliseconds, i.e. P300. Researchers suggest that ERP may be sensitive to the effects of MTBI if the cognitive demands for certain tasks require more attention and are significantly more challenging for the patients (Lavoie, Dupuis, Johnston, Leclerc, & Lassonde, 2004). Although Lavoie et al. (2004) showed that the P300 component may be more sensitive than other standard measurements such as imaging, EEG and even neuropsychological assessment to detect the neural impact of MTBI, the differences shown by symptomatic patients is not completely certain as there is as yet no evidence that these differences are associated with neurocognitive changes, even in the acute phase. Few studies have shown that symptom severity is related to P300 abnormalities and that even in the absence of observable symptoms P300 abnormalities can be observed (Lavoie et al., 2004).

Auditory evoked potentials are a subclass of event-related potentials where the “event” is sound (Stapells, 2004, 2005). These evoked potentials assess the pathway beginning at the eighth cranial nerve, through the auditory brainstem nuclei and ending in the contralateral Heschel’s gyrus (Adams et al., 1996). Abnormalities along this pathway suggest dysfunction in distinct brainstem nuclei (Ropper & Miller, 1985). According to Mandel (1989), up to 80% of patients with dizziness, vertigo, and hearing loss have been reported to have abnormal brain stem auditory evoked potentials, which indicate a slowing of central auditory processing. Although abnormal auditory evoked responses have been found in some MTBI patients post-injury, their prognostic value is yet to be demonstrated (King, 1997). Whilst abnormal auditory evoked responses do provide evidence of organic factors possibly being involved following MTBI, their presence does not confirm an organic basis for PCS (King, 1997). As with EEG information,



results of auditory evoked potentials data suggest that they are better at predicting morbidity in TBI than accurately predicting functional outcome (Lindsey, Pasaoglu, & Hirst, 1990).

### **3.3.5 Transcranial Doppler Ultrasound (TCD)**

Recently researchers headed by Neurologist, Dr Tegeler at the University of Wake Forest have begun to use transcranial Doppler ultrasound (TCD) in the evaluation of MTBI particularly in the sports arena (Tegeler et al., n.d.). As previously discussed, a mismatch in the brain metabolism (increased) and blood flow (decreased) has been suggested after MTBI. Since the response and function of cerebral vessels after MTBI is poorly understood, TCD may offer important information in that it is used to measure cerebrovascular blood velocities post-injury (Tegeler et al., n.d.). This work-in-progress pilot study shows that TCD may reveal pathophysiological mechanisms of MTBI that may persist even after cognitive functions have returned to baseline or 'normal' levels in athletes who have sustained MTBIs (Tegeler, 2004). Transcranial Doppler ultrasound may identify opportunities for acute treatments, and provide an additional objective measure to assist with return-to-play decisions in athletes (Tegeler et al., n.d.). It is safe and non-invasive and therefore may be used in young children.

## **3.4 OUTCOME FOLLOWING MTBI IN GENERAL**

It is evident from reviewing the literature that there is general confusion and debate around outcome following MTBI. Much of this confusion relates to methodological issues which play a major part in producing different outcomes in studies investigating MTBI (Kibby & Long, 1996). The populations studied are often heterogeneous with pre-morbid factors such as a history of neurological or psychiatric disorders, prior drug and alcohol abuse, prior head injuries and learning disabilities not controlled for in the sample groups (Alves, Macciocchi, & Barth, 1993; Dikmen & Levin, 1993). These factors serve to lengthen the period of recovery in patients and therefore influence the final outcome (see Section 3.5). Other factors such as inconsistent control groups, differences in injury severity classification, selection bias, subject attrition and varied time intervals from the time of injury to when measurement is taken, also contribute to the differences in outcome between studies (Kibby & Long, 1996; Macciocchi et al., 1998).

The varied outcome measures (such as neurocognitive tests, postconcussion inventories and outcome scales) employed to measure factors such as cognitive deficits, PCS and employment status, create other methodological issues. In addition to researchers using combinations of these measures in their studies, they also use different cognitive tests and measure different symptoms when measuring outcome (Kibby & Long, 1996). Researchers have expressed the need for consistency across studies in terms of cognitive measures, PCS, standard times of measurement post-injury, matched controls and standardized definitions for MTBI in order to improve the generalizability of outcome findings (Kibby & Long, 1996). Methodological issues pertaining to studies investigating MTBI in sport will be discussed in more detail in Chapter Six.

Many researchers believe that the effects of MTBI are subtle and that in general the cognitive sequelae and associated symptoms reduce to levels comparable with non-injured individuals within weeks or within three months post-injury (Barth et al., 1983; Collins, Grindel, et al., 1999). These effects, as identified earlier, are referred to as being *acute* (e.g. Alexander, 1995; Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005; Binder, Rohling, & Larrabee, 1997; Dikmen, McLean, & Temkin, 1986; Englander, Hall, Simpson, & Chaffin, 1992; Evans, 1992; Frencham, Fox, & Maybery, 2005; Mathias, Beall, & Bigler, 2004; Newcombe et al., 1994; Raskin, Mateer, & Tweeten, 1998; Reitan & Wolfson, 1999) and outcome is considered to be favourable. For others where the effects persist longer than three months, they are considered to be *chronic* and recovery is prolonged (e.g. Bernstein, 2002; Segalowitz, Bernstein, & Lawson, 2001; Van der Naalt, 2001; Vanderploeg et al., 2005). Generally, the recovery patterns of MTBI patients in the general population are consistent with the pattern of recovery amongst athletes who sustain MTBI as a result of participating in contact sport, in respect of neurocognitive outcome, in both the acute and chronic condition (e.g. Bleiberg et al., 2004; Hatfield et al., 2004; Hinton-Bayre & Geffen, 2004; Lovell et al., 2003; McCrory et al., 2000; Shuttleworth-Edwards et al., 2004; Stephens et al., 2005). In respect of PCS however, different outcomes have been demonstrated which is largely as a result of the different set of motivations in operation here between the different MTBI groups (Davis, 2002; Lees-Haley & Brown, 1993). Whilst athletes who have sustained MTBIs tend to underreport symptoms in order to return to play as soon as possible, MTBI patients in general tend to over report symptoms especially when the motive is one of compensation as a result of litigation (see Chapter Five). Cognitive deficits and symptoms

observed in both the acute and chronic condition following MTBI, specific to the contact sports, will be discussed in Chapter Six.

Generally, the measurement of outcome following MTBI is difficult to evaluate because there is little association between the injury severity and outcome amongst this population. Injury severity is determined by clinical symptoms which may or may not reflect the underlying pathophysiological processes involved (Macciocchi et al., 1998). Thus, the injury severity in two patients may be classified as being the same but their outcomes may be totally different. Moreover, many pre-injury factors such as age, education, history of neurological or psychiatric disorders, learning disorders and post-injury factors such as family support and compensation interact with cognitive functions and directly influence PCS, whilst they can also interact with injury severity and alter the recovery process significantly (Kibby & Long, 1996). Therefore, whilst one would expect individuals who suffer from a severe TBI to have a poorer outcome than those who suffer from a MTBI, there has been evidence to suggest that individuals reporting MTBIs also report specific problems post-injury which could be enhanced by pre-injury factors. For example in a study measuring outcome using the Glasgow outcome scale and problem orientated questionnaire in a sample of 2,962 patients aged 14 years and up, at one year post-injury, although 78% of the sample who suffered from severe TBI were disabled, 51% of survivors of MTBIs were also disabled, with increased dependency in 45% of the survivors who sustained a severe TBI and 28% of survivors with MTBI (Thornhill et al., 2000). Furthermore, the number of patients unfit for work amongst the MTBI group increased from 52 to 91 including 29 patients who were previously employed.

In a study by Brown et al (2004), which provided a population-based analysis of long-term survival following TBI in the United States, their findings demonstrated that persons with MTBI exhibited a small but statistically significant reduction in long-term survival compared to the general population. Moreover, although the fatality rate for individuals suffering from moderate to severe TBI was high, those who survived longer than six months had a similar long-term survival to those survivors of MTBI. Whilst pre-injury risk factors are predictive of a poorer outcome following MTBI, there have been some studies where MTBI patients had no pre-existing risk factors and still failed to achieve a favourable recovery (Thornhill et al., 2000).

Factors such as secondary gain or undetected pathophysiological processes could have played a role here (Sweet et al., 2003).

As previously discussed, because adults and children respond differently to TBI in terms of the underlying physiological and functional processes involved, both groups are expected to have variable outcomes (Giza & Hovda, 2004), where some studies have demonstrated children experiencing prolonged neuropsychological recovery compared with adults (Field, 2003; Pellman et al., 2006). With regards to MTBI amongst school-aged children, although there is still considerable debate with respect to outcome, generally a good recovery is expected in children with no pre-morbid conditions (Asarnow et al., 1995; Nortje and Menon, 2004; Ponsford et al., 1997). For example, researchers conducted a longitudinal study comparing children who sustained a MTBI with age-matched controls (Ponsford et al., 1997). The results revealed a good outcome for children generally, although children with a pre-morbid history of learning or behavioural problems exhibited deficits on neurocognitive and behavioural measures (Ponsford et al., 1997). Pre-morbid factors which pose as possible risk factors which serve to influence the overall outcome of the patient will be discussed in the next section.

### **3.5 RISK FACTORS INFLUENCING MTBI OUTCOME**

As indicated above, regardless of injury severity, many factors may affect or interact with injury severity to produce a better or worse outcome than expected (Macciocchi et al., 1998). Those factors considered to be of particular relevance are discussed below.

#### **3.5.1 Age**

Age is a strong predictor of recovery and therefore outcome, with older age been linked with poor recovery (King, 1997). Researchers Susman and colleagues (2002) suggest that it is not just the increasing incidence of complications amongst the aged that produce a poor outcome following MTBI but the characteristics of the aging brain. Generally the literature suggests a cut-off point at around 40 years of age (King, 1997) as being the point at which general recovery is slower in terms of PCS (Binder, 1986; Evans, 1992; Rutherford, Merrett, & McDonald, 1979) and neurocognitive functioning (Barth et al., 1983, 1989; Gronwall, 1989). Katz and Alexander (1994) found that amongst older patients older than 60 years who had suffered MTBI, 30% were

severely disabled 12 months post-injury. Recovery from cognitive deficits has been shown to be adversely affected by advancing age (Gronwall, 1989; Gronwall & Wrightson, 1974; Kibby & Long, 1996), with older age having a negative effect on cognitive functions such as naming, verbal fluency, visual and verbal memory, information processing and attention being the most vulnerable functions affected (Gronwall & Wrightson, 1974; Lezak et al., 2004). Russel and Smith (1961) found that the number of reported PCS following MTBI increased with age and that elderly patients also experienced significantly more depression and anxiety (Goldstein, Levin, Goldman, Clark, & Altonen, 2001). Age has also been found to have a poor correlation with returning to work following MTBI (Stambrook, Moore, Peters, Deviaene, & Hawryluk, 1990).

Similarly, the outcome of MTBI for children may also be negative when compared to adolescents and younger (as distinct from 'older') adults. Segalowitz & Brown (1991) found a significant relationship between the incidence of MTBI and several developmental factors such as hyperactivity, stuttering and mixed handedness. Other researchers found a significant difference in outcome between infants and toddlers (Ewing-Cobbs, Miner, Fletcher, & Levin, 1989). Although the level of severity was not highlighted in the study, the researchers found that children who sustained a TBI in infancy experienced difficulty developing receptive and expressive language whereas those who sustained TBI as toddlers exhibited a better rate of improvement in receptive language. Thompson, Francis, Steubing, & Fletcher (1994) investigated the change in factors affecting recovery following TBI in children through to adolescence. Their results confirmed the findings of previous studies which revealed that younger children with TBIs recover more slowly than older children with similar injuries.

According to researchers Anderson et al. (2001), a TBI that occurs in childhood may result in the alteration of the normal developmental process, on both a neurological and cognitive level, influencing the consolidation of cognitive skills. Whilst adult models form the basis of our knowledge of neurological disorders in children, they relate to a more static and mature brain. The brain of a child, on the other hand, is in a state of constant change and development. It is therefore reasonable to assume that a TBI in early childhood may influence ongoing development, although a high degree of variability in outcome amongst children exists (Anderson et al., 2001).

Evidence suggests that growth spurts occur in early infancy, and again around the ages of 7 to 10 years, with a final spurt in adolescence from 16 to 19 years (Fuster, 1993; Jernigan & Tallal, 1990; Klinberg, Viadya, Gabrieli, Moseley, & Hedehus, 1999; Sowell & Jernigan, 1998; Thatcher, 1991). These growth spurts are associated with 'critical periods' which represent stages in the developmental process during which an aspect of behavioural functioning may experience a major progression (Anderson et al., 2001). Thus, these critical periods have been conceptualized as opportune periods during which skills need to be consolidated so that the system involved can then establish interconnections with other systems (Anderson et al., 2001). For example, developmental researchers argue that a number of discrete growth spurts occur specifically in frontal lobe development, beginning at the age of six, again at the age of 10 years, with a final spurt in early adolescence (e.g. Bell & Fox, 1992; Cummings, 1993; Klinberg et al., 1999; & Thatcher, 1991) and that the timing of these developmental spurts is consistent with those described by neurophysiological researchers (Anderson et al., 2001). If for some reason this progression does not occur appropriately, then it may never occur (Anderson et al., 2001)

Developmental theorists such as Piaget emphasize a stage-like process of cognitive development in which children are required to pass through a series of preset developmental stages which coincides with cerebral maturation (Piaget, 1963). For example, at approximately seven years of age the transition to the stage of operational thought is reflected in the increased reasoning and problem-solving ability in children and is categorized by the emergence of neurocognitive functions in terms of executive skills such as reasoning, problem solving, organization and mental flexibility (Thomas, 1992). These functions become fully consolidated in the formal operational stage in early adolescence (Thomas, 1992). Whilst most children pass through these stages without crisis, where some disruption or impairment is present each transition to the next stage may be more problematic (Anderson et al., 2001).

### **3.5.2 Learning Disorders and prior psychiatric and neurological conditions**

Learning disability (LD) is a heterogeneous group of disorders which are typically identified in early childhood and refer to difficulties in the acquisition of listening, reading, writing, reasoning, or mathematical abilities (Collins, Grindel, et al., 1999). Studies have suggested a possible link between individuals with a history of LD and poor TBI outcome. Vieth, Johnstone and Dawson (1996) compared pre-and post-TBI data in an individual with a LD. The findings

showed a global and generally consistent decline in all cognitive areas. Other studies where individuals have a history of LD have reported a greater number of PCS post-injury (Kibby & Long, 1996). In a study by Haas, Cope, & Hall (1987), up to 50% of the sample of 80 TBI patients revealed poor pre-morbid academic performances. Similarly Segalowitz and Brown (1991) found a significant relationship between MTBI, learning disability and hyperactivity. Moreover, in a study by Collins, Grindel et al. (1999) investigating the relationship between prior MTBIs and LD amongst university student athletes, their findings demonstrated that a history of multiple concussions and LD are independently related to lower cognitive performances. Their study suggests an additive effect of LD and multiple episodes of MTBI on lowered functioning and ultimately poor outcome (Collins, Grindel, et al., 1999). Similarly, Donders and Strom (1997) examined the effect of traumatic brain injury on children with learning disabilities. On some neuropsychological measures the children with a history of LD deteriorated from their premorbid functioning as a result of the TBI, which illustrated that TBI can cause additional cognitive impairment in children with LD.

Prior neuropsychiatric conditions have also been seen to influence the level of recovery amongst patients. These patients often present with an increased amount of PCS and tend to have a poorer outcome (Kibby & Long, 1996). Bohnen, Twijnstra, & Jolles (1992), attempted to examine the association of pre-morbid emotional problems on PCS in MTBI patients and found that those patients with a prior history of MTBI or pre-morbid emotional problems reported significantly more symptoms than controls. Previous neurological insults such as tumours and strokes also serve to lengthen the time of recovery post-injury (Kibby & Long, 1996). Generally the more cells that have been damaged the longer it takes the individual to recover and the greater the risk for residual cognitive and psychosocial deficits (Kibby & Long, 1996).

### **3.5.3 Multiple mild traumatic brain injuries**

The effects of multiple MTBIs have been suggested as a possible risk factor for the persistence of chronic effects following MTBI (e.g. Macciocchi et al., 1998; Mrazik et al., 2000). The cumulative effects and risks associated with multiple MTBIs have been widely documented in the sports arena which presents as a high risk, highly predictable environment for repeated MTBI and will be discussed in detail in a subsequent chapter. For example amongst boxers multiple

sub-concussive blows to the head have been shown to produce long-term residual deficits even though each individual injury produces minimal or unobservable immediate effects (Casson, Sham, Campbell, Tarlau, & Didomenico, 1982; Heilbronner & Ravdin, 2004; Jordan, Matser, et al., 1996; Ryan, 1987). The increased number of bouts and length of exposure to the sport are factors which appear to exacerbate this process (Casson et al., 1984; Jordan, Matser, et al., 1996). Similarly, the long-term implications of the sub-concussive effects of heading the ball in soccer, is also a source of controversy (Rutherford et al., 2003). Whilst it seems obvious that multiple MTBIs should negatively influence outcome, results between studies are conflicting.

The relationship between multiple MTBI and dementia is also controversial. Both autopsy reports and experimental studies suggest that TBI, in particular severe and repeated mild to moderate head injury are risk factors for the development of dementias such as Parkinson's disease (Allam, del Castillo & Navajas, 2003) and Alzheimer's disease (AD) (Guo et al., 2000; Jellinger, 2004; Mortimer, French, Hutton, & Schuman, 1985). According to Carroll, Cassidy, et al. (2004), studies regarding the role of multiple MTBI as a risk factor for dementia should be considered as inconclusive at this time because of the conflicting findings. Whilst some studies found no relationship between dementia and multiple MTBI (Broe et al., 1990; Chandra, Kokmen, Schoenberg, & Beard, 1989; Fratiglinoni, Ahlbom, Viitanen, & Winblad, 1993; Mehta et al., 1999) others support the relationship (Graves et al., 1990; Mortimer et al., 1985; Mayeux et al., 1993; Schofield, Tang, et al., 1997).

The symptomatology and neuropathology of AD is also similar to that of dementia pugilistica (DP) or what is also known as chronic TBI, which is characterized by dysarthria, bradykinesia, cognitive impairment and personality change, as well as the presence of diffuse senile plaques and neurofibrillary tangles similar to those found in the brains of AD patients (Allsop, Haga, Bruton, Ishii, & Roberts, 1990; Rasmusson, Brandt, Martin, & Folstein, 1995). There is also evidence to suggest that TBI induces the deposition of amyloid  $\beta$  protein ( $A\beta$ ) in the brain which has been implicated in the formation of the diffuse plaques and tangles found in the cortex of individuals with DP and AD (Allsop et al., 1990; Roberts, Allsop, & Bruton, 1990). In a study by MacFarlane, Nicoll, Smith, & Graham (1999) between long-term TBI survivors and matched controls with the apolipoprotein-epsilon 4 allele (i.e. the genetic risk factor for AD), although there was no difference in  $A\beta$  deposits between survivors of TBI and controls,  $A\beta$  deposits

were more common amongst the apolipoprotein-epsilon 4 allele (APOE- $\epsilon$ 4) group suggesting that pathogenetic mechanisms other than A  $\beta$  deposition must explain the association of TBI and AD.

Although there have been many epidemiological studies to suggest an association between, multiple TBI, the apolipoprotein epsilon 4 allele genotype, amyloid  $\beta$  protein and the development of AD, researchers agree that further research is needed to clarify the relationship between these variables (Jellinger, 2004). Whilst some research supports an association between multiple TBI and AD (Casson et al., 1982; Guo et al., 2000; Jordan et al., 1992), researchers caution that AD cases do not always have a history of TBI and TBI victims invariably do not acquire AD (Lye & Shores, 2000). As multiple TBIs represent one of the many risk factors associated with the disease, all variables need to be considered when making a diagnosis. There are also methodological issues to consider, (e.g. establishing a universal definition for identifying, classifying and diagnosing TBI) when incorporating TBI as a possible risk factor which could ultimately effect the disease presentation.

#### **3.5.4 Genetic Factors**

Researchers support the relationship between the  $\epsilon$ 4 allele of the apolipoprotein E (ApoE) gene, identified as a risk factor in Alzheimer's disease (Guo et al., 2000; Jordan et al., 1997; Macfarlane, Nicoll, Smith, & Graham, 1999) and outcome after TBI (Lendon et al., 2003; Teasdale, Murray, & Nicoll, 2005; Teasdale, Nicoll, Murray, & Fiddes, 1997). Teasdale and his colleagues (1997) followed the outcome of 89 head injured patients. Their findings showed that patients with the APOE  $\epsilon$ 4 gene were more than twice as likely as those without the gene to have an unfavourable outcome six months post-injury. Similarly, Friedman et al. (1999) and Lichtman, Selinger, Tycko, & Marder (2000) in their studies showed that those patients with the APOE-  $\epsilon$ 4 allele experienced a worse outcome post-injury than those without the gene. Further, in a study by Jordan and his colleagues (1997) involving professional boxers, all boxers with severe chronic traumatic brain injury possessed at least one copy of the APOE- $\epsilon$ 4 allele. This was also supported in another boxing study (Kutner et al., 2000).

In a study by Mayeux et al. (1995) the relationship between the  $\epsilon$ 4 allele of the apolipoprotein E (ApoE) gene as a risk factor in Alzheimer's disease was examined amongst 236 community-

dwelling elderly persons. Results reported a ten-fold increase in the risk of AD associated with persons possessing both the APOE- $\epsilon$ 4 gene and a history of TBI compared with a two-fold risk with the APOE- $\epsilon$ 4 alone, although TBI in the absence of the APOE- $\epsilon$ 4 did not show an increased risk (Mayeux et al., 1995). In a study by Guo et al. (2002), however, the influence of TBI as a risk factor for AD appeared to be greater amongst people lacking the APOE- $\epsilon$ 4 gene compared to those having one or two  $\epsilon$ 4 alleles, suggesting a common biological underpinning associated with both these risk factors. Rasmusson et al. (1995) support this finding in their results which indicate that TBI may be a predisposing factor to AD in the absence of clear genetic contributions, although other unmeasured factors (e.g. pre-existing co-morbidities such as LD, psychiatric problems, neurological disorders and age) could also influence this association contributing to the overall pathogenesis of this disease.

Closely allied to the phenomenon of risk factors in outcome following TBI, is the postulated threshold theory of Brain Reserve Capacity (BRC) as it relates to differential presentation of neurological disease. 'Risk factors' ('vulnerability' factors in BRC terms) are empirically demonstrated phenomena that result in poorer outcome following TBI. Against the background of such research, the theory of Brain Reserve Capacity has been formulated, and allows for a more integrated level of conceptualization of such findings with important heuristic application in the MTBI research context.

### **3.6 BRAIN RESERVE CAPACITY THEORY**

The idea of cognitive reserve has been proposed by researchers to account for the inconsistencies and variability amongst individuals who sustain TBIs with similar levels of severity but where the outcome is different (Stern et al., 2003). The theory of Brain Reserve Capacity (BRC), reviewed by Satz (1993) and others (Randolph, 2001; Stern, 2002; Stern et al., 2003) represents as a threshold model to explain the concept of cognitive reserve. The theory postulates that a threshold factor or what Randolph terms as 'functional reserve' (Randolph, 2001), exists in each individual prior to the manifestation of symptoms resulting from dysfunction in the central nervous system or during normal aging (Richards & Sacker, 2003). Hence, once the BRC is depleted past this threshold, clinical or functional deficits emerge (Stern et al., 2003).

Accordingly researchers propose that variability amongst individuals occurs as a result of individual differences in the amount of neuronal damage or disease incurred which may serve to protect or cause vulnerability to functional impairment. The existence of pre-morbid vulnerability factors with the overlay of single or multiple traumas of neurological damage or cognitively challenging tasks, will result in an aggregation effect which further increases vulnerability to functional impairment (Shuttleworth-Edwards et al., 2004).

According to researchers there are both active and passive models of cognitive reserve which account for the variations amongst individuals (Stern, 2002). The passive model investigates the role of brain capacity as measured by head circumference or brain volume and supposes that individuals with a larger brain capacity can therefore afford to lose a greater number of neurons before they begin to show impairment (Lee, 2003; Stern et al., 2003). The active model focuses more on the efficiency or flexibility of neural networks. This type of reserve is used by healthy individuals as well as individuals with TBI when coping with the demands of a task (Stern et al., 2003). Hence, an individual who uses this network more efficiently in response to cognitive demands may have more cognitive reserve. Such is seen in individuals with higher levels of intelligence or educational attainment (Stern et al., 2003). Moreover, it has been suggested that early life physical activity may enhance the cognitive reserve by increasing the neural efficiency of the brain (Dik, Deeg, Visser, & Jonker, 2003). The two models are tightly interlinked such that given the same degree of efficiency, individuals with a larger brain capacity will suffer less impairment, and individuals with the same level of capacity, with a high degree of efficiency, will be able to tolerate a brain insult better (Lee, 2003).

The existence of pre-morbid variables (some of which have been identified above as risk factors associated with poor outcome following MTBI) such as low education and low IQ (Coffey, Saxton, Ratcliff, Bryan, & Lucke, 1999; Hill et al., 1993; Mortimer, 1994; Reitan & Wolfson, 1997), smaller head size (Di Sclafani et al., 1998; Mortimer, Snowdon & Markesbery, 2003; Schofield, Logroscino, Albert, & Stern, 1997), female gender (Zhang et al., 1990), the presence of a psychiatric or neurological disorder (Hibbard, Uysal, Kepler, Bogdany, & Silver, 1998), prior head injury (Mayeux et al., 1993; Salcido & Costich, 1992), aging (Satz, 1993; A.B. Jordan, 1997), learning disorders (Haas et al., 1987; Segalowitz & Brown, 1991), alcoholism (Temkin, Machamer, & Dikmen, 2003) or a combination of these factors present as vulnerability

factors predisposing individuals to the early onset of symptoms in association with neurological disease.

Thus, for example low education is a risk factor for accelerated aging and is also associated with a higher incidence of AD (Mortimer, 1994). In a study involving Roman Catholic nuns, those nuns who had low education levels demonstrated severe cognitive impairment, reduced motor function, and a higher impairment in activities of daily living (Snowdon, Ostwald, & Kane, 1989). Similarly, in a study by Schofield, Logrosino, et al. (1997), smaller brain size was seen to be significantly associated with the development of AD. More recently researchers Mortimer et al. (2003) suggested that higher education and larger head size, alone or in combination, may reduce the risk of a dementia in later life. In their study individuals with both smaller head circumference and lower education were four times more likely to be demented than those with other combinations of head size and education level. Conversely factors such as high levels of education and occupational attainment or intelligence, increased brain capacity and genetic predisposition (Lee, 2003), present as protective factors reducing the risk of functional impairment. For example, researchers suggest that increases in education, brain size and IQ may act as protective factors thereby delaying the onset of AD, resulting in lower incidence rates in the future (Mortimer, 1997). Hence, it is this variability in BRC that produces either a protective or vulnerability factor to symptom onset.

Jordan (1997), developed a model of inter-individual variability in aging, within which, as indicated above, she conceptualized the aging process as progressive mild brain insults, and this in turn can be linked to cumulative mild traumatic brain injury that may occur over a period of time within the sports context. As in the aging context, inter-individual variability in the presentation of symptoms is likely to exist in association with differential cognitive reserves. Thus, whilst certain individuals may not present with cognitive deficits others will demonstrate a fall-off in functioning due to factors (mentioned above) which cause a lowering of the threshold. There is also an aggregation effect of pre-morbid vulnerability factors or multiple neurological traumas which produces a vulnerability to reduce the BRC. With reference to this model, each subsequent injury, however minor, results in the further depletion of this reserve capacity thereby limiting the rate and perhaps the degree to which functional recovery can occur (Randolph, 2001). Integrating the aging effect with that of progressive mild traumatic brain injury, Satz

(1993) points out that the neuronal attrition associated with the aging process, accelerated by the cumulative effects of MTBI over the years together would lower the brain reserve threshold for early onset dementia. Such is seen typically seen amongst former of professional boxers, where abnormal CT scans and neurological deficits have provided evidence suggestive of early onset dementia (Casson et al., 1982; Jordan et al., 1992). Thus, whilst the early effects of multiple concussions may not be functionally evident, aggregation during aging may cause neuronal loss, resulting in a reduction in cerebral reserves, pushing the individual over the functional threshold into impairment.

In the BRC literature cited above (for example, Jordan, 1997; Satz, 1993; Stern, 2002; Stern et al., 2003), the theory has received elaboration largely in relation to the aging process and dementias. However, as suggested by Shuttleworth-Edwards et al. (2004) it is possible to elaborate the theory outside of the aging context to encompass the phenomenon of repeated MTBI in sport, in order to have more specific relevance to the present study, in the following way. Residual deficits have been demonstrated on testing amongst soccer athletes as a result of the concussive and sub-concussive trauma associated with frequent heading as well as multiple concussions sustained as a result of general play (Downs & Abwender, 2002; Matser, Kessels, Lezak & Troost, 2001; Witol & Webbe, 2003). More concerning is that the effects of multiple concussions are already in evidence amongst younger athletes (Collins et al., 2002; Iverson et al., 2004a). For example in a study examining the prolonged effects of MTBI in high school American football athletes, academic grades were significantly lower for athletes who experienced two or more concussions (Moser et al., 2005). Similarly, in a study by Collins, Grindel et al. (1999), athletes participating in American football with a history of two or more MTBI performed more poorly on cognitive tests and reported more symptoms than those with no previous MTBI. These studies support BRC theory by illustrating the cumulative additive effect of concussive and sub-concussive events in lowering the existing cognitive reserve to produce effects.

Further in terms of BRC theory and repeated MTBI, it can be extrapolated that the early effects of multiple concussions may not be functionally evident in the absence of aggregation with other vulnerability factors (for example, prior learning disability, older age, psychiatric or neurological disorder, or relatively low IQ), but may become evident when occurring in aggregation with one

or more vulnerability factors. Research reported above has suggested a ten-fold increase in the risk of AD associated with persons possessing both the APOE- $\epsilon$ 4 gene and a history of TBI compared with a two-fold risk with the APOE- $\epsilon$ 4 alone. Traumatic brain injury in the absence of the APOE- $\epsilon$ 4, however, did not show an increased risk (Mayeux et al., 1995). Similarly, in a study by Kutner et al. (2000) older American football players possessing the APOE  $\epsilon$ 4 exhibited significantly lower cognitive test scores than all other players studied, which suggests that the aggregate effect of repeated exposure to MTBI in addition to inherited factors such as the APOE  $\epsilon$ 4, as well as older age, produces a significant fall-off in cognitive functioning.

Other studies reported above investigating the relationship between TBI and LD in the general population have all suggested that LD in combination with a TBI result in a poorer outcome in terms of cognitive decline and a greater number of reported symptoms (Kibby & Long, 1996; Segalowitz and Brown, 1991; Vieth et al., 1996). More specifically in the sports context Collins, Grindel et al. (1999), cited above, investigated this relationship amongst a group of university athletes. The results suggested that both a history of multiple concussions and LD are associated with a reduction in cognitive performance. A significant relationship was found between the number of concussions and the number of years playing sport, which further supports the BRC theory that the greater the number the years of exposure to the sport in addition to the additive effect of a LD will push the individual over the cognitive threshold and produce symptoms. Similarly, Shuttleworth et al. (2004) reported on a subgroup of schoolboy Rugby Union players with a history of LD. Although the rugby subgroup had a higher estimated IQ than the field hockey subgroup who also had a LD, the percentage deficit for the rugby players clustered around 67%, compared with an almost zero indication of deficit for the field hockey players. Although the sample was small the results still suggest an aggregate effect due to the combined presence of a pre-morbid vulnerability factor with the overlay of single or multiple traumas.

Generally researchers exclude individuals with pre-morbid histories or vulnerability factors in their studies in order to prevent such variables from confounding test results. By including confounding variables in the research it becomes uncertain whether the effects are attributed to the injury alone or as a result of an aggregation effect with other vulnerability factors. Even if the level of IQ is controlled for in a sample along with other confounding variables, one still would not be able to account for the variation in individuals due to pre-existing levels of cerebral

reserve such as brain size, intellectual capacity and genetic brain disposition. Thus, it is expected that there will be differences in the presentation of symptomatology between individuals.

With regards to the present study, which is to examine the chronic and cumulative effects of MTBI over a high school contact sport playing career, it is expected that there will be differences with respect to individuals exposed to repeated MTBI through participation in a contact sport versus those who do not have this exposure, in terms of poorer performances on the challenging cognitive tasks and an increase symptom reporting for the contact and two or more concussion groups. It is therefore hypothesized that the additive effect of cumulative MTBI (as a result of being exposed to a contact sport over a high school playing career) with varying pre-existing levels of cognitive reserve would create a vulnerability in those contact sports players and individuals who have sustained multiple concussions resulting in the lowering of the cognitive threshold. This lowering would produce effects as evident on cognitive tests and self-reported symptoms, although the effects at the high school level are likely to be more subtle than in older athletes, due to shorter duration of exposure to the sport amongst high school athletes.

## CHAPTER FOUR

### ASSESSMENT OF COGNITION FOLLOWING MILD TRAUMATIC BRAIN INJURY IN GENERAL AND IN SPORT

From the discussion in chapter three, it is evident that sequelae following MTBI occur in the form of (i) cognitive deficits as identified on objective neuropsychological tests or (ii) self-reported postconcussive symptoms. The purpose of this chapter is to report specifically on the first of these, viz, cognitive deficits following MTBI as identified on objective tests measures. This chapter will first discuss general research issues relating to test characteristics as well as the criteria considered when selecting tests for the purpose of MTBI evaluation. This will be followed by a review of sensitive paper-and-pencil neuropsychological measures used in MTBI research in general including the sports arena. In addition to traditional paper-and-pencil measures that are routinely used in neuropsychological assessment, measures that have been specifically introduced for the evaluation of MTBI in sport will be reviewed, including sideline assessment techniques and the innovative computerized neurocognitive test batteries. The paper-and-pencil tests and computerized test battery that were employed for the purposes of this study will be highlighted in the different sections that follow below.

#### 4.1 GENERAL ASSESSMENT ISSUES

Neuropsychological assessment is a widely recognized method used to evaluate *objective* changes in cognitive function following MTBI that are not detected by routine clinical neurological examinations (Katz, Goldstein, Rudisin, & Bailey, 1993). According to researchers neuropsychological testing is more sensitive to the subtle effects of MTBI than any other diagnostic tool, and of obtaining a more objective evaluation of subjective complaints (Anderson, 1996; Rees, 2003). For severe and moderately severe TBI patients, neuropsychological evaluation is not usually performed in the acute stages, since the patient may still be in a coma or just out of one and may be minimally responsive, disorientated and unable to understand or to complete the different tasks (Lezak et al., 2004). For these patients neuropsychological assessments are usually performed at time intervals such as three or six month's post-injury as well as several years after the injury, whereas MTBI patients can be

assessed within days of injury, as well as at time intervals of weeks, months and years post-injury (Lezak et al., 2004).

#### **4.1.1 Test characteristics and assessment of cognitive domains**

Neuropsychological assessment involves the identification and measurement of psychological (cognitive and behavioural) deficits but is also concerned with the identification and description of preserved functions (Lezak et al., 2004). Neuropsychological tests occur individually or in test batteries in order to look for consistencies in symptoms as exhibited in variations among different test scores (Barr, 2001). They are standardized in their administration and use which means that the procedures, materials and scoring of the tests are fixed so that the same methods can be used by others at different times and places (Barr, 2001). Since the availability of premorbid test data on individuals is very rare, demographically based normative data assists with test interpretation (Lezak et al., 2004). Demographic variables are significantly related to test performance, and therefore most neuropsychological tests are normed according to demographic data such as age, education and gender.

Many researchers recommend the use of gender-based norms when evaluating neuropsychological test performances of both male and female groups, as research has demonstrated differences in performances between these groups, particularly when assessing MTBI in sports (e.g. Barr, 2003; Halpern, 1997; Putukian, Echemendia, & Machin, 2000; Ryan, Atkinson, & Dunham, 2004). Barr (2003) administered neuropsychological tests to a sample of 60 male and 40 female high school athletes. Although there were no differences in neuropsychological test scores between the different sports, significant differences were found between the males and females on testing, where females obtained higher mean scores on tests of processing speed, mental tracking and verbal initiation. Similarly, in a study by Putukian et al. (2000) investigating the effects of heading on cognitive functioning in male and female soccer athletes, there was a significant gender effect on measures of attention and concentration, with the females performing more optimally than the males. Further, Ryan et al. (2004) investigated sport-specific performances on perceptual-motor and verbal fluency tasks. The female athletes displayed faster and more accurate performances on the perceptual-motor tasks and on one verbal fluency task compared with the male athletes. The above studies demonstrate why

separate normative data are necessary for the different gender groups. By understanding the differences and similarities across the genders on various neurocognitive measures group differences in subsequent studies can be determined (Ryan et al., 2004).

Neuropsychological assessment tools for children and adolescents have also been developed within adult practice and have tended to follow adult models closely (Anderson et al., 2001). This means that the existing neuropsychological tools have been adapted and modified for use on child population groups, although research investigating the effects of MTBI amongst children and adolescents is very limited, especially in the sports field. Although there have been many studies investigating the effects of MTBI with respect to neuropsychological sequelae in high school athletes (e.g. Barr, 2003; Collins, Field, et al., 2003; Field et al., 2003; Guskiewicz et al., 2000; Lovell et al., 2003; Moser et al., 2005; Iverson et al., 2006; Stephens et al., 2005; Webbe & Ochs, 2003; Wilberger et al., 1991; Witol & Webbe, 2003), to the author's knowledge there have been no studies investigating these effects in child athletes, other than that of Gaetz et al. (2000), who investigated the cumulative persistent effects of MTBI in junior ice hockey players, although no age range was given for this sample group.

Researchers have identified core cognitive domains in their investigations of MTBI, which have shown to be sensitive to the effects of MTBI. These include information processing speed (e.g. Bohnen, Twijnstra & Jolles, 1992; Gentilini, Nichelli, & Schoenhuber, 1989; Gronwall & Wrightson, 1974, 1975; Mathias et al., 2004; Pellman et al., 2004), reaction time (e.g. Levin, Lippold, et al., 1987; O'Connor & Burns, 2003), memory (e.g. Binder et al., 1997; Frenchman et al., 2005; Hugenholtz, Stuss, Stethem, & Richard, 1988; Leininger, Gramling, Farrel, Kreutzer, & Peck, 1990; Matser et al., 1998, 1999, 2001; Stuss et al., 1985), attention (e.g. Frenchman et al., 2005; King, 1997; Parasuraman, Mutter, & Molloy, 1991; Raskin et al., 1998; Witol & Webbe, 2003; Webbe & Ochs, 2003) and executive functioning (e.g. Frenchman et al., 2005; Shallice & Burgess, 1991). There are also a number of domains that are much less sensitive to the effects of MTBI (e.g. language, simple motor functioning and visuospatial functioning) (Downs & Abwender, 2002; Randolph, 2001; Rutherford, McCrea, & Barr, 2005).

It should be noted that there is great variability between studies with regards to the specification of the domains assessed and the tests employed to evaluate those domains. In many cases

researchers have modified the domains to accommodate the different tests used in their studies. For example, in some studies tests of reaction time have been included with tests of processing speed, under the domain of information processing (O'Connor & Burns, 2003). Other studies have included a wide variety of tests which have normally been used to measure different aspects of a domain but for maximum effect have been included together under one function. For example, in some studies the domain of memory which includes many aspects such as auditory and visual memory, learning and delayed memory, and working memory (Burton, Ryan, Axelrod, Schellenberger, & Richards, 2003; Binder et al., 1997), has incorporated all these different aspects into a single domain (Frenchman et al., 2005; Miller & Rohling, 2001).

Most neuropsychological tests are multi-functional in that they rely on a number of skills for successful task completion (Anderson et al., 2001). For example, the Complex Figure-Copy task is used as a measure of both construction ability (Anderson et al., 2001) and of visual perception (Raskin et al., 1998). Similarly, the Stroop Color Word Interference Test is used as a measure of both executive function (Raskin et al., 1998), and of attention (Bohnen, Twijnstra, et al., 1992; Binder et al., 1997; Frenchman et al., 2005; Putukian et al., 2000). Over the years many neuropsychological tests have been revised in terms of their format, structure and presentation (for example, the Wechsler Adult Intelligence Scale, including its various subtests such as the Digit Symbol Substitution Test). This involves the modification of the test in terms of an increase or reduction in the length or in the number of trials and scoring system, even though the basic premise of the test remains intact across all test versions. The use of different test versions could create potential methodological problems in research when comparisons across studies are made. This is due to the fact that one would expect variations in performances between groups and between different test versions owing to an increase or decrease in the length of a test, for example, even though the basic premise of the test remains consistent across all test versions. However, researchers Shuttleworth-Jordan and Bode (1995a, 1995b) illustrate that such variations due to test length cannot be assumed until subjected formally to research such as in their comparison between the original 68-pair version of the Wechsler Adult Intelligence Scale-Revised (WAIS-R), Digit Symbol Substitution test and a modified shortened version of 43 pairs (see review on Digit Symbol Substitution test, p.78). Here, differences in the length of exposure to the digit-symbol pairs did not produce any significant recall differences on the adjunctive

measure (Digit Symbol Incidental Paired Associate Recall-Immediate) between the groups studied.

Certain paper-and-pencil tests, particularly tests which measure memory and visual-processing speed (e.g. the Digit Symbol Substitution Test, Digit Symbol Incidental Recall, the Trail Making Test and Digit Span test) are sensitive to the effects of practice as a result of repeat testing, with the greatest effects likely to occur between the first and second test administrations (Lezak et al., 2004). This, as discussed above, presents as a problem for researchers evaluating MTBI in athletes and where serial testing over short time intervals is necessary. The problem of practice effects is significant when evaluating memory because the repeat testing of the same test leads to the test material being learnt which enhances the patient's performance on subsequent test administrations. Conversely the absence of any practice effect on tests where the effect is expected is equally significant as this may suggest a deficit in the learning process as a result of an acquired brain injury (Lezak et al., 2004).

Whilst measures of memory and processing speed may be sensitive to practice effects, research reports that these cognitive domains as well as the domains of attention and reaction time have consistently shown to be the most sensitive to the effects of diffuse brain injury (e.g. Erlanger, Kutner, Barth, & Barnes, 1999; Frenchman et al., 2005; Hinton-Bayre et al., 2004; Tromp & Mulder, 1991; Lezak, 1995; Lezak et al., 2004; Matser et al., 1998,1999,2001; Pellman et al., 2004; Raskin et al., 1998; Shuttleworth-Edwards et al., 2004).

#### **4.1.2 Test selection criteria**

Traditional assessment measures take the form of paper-and-pencil tests and only recently have computer-based neuropsychological tests been introduced for assessment purposes, and this has been a particularly strong initiative for the evaluation of MTBI in the sports arena. As previously mentioned computer-based neuropsychological test batteries were developed in order to address some of the limitations and difficulties encountered with paper-and-pencil measures in the sports context, which include the known risks of practice effects as a result of repeat testing over short test intervals and the time and cost involved in assessing large numbers of athletes. Computer-based neuropsychological tests control for these effects by allowing for the randomisation of test

stimuli thereby minimizing practice effects (Lovell & Collins, 2002). Moreover, apart from being efficient, reliable and cost-effective, computerized neuropsychological tests provide more accurate measurement of processes such as reaction time and processing speed in milliseconds. Currently, there are a number of computer-based neuropsychological tests available particularly in the sports management context, which will be discussed in more detail in a subsequent section. Whilst most paper-and pencil measures have been extensively validated and have included test-reliability in their manuals, as well as shown sensitivity and specificity, only few studies have investigated these properties in computerized neurocognitive test batteries (Randolph et al., 2005). This will be reviewed in more detail when evaluating the computerized neurocognitive test batteries currently used in the investigation of MTBI in sport.

The foundation of all forms of scientific measurement, including neuropsychological testing is provided by its reliability and validity. Reliability and validity estimations are related in that both attempt to increase the truthfulness and utility of the measurement process (Franzen, Frerichs & Iverson, 2004). Reliability refers to the consistency of the test scores obtained for an individual after repeat observations or under different testing situations (Barr, 2001), which includes the investigation of practice effects or improvement in test performance as a result of repeat testing (Randolph et al., 2005). Validity refers to how well a test measures what it is supposed to be measuring, which concerns whether the content of the test provides an accurate representation of the symptom in question and how the test in question compares with others evaluating the very same symptom (Barr, 2001), this means that the test needs to be a valid index of cognitive impairment (Franzen et al., 2004). Therefore, premorbid factors such as learning difficulties, previous head injuries and prior psychological conditions need to be controlled for and must be ruled out when evaluating test results as they represent potential factors which are likely to cause fluctuations in test scores (Rees, 2003; Weight, 1998).

The sensitivity and specificity of neuropsychological instruments are also very important concerns. "Sensitivity refers to the probability of correctly detecting abnormal functioning in an impaired individual and specificity refers to the probability of correctly identifying a normal individual or an individual from another clinical population intact with respect to the test under consideration" (Lezak et al., 2004, p. 149). According to Barr and McCrea (2001), one may want to emphasize the sensitivity of the instrument over the specificity in the sports context, in order to identify as many injured participants as possible. Although most neuropsychological tests are

sensitive to cognitive impairment, they may lack specificity in that the impairment they seek to measure may not be related to the brain injury itself but to other factors such as mental illness, depression, alcohol abuse etc. (Macciocchi & Barth, 2004). Thus, the extent to which a test is seen to be not specific to the particular condition it seeks to measure must also be carefully considered in the interpretation of test results.

## **4.2 COGNITIVE EVALUATION OF MTBI IN GENERAL INCLUDING MTBI IN SPORT**

### **4.2.1 Framework for evaluation of cognitive functions**

World renowned neuropsychologist, Dr Muriel Lezak, has reviewed much of the research, theory and assessment techniques pertaining to neuropsychological assessment which has been documented in her various editions of *Neuropsychological Assessment* (Lezak, 1976, 1983, 1995; Lezak et al., 2004). Lezak et al. (2004) conceptualises human behaviour in terms of three systems which are integral to all functioning: 1) *cognition*, which involves how information is managed, 2) *emotionality*, which involves feelings and levels of motivation and 3) *executive functions*, which involves how behaviour is expressed. Although MTBI usually affects all three systems, in neuropsychology the cognitive functions have received more interest partly because they are readily identified and measured, as well as that they feature more prominently in their symptomatology (Lezak et al., 2004). Emotional (personality) variables, on the other hand, have not shown the levels of reliability nor predictability that cognitive functions have, and impairments in efficiency and executive functions are usually evaluated in terms of their effects on cognitive activities or personality characteristics instead of being investigated independently (Anderson et al., 2001; Lezak et al., 2004).

Thus, broadly the notion of cognition refers to the management of information (rather than its emotional or behavioural expression), and more specifically can be seen to refer to the brain's ability to analyse, store, retrieve and manipulate information in order to solve problems (Tromp & Mulder, 1991). Mild traumatic brain injury can produce distinct deficits in cognitive functioning, causing impairments in all these aspects of information processing (Tromp & Mulder, 1991). There are four basic classes of cognitive functions as conceptualised by Lezak

(1995), as follows: 1) *receptive functions* which involve the ability to select, acquire, classify and integrate information; 2) *memory and learning* involving the retrieval and storage of information; 3) *thinking* which deals with the mental organization and reorganization of information and 4) *expressive functions* which refer to the means in which information is communicated or acted upon (Lezak, 1995). Whilst it is easier to conceptualize each class as involving an independent or separate function, one should be aware that they consist of many functions which work either separately or interact with the other classes. Lezak (1995) classified attention variables as *mental activity variables* placed separately from the other four classes because they underlie and maintain the activity of these functions and therefore will be discussed first. Together these classes will provide a framework within which to review the tests used specifically for MTBI research. Some tests will be reviewed separately whilst others will be reviewed collectively under one subcategory.

#### **4.2.1.1 Mental activity variables**

It has been suggested that the main cause of mental slowness after MTBI is as a result of the delayed access to stored knowledge (Tromp & Mulder, 1991). Because of the continuous interplay between cognitive processes and stored knowledge, slowness in the retrieval process will manifest itself in every task (Stillings et al., 1987). Deficits outside the memory system such as attention, concentration, information processing speed and self-monitoring can affect memory performance (Ganor-Stern, Seamon, & Carrasco, 1998). It is for this reason that any assessment of memory must be preceded by an assessment of attention (Lezak et al., 2004).

Mental activity variables underlie and concern the efficiency of mental processes and are classified as (i) *level of consciousness*, (ii) *attention* and (iii) *activity rate or processing speed* (Lezak et al., 2004).

(i) *Consciousness* refers to the state of being aware and to some extent also involves orientation as having a sense of knowledge about one's self and surroundings (Adams et al., 1996). Transient loss of orientation is a common result of global dysfunction of brain function (High, Levin, & Gary, 1990) and so is the level of consciousness which ranges on a continuum from drowsiness to coma (Lezak et al., 2004). Consciousness should not be confused with attention, and arousal, although the inter-connection between the terms cannot be ignored. In some cases

loss of consciousness may accompany a MTBI, although as discussed, this period is usually brief and short-lived (< 30 to 60 minutes) (International Classification of Diseases, 2006). All neuropsychological testing is performed on patients when they are fully conscious.

(ii) According to researchers measures of *attention* may be the most sensitive indicators of deficit following MTBI (Binder et al., 1997; Frenchman et al., 2005; Raskin et al., 1998). Arousal and attention play important roles in most cognitive tasks, since it is the combination of the two functions that enables the registration, awareness and cognitive processing of one's environment. Arousal concerns both the wakefulness of the individual as well as the preparedness of the individual to respond to a sudden stimulus (Adams et al., 1996). Marked changes in arousal are common after MTBI and researchers have noted that the disruption of arousal mechanisms is a primary contributor to the changes commonly observed in reaction times, information processing speed, and the different aspects of attention in MTBI patients (Gronwall, 1987). Attention involves being able to process one's environment efficiently and can be divided into four types: a) sustained attention, b) selective attention, c) divided attention and d) attention control.

a) Sustained attention involves maintaining concentration over time and the term vigilance has commonly been used to describe this (Parasuraman et al., 1991). The *Digits Forward* test (Wechsler, 1981) is an example of a measure of sustained attention. It forms part of the Digit Span subtest of the Wechsler Adult Intelligence Scales (WAIS) and is sensitive to deficits in sustained attention and concentration. Digits Forward involves the repetition of a random sequence of numbers ranging from two to nine. According to researchers MTBI patients with mental tracking difficulties are more than likely to correctly recall all the digits of the Digits Forward test but in the wrong order (Lezak et al., 2004). Poor performances on the Digits Forward subtest have been demonstrated in studies investigating the effects of MTBI in patient populations. For example, in a study investigating disturbances in attention in a group of children and adolescents who suffered from TBI of varying degrees of severity, performances on the Digit Forwards subtest were reduced following MTBI (Ewing-Cobbs et al., 1989).

Examples of studies investigating the cognitive effects of MTBI in contact sports who have employed the Digit Forwards test as part of their overall test battery include Barr (2003); Collins, Grindel, et al. (1999); Field et al. (2003); Guskiewicz et al. (1997, 2002); Hatfield et al. (2004);

Heilbronner et al. (1991); Kaste et al. (1982); Levin, Lippold et al. (1987); McLatchie et al. (1987); Matser et al. (1998, 1999); Pellman et al. (2004); Porter (2003); Porter and Fricker (1996); Ravdin et al. (2003); Rutherford et al. (2005); Shuttleworth-Edwards et al. (2004); Stewart et al. (1994). Whilst few studies have demonstrated the Digit Forwards test's sensitivity to the effects of MTBI, with contact sports players performing worse following concussion (e.g. Hatfield et al., 2004; Matser, 1999) most studies have not (e.g. Barr, 2003; Collins, Grindel, et al., 1999; Field et al., 2003; Guskiewicz et al., 1997, 2002; Heilbronner et al., 1991; Matser, 1998; McLatchie et al., 1987; Pellman et al., 2004; Ravdin et al., 2003; Rutherford et al., 2005). The Digits Forwards test has also shown to be vulnerable to practice effects which means that instead of decrements in performance occurring following injury, improvements as a result of practice have taken place over subsequent test trials (e.g. Guskiewicz et al., 1997; Pellman et al., 2004; Putukian et al., 2000).

b) Selective attention concerns the ability to focus on a particular stimulus in the presence of distractors. The *Stroop Color Word Test* (Golden, 1978) is an example of a measure of selective attention. It is both a test of attention, cognitive flexibility and executive function. It consists of many formats, with the number of trials ranging from two to four. Depending on the number of trials, the subject is instructed to read as quickly as possible colour words printed in ink of different colours, and then to name the printed colours on another sheet. Although the Stroop test has shown to be sensitive to subtle deficits in attention following MTBI in some studies (e.g. Bohnen, Twijnstra, et al., 1992) in others, as a single measure, it has not shown to be sensitive when evaluating MTBI independently of other tests (e.g. Rojas & Bennett, 1995).

Examples of studies investigating the cognitive effects of MTBI in contact sport who have employed the Stroop test as part of their overall test battery include Guskiewicz et al. (1997, 2002); Hatfield et al. (2004); Jordan, Matser et al. (1996); Killam et al. (2005); Matser et al. (1998, 1999, 2001); Putukian et al. (2000); Ravdin et al. (2003); Rutherford et al. (2005); Wilberger et al. (1991). The sensitivity of the Stroop test in the evaluation of MTBI in sport has also proved to be inconsistent. Whilst some studies have demonstrated its sensitivity in evaluating the effects of concussion, with contact sports players performing worse following concussion (e.g. Jordan, Matser et al., 1996; Putukian et al., 2000; Wilberger et al., 1991) others

have not (e.g. Guskiewicz et al., 1997, 2002; Hatfield et al., 2004; Killam et al., 2005; Matser et al., 1998, 1999, 2001; Ravdin et al., 2003; Rutherford et al., 2005).

c) Divided attention involves dividing one's attention between salient stimuli. *The Paced Auditory Serial Addition Test (PASAT)* (Gronwall & Wrightson, 1981) is an example of a measure of divided attention. It is a task of both attention and working memory. It requires that the patient add 60 pairs of randomized digits so that each digit is added to the digit immediately preceding it (Lezak et al., 2004). Researchers Gronwall and Wrightson (1981) found that MTBI patients showed signs of reduced efficiency when tested on the PASAT but that performances returned to normal within one month post-injury. In both Binder et al. (1997) and Frenchman et al.'s (2005) meta analytical reviews of studies investigating MTBI in general, it was evident that the PASAT has been consistently used and has shown to be a very sensitive measure of attention.

Consistent with Binder et al. (1997) and Frenchman et al.'s (2005) evaluation many studies investigating the cognitive effects of MTBI in contact sport have employed the PASAT as part of their overall test battery. Examples of these studies include, Abreau et al. (1990); Barth et al. (1989); Brooks, Kupsik, et al. (1987); Butler et al. (1993); Downs & Abwender (2002); Ferace et al. (2003); Jordan, Matser, et al. (1996); Levin, Lippold, et al. (1987); Macciocchi et al. (1996, 2001); Matser et al. (1998, 1999, 2001); McLatchie et al. (1987); Webbe & Ochs (2003); Witol & Webbe (2003). Whilst some studies have demonstrated the PASAT's sensitivity to the effects of MTBI with contact sports players performing worse following concussion (e.g. Macciocchi et al., 1996; Webbe & Ochs, 2003; Witol & Webbe, 2003), others have not (e.g. Abreau et al., 1990; Barth et al., 1989; Brooks, Kupsik, et al., 1987; Downs & Abwender, 2002; Ferace et al., 2003; Jordan, Matser et al., 1996; Macciocchi et al., 2001, Matser et al., 1998, 1999, 2001; McLatchie et al., 1987)

d) Attention control involves shifting appropriately between stimuli (Adams et al., 1996). *The Trail Making Test* (Reitan & Wolfson, 1985) is an example of a measure of attention control. Tasks assessing divided attention may be similar to tasks assessing selective attention in that both tasks require the presence of multiple stimuli, although it is often difficult to distinguish a deficit in the ability to simultaneously process multiple stimuli from a reduction in information processing speed or attention capacity (Gronwall, 1987). The Trail-Making Test consists of two

parts, Part A and Part B. Whilst Part A is more a measure of attention and working speed, Part B evaluates cognitive flexibility, processing speed and executive function. Part A involves the connecting of circled numbers that are randomly printed on a page as fast as possible. Part-B follows Part A and involves connecting consecutively numbered and lettered circles on a sheet of paper by alternating between the two sequences. Whilst many MTBI patients will have no difficulty on the Trail Making test (e.g. Lange, Iverson, Zakrzewski, Ethel-King, & Franzen, 2005; Nilson, Barregard, & Backman, 1999), others have demonstrated slower performances when compared with controls and that this slowing increases with levels of injury severity (e.g. Leininger et al., 1990; Raskin et al., 1998). In a study by Cicerone and Azulay (2002) investigating persistent effects following MTBI at 3 to 42 months post-injury, the Trail Making Test was one of the most sensitive measures to the effects of MTBI employed in the study, showing good specificity in identifying impairment. Whilst some researchers have found the Trail Making Test - Part B to be more sensitive to the effects of MTBI, than Part-A, for example, Raskin and his colleagues (1998) found that 28% of their MTBI population performed below normal levels on Part-B compared with 15% below normal levels on Part-A, others have found greater sensitivity with Part-A, for example Cicerone and Azulay (2002) identified attention impairments with a 90% confidence on the Trail Making Test-Part A.

Examples of studies investigating the cognitive effects of MTBI in contact sport who have employed the Trail Making Test as part of their overall test battery include Barr (2003); Barth et al. (1989); Casson et al. (1984); Collins, Grindel, et al. (1999); Farace et al. (2003); Guskiewicz et al. (1997, 2002); Hatfield et al. (2004); Heilbronner et al. (1991); Jordan, Matser et al. (1996); Kaste et al. (1982); Matser et al. (1998, 1999, 2001); Moser et al. (2005); Porter (2003); Porter & Fricker (1996); Levin, Lippold, et al. (1987); Macciocchi et al. (1996, 2001); Makdissi et al. (2001); Putukian et al. (2000); Ravdin et al. (2003); Ross et al. (1987); Rutherford et al. (2005); Shuttleworth-Edwards et al. (2004); Shuttleworth-Jordan et al. (1993); Stewart et al. (1994); Tysvaer & Lochen (1991); Webbe & Ochs (2003); Wilberger et al. (1991); Witol & Webbe (2003).

Many studies have demonstrated the sensitivity of the Trail Making Test to the effects of MTBI with contact sports players performing worse following concussion (e.g. Barr, 2003; Casson et al., 1984; Farace et al., 2003; Field et al., 2003; Guskiewicz et al., 2002; Hatfield et al., 2004;

Kaste et al., 1982; Macciocchi et al., 1996; Matser et al., 2001; Ravdin et al., 2003; Shuttleworth-Jordan et al., 1993; Tysvaer & Lochen, 1991; Witol & Webbe, 2003). Some studies have found Part B to be more sensitive than Part A (e.g. Collins, Grindel, et al., 1999; Rutherford et al., 2005; Shuttleworth-Edwards et al., 2004; Webbe & Ochs, 2003). Examples of studies which have not found the Trail Making Test to be sensitive to the effects of MTBI include 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., 1998,1999; Moser et al., 2005; Porter & Fricker, 1996, 2003; Putukian et al., 2000; Ross et al., 1987; Wilberger et al., 1991). The Trail Making Test is also sensitive to the effects of practice as some studies have shown improvement in test performances over time (e.g. Guskiewicz et al., 1997; Pellman et al., 2004; Shuttleworth-Jordan et al., 1993).

iii) *Activity rate or information processing speed* involves the speed at which mental activities are performed and the speed of motor responses (Lezak et al., 2004). According to Rabbitt, Osman, Moore, & Stollery (2001) some researchers have speculated that information-processing speed (“neurological speed”) is the sole functional determinant of individual differences in efficiency on all cognitive tasks, including intelligence tests, and so must be the “biological basis of intelligence” (Eysenck, 1986). Mental slowing shows up in slowed reaction times and in overall slow performance times in the absence of a specific motor disability. This is the most common characteristic of both aging and diffuse brain damage, typically occurring in closed TBI (Hinton-Bayre, Geffen, & Friis, 2004; Tromp & Mulder, 1991). Thus, reaction times can be inferred from sluggish performances on speeded attention tasks which measure information processing speed (Lezak et al., 2004). An extensive literature review by Frenchman and her colleagues (2005) revealed that measures of speed of processing were also seen to be very sensitive to dysfunction in MTBI patients.

The *Digit Symbol Substitution Test (DSST)* (Wechsler, 1981) is reputed to be one of the most sensitive measures of subtle cognitive impairment and it is therefore a good indicator of diffuse brain injury associated with MTBI (Erlanger, Kutner, Barth, & Barnes, 1999; Frenchman et al., 2005; Lezak, 1995). It is also extremely sensitive to dementia, being one of the first tests to show a cognitive decline in functioning (Lezak et al., 2004). The Digit Symbol Substitution Test is a test of visuo-perceptual processing requiring visual attention, sustained concentration, response

speed, visual shifting and visuomotor coordination (Lezak et al., 2004), although half of the total score on the DSST is influenced by copy speed alone (Joy, Fein, Kaplan, & Freedman, 2000; Kreiner & Ryan, 2001; Storandt, 1976). Owing to its known sensitivity, the DSST was employed as one of the paper-and-pencil measures in the present study.

The Digit Symbol Substitution Test forms part of the Wechsler Intelligence Scale (1955). Variations on this original battery have been developed through the years, (see review in Shuttleworth-Edwards, 2002) including the South African Wechsler Adult Intelligence Scale (SAWAIS, 1969), the Wechsler Adult Intelligence Scale-Revised (WAIS-R; Wechsler, 1981), the Wechsler Adult Intelligence Scale-Revised as a neuropsychological instrument (WAIS-R-NI; Kaplan, Fein, Morris, Delis, 1991) and the Wechsler Adult Intelligence Scale - 3<sup>rd</sup> edition (WAIS-III; Wechsler, 1997). Within these different test batteries the basis of the Digit Symbol Substitution Test remains intact although there are some differences between the various versions in respect of administration time and length of task. For example the SAWAIS, WAIS-R and WAIS-R-NI all have a copy time of 90 seconds in contrast to the WAIS-III, which is a longer test, and has a copy time of 120 seconds. Each test consists of rows (depending on the version it can be two, three, four or more rows) of numbers one to nine which are paired with blank blocks. Above this grid is a key which pairs the numbers, one to nine, with a different symbol. After completing a practice task of seven or eight blocks the subjects are required to complete the task line by line, without omitting any blocks, as quickly as possible up until the point at which the time limit has been reached. The total score is the sum total of the number of correctly completed blocks filled within the given time limit. The test is scored according to standardized norms as set out in the appropriate WAIS manuals.

The Digit Symbol Substitution Test has not only formed part of neurocognitive batteries in their investigation of the effects of MTBI in general, but also it has frequently been used in the investigation of MTBI in contact sports (e.g. Barr, 2003; Brooks, Kupsik, et al., 1987; Casson et al., 2004; Dicker & Maddocks, 1988; Guskiewicz et al., 2002; Heilbronner et al., 1991; Hinton-Bayre et al., 1997, 1999, 2004; Jordan, Matser et al., 1996; Levin, Lippold, et al., 1987; Macciocchi et al., 1996; Maddocks et al., 1995; Maddocks and Saling, 1996; Makdissi et al., 2001; Matser et al., 1998, 1999, 2001; McCrory et al., 2000; Porter, 2003; Porter and Fricker, 1996; Ross et al., 1987; Rutherford et al., 2005; Shuttleworth-Edwards et al., 2004).

Some studies have demonstrated its sensitivity to the effects of MTBI, with contact sports players performing worse following concussion (e.g. Casson et al., 1984; Dicker & Maddocks, 1988; Hinton-Bayre et al., 1999; Macciocchi et al., 1996; Maddocks and Saling, 1996; Master et al., 1999; McCrory et al., 2000; Shuttleworth-Edwards et al., 2004), whilst others have not (e.g. Brooks, Kupsik, et al., 1987; 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; Ross et al., 1987; Rutherford et al., 2005). Although the DSST is very sensitive to subtle cognitive deficits associated with MTBI as discussed above, it too is prone to practice effects. For example, researchers Barr (2003); Hinton-Bayre et al. (1997); Maddocks & Saling (1996); Makdissi et al. (2001), all reported practice effects in their studies with athletes' performances improving over subsequent trials.

#### **4.2.1.2 Receptive functions**

Arousal and attention play an important role in sensory reception and perception, for it is the combination of the two former functions that enables one to actively register and become aware of one's environment (Adams et al, 1996). Sensations are rarely experienced in themselves but rather become perceptions already equipped with previously learned meanings (Lezak et al., 2004). Perception involves the complex continuous processing of sensation as well as their filtering from consciousness and like other cortical functions is extensively distributed throughout the brain making them particularly vulnerable to injury (Lezak et al., 2004). It includes activities such as awareness, recognition, discrimination, patterning and orientation. Although perceptual deficits secondary to TBI are uncommon (Adams et al., 1996), the most common perceptual deficits are those involving visual perception (visual inattention, visual scanning deficits, colour perceptual deficits, visual recognition difficulties or agnosia, visual disorganization, and visual interference) (Lezak et al., 2004). Whilst some tests, for example the DSST, also involve a degree of visual perceptual ability in conjunction with speed, there are few tests in MTBI research used to measure visual perception per se. One such test will be reviewed here, the *Rey Complex Figure Test* (Rey, 1941).

The Rey Complex Figure Test is used to measure both perceptual organization and visual memory and is frequently used as part of the neuropsychological assessment of MTBI patients.

The copy task of the Rey Complex Figure, which is used to measure perceptual organization, involves copying the complex figure onto a sheet of paper. In a study by Leninger, Gramling, Farrel, Kreutzer and Peck (1990), MTBI patients achieved an average score significantly below the mean control group score. Similarly, in another study, Raskin and colleagues (1998) found that 15% of their sample of MTBI patients scored below the normal range on the Rey Complex Figure copy task. Although the Rey Complex Figure-Copy task is sensitive to the effects of MTBI, great variability in test performances exist amongst this patient group (Lezak et al., 2004).

Examples of studies investigating the cognitive effects of MTBI in contact sport who have employed the Rey Complex Figure Test as part of their overall test battery include Brooks, Kupsik, et al. (1987); Jordan, Matser, et al. (1996); Matser et al. (1998, 1999, 2001); McLatchie et al. (1987); Porter (2003); Porter and Fricker (1996); Rutherford et al. (2005); Stewart et al. (1994); Webbe and Ochs (2003); Witol and Webbe (2003). Some studies have demonstrated its sensitivity to the effects of MTBI with contact sports players performing worse following concussion (e.g. Jordan, Matser, et al., 1996; Matser et al., 1998, 1999, 2001; McLatchie et al., 1987) whilst others have not (e.g. Brooks, Kupsik, et al., 1987; Rutherford et al., 2005; Webbe and Ochs, 2003; Witol and Webbe, 2003).

#### **4.2.1.3 Memory and Learning**

Memory is defined as the ability to remember; the process by which previous experience influences an individual's current cognitive, emotional or behavioural state and is central to all cognitive functions (Adams et al., 1996). Memory can be broken down into two distinct systems: declarative or explicit memory which deals with facts and events (the kind of information that is available as conscious recollections) and nondeclarative or implicit memory that has been learned and is non-conscious (not requiring access to the original experience or memory content) (Squire & Butters, 1992). Memory problems are the most common complaint amongst MTBI patients (Adams et al., 1996; Frenchman et al., 2005; Lezak et al., 2004; Raskin et al., 1998). The severity of the memory impairment is related to the magnitude of the lesion site and the severity of the brain injury, and can result from injuries from many different parts of the brain (Levin et al., 1989).

Memory deficits amongst MTBI patients take three different forms: 1) patients manifest an inability to recall events experienced during a short period prior to the injury, retrograde amnesia; 2) patients manifest an inability to recall events experienced immediately following the ending of coma, posttraumatic amnesia (PTA) and 3) patients also manifest a disturbance in memory function that persists beyond the latter point (Richardson, 1990). Both the duration of retrograde amnesia and PTA vary with each other and with the severity of injury. Retrograde amnesia is a selective impairment in episodic memory whilst PTA predominantly involves a failure to consolidate new information in long-term memory (Richardson, 1990). Even after experiencing retrograde amnesia and PTA many patients will continue to complain of memory difficulties that persist beyond these amnesic periods. For example, Rimel and colleagues (1981) found that 59% of the MTBI patients in their study complained of impaired memory three months after the injury. Whilst MTBI patients may complain of memory problems post-injury they may not exhibit these problems on formal testing. Imaging studies have provided some explanation for this. According to McAllister et al. (1999), in response to the increasing working memory processing loads, different brain activation patterns are present between MTBI patients and controls, with an increase activation seen in MTBI patients during high processing load tasks. This may explain why MTBI patients one month post-injury complain of memory problems when faced with memory tasks with a high degree of mental challenge, even though the effects are not evident on formal testing.

Because memory deficits are diverse and varied, no one test is adequate to assess the extent of MTBI on memory functioning (Lezak et al., 2004). Although some researchers have pooled both visual and verbal memory tests into one 'memory' domain (Binder et al., 1997; Frenchman et al., 2005), it is more discriminating from a neuropsychological perspective to divide memory into visual memory, verbal memory and working memory, focussing on the tests which have shown to be the most sensitive to the effects of MTBI.

#### (i) Verbal Memory

According to researchers MTBI patients are very sensitive to tests of verbal memory (Dikmen et al., 1986; Leininger et al., 1990, Raskin et al., 1998). Certain tests have proved to be more sensitive than others. Examples of these measures of verbal memory and learning include the

*Rey Auditory-Verbal Learning Test (RAVLT)* (Rey, 1964), the *California Verbal Learning Test (CVLT)* (Delis, Kramer, Kaplan, & Ober, 1987), the *Selective Reminding Procedure* (Buschke & Fuld, 1974), the *Wechsler Memory Scales Revised (WMS-R) Verbal Paired Associates* test, and the *WMS-Logical Memory* test (Wechsler, 1981).

The RAVLT, CVLT and the Selective Reminding tests are all verbal learning tests where the individual has to learn a series of words over a set number of trials. The original WMS Verbal Paired Associates test is also a verbal learning test which consists of ten word pairs, six easy word pairs and four hard word pairs. The WMS-R version consists of eight word pairs, four easy and four difficult. In both versions the word pairs are read three times, with a memory trial following each reading (Lezak et al., 2004). The WMS Logical Memory test consists of two stories which are read by the examiner, stopping after each story for an immediate free recall, and then a delayed recall of approximately 30 minutes (Lezak et al., 2004).

Significant scores on the delayed version of the WMS Logical Memory test differentiated MTBI patients, who had apparently recovered from their injuries, from normal controls (Stuss et al., 1985). In some studies the WMS Logical Memory test has shown to be more sensitive than both the WMS Verbal Paired Associates and verbal memory tasks (Guilmette and Rasile, 1995), whilst in others the associate learning task has shown more sensitivity (Brooker, 1997). In a study by Frenchman et al. (2005), however, MTBI patients performed significantly worse on all measures of verbal memory, than on any other measures, these included the WMS-R Verbal Paired Associates test, the WMS-Logical Memory test and the CVLT.

Examples of studies investigating the cognitive effects of MTBI in contact sport who have employed the WMS Logical Memory and WMS Verbal Paired Associates as part of their overall test battery include Brooks, Kupshik, et al. (1987); Casson et al. (1984); Jordan, Matser, et al., 1996; Kaste et al. (1982); Matser et al. (1998, 1999, 2001); McLatchie et al. (1987); Pettersen & Skelton (2000); Porter (2003); Porter and Fricker (1996); Ross, Casson, Siegel, & Cole (1987); Shuttleworth-Edwards et al. (2004); Wilberger et al. (1991). Some studies have demonstrated the WMS Logical Memory and WMS Verbal Paired Associates sensitivity to the effects of MTBI, with contact sports players performing worse following concussion (e.g. Casson et al., 1984; Jordan, Matser, et al., 1996; Matser et al., 1998, 1999; Petersen & Skelton, 2000; Ross et al.,

1987; Shuttleworth-Edwards et al., 2004) whilst others have not (e.g. Brooks, Kupsik, et al., 1987; Kaste et al., 1982; Matser et al., 2001; McLatchie et al., 1987; Wilberger et al., 1991).

(ii) Visual Memory

a) The *Complex Figure-Recall task* (Rey, 1941; Osterrieth, 1944), b) the *WMS-Visual Reproduction task* (Wechsler, 1945) and c) the *Digit Symbol Paired Associate Incidental Recall* (Kaplan, Fein, Morris, & Delis, 1991) have all shown to be sensitive to the effects of MTBI.

a) The Complex Figure-Recall task follows immediately after the copy trial, or on delay or both where the subject has to recall the complex figure from memory (Lezak et al., 2004). Researchers have shown that MTBI patients experience some difficulty on the recall trials of the Complex Figure- recall task. In a study by Leninger et al. (1990), MTBI patients showed deficits on the three minute delay recall trial within the first 21 months post-injury. Similarly, Raskin et al. (1998) found that 14% of their MTBI group scored below the normal range on the Complex Figure recall task. Examples of studies investigating the cognitive effects of MTBI in contact sport who have employed the Complex Figure Task as both a measure of perceptual organization and visual memory have been discussed above.

b) With the WMS-Visual Reproduction task the subject is presented with three cards all containing different designs. The cards are shown for a period of five seconds after which they are removed from sight and the subject has to draw what they can remember of the designs (Lezak, et al., 2004). Significantly poorer performances were shown by MTBI patients on the WMS-Visual Reproduction task in the acute phase post-injury, which differentiated themselves from control subjects (Stuss et al., 1985). Examples of studies investigating the cognitive effects of MTBI in contact sport who have employed the WMS-Visual Reproduction task as part of their overall test battery include Matser et al. (1998, 1999, 2001) and Shuttleworth-Edwards et al. (2004). Most studies have demonstrated its sensitivity to the effects of MTBI with contact sports players performing worse following concussion (e.g. Matser et al. 1998; 1999; Shuttleworth-Edwards et al., 2004), whilst other studies have not (e.g. Matser et al., 2001).

c) Given the sensitivity of the DSST, during the past 20 years neuropsychologists have added an adjunctive procedure to the DSST in order to isolate the memory effects, namely the *Digit*

*Symbol Incidental Recall* which is an unwarned task used to measure incidental learning (Shuttleworth-Edwards, 2002). This too is a very sensitive test and like the Digit Symbol Substitution test is often used as a screening test for dementia (Shuttleworth-Edwards, 2002). The Digit Symbol Incidental Recall is more frequently used in sports research than the other two visual memory measures mentioned above (Complex Figure-Recall and the WMS-Visual Reproduction task) because of being an adjunctive task to the very sensitive Digit Symbol Substitution Test often used in sports research. There are two types of Digit Symbol Incidental Recall tasks that have been used in research: *The Digit Symbol Paired Associate Recall* which requires the individual to remember as many of the symbol pairs as they are able and the *Digit Symbol Free Recall* which requires the individual to write down as many of the symbols as they can remember. Researchers have argued that of the two incidental recall tasks, the paired associative recall is more sensitive to subtle cognitive dysfunction and is therefore an obvious test of choice in the investigation of MTBI (Hart, Kwentus, Wade, & Hamer, 1987; Shuttleworth-Jordan & Bode, 1995a & 1995b). The Digit Symbol Incidental Paired Associate Recall- Immediate and Delayed was also used for the purposes of this study.

The Digit Symbol Incidental Paired Associate Recall task follows the 90 second administration of the Digit Symbol Substitution Test (SAWAIS, WAIS-R-NI and WAIS-R Short Form versions) or follows the 120 second administration in respect of the WAIS-III version. In the SAWAIS, WAIS-R-NI and WAIS-R versions following the 90 second administration, after recording the last block completed by the subject, there is a practice requirement where the subject is then asked to complete up to the end of the third row of the grid (i.e. up to 68 pairs, or 67 pairs in the SAWAIS version). This is to ensure that all subjects have had enough exposure to all the digit-symbol pairs (Wechsler, 1997). Following this, the subject is then asked to recall the digit-symbol pairs from memory. In respect of the WAIS-III version the practice requirement is the end of the first four rows (73 pairs), and with the WAIS-R and SAWAIS form to the end of the third row (68 or 67 pairs), whereas in the shortened WAIS-R form, which was developed using the SAWAIS form (Shuttleworth-Jordan & Bode, 1995b), the practice requirement is to the end of the second row (42 pairs on the SAWAIS and 43 pairs on the WAIS-R form). Whilst this is more easily achieved by most subjects within the 90 second time limit in contrast to the longer 67 or 68-pair version, as previously mentioned differences in the length of exposure to pairs between the shortened WAIS-R form and the longer SAWAIS and WAIS-R-NI versions

have caused concern as to whether there would be differences between subjects in the number of symbols recalled. In order to evaluate this, researchers Shuttleworth-Jordan & Bode (1995a, 1995b) performed comparisons between the 43-pair shortened WAIS-R version and the 68-pair WAIS-R version on an older population between the ages of 50-90 years. Their findings revealed that no recall differences between the different versions were shown, which meant that regardless of length of exposure to the symbol pairs, on either the shortened or longer Digit Symbol Substitution Test forms, the incidence of recall remained consistent across the groups. Therefore the shorter version appears to be useful in clinical and particularly research settings in that it is efficient and saves time.

Although the Digit Symbol Incidental Paired Associate Recall task, particularly the Delayed Recall task, is very sensitive to subtle cognitive deficits associated with concussion it is also prone to practice effects (Lezak et al., 2004). For example, in a study by DeMonte, Geffen, & Kwapil (2005) examining the test-retest reliability and practice effects of the Rapid Screen of Concussion test and the Digit Symbol Substitution Test on 20 mild traumatic brain injured male patients, practice effects were demonstrated on both the Digit Symbol Substitution Test and on the delayed recall task.

Examples of studies investigating the cognitive effects of MTBI in contact sport who have employed the Digit Symbol Incidental Paired Associate Recall task as part of their overall test battery include Heilbronner, Henry, & Carson-Brewer (1991); Hinton-Bayre et al. (1997); Makdissi et al. (2001); Shuttleworth-Edwards et al. (2004). Some studies have demonstrated its sensitivity to the effects of MTBI with contact sports players performing worse following concussion (Heilbronner et al., 1991; Shuttleworth-Edwards et al., 2004), whilst other studies demonstrated practice effects with contact sports players demonstrating improved performances on subsequent trials postconcussion (e.g. Hinton-Bayre et al., 1997; Makdissi et al., 2001).

### (iii) Working Memory

*The Digits Backward* test forms part of the Digit Span subtest of the Wechsler Intelligence Scale (WIS) test battery and is an example of a test which is sensitive to deficits in mental tracking, cognitive flexibility and working memory, although attentional deficit has also been implicated with this test. Digits Backward involves a sequence of numbers starting from two to eight which

are given in a particular order and on hearing them the subject is requested to repeat them in the reverse order. Studies using the Digits Backwards test in their investigations of MTBI have produced inconsistent results, where some studies have shown particular deficits on this test (Iverson, Lange, & Franzen, 2005) others have shown minimal effects (Raskin et al., 1998).

Examples of studies investigating the cognitive effects of MTBI in contact sports who have employed the Digit Backwards test as part of their overall test battery include Barr (2003); Collins, Grindel, et al. (1999); Field et al. (2003); Guskiewicz et al. (1997, 2002); Hatfield et al. (2004); Heilbronner et al. (1991); Kaste et al. (1982); Levin, Lippold, et al. (1987); McLatchie et al. (1987); Matser et al. (1998, 1999); Porter (2003); Porter and Fricker (1996); Pellman et al. (2004); Ravdin, Henry, & Carson-Brewer (2003); Rutherford et al. (2005); Shuttleworth-Edwards et al. (2004). Whilst few studies have demonstrated the Digit Forwards test's sensitivity to the effects of MTBI with contact sports players performing worse following concussion (e.g. Hatfield et al., 2004; Matser, 1999; Shuttleworth-Jordan et al., 1993; Shuttleworth-Edwards et al., 2004) most have not (e.g. Barr, 2003; Collins, Grindel et al., 1999; Field et al., 2003; Guskiewicz et al., 1997, 2002; Heilbronner et al., 1991; Kaste et al. 1982; Matser, 1998; McLatchie et al. 1987; Pellman et al., 2004; Ravdin et al., 2003; Rutherford et al., 2005).

#### **4.2.1.4 Thinking**

Thinking is regarded as a function of the entire brain that defies localization (Gloning & Hoff, 1969) and includes a complex number of cognitive functions such as computation, reasoning, and judgment, concept formation, abstracting and generalizing, ordering, organizing, planning and problem solving (Lezak et al., 2004). Although most tests of problem solving and concept formation require reasoning for successful task completion, some tests of reasoning are reflected in the WAIS battery and include the subtests of *Comprehension*, *Arithmetic*, *Picture Completion* and *Picture Arrangement* (Wechsler, 1981). Both the *Comprehension* and *Arithmetic* subtests are included in the Verbal Scales whilst the *Picture Arrangement* and *Picture Completion* subtests are included in the Performance Scales. The *Comprehension* and *Arithmetic* subtests are given orally and consist of questions which are presented in order of increasing difficulty. The *Comprehension* subtest consists of some easy questions which involve practical judgement and others which are more difficult involving the meaning of proverbs (Lezak et al., 2004). The

Arithmetic subtest involves problems presented in story form of increasing difficulty. Both the Picture Completion and Picture Arrangement subtests are presented visually on cards in order of increasing difficulty. With the Picture Completion subtest, the subject is presented with a series of familiar pictures with some missing feature. The subject is then instructed to tell the examiner what is missing from the picture. The Picture Arrangement subtest consists of sets of black and white cards depicting cartoon pictures which are scrambled. The subject is then instructed to put the cards into a chronological sequence so that they tell a story that makes sense (Lezak et al., 2004).

The subtests mentioned above have all shown to be relatively insensitive to the effects of MTBI (Raskin et al., 1998), and the Comprehension and Picture Completion subtests in particular are the least likely to show impairment following injury (Lezak et al., 2004; Raskin et al., 1998). This is largely due to the fact that many of these cognitive skills hold in the face of diffuse brain injury, that is, these tests seem to be relatively resistant to brain damage (Reitan and Wolfson, 1997). For these reasons researchers employ these tests for the purposes of providing an estimate of pre-morbid IQ. For example, researchers Shuttleworth-Edwards and colleagues (2004) used both the Comprehension and Picture Completion subtests in their study evaluating the chronic effects of concussion amongst Open and Under 21 Rugby Union athletes in South Africa, in order to establish a pre-morbid estimate of functioning in the groups.

#### **4.2.1.5 Expressive functions**

These include functions such as speaking, drawing, writing, manipulating, physical gestures, facial expressions or movements where mental activity is inferred from them (Lezak et al., 2004). Apraxias, constructional disorders and aphasias are included in this group.

Apraxias involve the impairment of learned voluntary acts despite having adequate strength and full muscle use (Lezak et al., 2004). Deficits occur as a result of disrupted pathways that connect the processing of information with centres for motor planning or when there has been a breakdown in motor programming and executive functioning integral to the performance of complex learned acts (De Renzi, Faglioni & Sorgato, 1982). Constructional disorders are often seen as apraxias but are rather disturbances in formulative activities such as assembling, building and drawing and frequently appear with defects of spatial perception (Benton, 1969). Classical

syndromes of aphasia occur in about 2% of closed head injured patients whilst in penetrating head injuries or patients with mass lesions this rate is higher (Levin et al., 1982). Despite the rarity of these syndromes varying degrees of less pervasive language disturbance are common after MTBI (Levin et al., 1982). The pattern of deficits vary according to the areas of the brain that are injured but the most commonly affected areas are language comprehension, naming, word-finding and word-generation. *The Controlled Oral Word Association Test (COWAT)* (Benton & Hamsher, 1978) is a test of verbal fluency. It consists of three one-minute word-naming trials, for example C-F-L or F-A-S, selected on the basis of the frequency of English words beginning with these letters. MTBI patients are often associated with poor performances on verbal fluency (Raskin & Rearick, 1996). In a study by Raskin and colleagues (1998), 17% of the MTBI population studied showed deficits on the COWAT.

Examples of studies investigating the cognitive effects of MTBI in contact sports that have employed the COWAT test as part of their overall test battery include Barr (2003); Collins, Grindel, et al. (1999); Field et al. (2003); Guskiewicz et al., (2002); Ravdin et al. (2003); Levin, Lippold, et al. (1987); Matser et al. (1998, 1999, 2001); Pellman et al. (2004); Rutherford et al. (2005). Whilst a few studies have demonstrated the COWAT's sensitivity to the effects of MTBI with contact sports players performing worse postconcussion (e.g Levin, Lippold, et al. 1987; Ravdin et al., 2003) most studies have not (e.g. Barr, 2003; Collins, Grindel, et al., 1999; Field et al., 2003; Guskiewicz et al., 2002; Matser et al., 1998, 1999, 2001; Pellman et al., 2004; Rutherford et al., 2005).

#### **4.2.1.6 Executive functions**

Stuss (1987) defined executive functioning as the ability "to extract and use information from the posterior parts of the brain systems and to anticipate, select, plan, experiment, modify and act on such information in novel situations" (p.175). This pattern is typically ascribed to injuries of the frontal lobes. Although cognitive deficits usually involve specific functions, deficits in executive functioning tend to show up globally and affect all aspects of behaviour (Lezak et al., 2004). Lezak and her colleagues (2004) conceptualize the executive functions as having five components. Whilst each involve a distinct set of activity-related behaviours, all are necessary for appropriate conduct (Lezak et al., 2004). 1) *Volition* is the capacity to formulate a goal or

intention. 2) *Planning* refers to the organization of the steps needed to achieve a goal or intention. 3) *Purposive action* refers to the programming of the activity, requiring the individual to initiate, maintain, switch and stop sequences of behaviour into a structured and organized manner. 4) *Self-regulation* requires that the individual be perceptually and cognitively flexible in response to the demands of a changing environment. 5) *Effective performance* depends on the ability of the individual to effectively monitor and self-regulate their actions in response to their environment (Lezak et al., 2004).

Deficits in executive function exhibit themselves emotionally in the form of a lack of self-control, emotional lability, impulsivity, irritability and rigidity, and have been described as typical personality changes (Adams et al., 1996). Other deficits in the form of erratic performances, poor mental shifting and the ability to plan and organize actions have also been shown (Shallice & Burgess, 1991). As previously mentioned, impairments in executive functions are usually evaluated in terms of their effects on cognitive activities instead of being investigated independently and more often the examiner must rely on their observations of these patients (Anderson et al., 2001; Lezak et al., 2004). Many tests mentioned above, sensitive to MTBI are sensitive to deficits in executive functioning. For example, in a study by Brooks, Fos, Greve and Hammond (1999) investigating the nature of functional deficit after MTBI, tests specifically sensitive to executive functioning (e.g. Trail Making Test, COWAT, and PASAT) differentiated MTBI patients from controls. Whilst the efficiency of executive functions is mediated by lower order functions, in order to measure executive functioning, it is essential to evaluate it in the context of all other functions (Anderson et al., 2001).

### **4.3 COGNITIVE EVALUATION AND MANAGEMENT OF MTBI IN SPORT**

In addition to the paper-and-pencil measures used in the evaluation of MTBI in sport, the use of computerized neurocognitive assessment batteries have gained in popularity in the last few years. At both International Symposia on Concussion in Sport hosted in Vienna in 2001 (Aubry et al., 2002) and Prague in 2004 (McCrory, Johnston, et al., 2005), the Concussion in Sport Group concurred that neuropsychological evaluation should play an integral part in the overall management and treatment of athletes who have sustained concussions in sport (Aubry et al.,

2002; McCrory, Johnston, et al., 2005). The Concussion in Sport Group also approved the use of computerized neuropsychological test batteries over the paper-and pencil measures. Their support was founded in that computerized neuropsychological test batteries were superior in that they are able to utilize multiple test forms thereby eliminating practice effects and were also able to be administered in large groups at one time (Aubry et al., 2002).

There are three levels of neuropsychological evaluation used in the management of sports-related concussion, these will be described below followed by a review of the various computerized neuropsychological test batteries used to evaluate MTBI in sport.

### **4.3.1 Levels of neuropsychological evaluation of MTBI in sport**

#### **4.3.1.1 The sideline examination**

Current guidelines for the management of concussion rely on the assessment of cognitive status immediately post-injury, which will determine whether an athlete returns to play in the game. The most widely used sideline assessment is the Standardized Assessment of Concussion (SAC) (McCrea, Kelly, Kluge, Ackley, & Randolph, 1997). It was developed in response to the recommendations of the American Academy of Neurology Practice Parameter and the Colorado Guidelines and is a brief scale constructed for the purpose of detecting and quantifying the acute neurocognitive effects of concussion. It consists of a 30-point scale and measures include orientation, immediate memory, concentration and delayed recall where lower scores indicate more severe cognitive impairment (McCrea et al., 1997). The SAC takes approximately five minutes to administer and was designed for use by the athletic trainer, coaches and sideline medical personnel. It is not intended as a substitute for formal neurological or neuropsychological evaluation of the athlete however, but has demonstrated both reliability and validity in detecting changes in mental status following concussion, and shows sensitivity and specificity comparable with neuropsychological testing up to three days post-injury (Barr, & McCrea, 2001; McCrea et al., 1997; McCrea et al., 2003).

In a study investigating the clinical utility of the SAC, 568 athletes were administered the SAC prior to the athletic season. Thirty-three of these players experienced concussions and underwent additional testing 48 hours post-injury. The results showed that the concussed athletes scored

significantly lower than non-concussed controls on all SAC measures and significantly below their own pre-injury baseline measures (McCrea et al., 1998). Sensitivity and specificity of the SAC has been demonstrated in a study which determined the immediate effects of concussion in high school and college athletes (Barr & McCrea, 2001). The results indicated a decline of one point on the SAC at retesting which classified injured and non-injured athletes, with a sensitivity of 94% and a specificity of 76% (Barr, & McCrea, 2001). Although the SAC continues to be used extensively throughout the sports field and has three equivalent forms (McCrea, 2001) it has been shown to be vulnerable to both practice effects and ceiling effects which arise when a task is insufficiently challenging (Bender, Barth, & Irby, 2004).

Recently at the Second International Symposium on Concussion in Sport hosted in Prague (McCrory, Johnston, et al., 2005) the Sport Concussion Assessment Tool (SCAT) was developed through a collaborative process and with the intent that it could be used for patient education as well as for the physician's assessment of concussion (McCrory, Johnston, et al., 2005). The SCAT was developed by combining existing concussion management tools into one standardized tool, which was evaluated for content (criterion) validity, which refers to the extent to which the components of the test reflects the particular purpose for which it is being used (Rust & Golombok, 1989), on the basis of scientific literature (McCrory, Johnston, et al., 2005). The measures used included: (i) the Sideline Evaluation for Concussion-Colorado Head Injury Foundation, (ii) the Management of Concussion Sports Palm Card-American Academy of Neurology & Brain Injury Association, (iii) the Standardized Assessment of Concussion (SAC), (iv) the Sideline Concussion Check-UPMC, Thinksafe, Sports Medicine New Zealand Inc and the Brain Injury Association, (v) the McGill Abbreviated Concussion Evaluation (ACE), (vi) the National Hockey League Physician Evaluation Form, (vii) the UK Jockey Club Assessment of Concussion and (viii) the Maddocks Questionnaire. In line with the SAC, the SCAT consists of a 24-symptom checklist and measures of orientation, immediate memory, concentration and delayed recall where lower scores indicate more severe cognitive impairment (McCrory, Johnston, et al., 2005). Although the authors evaluated the SCAT for face and content validity on the basis of scientific literature and clinical experience (McCrory, Johnston, et al., 2005), studies investigating the clinical utility as well as limitations of the SCAT have yet to be published.

#### **4.3.1.2 Baseline neuropsychological examination**

Individualized evaluation in the form of pre and post-season neuropsychological evaluation in conjunction with individualized medical management has become the “cornerstone” in safe return to play following concussive injury (Collins et al., 2004). According to researchers the baseline battery is arguably the most important assessment tool in the decision-making process in managing sports-related concussion (Randolph, 2001). Pre-season baseline evaluation of the athletes is recommended if neuropsychological testing is going to be used. Baseline testing enables the neuropsychologist to make informed decisions about the presence or absence of potentially subtle changes in cognitive functioning over time by using the athlete’s pre-injury functioning as a starting point (Barr, 2001). Thus, without the benefit of knowing how an individual performs prior to a concussion it is difficult to determine whether testing deficits are due to the effects of the concussion or to other factors which have nothing to do with the injury (Wojtys et al., 1999). This is important because each individual varies considerably with regards to their level of performance on tests of memory, attention, concentration, mental processing speed and motor speed. Moreover, some athletes could be performing badly as a result of pre-injury learning disabilities or as a result of the effects of a previous concussion (Wojtys et al., 1999). Baseline testing also minimizes many of the cultural and other issues associated with testing. It allows for the control of many confounding variables and provides intra-individual comparisons (Echemendia, 2004). Research suggests that younger athletes may take longer to recover from concussion than older athletes and whilst they are maturing at faster rates than their older counterparts, more frequent updates of baseline testing will be required (Guskiewicz et al., 2004).

Prior to computerized neurocognitive tests, many paper-and-pencil tests, mentioned above, sensitive to the effects of MTBI (e.g. PASAT, Digit Symbol Substitution Test, Stroop test, Trail Making Test, WMS Logical Passages and Visual Reproduction) were and are still used for the purpose of baseline testing. Researchers Macciocchi et al. (1996) and Lovell & Collins (1998), used paper-and-pencil tests in the first large-scale sports studies and subsequently others followed adopting baseline neuropsychological testing in their management programmes. The baseline examination typically took 20-30 minutes and the tests used targeted those domains most sensitive to concussion (e.g. memory, attention, and speed and flexibility of cognitive processing). Although the paper-and-pencil measures provided information about the

neurocognitive functioning of athletes, no uniform scaling or composite battery scores were created for these paper-and-pencil measures which made up the baseline test batteries (Randolph et al., 2005). In response to these and other problems this led to the development of the computerized neurocognitive test batteries which will be described in more detail in the next section. A self-reported symptom checklist is also typically used in the pre-injury assessment and is among the more obvious and recognizable ways to assess the effects of concussion (Guskiewicz et al., 2004). Examples of postconcussive inventories used in the evaluation of MTBI in sport will be discussed in the next chapter.

#### **4.3.1.3 The postconcussion assessment**

The postconcussion evaluation involves a repeat of the baseline examination, although usually different forms of tests are used to prevent practice effects and to ensure for test reliability and validity. A self-reported symptom checklist is also used in the post-injury assessment. Based on current recommendations the athlete should be symptom free and should cognitively be at or above his baseline level for at least a week both at rest and during exertion to be allowed to return-to-play (Guskiewicz et al., 2004). Researchers suggest that neurocognitive data and post-concussive symptoms should be given equal consideration when determining recovery from injury (Collins et al., 2004).

#### **4.3.1.4 The comprehensive neuropsychological examination**

The standard clinical neuropsychological examination takes approximately three to six hours and consists of tests designed to measure deficit or impaired functioning in respect of general intelligence as well as multiple neurocognitive domains such as those described above in the previous section. These would include *attention* (e.g. the Trail Making Test), *information processing speed* (e.g. the Digit Symbol Substitution Test), *receptive functions* (e.g. Rey Complex Figure), *verbal memory and learning* (e.g. Rey Auditory Verbal Learning Test and WMS Logical Memory), *visual memory* (e.g. the Digit Symbol Paired Associate Incidental Recall – Immediate and Delayed), *thinking or reasoning* (e.g. WAIS subtest –Picture Completion), *expressive functions* (e.g. the Controlled Oral Word Association Test), *executive functions* (e.g. the Trail Making Test-Part B), as well as an assessment of psychological and emotional functioning. In most cases of concussion complete recovery takes place within a short

period of time (acute recovery) and so this type of assessment is unwarranted. If however, there is some question of permanent damage, or the persistence of symptoms (chronic effects), or if an athlete has suffered multiple injuries or has not returned to baseline levels and his tests scores are difficult to interpret, then a comprehensive assessment would be used (Randolph, 2001).

#### **4.3.2 Computerized neurocognitive screening tests used in the evaluation of sports concussion**

As previously mentioned, computerized and web-based neurocognitive test batteries were developed in order to address the limitations of traditional paper-and pencil measures and to assist sports clinicians with return-to-play decisions. They constitute a more sophisticated and challenging neurocognitive test procedure than the sideline assessment (e.g. SCAT), and a less comprehensive and more time efficient procedure than the three to six hour comprehensive neuropsychological assessment. The same test criteria in respect of validity, reliability, sensitivity and specificity which apply to paper-and-pencil test measures applies to computerized neurocognitive test batteries. Thus, when considering the validity of a computerized test, one needs to assess how well a conventional neuropsychological measure correlates with the computerized version of that measure. Some advantages of computerized testing previously discussed are as follows: (i) computerized test batteries allow for the evaluation of a large number of athletes with minimal labour required; (ii) the data received can be easily stored and accessed at a later date; (iii) it offers a more accurate measurement of processes such as reaction time and processing speed in milliseconds can be made which will increase its validity; (iv) it allows for the randomisation of test stimuli that could help improve reliability across multiple administration periods, thereby minimizing practice effects and lastly v) computer-based testing allows for the rapid production of clinical information into an easy-to-read computer-generated clinical report (Lovell & Collins, 2002).

There are a number of computer-based neuropsychological tests available for use in the sports arena these include CogSport (CogState Ltd, Melbourne), Headminder or Concussion Resolution Index (Headminder Inc., New York), ANAM (Automated Neuropsychological Assessment Metric) (developed by the US Department of Defense) and ImPACT (Immediate Postconcussion Assessment and Cognitive Testing) (ImPACT Inc., Pittsburgh, PA), which was used in the

present study. Most of these programmes were modelled on traditional neuropsychological tests and are designed to measure the cognitive domains sensitive to the effects of MTBI, for example attention, reaction time, working speed, speed of information processing and memory (Podell, 2004).

#### **4.3.2.1 CogSport**

CogSport represents one test from a group of computerized neurocognitive tests developed by the company CogStage Ltd. CogSport is a web-based programme designed to evaluate cognitive changes following MTBI (Collie et al., 2003). The programme is not modelled on traditional neuropsychological tests but rather makes use of playing cards as a visual stimulus to evaluate these changes. The programme consists of seven subtasks (Simple Reaction Time, Choice Reaction time, Congruent reaction time, Monitoring, One-back, Matching and Learning) which measure cognitive functions including motor function, decision making, simple and divided attention, complex attention, working memory, learning and memory (Collie et al., 2003). The programme takes approximately 15 to 20 minutes to complete, consists of multiple test forms and stores data for comparison (Podell, 2004).

Few studies have investigated the reliability and validity of CogSport. The test-retest reliability over short time periods of one hour and one week ( $r = .31$  to  $0.82$ ) has been demonstrated in studies (Collie et al., 2003; Straume-Naesheim, Andersen, Dvorak, & Bahr, 2005), as well as the validity, where positive correlations with two independent measures of processing speed (Trail Making Test,  $r = 0.23$  to  $0.44$  and Digit Symbol Substitution Test,  $r = 0.42$  to  $0.86$ ) were made (Collie et al., 2003). Although no controlled studies have demonstrated the sensitivity of CogSport to the effects of concussion (Randolph et al., 2005), one study has assessed the utility of one component (Simple Reaction Time) of the test battery (Makdissi et al., 2001). In a study by Makdissi et al. (2001), baseline assessments were conducted on two occasions on 240 athletes using the CogSport battery as well as two paper-and-pencil tests (Digit Symbol Substitution Test and the Trail Making Test). The results showed greater sensitivity to concussion obtained from one simple reaction time test from the CogSport battery but no changes on the paper-and-pencil measures were evident (Makdissi et al., 2001).

Limitations of the CogSport programme include the lack of a postconcussion symptom checklist and that there has been some evidence of practice effects amongst athletes where testing has taken place over brief intervals which questions its reliability (Collie et al., 2003; Straume-Naesheim et al., 2005).

#### **4.3.2.2 Concussion Resolution Index (Headminder)**

The Concussion Resolution Index is a web-based computerized neurocognitive programme designed to evaluate the resolution of symptoms following concussion (Erlanger et al., 2003, Podell, 2004). The test is modelled on traditional paper-and-pencil neuropsychological tests and consists of six subtests which measure the cognitive domains of reaction time, visual recognition, and speed of information processing. Three indexes, Simple Reaction Time (SRT), Complex Reaction Time (CRT), and Processing Speed (PS) are derived from the six subtests. The test takes approximately 20 to 25 minutes to complete, consists of multiple test forms and data can be stored for test comparisons (Podell, 2004).

Researchers have reported correlations between the Concussion Resolution Index scores and traditional paper-and-pencil measures (Symbol Digit Modality Test, Grooved Pegboard, WAIS-III Symbol Search subtest and Trail Making Test-Part A) (Erlanger et al., 2001). The CRI has shown to have good test-retest reliability (Processing Speed,  $r = 0.82$ ; Simple Reaction Time,  $r = 0.70$  and Complex Reaction Time,  $r = 0.68$ ) over a short interval of two weeks in a large sample of athletes ( $n = 175$ ) aged 13-35, where it was found to be a reliable and valid method for determining changes in psychomotor speed and speed of information processing following sports concussion in comparison to the more traditional methods, although practice effects were reported between test intervals on the Processing Speed Index (Erlanger et al., 2003). The developers also demonstrated concurrent validity (the correlation of the test battery with other constructs measuring the same thing) for the CRI with other paper-and-pencil measures (Digit Span, Digit Symbol, Symbol Search, Symbol Digit Modalities Test, Trail Making Test-Parts A and B, Grooved Pegboard, Stroop Colour Word Test), with the Processing Speed Index having the highest correlation with Symbol Digit Modalities Test, Grooved Pegboard, Symbol Search, and the Stroop Colour Word Test (Erlanger et al., 2003). Although there are no studies investigating the sensitivity of the CRI to the effects of concussion, current data suggests that

components of the programme may be sensitive to detecting impairment in some athletes, moreover the findings in the above validation study suggest that the CRI is sensitive to the effects of MTBI (Erlanger et al., 2003). Limitations of the programme include the limited number of cognitive domains that it evaluates (reaction time and processing speed) and although some neurophysiological symptoms are evaluated it does not include a postconcussion checklist that evaluates other symptoms specific to concussion, for example, cognitive symptoms (e.g. 'difficulty remembering') or psychological/emotional symptoms (e.g. 'feeling emotional').

#### **4.3.2.3 ANAM (Automated Neuropsychological Assessment Metric)**

ANAM was originally designed for clinical and research purposes as an assessment tool that could be used when repeated measures were required (Reeves et al., 1989). It has traditionally been used to assess the effects of pharmacological treatments on cognitive functioning and only more recently has it been used to evaluate concussion (Bleiberg, Garmoe, Cederquist, Reeves, & Lux, 1993; Bleiberg, Garmoe, Halpern, Reeves, & Nadler, 1997). The programme consists of the following five subtests, Simple Reaction Time, Sternberg Memory, Math Processing, Spatial Processing and Matching to Sample, which assess the cognitive domains of attention and concentration, visual memory, working memory and visuo-spatial processing (Reeves et al., 1989). The test takes approximately ten minutes to complete and consists of a large pool of test items which using pseudorandomization permits each test multiple test forms, thereby allowing for repeat testing over extended periods (Podell, 2004).

Although there is currently no published data on measures of reliability for ANAM (Randolph et al., 2005), its developers have demonstrated good construct validity with other paper-and-pencil measures such as the PASAT, the Trail Making Test, Stroop Colour Word test and the Hopkins Verbal Learning Test (Bleiberg, Kane, Reeves, Garmoe, & Halpern, 2000). Only one published study so far has measured the sensitivity of ANAM to the effects of MTBI (Bleiberg et al., 2004), using boxers who sustained concussions. All participants were measured at 0 to 23 hours, one to two days, three to seven days and at 8 to 14 days post-injury. One subtest (Spatial Processing) yielded a significant group difference between 0 to 23 hours and one to two days post-injury, and another subtest (Mathematical Processing) a significant difference on the 0 to 23 hours test interval. In addition to the fact that ANAM's sensitivity to concussion has not been

fully demonstrated, and that the programme itself has not been designed for use in sports concussion, further limitations of the programme include the lack of a symptom checklist and that the programme does not generate a report nor display earlier data on individual's previously evaluated (Podell, 2004).

#### **4.3.2.4 ImPACT (Immediate Postconcussion Assessment and Cognitive Testing)**

The ImPACT programme was developed out of a research context in order to evaluate cognitive outcome following concussion (Collins, Lovell, et al., 1999). ImPACT was designed to assess multiple aspects of neuropsychological functioning, including attention span, sustained and selective attention, reaction time, and both verbal and visual dimensions of memory (Maroon et al., 2000). Due to the time and costs involved in the use of traditional neuropsychological testing with athletes, the ImPACT computerized programme presented as a fast and efficient way to assess large groups of individuals within a short period of time.

Currently there are three versions available of the ImPACT test. The 4<sup>th</sup> version is to be released in 2006 as well as the ImPACT web-based version which allows accessibility to the test via any internet linked computer. The first version ImPACT 1.0 was developed in 1999 and has been used in many studies relating to concussion outcome (e.g. Collins, Iverson, et al., 2003; Iverson, Lovell, Collins, & Norwig, 2002; Lovell et al., 2003). Although the basic neurocognitive battery has remained the same in all versions of ImPACT, additional refinements have been made with the development of newer versions. For example, the visual memory composite (design memory) was an addition to Version 2.0 showing refinement over version 1.0. In addition, one of the working memory tasks (X's and O's) was expanded and modified in version 2.0, making it more difficult than in Version 1.0. Version 2.0 also produces two memory composite scores (Verbal Memory and Visual Memory) whilst version 1.0 has only one memory composite score (Iverson, Lovell, & Collins, 2003). Version 3.0 showed additional refinement over Version 2.0 by introducing a Reliability Change Index which assists with the tracking of recovery from concussion by allowing for comparisons between different tests, players and repeated assessments, thus providing a more quantitative basis for return to play decision-making (Iverson, Lovell, Collins, & Norwig, 2002). Percentile scores are also indicated on Version 3.0.

ImPACT is currently available in several languages namely Spanish, French, Italian, Swedish, Czech, German, Japanese and Portuguese.

ImPACT 2.0 was used in this study due to fact that ImPACT 3.0 was only released in 2003 after all testing was completed. Version 2.0 consists of six test modules that assess aspects of cognitive functioning relating to attention, memory, reaction time and processing speed and which contribute scores to multiple composite scores. The modules are as follows: (i) Word Memory which measures attention and verbal recognition processes, (ii) Design Memory measuring attention and visual recognition processes, (iii) X's and O's, which measures visual working memory, visual processing speed and visual memory, (iv) Symbol Matching measuring visual processing speed, learning and memory, (v) Colour Match measuring impulse control and response inhibition, and (vi) The Three Letter Module, which measures working memory and visual motor response speed. Aspects from these six modules are isolated and combined to produce five composite scores: 1) Verbal Memory, 2) Visual Memory, 3) Visual Motor Speed, 4) Reaction Time and 5) Impulse Control. In addition to the Neurocognitive Test Battery, the ImPACT programme also consists of a demographic questionnaire and a Postconcussion Symptom Scale consisting of 22 symptoms commonly experienced after concussion (see Methodology for a full description of the different test modules of the ImPACT programme Version 2.0).

#### (i) Reliability of ImPACT

The ImPACT test consists of a near infinite number of random forms thereby minimizing practice effects and has shown good test-retest reliability (Maroon et al, 2002). In a recent study concussed athletes were followed up for one-week post-injury and compared with controls (Lovell et al, 2003). The control group's scores on the memory composite did not increase with multiple testing which demonstrated that the ImPACT memory composite score was not subject to practice effects. Moreover, the concussed athletes performed much more poorly on the Verbal Memory test at 36 hours, four and seven days post-injury compared to their baseline scores. In a study investigating the effects of concussion in a group of 24 high school athletes (Iverson, Lovell, Collins, & Norwig, 2002), the ImPACT programme was administered four times, two to eight days apart. The Memory composite produced test-retest correlation coefficients ranging from 0.66 to 0.85 between the test time sessions one to two, two to three, and three to four.

Across the same assessment comparisons, test-retest correlation coefficients for the Processing Speed composite ranged from 0.75 to 0.88 and for the Reaction Time composite 0.62 to 0.66. Although the Reaction Time composite was highly consistent across all testing intervals, the Memory and Processing Speed composites varied between test intervals, with times one to two being slightly weaker than between intervals two to three, and three to four. Thus, whilst there was some improvement after the first test interval, there was little practice effect shown after subsequent administrations. This is an essential feature of the ImPACT programme because it is designed to be used repeatedly over short time intervals and through the randomisation of the stimuli presentation it allows for a reduction in practice effects (Iverson, Lovell, Collins, & Norwig, 2002).

#### (ii) Sensitivity, Specificity and Validity of ImPACT

In a study investigating the diagnostic utility of ImPACT and the Postconcussion Symptom Scale, high school athletes were examined within 72 hours of sustaining a concussion and compared to non-concussed athletes (Schatz et al., 2005). The results showed that the combined sensitivity of ImPACT and the symptom score (i.e. the probability that a test result will be positive when a concussion is present) was 81.9%, and the specificity (i.e. the probability that a test result will be negative when a concussion is not present) was 89.4%. Thus, the study demonstrated that ImPACT is both sensitive and specific to the assessment of neurocognitive and neurobehavioural sequelae of concussion (Schatz et al., 2006). Similarly, in further studies illustrating the sensitivity of ImPACT, the neurocognitive recovery curve of high school athletes diagnosed with a Grade I concussion was evaluated (Lovell, Collins, Iverson et al., 2004). Athletes with concussions showed a decline in memory functioning one to three days post-injury followed by a return to baseline five to ten days post-injury. In another study, athletes reporting headaches had slower reaction times and poorer memory scores than concussed athletes not reporting headaches (Collins, Iverson et al., 2003). Furthermore, athletes self-reporting symptoms of 'fogginess' at one week post-injury had slower reaction times, reduced processing speed and poorer memory scores than concussed athletes who didn't report fogginess (Iverson et al., 2004b).

Several validity aspects of ImPACT have also been assessed, illustrating its ability to separate injured from non-injured athletes. In a sample of 120 high school and college athletes criterion validity was examined by determining whether the composite scores of ImPACT were sensitive

to the effects of concussion. Concussed athletes reported significantly more symptoms, and performed worse on both Memory and Reaction Time indices (Iverson et al., 2002a). In a sample of 72 amateur athletes seen within 21 days of sustaining a concussion, construct validity was examined by investigating the relationship between a traditional measure, such as the Symbol Digit Modalities test, and ImPACT. Decreased performance on the Symbol Digit Modalities test was significantly correlated with the ImPACT Processing Speed ( $r = 0.70$ ) and Reaction Time Indices ( $r = -0.60$ ) (Iverson, Gaetz, Lovell, & Collins, 2005). Divergent validity, which is demonstrated through a low correlation with a test that measures a different construct (Kerlinger, 1986), was examined through an intercorrelation matrix of composite scores at both pre- and post-season intervals (Iverson, Lovell, & Collins, 2002a). At pre-season a small correlation between the Reaction Time and Processing Speed indices ( $r = -0.35$ ) was observed, whereas at post-season there were correlations between symptoms and Memory ( $r = -0.38$ ), Memory and Reaction Time ( $r = -0.27$ ), Memory and Processing Speed ( $r = .35$ ) and Reaction Time and Processing Speed ( $r = 0.32$ ). These small non-significant correlations indicate that the composite scores do not have much shared variance and hence appear to be measuring different constructs (Iverson et al., 2002a).

A test has convergent validity if it has a high correlation with another test that measures the same construct, whilst discriminate validity is the ability to differentiate the construct from other similar constructs (Kerlinger, 1986). Convergent and discriminate validity of ImPACT was examined by evaluating the relationship between the ImPACT composite scores to the Symbol Digit Modalities test, Trail Making Test- Parts A and B and the Brief Visual Spatial Memory test-Revised (BVMT-R) total and delayed memory scores in a group of 25 concussed high school and college athletes (mean age = 17.4 years). Overall, high correlations between the Visual and Verbal Memory Composites from ImPACT ( $r = 0.75$ ), Trail Making Test Parts-A and B and the Symbol Digits Modalities test ( $r = -0.70$ ) and the total and delayed score from the BVMT-R was demonstrated, illustrating the monotrait-momomethod (Iverson, Franzen, Lovell, & Collins, 2004). The monotrait-heteromethod illustrated medium correlations between the BVMT-R total score and the Verbal and Visual Memory composite scores from ImPACT ( $r$ 's = 0.50). A high correlation was demonstrated between the delayed memory score of the BVMT-R and the two ImPACT memory composite scores ( $r$ 's = 0.85). There was also a medium correlation between ImPACT Processing Speed and the Trail Making test-Parts A ( $r = 0.49$ ) and

B (-0.60), as well as the Symbol Digits Modalities test ( $r=0.068$ ). Therefore the above findings confirm the correlations of the ImPACT composite scores with traditional paper-and-pencil measures, designed to measure specific aspects of neurocognitive functioning (Iverson, Franzen, et al., 2004). Specifically, the construct validation for the ImPACT programme with traditional neuropsychological tests designed to measure processing speed (for example, the Symbol Digit Modalities test), serve to confirm the sensitivity of both measures in evaluating the effects of concussion.

### (iii) Normative scores and classification ranges

Normative data for young adults was based on a sample of 545 adolescents between the ages of 13 and 18 (Iverson et al., 2002b). Individuals were stratified according to age, gender and education related problems or special education placement. Individuals with any reported education problems (e.g. diagnosed with a learning disability, ADHD, attended special classes) were subsequently dropped from the normative sample but included in the 'special education' group. The remaining 341 boys and 83 girls were included in the normative sample group. The normative data were presented by gender as there was a trend for the girls to perform better on the Verbal Memory composite ( $p < .01$ ,  $d = .32$ ) and the Processing Speed composite ( $p < .055$ ,  $d = .24$ ) than the boys. The total sample group was also analysed for age effects. Comparisons revealed significantly higher Processing Speed score for the 16, 17 and 18 year olds compared to the 13 and 14 year olds, therefore a distinction was made between the age groups 13 to 14 years and 16 to 18 years. The sample was then sorted into two groups for age and gender, for those between the ages of 13 to 15 years (the 15 year olds were included in this sample because they did not differ from either the older or younger subjects) and those between the ages of 16 to 18 years. The normative tables consisted of boys aged 13 to 15 years,  $n = 183$  and boys aged 16 to 18 years,  $n = 158$  and for girls aged 13 to 18 years,  $n = 83$  (Iverson et al., 2002b). The natural distribution of these scores within these groups were examined and percentile ranks assigned. The raw scores were not converted to standard scores. The classification ranges for the index scores for regular education high school boys aged 16 to 18 years are presented in Table 4.3.2.4.1.

The ImPACT programme has been used predominantly in the acute assessment of concussion in the contact sport of American football (e.g. Collins, Field, et al., 2003; Field et al., 2003; Iverson et al., 2002; 2004a, 2004b, 2006; Lovell et al., 2003, 2004; McClincy et al., 2006; Mihalik et al.,

2005; Pellman et al., 2006), with relatively fewer studies investigating the chronic or persistent effects of concussion (e.g. Iverson et al., 2002, 2004b, 2006; Lovell et al., 2003, 2004; McClincy et al., 2006).

**Table 4.3.2.4.1**  
**Approximate Classification Ranges for Index Scores: Boys Ages 16-18 years**

	<b>Verbal Memory</b>	<b>Visual Memory</b>	<b>Processing Speed</b>	<b>Rxn Time</b>
<b>Impaired</b>	≤ 68	≤ 51	≤ 26.4	≥ .74
<b>Borderline</b>	69 - 74	52 - 59	26.5 - 29.6	.73 - .64
<b>Low Average</b>	75 - 79	60 - 70	29.7 - 33.6	.63 - .59
<b>Average</b>	80 - 92	71 - 88	33.7 - 42.5	.58 - .50
<b>High Average</b>	93 - 98	89 - 93	42.6 - 47.7	.49 - .47
<b>Superior</b>	99	94 -96	47.8 - 51.1	.46 - .43
<b>Very Superior</b>	100	97 - 100	≥ 51.2	≤ .42

(ImPACT version 2.0 Clinical user's manual)

### 4.3.3 Synthesis of cognitive test evaluation in MTBI in general

From the above review it would appear that the cognitive functions of information processing speed, attention, reaction time and memory seem to be the most sensitive to the effects of diffuse brain injury (Binder et al., 1997; Frenchman et al., 2005). The tests isolated for use in the present study included the paper-and-pencil measures, the Digit Symbol Substitution Test which evaluates processing speed and its adjunctive task, the Digit Symbol Paired Associate Incidental Recall, which evaluates visual memory, as well as the computerized neurocognitive test battery, ImPACT, which evaluates visual and verbal memory, reaction time and processing speed. The paper-and-pencil measures were chosen owing to their frequent use and increased sensitivity to the effects of MTBI in the sports arena (e.g. Casson et al., 1984; Dicker & Maddocks, 1988; Hinton-Bayre, Geffen, Geffen, McFarland, & Friis, 1999; Macciocchi et al., 1996; Maddocks and Saling, 1996; Master et al., 1999; McCrory et al., 2000; Shuttleworth-Edwards et al., 2004). In addition, the ImPACT computerized neurocognitive test battery was employed for the purposes of this research because it appeared to be the most comprehensive and widely used computerized assessment tool in large-scale evaluations of sports MTBI (e.g. Collins, Field, et

al., 2003; Field et al., 2003; Iverson et al., 2002; 2004a, 2004b, 2006; Lovell et al., 2003, 2004; McClincy et al., 2006; Mihalik et al., 2005; Pellman et al., 2006).

Satz (1993) postulates that cognitive deficit would remain undetected (sub-threshold) due to the protector factor of a greater BRC (for example, high IQ, high level of education and large brain size), until such time that a sufficiently *challenging* task presents itself. In line with BRC theory, neurophysiological studies illustrate that following MTBI there is an increase in the glucose metabolic activity rate in the brain (Giza & Hovda, 2004; Haier et al., 1988). As discussed previously, fMRI studies showed larger activation in the brain's of MTBI patients being scanned whilst simultaneously taking sensitive neuropsychological tests due to a reduction in the brain's ability to process information efficiently, thereby placing additional demands (challenges) on the neural networks involved in successful task completion (Lovell & Collins, 2002; McAllister et al., 1999). Thus, cognitive deficit would not be detected were the neuropsychological measures not sufficiently robust to allow the individual to fall below the cognitive threshold level and present with symptoms. From the above review it can be postulated that tests measuring the cognitive domains of processing speed and reaction time, attention and memory are the most sensitive to the effects of MTBI. In light of this, tests isolated for the purposes of the present study that have demonstrate sensitivity to MTBI, (Digit Symbol Substitution Test, Digit Symbol Incidental Paired Associate Recall-Immediate and Delayed and tests of visual motor speed, reaction time, visual and verbal memory in the computerized ImPACT programme) will be those that are likely to provide optimal challenge on the neural networks involved in task completion in a cohort of contact sports players. Accordingly this should cause them to fall below the threshold of symptom presentation and allow for the identification of incipient cognitive deficits should they be present.

## CHAPTER FIVE

### ASSESSMENT OF SELF-REPORTED POSTCONCUSSIVE SYMPTOMS FOLLOWING MILD TRAUMATIC BRAIN INJURY IN GENERAL AND IN SPORTS

The purpose of this chapter is to report specifically on the self-reported postconcussive symptoms (PCS) following MTBI as identified on postconcussion symptom inventories and scales. This chapter begins by reviewing the etiology of PCS which includes the Diagnostic and Statistical Manual for Mental Disorders - 4<sup>th</sup> Edition's (DSM-IV) diagnosis of postconcussional disorder. This is followed by an investigation into postconcussive symptoms in sport which begins by reviewing two inventories, one a paper-and-pencil measure and the other the ImPACT Postconcussion Scale, which forms part of the computerized neurocognitive programme ImPACT. Finally, studies investigating the self-reported PCS as a consequence of MTBI in sport will be reviewed.

#### 5.1 THE ETIOLOGY OF PCS

Whilst most MTBI patients tend to make a favourable recovery, a significant number continue to report symptoms which may persist for months or even years after the injury. These symptoms are collectively known as postconcussive symptoms (PCS) which may present as a single symptom or collectively as a group of symptoms usually following MTBI in both the acute and chronic phases (Evans, 2004). About 50% of all MTBI patients develop PCS (Bazarian & Atabaki, 2000; Mandel, 1989) and, although there is some consistency in patients with the reporting of early symptoms, the number and combination of the symptoms reported varies considerably (Dikmen & Levin, 1993). Full recovery of PCS usually takes place within three months following MTBI (Alves, Colohan, O'Leary, Rimel, & John, 1986; Englander et al., 1992; Evans, 1992, Levin, Lippold, et al., 1987), although researchers state that approximately 10 to 15 % of MTBI patients may continue to suffer from PCS for many months or years post-injury (Davis, 2002; Satz et al., 1999). Where PCS persist this is known as the postconcussion syndrome or persistent postconcussive symptoms (Dikmen & Levin, 1993; Ryan & Warden, 2003).

The term postconcussion syndrome was first used by Strauss and Savitsky in 1934. Patients experiencing persisting symptoms related to individual recovery curves or individual vulnerability, and which last longer than three months are commonly referred to as the 'Miserable Minority' (Ruff, 2005; Ruff, Camenzuli, & Mueller, 1996). Studies indicate that this group accounts for between 20 to 66% of all MTBI patients (Alves et al., 1986; Englander et al., 1992; Jones, Violen, Laban, Schynoll, & Krome, 1992; Rutherford, 1989; Ryan & Warden, 2003; Wrightson & Gronwall, 1989). Owing to the vast number of individuals who sustain MTBIs annually this group would translate into a large group, whose lives may be significantly affected by their symptom presentation (Dikmen & Levin, 1993). Loss of consciousness and PTA are not requirements of the postconcussion syndrome. Although the duration of anterograde amnesia correlates with the extent of cerebral injury and loss of consciousness, it does not necessarily correlate with the development of the postconcussive syndrome (Mandel, 1989).

Whilst in the past many clinicians have considered PCS to be incorporated into one syndrome, i.e. the postconcussion syndrome, more recently researchers have regarded them in the form of distinct symptom clusters (King, 1997). In Levin, Lippold, et al.'s study (1987) factor analysis of PCS revealed three different presentations: (i) those with no or minimal PCS; (ii) mainly cognitive and depression presentations and (iii) primarily somatic symptoms. Others like Bohnen, Twijnstra, et al. (1992) in their study identified two main clusters of PCS: 1) cognitive symptoms, for example headaches, dizziness, fatigue, poor concentration and 2) emotional-negative symptoms, for example depression, emotional lability. Whilst Cicerone and Kalmer (1995) in their analysis of MTBI patients, identified four PCS factors: cognitive, somatic, affective and sensory. Depending on the PCS presentation of their patients, each would fall into four identified clusters: (i) those with minimal symptoms; (ii) primarily cognitive-affective symptoms; (iii) primarily somatic symptoms, and (iv) global symptoms. Of the four clusters, patients falling in the first two had better outcomes than the latter two clusters. S.D. Anderson (1996) attempted to categorize PCS into three domains: 1) Neurocognitive - impaired attention and poor concentration, memory difficulties, reduced mental flexibility, slowed reaction time, speech difficulties, impaired decision-making and mental fatigue; 2) Somatic or Physical - headaches, dizziness, insomnia, loss of appetite, drowsiness, blurred vision, sensitivity to noise, sensitivity to medication or alcohol, clumsiness and postural changes; and 3) Emotional or Neuropsychiatric - depression, emotional lability, anxiety, aggression, apathy, personality

changes and lowered frustration tolerance (S.D. Anderson, 1996). Although there is some evidence for the use of symptom clusters researchers caution not to draw any significant conclusions from them since the clusters identified need to be empirically based and cross-validation studies are necessary to confirm them (King, 1997).

Researchers draw a distinction between early onset PCS, those appearing immediately or the morning after the injury and late onset PCS, those which develop a few weeks later (King, 1997). The early onset symptoms include headaches, dizziness, vomiting, nausea, drowsiness, and blurred vision whilst those developing later include irritability, anxiety, depression, poor memory, poor concentration, sleep disturbance, fatigue and hearing or visual disturbance (King, 1997). Some researchers argue the psychogenic basis for the late onset of symptoms, whilst others propose that some organic symptoms only occur under extreme emotional or cognitive stress (King, 1997), such as entering into stressful work environments after being in a stress-free hospital environment (Rutherford, 1989). Moreover, others still argue that PCS may begin on an organic basis but that psychological factors contribute to their persistence (Levin et al., 1982). The attribution of PCS to organic or psychological factors is very difficult to assess for both the clinician and the patient as it is acknowledged that the operation of psychological factors involves complex interactions between organic and psychogenic mechanisms (Lishman, 1988). This can be seen with symptoms such as dizziness, loss of balance or hearing loss which can have persistent disabling effects but may be the result of cranial nerve damage rather than damage to the brain itself (Dikmen & Levin, 1993). Similarly, headaches may be related to injury to the musculoskeletal system rather than to brain injury (Dikmen & Levin, 1993). The debate on whether chronic symptoms have an organic basis continues today (Sweet et al., 2003).

As previously discussed, a number of variables have been identified as predictive of persistent PCS: (i) Age is a variable which is associated with slower neuropsychological recovery and slower PCS recovery, particularly in individuals over the age of 40 years (Barth et al., 1983; Binder, 1986; Evans, 1992; Ferguson et al., 1999; King, 1997; Ryan & Warden, 2003), (ii) Woman are reported to recover slower from PCS than men and have reported higher incidence rates of PCS as oppose to men (Davis, 2002; Rutherford et al., 1979), (iii) alcohol and substance abuse are both associated with delayed recovery (Lishman, 1988) and (iv) a prior history of MTBI also increases the likelihood of persistent PCS (Carlsson, Svardsudd, & Welin, 1987;

Lishman, 1988; Ryan & Warden, 2003; Sortir, 2001). Other factors which could result in the persistence of symptoms include premorbid psychopathology, compensation claims, which provide a strong motivation for the aggravation of disability (Davis, 2002; Lees-Haley & Brown, 1993), and emotional factors such as coping styles (Alexander, 1995; Bohnen, Jolles, & Twijnstra, 1992; King, 1997; Lishman, 1988; McAllister & Archiniegas, 2002).

### **5.1.1 Diagnostic and Statistical Manual for Mental Disorders (DSM-IV) diagnosis of postconcussional disorder**

The DSM-IV incorporated the first experimental definition for postconcussional disorder (PCD), which includes those patients who present with persisting symptoms for more than three months post-injury (see Table 5.1.1.1). According to the DSM-IV (1994), PCD “is an acquired impairment in cognitive functioning, accompanied by specific neurobehavioural symptoms, that occurs as a consequence of closed head injury of sufficient severity to produce a significant cerebral concussion” (p.760). The DSM-IV criteria in line with the symptom clusters of previous authors, incorporates somatic, cognitive and emotional symptoms, which are outlined in Table 5.1.1.1 (Ruff, 2005).

The difficulty in defining PCD parallels the difficulty in finding a unified definition of MTBI. Ruff (2005) recommends the following additions to the above definition and suggests modifiers which can be used to capture the different types for the DSM-IV diagnosis of PCD (see Table 5.1.1.2). Firstly, it is suggested that the term postconcussion syndrome be replaced with postconcussional disorder because the former term indicates a single uniform syndrome which is not the case. Secondly, PCD can be caused by neuropathology, psychopathology and secondary gain in the form of consciously reduced effort or malingering (“in which the desire for compensation may lead to the prolongation or production of symptoms”, DSM-IV, 1994, p.761), and any combination thereof (Ruff, 2005).

Although the DSM-IV’s recognition of PCD has provided some validation for the presentation of such symptoms which accompany MTBI, the criteria as set out by the DSM-IV has met with criticism. Both the DSM-IV and the modifiers for PCD do not differentiate between PCS and neurocognitive deficits measured objectively on formal testing but rather seem to merge both effects into one diagnosis. Although the neurocognitive deficits and PCS sequelae following

MTBI may present either separately or together, the course and recovery of both are very different and should therefore be viewed as such. For example, whilst some studies have demonstrated that cognitive recovery coincides with PCS resolution (Bohnen, Twijnstra, et al., 1992; Gronwall & Wrightson, 1975; Levin, Lippold, et al., 1987) others have found that PCS continue to persist long after cognitive sequelae have resolved and that no association between neurocognitive status and the persistence of PCS symptoms exists (Ferguson et al., 1999; Macciocchi et al., 1996; MacFlynn, Montgomery, Fenton, & Rutherford, 1984; McLean, Temkin, Dikmen, Wyler, & Gale, 1984; Ruff et al., 1989; Thompson, Sebastianelli and Slobounov, 2005). Furthermore, in MTBI in sport where known underreporting of symptoms occurs, in some cases researchers have reported the resolution of postconcussive symptoms prior to cognitive recovery (Collins, Grindel, et al., 1999; Lovell et al., 2003; McClincy et al., 2006).

**Table 5.1.1.1**  
**Research criteria for Postconcussional Disorder**

- A. A history of head trauma that has caused significant cerebral concussion.
- Note: The manifestations of concussion include loss of consciousness, posttraumatic amnesia, and, less commonly, posttraumatic onset of seizures. The specific method of defining this criterion needs to be established by further research
- B. Evidence from neuropsychological testing or quantified cognitive assessment of difficulty in attention (concentrating, shifting focus of attention, performing simultaneous cognitive tasks) or memory (learning or recalling information).
- C. Three (or more) of the following occur shortly after the trauma and at least three months:
- a. becoming fatigued easily
  - b. disordered sleep
  - c. headache
  - d. vertigo or dizziness
  - e. irritability or aggression on little or no provocation
  - f. anxiety, depression, or affective lability
  - g. changes in personality (e.g., social or sexual inappropriateness)
  - h. apathy or lack of spontaneity
- D. The symptoms in Criteria B. and C. have their onset following head trauma or else represent substantial worsening of pre-existing symptoms.
- E. The disturbance causes significant impairment in social or occupational functioning and represents a decline from a previous level of functioning. In school-age children, the impairment, the impairment may be manifested by a significant worsening in school or academic performance dating from the trauma.
- F. The symptoms do not meet criteria for Dementia Due to Head Trauma and are not better accounted for by another mental disorder (e.g., Amnesic Disorder Due to Head Trauma, Personality change Due to Head Trauma).

Another criticism of the DSM-IV definition is that it fails to include a number of symptoms which have been reported amongst MTBI patients post-injury, such as nausea, tinnitus, impaired coordination and balance, sensitivity to light and sensitivity to noise. In addition, although it includes cognitive symptoms such as deficits in memory, attention and concentration, it excludes other cognitive deficits such as processing speed, executive functioning and speech problems, as well as other factors such as insomnia, fatigue, drug and alcohol abuse (S.D. Anderson, 1996). The elimination of criteria from the above definition could result in misdiagnosing a MTBI patient who is experiencing memory and executive functioning problems post-trauma, as having dementia and not PCD (S.D. Anderson, 1996).

**Table 5.1.1.2**  
**Modifiers for the Postconcussional Disorder (PCD)**

PCD with objective neuropathological features	This classification identifies those patients with a combination of neuropsychological deficits and positive traumatic brain injury-related neuroimaging. Levin et al. (1987a) and Borgaro (2003), refer to these MTBI patients as complicated
PCD with neurocognitive features	Reliably documented on neuropsychological testing, a cluster of cognitive deficits that is consistent with MTBI without significant emotional overlay; lack or absence of positive neuroimaging
PCD with psychopathological features	Persistent emotional sequelae that dominate the clinical picture (e.g. acute stress reaction, depression, panic attacks); neuropsychological deficits disproportionate with duration of loss of consciousness or posttraumatic amnesia and if available neuroimaging is negative, symptoms tend to persist or even worsen over time
PCD with neurocognitive and psychopathological features	A mixture of documented neurocognitive deficits and acquired emotional residua that demonstrate some improvements over time; lack or absence of positive neuroimaging

(Ruff, 2005)

Although PCS represent common features amongst MTBI patients, they may not be specific to MTBI and may reflect the presence of other disorders. For example postconcussive symptoms such as irritability, insomnia, fatigue and feelings of being slowed down suggest depression even when the patient may not support this explicitly (Suhr & Gunstad, 2002). This raises concerns as

to the validity, specificity and sensitivity of self-reporting PCS inventories in screening for symptoms specific to MTBI and not to other pre-morbid conditions such as depression. For these reasons it is important to exclude participants on the basis of any confounding variables for example, any psychiatric or neurological disorder when investigating the sequelae of MTBI. An understanding around the culturally based differences in symptom reporting is also essential in the evaluation of PCS (Ruff, 2005).

## 5.2 POSTCONCUSSIVE SYMPTOMS IN SPORT

Although the presence of postconcussive symptoms are uncontested sequelae following MTBI, there are no studies which have reported on the incidence of postconcussive symptom reporting amongst athletes. The lack of incidence reports may be due to the dearth of studies investigating these effects in MTBI in sport. Arguably most researchers investigating the neuropsychological effects following MTBI have focussed on the cognitive sequelae given that these effects are easily evaluated using objective assessment tools. The assessment of postconcussive symptoms however, is more difficult to validate using self-reporting inventories and particularly because of the known underreporting of symptoms by athletes that perpetuate the sports arena (Lovell, 2002).

Part of the process of diagnosing a concussion comes from the immediate evaluation of these symptoms on the sideline, which ultimately assists with the management of the concussion. At both the International Symposia on Concussion in Sport hosted in Vienna (Aubry et al., 2002) and Prague (McCrory, Johnston, et al., 2005), the Concussion in Sport Group highlighted acute signs and symptoms associated with MTBI which are crucial for the immediate assessment of the injury. These symptoms can be grouped into symptom clusters mentioned previously, consisting of neurocognitive, somatic and emotional or behavioural domains. Neurocognitive symptoms include symptoms such as feeling 'dinged' or 'foggy' or 'dazed', poor concentration, or easily distracted and amnesia. Somatic symptoms include headache, balance problems, nausea, confusion, vomiting, hearing problems, slurred speech, seizures, slowness in answering questions, vacant stare or dizziness and visual problems and emotional symptoms include displays of inappropriate emotion such as laughing or crying, irritability or emotional changes. Whilst the Concussion in Sport Group reports on the acute symptoms for the immediate

management of concussion, there is no mention of the long-term management of the athlete with respect to persistent postconcussive symptoms (Aubry et al., 2002; McCrory, Johnston, et al., 2005).

### **5.2.1 The measurement of postconcussive symptoms in sport**

Most inventories or scales which measure emotional or subjective variables have evolved out of clinical experience, whilst others have been developed with a particular population in mind for specific research purposes (Lezak et al., 2004). There are however, a number of difficulties using PCS scales, inventories or questionnaires in the evaluation of PCS in general. Many inventories or scales are developed without the benefit of psychometric scaling techniques and are not rigorously standardized. The main premise of a scale or inventory used in clinical studies is that it is sufficiently robust to measure symptoms which separate members of the population that they intend to investigate from members of the population at large (Lezak et al., 2004). This is difficult considering that it is not always clear whether the symptoms reported occur pre-morbidly or are attributed to the MTBI itself. Moreover, no indication is given on the inventories or scales as to what extent the symptoms cause disability to the individual concerned (King, 1997). Some researchers have addressed these problems by developing checklists that ask the patient to report the absolute or relative increase or decrease in the symptoms following a TBI (Bohnen, Jolles, et al., 1992). Others ask patients to rate their symptoms according to the extent at which they impede their everyday functioning (Englander et al., 1992). Whilst this may offer some control against pre-morbid symptoms, these tests still need to be validated against a range of clinical and ecological measures (King, 1997).

Generally two types of inventories have been employed in both clinical and research settings, these include symptom checklists (Echemendia et al., 2001; Guskiewicz, Ross, & Marshall, 2001) and symptom scales (Lovell et al., 2003). A symptom checklist consists of a set of concussion-related symptoms and allows the athlete to report on the presence of the symptom by reporting "yes" or "no" (Guskiewicz et al., 2004). A symptom scale on the other hand involves a Likert scale that allows the athlete to rate the severity or frequency of postconcussive symptoms from mild, moderate to severe, on a scale ranging from 0 to 5 or 6. These scores are then summed to form an overall quantitative measure of severity and provide a good measure against

which to track post-injury symptom recovery (Guskiewicz et al., 2004). Obtaining a baseline symptom score is useful in order to establish pre-existing symptoms. Following concussion serial administration of the symptom checklist is recommended in order to track symptom resolution over time (Guskiewicz et al., 2004), although as previously mentioned this is difficult to establish in athletes who underreport symptoms. Researchers Collins and colleagues (2004) suggest that neurocognitive data and postconcussive symptoms should be given equal consideration when determining recovery from injury.

Two postconcussion inventories were employed for the purposes of this research and will be reviewed here. One inventory formed part of the ImPACT computerized neurocognitive battery (ImPACT Postconcussion Scale) and the other, a paper-and-pencil inventory, developed from an inventory used in previous research investigating MTBI in sport in respect of Rugby Union in South Africa (Shuttleworth-Edwards et al., 2004).

#### **5.2.1.1 ImPACT Postconcussion Scale**

The ImPACT Postconcussion Scale (see Table 5.2.1.2.1), which has been widely utilized throughout organized sport (e.g. Aubry et al., 2002; Collins, Field, et al., 2003; Collins, Iverson, et al., 2003; Iverson et al., 2004a, 2004b, 2006; Lovell & Collins, 1998; Lovell et al., 2003; McClincy et al., 2006; Mihalik et al., 2005; Pellman et al., 2006) was employed for the purpose of this study. Although it forms part of the ImPACT programme it is one the few PCS scales that has been validated for use in concussion. The psychometric and clinical properties of this scale will be reviewed here.

The ImPACT Postconcussion Symptom Scale was also developed for the purposes of concussion management by providing a formal method of documenting PCS following concussion (Lovell, 1996) and to assist medical health professionals regarding the resolution of symptoms over time. It consists of 22 items which include a range of cognitive, physical and emotional symptoms, each of which is assigned a numeric value. The scale itself is based on a seven point Likert scale with scores ranging from 0 = not experiencing the symptom to 6 = severely experiencing the symptom. The dependent measure is the total score obtained from the 22-item scale (Iverson, Lovell, & Collins, 2003). Symptoms employed in the scale are specific to the sports context,

with terms such as “fogginess” included based on the recurrent use of the term by athletes (Lovell et al., in press).

**Table 5.2.1.1.1**  
**The ImPACT Postconcussion Scale**

Symptom	None	Mild		Moderate		Severe	
Headache	0	1	2	3	4	5	6
Nausea	0	1	2	3	4	5	6
Vomiting	0	1	2	3	4	5	6
Balance problems	0	1	2	3	4	5	6
Dizziness	0	1	2	3	4	5	6
Fatigue	0	1	2	3	4	5	6
Trouble falling asleep	0	1	2	3	4	5	6
Sleeping more than usual	0	1	2	3	4	5	6
Sleeping less than usual	0	1	2	3	4	5	6
Drowsiness	0	1	2	3	4	5	6
Sensitivity to light	0	1	2	3	4	5	6
Sensitivity to noise	0	1	2	3	4	5	6
Irritability	0	1	2	3	4	5	6
Sadness	0	1	2	3	4	5	6
Nervousness	0	1	2	3	4	5	6
Feeling more emotional	0	1	2	3	4	5	6
Numbness or tingling	0	1	2	3	4	5	6
Feeling slowed down	0	1	2	3	4	5	6
Feeling mentally “foggy”	0	1	2	3	4	5	6
Difficulty concentrating	0	1	2	3	4	5	6
Difficulty remembering	0	1	2	3	4	5	6
Visual problems	0	1	2	3	4	5	6

(Lovell et al., in press)

(i) Sample

The scale was developed in the context of a clinical setting, when in the late 1980’s the scale was adopted by the Pittsburgh Steelers concussion management programme. Normative data was obtained from over a thousand high school and university student athletes. Initially, the total high school and university group employed for the project included a sample of 2304 students (894 high school students and 1,295 university students). This group also included a sample of 115 high school and university students in the acute recovery period post-concussion. This cohort was stratified according to the level of education, gender and learning or special education status.

The “regular education” group consisted of 707 high school students (588 males and 119 females) and 1039 university students (803 males and 236 females). The “special education” sample which included any person who had reported learning problems or attended special classes, consisted of 187 high school students (156 males and 31 females) and 256 university students (196 males and 60 females). A separate clinical sample of 260 high school (85%) and university athletes, (83.5% male and 16.5% female) were evaluated within five days of sustaining a concussion. From this sample a sub-sample of 52 athletes was selected who had sustained concussions and were evaluated three times within specified intervals, of 72 hours, four to eight days, 7 to 30 days (Lovell et al., in press). A concussion was diagnosed if the athlete reported any symptoms such as headache, dizziness, nausea etc. or if he or she demonstrated a decline in overall cognitive functioning as indicated by a poor performance on a mental status examination (Lovell, Collins, Iverson, et al., 2004).

(ii) Normative scores & classification ranges

Descriptive statistics and psychometric analysis revealed that although no significant differences in the total symptom scores between the regular education high school and regular education university students were evident (Mean = 5.31, SD = 9.21 and Mean = 5.28 and SD = 8.36), there was a significant difference between males and females in both groups. Consistent with other studies (Rutherford et al., 1979) the results showed that young woman reported more symptoms than young men in both the high school ( $p = 0.03$ ) and university sample groups ( $p = 0.001$ ). This was not evident in the concussed sample, where there were no differences with regards to total symptom scores and gender. As a result, normative data for the ImPACT Postconcussion Scale was presented by gender, although it was noted that the distribution of total scores were skewed for this population because young, healthy individuals tended to report fewer symptoms (Lovell et al., in press). In order to control for this, classification categories with raw score and percentile rank ranges were created that reflect proportions of normative individuals (for the purpose of this study only the tables relating to regular education high school boys will be presented here). Thus, in Table 5.2.1.2.2, 40.5% of high schoolboys obtained a zero score on the scale, i.e. ‘low-normal’, 90% scored 13 or less, and only 11% scored 14 or higher.

**Table 5.2.1.1.2**  
**Classifications, raw scores, and percentile ranks based on a sample of 588 regular education high school boys**

<b>Classification</b>	<b>Raw scores</b>	<b>Percentile ranks</b>
<b>Low-Normal</b>	0	40.5
<b>Normal</b>	1-6	49-76
<b>Unusual</b>	7-13	79-90
<b>High</b>	14-21	91-95
<b>Very High</b>	22+	>95

(Lovell et al., in press)

Since the concussed group had more high school students in the sample than university students (85%), when comparing the concussed athletes to the non-concussed, healthy sample group, both high school and university student groups were combined. The breakdown of the percentages into the total score classification ranges, for both the normative and concussed samples are presented below in Table 5.2.1.2.3

**Table 5.2.1.1.3**  
**Percentage of normative subjects and concussed athletes falling in each classification range**

<b>Classification</b>	<b>Normative Sample</b>		<b>Concussed Sample</b>	
	<b>Young Men</b>	<b>Young Woman</b>	<b>Young men</b>	<b>Young woman</b>
Low-normal	42%	28%	6.5%	2.3%
Broadly normal	32%	45%	14.7%	2.3%
Borderline	15%	17%	11.5%	23.2%
Very High	8%	7%	32.7%	25.6%
Extremely High	2%	2%	34.6%	25.6%

(Lovell et al., in press)

When the two lowest classification ranges are combined (i.e. Broadly normal range) for the normative sample, 74% of young men and 73% of young women fell into this range, in comparison to 21% of young men and 26% of young women in the concussed sample. If the two highest ranges are combined, 10% of young men and 9% of young women from the normative sample fall into this range in comparison to 67% of young men and 51% of young women of the

concussed sample who fell into this combined range. Thus, the concussed group reported more symptoms than the normative group across all ranges.

### (iii) Scale Reliability

The internal consistency reliability of the ImPACT Postconcussion Scale ranged from 0.88 to 0.94 across the samples of high school and university students. The confidence interval represents a range of scores around an observed score in which the individual's true score is believed to fall (Lovell et al., in press). For the normative sample of young men, the 80% confidence interval was +/- 3.4 points and for young woman +/- 4.4 points. Whilst for the concussed athletes, the internal consistency of the scale was high ( $r = 0.93$ ), the standard error of measurement was 5.3 point and the 80% confidence interval 6.8 points (Lovell et al., in press).

The frequencies of symptom endorsements for the ImPACT Postconcussion Scale in concussed athletes illustrates that 60 to 79% of the sample reported symptoms of headaches, fatigue, feeling slowed down, drowsiness, difficulty concentrating, feeling mentally foggy and dizziness, whilst less than 25% of the sample reported symptoms of nervousness, feeling more emotional, sadness, numbness or tingling and vomiting. Across the intervals there was a consistent trend for the athletes to report fewer symptoms (Lovell et al., in press).

The authors assert that whilst the ImPACT Postconcussion Scale is an important tool in the evaluation of concussion, it should be used in combination with neuropsychological testing and other appropriate medical management, to assist with return-to-play decisions. In future authors hope to identify reliable subscales or symptom clusters within the scale which will be normed and evaluated to help provide information regarding how to interpret statistically reliable change (Lovell et al., in press).

#### **5.2.1.2 Paper-and-pencil Postconcussion Symptom Questionnaire**

A Postconcussion Symptom Questionnaire as a paper-and-pencil measure (see Table 5.2.1.2) was developed for the purpose of this study to provide a formal method of listing PCS following MTBI in sport. It consists of common symptoms reported amongst athletes after concussion and

was developed from a paper-and-pencil questionnaire used in prior research in South Africa on Rugby Union athletes (Shuttleworth-Edwards et al., 2004).

**Table 5.2.1.2**  
**The Postconcussion Symptom Questionnaire**

Symptom	None	Minor		Somewhat		Severe
Headache	0	1	2	3	4	5
Nausea	0	1	2	3	4	5
Vomiting	0	1	2	3	4	5
Balance Problems	0	1	2	3	4	5
Dizziness	0	1	2	3	4	5
Fatigue	0	1	2	3	4	5
Trouble Falling sleep	0	1	2	3	4	5
Sleeping More Than Usual	0	1	2	3	4	5
Sleeping Less Than Usual	0	1	2	3	4	5
Drowsiness	0	1	2	3	4	5
Sensitivity to Light	0	1	2	3	4	5
Sensitivity to Noise	0	1	2	3	4	5
Irritability	0	1	2	3	4	5
Sadness	0	1	2	3	4	5
Nervousness	0	1	2	3	4	5
Feeling More Emotional	0	1	2	3	4	5
Numbness or Tingling	0	1	2	3	4	5
Feeling Slowed Down	0	1	2	3	4	5
Feeling Mentally "Foggy"	0	1	2	3	4	5
Difficulty Concentrating	0	1	2	3	4	5
Difficulty Remembering	0	1	2	3	4	5
Visual Problems	0	1	2	3	4	5
Poor Appetite	0	1	2	3	4	5
Difficulty Hearing	0	1	2	3	4	5
Aggression	0	1	2	3	4	5
Speech Problems	0	1	2	3	4	5

The original questionnaire consisted of 31 items on a six point Likert scale and was reduced to 26 items using the same six point scaling for the present study. In the original questionnaire questions that appeared to be repetitive, for example 'do you have problems with your speech?', 'do you slur your words?', 'do you stutter or stammer?' and 'do you stumble over your words?' were refined and reduced to a single item, i.e. 'speech problems'. In addition, items which seemed inappropriate to be used on an adolescent group (e.g. are you experiencing sexual problems?) were also excluded. These exclusions resulted in the inventory being streamlined and reduced to 26 items, of which 22 items are the same as those in the ImPACT Postconcussion Scale, and are worded and ordered in the same way. Of the four symptoms included in the

Postconcussion Symptom Questionnaire that do not form part of the ImPACT Symptom Scale (poor appetite, difficulty hearing, speech problems and aggression), two symptoms demonstrated their sensitivity to the effects of concussion in research in South Africa, where professional and high school Rugby Union players exhibited symptoms of speech problems and aggression (Shuttleworth-Edwards et al., 2004). Although there are no validity studies on this revised postconcussion inventory designed especially for the present study, the inventory contains all the items in the ImPACT Symptom Scale which has established validity, and the basic inventory served to discriminate Rugby Union players from controls in the research of Shuttleworth-Edwards et al. (2004).

### **5.2.2 Studies investigating postconcussive symptoms as a consequence of MTBI in sport**

In comparison to studies investigating the effects of MTBI on cognition, there are relatively few studies which have specifically targeted the study of PCS as a consequence of MTBI in sport. The following review will attempt to highlight those studies which have included any postconcussive symptom reporting amongst athletes as part of their investigation into the effects of MTBI in sport. Examples of these studies include Abreau et al. (1990); Barnes et al. (1998); Collins, Grindel et al. (1999); Collins et al. (2004); Collins, Field et al. (2003); Field et al. (2003); Ferguson et al. (1999); Gaetz et al. (2000); Guskiewicz et al. (2000, 2003); Hatfield et al. (2004); Hinton-Bayre et al. (1999); Iverson et al. (2002, 2004a, 2004b, 2006); Jordan et al. (1997); Jordan, Green, et al. (1996); Killam et al. (2005); Lovell et al. (2003); Macciocchi et al. (1996, 2001); McClincy et al. (2006); Mihalik et al. (2005); Moser et al. (2005); Pellman et al. (2006); Putukian et al. (2000); Shuttleworth-Jordan et al. (1993); Shuttleworth-Edwards et al. (2004); Wilberger et al. (1991).

Most of the above studies have incorporated individual symptoms (or symptom clusters) which are reported to be the most commonly experienced after MTBI, in their postconcussion inventories (e.g. Abreau et al., 1990; Barnes et al., 1998; Collins, Grindel et al., 1999; Ferguson et al., 1999; Field et al., 2003; Gaetz et al., 2000; Guskiewicz et al., 2000; Hatfield et al., 2004; Hinton-Bayre et al., 1999; Iverson et al., 2002, 2004a, 2006; Jordan, Green et al., 1996; Jordan et al., 1997; Killam et al., 2005; Lovell et al., 2003, Lovell, Collins, Iverson, et al., 2004; McClincy et al., 2006; Moser et al., 2005; Pellman et al., 2006; Shuttleworth-Jordan et al., 1993; Shuttleworth-Edwards et al., 2004; Wilberger et al., 1991). Some studies report using an

inventory but then do not report on the symptoms experienced by the athletes (e.g. Guskiewicz et al., 2005; Hatfield et al., 2004; Putukian et al., 2000; Macciocchi et al., 1996, 2001). A few studies have focussed on specific individual symptoms in their studies (Collins, Field, et al., 2003; Iverson et al., 2004b; Mihalik et al. 2005).

Examples of studies which have investigated the acute effects in respect of postconcussive symptoms following MTBI include Collins, Field, et al. (2003); Field et al. (2003); Guskiewicz et al. (2000, 2003); Hinton-Bayre et al. (1999); Iverson et al. (2004b); McClincy et al. (2006); McCrory et al. (2000); Mihalik et al. (2005); Pellman et al. (2006) and Putukian et al. (2000). Examples of studies which have investigated the chronic effects in respect of PCS include Abreau et al. (1990); Barnes et al. (1998); Ferguson et al. (1999); Gaetz et al. (2000); Guskiewicz et al. (2005); Hatfield et al. (2004); Iverson et al. (2006); Jordan et al. (1997); Killam et al. (2005); Moser et al. (2005) and Shuttleworth-Edwards et al. (2004). Examples of studies which have evaluated both acute and chronic effects with respect to self-reporting PCS include Collins, Grindel et al. (1999); Iverson et al. (2002; 2004a); Jordan, Green, et al. (1996), Wilberger et al. (1991); Lovell et al. (2003); Lovell, Collins, Iverson, et al. (2004); Macciocchi et al. (1996, 2001) and Shuttleworth-Jordan et al. (1993).

Consistent with previous studies investigating PCS in MTBI in general, distinct symptom clusters can be identified in respect of the above mentioned studies incorporating postconcussion inventories. For simplicity sake the symptoms included in the inventories will be clustered into neurocognitive, somatic and emotional or behavioural domains. Although some symptoms seem to be saying the same thing (e.g. fatigue and tiredness) they will be included here in order for the reader to gauge the different ways in which these symptoms are presented in the inventories. Symptoms measuring neurocognitive effects include attention deficit, memory and concentration problems, forgetting, trouble thinking, amnesia, and difficulty remembering. Symptoms which measure somatic effects include headache, dizziness, hearing impairment, neck pain, blurry or double vision, sensitivity to light, fatigue, tiredness, loss of consciousness, numbness/tingling, speech problems, nausea, vomiting, balance problem drowsiness, sensitivity to noise, feeling slowed down, feeling mentally 'foggy', LOC, disorientation, photophobia, 'blacking' out, having your 'bell rung' or 'seeing stars,' and unsteadiness. Lastly, symptoms measuring emotional/behavioural effects include depression, irritability, lack of energy, agitation,

aggression, delusions, hallucinations, dysphoria, anxiety, euphoria, apathy, disinhibition, lability, sleep disturbance, trouble falling asleep, sleeping more than usual, sleeping less than usual, sadness, nervousness, feeling more emotional, confusion, slower thinking or reactions, feeling 'different' than before experiencing the hit and feeling dazed.

Of all the symptoms reported in the investigation of MTBI in sport, the symptom of headache appears to be the most commonly reported symptom by athletes in both the acute and chronic condition following concussion (e.g. Collins, Grindel et al., 1999; Gaetz et al., 2000; Guskiewicz et al., 2003; Macciocchi et al., 1996; Wilberger et al., 1991). Consistent with researchers reporting of early onset PCS (those symptoms which appear immediately after the injury or the day after) in the general MTBI population, researchers investigating PCS in sport report symptoms of headache, dizziness, nausea, fogginess, blurred vision and poor concentration in athletes in the acute condition following concussion (e.g. Collins, Grindel et al., 1999; Field et al., 2003; Guskiewicz et al., 2000, 2003; Hinton-Bayre et al., 1999; Iverson et al., 2002, 2004b; Lovell et al., 2003; Macciocchi et al., 1996, 2001; Mihalik et al., 2005; Pellman et al., 2004; Wilberger et al., 1991). Similarly, consistent with researchers reporting of late onset symptoms (those symptoms which develop a few weeks after the injury) in the general MTBI population, researchers investigating PCS in sport report symptoms of poor memory, poor concentration, delusions, hallucinations, depression, anxiety, euphoria, apathy, irritability, sleep disturbances, aggression and hearing disturbances in athletes in the chronic condition following concussion (e.g. Ferguson et al., 1999; Gaetz et al., 2000; Jordan et al., 1997; Shuttleworth-Edwards et al., 2004).

### **5.2.3 Synthesis of PCS evaluation in MTBI in general**

From the above review it would appear that generally few studies relative to those investigating the neurocognitive effects of MTBI in sport, have employed postconcussion inventories in their investigations. Moreover some of the studies which have employed inventories have failed to remark on the symptoms reported in their athlete samples, which is alarming considering that the presence of PCS sequelae are as much a part of the 'concussion syndrome' as the cognitive deficits themselves. Whilst most inventories are employed for management purposes for the acute investigation of MTBI in sport, which follows that most investigation into the effects of

MTBI have investigated acute effects over chronic effects, in the general MTBI population the opposite case has transpired. Most patients are evaluated some time after their injury at which point acute symptoms would have resolved. Furthermore, different mechanisms of motivation are in evidence for reporting symptoms in both patient and athlete populations. In the patient population symptoms are often over reported particularly in the case of litigation where the motive is one of compensation. Amongst athletes however, the motivation is not to lose their position on the team and to return to play as soon as possible, which is why the underreporting of symptoms is so prevalent amongst this group since the resolution of symptoms is a requirement for return-to-play (Aubry et al., 2002).

According to Lezak (2004) the main premise of a scale used in clinical studies to evaluate postconcussive symptoms is to be sufficiently robust to measure symptoms which separate members of the population that they intend to investigate from members of the population at large (Lezak et al., 2004). The ImPACT Postconcussion Scale which represents one of the few scales that has been validated for use in the evaluation of concussion in athletes and the paper-and-pencil Postconcussion Symptom Questionnaire, which is a virtual replication of the ImPACT Postconcussion Scale, barring four symptoms of aggression, speech problems, difficulty hearing and poor appetite, were therefore selected on the basis that these measures would be sensitive to the effects of concussion and that those athletes demonstrating postconcussive sequelae as a result of concussion(s) will report on those symptoms included in these inventories.

## CHAPTER SIX

### MILD TRAUMATIC BRAIN INJURY IN SPORT

This chapter focuses specifically on mild traumatic brain injury (concussion) in contact sport. It begins by reporting on the incidence of concussion in contact sports in general and then focuses specifically on the sports of boxing, the martial arts, ice hockey, soccer, American football, Australian Rules Football, Rugby League, and Rugby Union. This section is followed by a review of existing research regarding the presence and potential for both acute and chronic consequences of concussion in contact sport, in respect of both neurocognitive and postconcussive symptom sequelae, although in some studies investigating the chronic effects in athletes the neurological outcome is also reported on. Since the focus of this research is on the chronic and cumulative effects of MTBI, the review on studies investigating the acute effects is extensive but not completely exhaustive as the intent was to provide a more comprehensive review of chronic investigations. There are a number of studies that have investigated the recovery curve of concussed athletes relative to controls, where recovery to baseline levels might be construed as confirmation for total recovery and therefore the absence of persistent cumulative neuropsychological effects of concussion (e.g. Barth et al., 1989; Barr, 2003; McCrea et al., 2003). However, none of these studies set out with the purpose of confirming the presence of chronic effects, and generally have methodological issues that prohibit clear-cut conclusions in this regard, such as inadequate control for IQ and education between contact and non-contact sports groups, gender differences, and poor test-retest reliability and practice effects on the instruments used.

A summary table of all the studies cited in the review appears at the end of the chapter and which summarizes all studies according to the type of study (acute or chronic and cross-sectional or prospective), sample constitution, cognitive and postconcussive symptom measures and limitations of the study. For the purpose of the study acute effects refer to studies which investigate the effects that for the most part resolve or plateau out within a period of three months. Studies which investigate the chronic effects refer to those effects presumed to be permanent or residual and which are not associated with the acute consequences of a reported concussion. In these tables, the term cross-sectional refers to studies which collect comparative data at one point in time in respect of targeted variables such as cognitive test performance and

PCS. In comparison, prospective studies refer to those involved in the collection of comparative data in a forward direction at several points over time on one or more variables. With regards to the limitations of the studies, the table comments on factors according to sample size, use of controls groups and control for age, gender, education or IQ and level of play. Further limitations will be discussed in the review.

Research reporting on the chronic neurocognitive and postconcussive symptom outcome following concussion in sport has increased in the past two decades. However, this type of largely quasi-experimental research has been beset with methodological problems in the form of poor subject control, lack of appropriate control groups, subject selection bias, poor control for significance as well as other problems which will be addressed in the final section of the chapter.

## **6.1 EPIDEMIOLOGY OF MTBI IN SPORT**

Mild traumatic brain injury (typically referred to as concussion in the sports arena) is a common feature amongst both amateur and professional sports alike and although once considered to be a 'routine risk' associated with participation in the game, the impact of these injuries has gained significant interest and concern amongst sports and health professionals in the past three decades (e.g. Barth et al., 1989; Echemendia & Julian, 2001; Lovell, 1999). Within each sport there is a pattern of injury that is unique to that particular sport associated with the dynamics of play, making it difficult for researchers to draw comparisons between the different sports studies. Moreover, the incidence of concussion in some sports may be over-estimated where a high incidence of concussion is expected (e.g. boxing) whilst in other sports the incidence may be under-estimated due to ignorance about the risks associated with the sport or fear of jeopardizing positions on a team (e.g. American football and Rugby Union) (Koh, Cassidy, & Watkinson, 2003). Due to the known underreporting of concussions, as well as the differences in the various definitions of concussion, the true incidence of concussion amongst the contact sports and amongst the different age and gender groups is difficult to ascertain.

According to researchers it is estimated that between 50,000 to 300,000 sports-related mild traumatic brain injuries (MTBI) occur each season in the United States and over 80% of these injuries occur amongst high school players (Mihalik et al., 2005; Schatz et al., 2006). Surveys of

high schools in both Canada and New Zealand indicated that approximately 30% of all high school students reported at least one concussion before their graduation with the average age of the reported concussive event being 10 years (Segalowitz & Brown, 1991; Segalowitz & Lawson, 1995). Powell and Barber-Foss (1999) in their study of 235 high schools in the United States over a three-year period from 1995 to 1997, estimated that 62,816 cases of MTBI (concussion) occur annually. American football accounted for 63% (i.e. 40,000 or 3.9%) of these cases, wrestling 10.5%, girl's soccer 6.2%, boy's soccer 5.7%, girl's basketball 5.2%, boy's basketball 4.2%, softball 2.1%, baseball 1.2%, field hockey 1.1% and volleyball 0.5% (Powell and Barber-Foss, 1999). In a sample of 1,372 979 children from Canada, between the ages of six to 16 years, most incidences of concussion occurred in basketball with 16.5%, followed by soccer (13.2%), gymnastics (11.3%), American football (7.7%), field hockey (6.8%), baseball (6.4%), skating (3.0%), wrestling (2.2%), volleyball (1.8 %), and lacrosse (0.8%). Other sports included skiing (5.8%), snowboarding (2.6%) and high jump (4.0%) (Willer, Dumas, Hutson, & Leddy, 2004).

Concussions presented to a United States emergency department between the years of 1990 to 1999 were estimated to be around 4,820 from ice hockey, 21,715 from soccer and 68,861 from American football (Delaney, 2004). Recent data from the National Collegiate Athletic Association (NCAA) Injury Surveillance System in the United States, during the 2002-2003 season, showed that concussion accounted for 12.2% of injuries in collegiate ice hockey, 8% in football and 4.8% in soccer (Dick, 2003). Koh and his colleagues (2003) in their systematic review of the literature on concussion in contact sports found that overall ice hockey showed the highest incidence of concussion amongst the team sports (American football, rugby and soccer) for high school male athletes, although ice hockey and rugby had similar rates at the professional level. Soccer had the lowest frequency of concussion amongst high school male athletes and boxing showed the highest frequency of concussion overall (Koh et al., 2003).

According to the Centers for Disease Control and Prevention (Thurman, Branche, & Sniezek, 1998), adolescents represent the highest concussion risk group, with males being at greater risk than females. Adolescent males tend to present with a higher proportion of head injuries owing to the fact that boys are involved in more high-risk collision or contact sports (Baker & Patel, 2000). Based on their investigations of concussion in a Canadian school, Willer and his

colleagues (2004) estimated that the probability of a female sustaining a concussion at any time during her school career was less than 1% whereas the probability for males was more likely to be 1.9%. Moreover, males were 2.23 times more likely to experience a head injury with symptoms than females. For males the rate of head injuries with symptoms of concussion tended to increase and then decrease with age over the age range from 6 to 16 years, whereas the rate of symptoms of concussion was more consistent across the ages for females (Willer et al., 2004). This may change subsequently in the years to come as more females are entering into male-dominated sports. Data from the Consumer Product Safety Commission National Electronic Injury Surveillance system (NEISS) database from 1990 to 2004 demonstrated different concussion injury rates for males and females where in softball and field hockey a greater number of concussions were reported for younger age groups (0 to 24 years) whilst males (25 to 65 years) experienced more injuries (Brooks, 2004). With respect to some high school sports, however, the risk of concussion is slightly higher in females as it is for males (Cassidy et al., 2004). The risk of concussion per athlete-game-exposure in males was 0.6 for soccer (0.7 for females), and 0.3 for basketball (0.4 for females) (Cassidy et al., 2004). This was also evident in Powell and Barber-Foss' (1999) study in the United States where the incidence of concussion amongst the female's high school soccer and basketball teams was higher than that of the male groups. Rates of concussion amongst collegiate female athletes have not as yet been studied (Brooks, 2004).

### **6.1.1 Incidence of MTBI in boxing and the Martial Arts**

Boxing dates as far back as Ancient Greece, where boxing like wrestling was considered a honourable sport for Greek boys (Pearn, 1998). Modern boxing has its origins in Great Britain (Pearn, 1998). The history of boxing is one of reticent but progressive rule changes beginning with the Broughton's rules in 1743, to the Amateur Boxing Association Rules in 1952, as well as the introduction of weight divisions between 1891 and 1909 by the National Sporting Clubs which encouraged lighter or younger boxers to compete (Pearn, 1998). The number of professional boxers worldwide is currently unknown although in 1980 it was estimated that the number of professional licensed boxers was around 25,000 (Morrison, 1986). In the United States the number of professional boxers was estimated at 5,000 and the number of amateur boxers 25, 000 (Morrison, 1986). About 15,000 of these amateur boxers are registered with the

Amateur Boxing Federation. The lifespan of an amateur boxer lasts about five years and on average they fight 20 fights per year (Morrison, 1986).

The incidence of concussion amongst boxers is obviously very high due to the nature of the sport, which is to render the opponent unconscious (Macciocchi et al., 1998). Documented incidence rates of concussion amongst boxers are not reported in the literature. In 1984 the British Medical Association passed a resolution which supported the abolishment of boxing in view of the proven ocular and TBI resulting from professional boxing. American, Australian and Canadian medical organizations also supported this line and countries such as Sweden, Czechoslovakia and Norway have made it illegal (Butler, Forsythe, Beverly, & Adams, 1993). Since then the British Medical Association and the American Academy of Paediatrics have called for total bans on organized boxing for children younger than 16 years of age. On the opposite end of the age spectrum, in amateur boxing there is an upper age limit of 32 years (Pearn, 1998). Boxing has grown in popularity amongst the female population. The risks are the same as for men in terms of the length of exposure but some professionals caution that the boxing exposure threshold is almost certainly lower in females than males (Pearn, 1998).

Consistent with the fighting trends seen in boxing are the martial arts which vary in terms of the number of different forms and include Taekwondo, Jeet Kune Do, Kung Fu, Karate, Kickboxing, Judo and Jujutsu. Whilst the different forms differ in respect of their fighting and self-defence techniques, all demand a high degree of hand strikes, punches as well as kicking. Generally amongst the marital arts men experience higher injury and severity rates than woman (Zemper & Pieter, 2004) with 63% of the severe injuries being cerebral concussions (Birrner, 1996). Although the incidence of concussion amongst the martial arts is also expected to be high due to the nature of the sport, to the authors knowledge very few incidence reports have been published except for amongst athletes participating in Taekwondo. In an adult Taekwondo competition in London, the men sustained more concussions than the woman at 7.04 per 1000 athlete exposures and 2.42 per 1000 athlete-exposures, respectively (Pieter and Zemper, 1998). Similarly, in a Taekwondo competition in London consisting of 3,341 boys and 917 girls between the ages of 6 to 16 years the incidence of concussion was 5.11 per 1000 athlete exposures for boys compared with 4.55 per 1000 athlete exposures for girls (Pieter and Temper, 1999). Further, the incidence of head blows and concussions in participants between the ages of 11 to 19 years in a Taekwondo

competition in Korea was extremely high at 226 and 50 per 1000 athlete exposures (Koh and Cassidy, 2004).

### **6.1.2 Incidence of MTBI in ice-hockey**

The game of ice hockey (hockey) began in the mid 1800's in Canada and currently, the most prominent ice hockey nations are Canada, Czech Republic, Finland, Russia, Slovakia, Sweden and the United States (Wikipedia, n.d.). Ice hockey is one of the four major North American professional sports, represented by the National Hockey League (NHL) at the highest level (Wikipedia, n.d.). The game itself is very fast-paced, with players gliding across the ice at swift rates, using their sticks to contest for the puck (rubber disk) often sending it across the ice at rates of 160 kilometres per hour. Ice hockey is played with two teams on the ice, six players in each team (one goalkeeper, three forwards and two defensemen) and players are allowed to be substituted whilst play is in progress. Most ice hockey games are played in three 20-minute periods separated by 10 to 15 minute intermissions, although the rules are different for professional and amateur athletes (Wikipedia, n.d.).

Researchers report that the majority of concussions sustained by ice hockey players are as a result of high speed collisions (Lovell, Echemendia, & Burke, 2004). At the 2002 Sports Concussion Conference hosted in Pittsburgh, the director of the NHL Concussion Programme, Dr Burke, reported that most concussions occurring in ice hockey were due to contact with the boards around the rink, glass and ice accounting for 42% of all concussions (Solomon et al., 2006). Other mechanisms resulting in concussive injury included an elbow or forearm to the head (21%), shoulder to head contact (19%), fighting on the ice (12%) and a puck or stick to the head (6%) (Solomon et al., 2006).

Honey (1998) reviewed all articles reporting on the incidence of ice hockey injuries from the years 1966 to 1997. The studies confirmed that the most common injury reported was brain injury, with the incidence of concussion ranging from 0.0 to 2.8 per 1000 playing hours for athletes between the ages of 5 to 14 years, from 0.0 to 2.7 per 1000 playing hours for high school athletes and from 0.0 to 6.6 per 1000 playing hours for players on elite teams. The review confirmed that the incidence of concussion increases with higher levels of play. In 21

professional ice hockey teams of the NHL in North America, during the 1989 to 1990 season, concussion was reported to be the leading cause of head injury amongst the players at 30%, with the majority of injuries occurring during games (Biasca, Simmen, Bartolozzi, & Trezn, 1995). Similarly, Tegner and Lorentzon (1996) reported on the incidence of concussion amongst Swedish elite ice hockey players. The study consisted of two parts with one retrospective questionnaire assessing previous concussions and one prospective questionnaire covering four seasons (1988 to 1989 and 1991 to 1992). The findings demonstrated that in the retrospective study, 22% of the players (51 out of 227 players) reported at least one concussion and in the prospective study, 52 concussions were reported. Body checking (when a player bumps against an opponent with his hip or shoulder to block him or throw him off balance) was the most common cause of concussion with most concussions occurring during games rather than during practices. Two athletes in their study stopped playing due to permanent brain damage associated with multiple concussions. Researchers Wennberg & Tator (2003) collected incidence reports of concussion via media injury reporting of the NHL from the years of 1986 through to 2002. Between the years 1986 to 1996 it was estimated that there were 7 to 17 concussions reported per season (4 to 8, per 1000 games). Whilst from 1997 the incidence rates increased from 27 to 74 per season (13 to 30 per 1000 games). The authors attribute the increase in the number of concussions to a greater awareness to the dangers associated with concussion in the sport. At the 2004 Sports Concussion Conference hosted in Pittsburgh, the incidence of concussion for the NHL between the years of 1997 to 2003 ranged from 71 to 128 per year (Solomon et al., 2006)

Ice hockey is played in all age groups with thousands of amateur players taking part at the high school level. Gerberich et al. (1987) investigated the injury rate amongst high school ice hockey players in the United States. Head and neck injuries accounted for 22% of all injuries sustained. In a sample of 103 athletes between the ages of 7 to 18 years admitted to a children's emergency hospital in the United States, 20% of all injuries were concussions (Reid & Losek, 1999). Further injury rates were documented for 807 boys and girls, at four boys and one girl's youth ice hockey tournament in the United States, between the 1993 to 1994 season (Roberts, Brust, & Leonard, 1999). All players were between the ages of 9 to 19 years. The findings demonstrated that generally the boys sustained more concussions than the girls, which consisted of 15 % of all injuries sustained. According to researchers Hatfield et al. (2004), there are no formal comparison injury rates between adolescents participating in ice hockey and other high school

sports, although the authors go on further to say that given the nature of the game one would expect the incidence rates to be as high as American football.

### **6.1.3 Incidence of MTBI in soccer or football**

Soccer, also referred to as football, is rated as one of the most popular sports in the world with approximately 200 000 professional and 240 amateur players (Junge, Cheung, Edwards, & Dvorak, 2004). The International Federation of Football Association (FIFA) conducted a wide-scale survey in 2002 and reported then that there are approximately 243 million active soccer players in 204 countries, of which approximately 31 are registered with FIFA (2002).

Research suggests that a substantial amount of soccer injuries involve trauma to the brain (Barnes et al., 1998; Boden, Kirkendall, & Garrett, 1998) accounting for up to 22% of all injuries in soccer (Kirkendall et al., 2001). Whilst studies investigating the incidence of MTBI in soccer are limited, there also appears to be great discrepancies between the studies in terms of the incidence rates of concussion amongst male and female players. An explanation for this would be the willingness of the athletes to report concussions, different methods and systems of reporting, different concussion criteria as well as different times of injury and positions played (Rutherford et al., 2003).

Soccer represents the sport with the highest incidence rate of concussions amongst females (Cassidy et al., 2004). Dick (1994) employing data from the National Collegiate Athletic Association (NCAA) in the United States over 3 to 6 years, found that the concussion rate per 1000 athlete exposures was 0.25 for male soccer players and 0.24 for female soccer players. According to Barnes et al. (1998), it is estimated that over 10 years of soccer play male soccer players had a 50% chance of sustaining a concussion, whilst female soccer players a 22% chance. Moreover, soccer players are three times as likely to suffer another concussion if they have sustained a previous concussion ( $p < 0.05$ ) while female soccer players are 2.5 times as likely to suffer a concussion as male soccer players ( $p < 0.05$ ) (Delaney, Lacroix, Leclerc, & Johnston, 2002). Boden and his colleagues (1998) in the United States looked at the incidence of concussion amongst elite collegiate players over two seasons. Fifty-nine percent of concussions were observed in males and 41 percent in females with concussions resulting primarily from

contact with another player's head. This translates to an incidence rate of 0.6 per 1000 game exposures amongst males and 0.4 per 1000 game exposures for female soccer players. In a study by Powell & Barber-Foss (1999), high-school boys had an incidence of 0.57 per 1000 game exposures compared with an incidence of 0.71 per 1000 game exposures amongst high school girls. Recent NCAA injury statistics showed that injury rates amongst men's soccer were 20.1 per 1,000 athlete exposures, with nearly 10% of all soccer injuries amongst collegians being concussions and concussions being the dominant head injury in both college and high school soccer players (Webbe & Ochs, 2003; Powell & Barber-Foss, 1999). Despite the limited studies investigating incidence of concussion in soccer, it would appear that the number of incidences amongst male and female soccer players have increased in the last few years especially since the NCAA reported on data from the 1993 to 1994 collegiate soccer season.

#### **6.1.4 Incidence of MTBI in American football**

American football originated from soccer football which was brought to America in the 1800s (Metzl, 1999). Although the first "football" match was played in 1869, the game was played using soccer rules. It took a period of 15 years before the game known as American football evolved largely through the incorporation of rules from rugby-football. The game consists of two teams with 11 players to each team. Physical collisions occur between the 11 opposing players, between players on the same team and between players and the ground on each and every play (Porter, 1999). In 1904 President Theodore Roosevelt threatened to ban American football due to the fatalities of 19 athletes. This led to the formation of the National Collegiate Athletic Association (NCAA) which established rules for safer athletic competition (Cantu, 1997b). Fatalities in American Football reached an all time high in 1954 with 30 deaths, and 819 deaths between the years of 1931 to 1986 (Cantu, 1997b). Between the years 1973 to 1983 fatalities in American football, mainly from head injury, exceeded deaths in all other sports combined (Cantu, 1997b).

In relation to other sports in the United States, American Football has the highest number of concussions each year owing to the large number of participants at the high school and collegiate levels (Dick, 2003). According to researchers concussion incident rates in high school football were as high as 15 to 20% for all players in a season in the 1970's (Gerberich, Priest, Boen,

Straub, Maxwell, 1983). This incidence rate was reduced to 10% in the late 1980's and more recently rates of between 3.6 to 5.6% have been recorded amongst high school players in the United States (Guskiewicz et al., 2003). In a population of high school football players, Wilberger (1993) reported a greater than 12% incidence of two concussions in the same season, where as at the college level estimates ranged from 5.3% to 8.2% per four-year career. McCrea et al. (1997), reported 33 (5.8%) concussions amongst a group of 253 high school and 215 college football players. This translates to an incidence of 4.3% concussions amongst the high school group. In a more recent survey amongst 450 high school football players however, the incidence of concussion was as high as 47.2 of 100 (Langburt et al., 2001). These results are even higher than those recorded in the 70's (Gerberich et al., 1983). The discrepancy in these results could be due to the different concussion guidelines that the various authors adhere to. Gerberich et al's study in the late 70's used the definitions given by the Committee on Head Injury Nomenclature of the Congress of Neurologic Surgeons, whereas Langburt et al. in their study adhered to Cantu's guidelines. The Congress of Neurologic Surgeons committee defines a grade I concussion as having 'no loss of consciousness', but there is a loss of awareness or transient confusion according to Cantu's definition which can last for up to 15 minutes (Langburt et al., 2001). Therefore, an athlete experiencing momentary dizziness or confusion would be classified as a grade I concussion on Cantu's scale but not necessarily in Gerberich's study, which meant that they underestimated the frequency of low-grade concussions which went unreported.

In comparison to the injury rates reported above for high school athletes, the injury rates are lower amongst collegiate players (4.8% and 4.0%) and also lower than those injury rates sustained by collegiate athletes in the 1980's (Barth et al., 1989; Dick, 2002). Whilst there are many studies reporting on the incidence rate of concussion amongst high school and collegiate athletes, according to the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury (Cassidy et al., 2004), there is a lack of studies on the rate of concussion incidence amongst professional football players.

Guskiewicz and his colleagues (2003) noted that the rate of concussion injuries in games was markedly higher than in practices. In their study of the 196 athletes who reported concussions, more than half of their total concussion injuries ( $n = 101$ ) occurred in games. This finding is

possibly attributed to the intensity, aggression, force and speed at which games are played relative to the practices. Other researchers also support this finding (Delaney et al., 2002; McCrea et al., 1997). According to researchers the risk of sustaining a concussion in American football has been reported to be four to six times greater if the player has already sustained a concussion (Kelly & Rosenburg, 1997; Guskiewicz et al., 2003). Collins et al. (2002) in their study on high school athletes found that those athletes with three or more prior concussions had an eight-fold greater risk of experiencing LOC and a five and a half fold greater risk of experiencing anterograde amnesia after a subsequent concussion.

### **6.1.5 Incidence of MTBI in Australian Football League (Australian Rules Football)**

Australian Football League (AFL) or Australian Rules Football was originally developed by Irish settlers and shares many of the characteristics of Gaelic football. It is a sport that is unique to Australia and was first recorded in 1877 (Hinton-Bayre & Geffen, 2004). Australian Football League is a very popular sport with over 385 000 participants recorded in 1993 (National Health and Research Council of Australia, 1994). In Australia three variations of football are played: Rugby Union, Rugby League and Australian Football League. An AFL game consists of two teams with 21 players per side. Eighteen players in each team remain on the field at any one time. All players move freely around the field as there is no off-side rule although there are set field positions for players around the field. The ball is moved around the field either by kicking, hand-balling or running with the ball, throwing is not permitted (Hinton-Bayre & Geffen, 2004). As with Rugby Union and Rugby League there is continual contesting for the ball in which players can be knocked out of the way or tackled. Concussions generally result from direct player contact or from a player striking their head on the ground. Players generally leap into the air to catch the ball and can use another player as a step to gain extra height in order to mark the ball (Hinton-Bayre & Geffen, 2004).

Data collected over the 1993 amateur football club season rated concussion as the most common injury (15%) (Shawdon & Bruker, 1994). The incidence of concussion is very prevalent amongst players of this sport where studies have shown that 60% of professional AFL players had a history of concussion (Maddocks, Saling & Dicker, 1995). The total injury incidence in AFL is 131 injuries per 1000 playing hours with the concussion incidence amongst elite players being

four concussions per 1000 player hours (McCrorry & Berkovic, 1998b). According to researchers this concussion rate has increased from 2.2 to 4.0 per 1000 playing hours in the past five years (McIntosh, McCrorry & Comerford, 2000), although others put the incidence rate at 3.3 per thousand playing hours (McCrorry et al., 2000). Researchers McMahon, Nolan, Bennett, & Carlin (1993) assessed the incidence of AFL injuries amongst children and adolescents and estimated that the functional impairment injury rate amongst children under the age of 10 is very low (4.89 per 1000 playing hours) but higher amongst adolescents (7.32 per 1000 playing hours).

#### **6.1.6 Incidence of MTBI in Rugby League**

Rugby Football originated in the 1820's in England but the separation of Rugby League from Rugby Union only took place at the end of the nineteenth century in 1895 (Hinton-Bare & Geffen, 2004). Rugby League is an extremely physical game which requires a combination of speed, stamina, strength and agility (Gibbs, 1993). The game consists of 17 players each side, with 13 players on the field at any one time. The game is divided into two 40 minute halves and each team is allowed six tackles or "downs" with the ball. The ball cannot be thrown forward but can be carried forward or kicked down field. Injuries as a result of direct trauma are common with players being frequently knocked over backwards and sustaining whiplash (Gibbs, 1993). Estimates of concussion amongst Australian men's Rugby League range from 1.6 to 3 per 1000 playing hours (Gibbs, 1993). Whilst other studies from the UK yielded variable rates of 0.6 to 8 per 1000 playing hours (Garraway & Macleod, 1995). Recently data amongst 175 professional Rugby League players showed that the incidence of concussion ranged between 5.9 to 9.8 per 1000 playing hours (Hinton-Bayre et al., 2004). According to researchers the incidence of concussions amongst Rugby League (8.5%) is over twice the incidence of AFL (3.6%) (Seward, Orchard, Hazard, & Collinson, 1993) with Rugby League producing the highest incidence of injury and head trauma (Hinton-Bayre & Geffen, 2004). Although studies suggest that the level of serious injuries is still lower than in Rugby Union (Walker, 1985).

#### **6.1.7 Incidence of MTBI in Rugby Union**

In 1823 a schoolboy, William Webb Ellis, whilst playing soccer in England picked up the ball and ran with it across the goal line (Sparks, 1981). His disregard for the rules of soccer led to the

development of Rugby football which later divided into several well known forms Rugby Union; Rugby League, American football and Australian Football League (Micheli & Riseborough, 1974). Rugby Union is a very popular field-based, full body contact team sport and falls second to soccer in terms of the number of nations in which it is played (Junge et al., 2004; Bathgate, Best, Craig, & Jamieson, 2002). Currently the International Rugby Board encompasses 92 national unions (Duthie, Pyne, & Hooper, 2003). It has been estimated that there are 200 000 Rugby Union players in England, New Zealand, France and South Africa and 100 000 players in the United States and Japan. Wales has approximately 35 000 players and Scotland 14 000 players (MacLeod, 1993). In South Africa, Rugby Union forms an integral part of the 'culture' and has traditionally (pre-apartheid) been the main winter sport for 'white' males. In the wake of the post-apartheid era, however, the sport has gained increasing popularity across the population groups, although amongst the black South African, soccer remains the most popular sport (Shuttleworth-Edwards et al., 2004). Most boys are encouraged at a young age to take part in the sport, which is associated with masculinity, strength, power and status. In August of 2001, however Rugby Union was formally launched for woman by the South African Rugby Union (SARFU) (Shuttleworth-Edwards et al., 2004).

The physiological demands of Rugby Union, like any other football code are complex, involving repeated high intensity sprints and a high frequency of contact (Duthie et al., 2003). All players accept the great risks of injury associated with participation in the game, where most players are largely unprotected from the impact forces and protective equipment has limited effectiveness in preventing injuries (Marshall et al., 2004). A Rugby Union match is played with two teams of 15 players each, eight forwards (ball winners) and seven backs (ball carriers). The forwards demand strength and power and are in close contact to the opposition, they are also more often prone to concussion than the backs (Junge et al., 2004). A match consists of two halves of 40 minutes each with a five to ten minute half time break. The ball is in play typically for an average of 30 minutes; the remaining time is made up of injury time, conversions, penalty shots or when the ball is out of play (McLean, 1992).

The rugby season in South African typically falls in the winter months between April to September each year. During those months it is predicted that a high proportion of Rugby Union players will sustain significant injuries. Because of the high incidence of spinal cord injuries in

Rugby Union spanning more than 20 years, changes in the rules of the game were made in most Rugby Union playing countries, with New Zealand changing its rules in 1980 and 1984, Britain in 1979, 1984 and 1985, Australia in 1985 and South Africa in 1990 (Noakes & Jakoet, 1995). Researchers have questioned the effectiveness of these rule changes, however, since the incidence of injury is still very high amongst players in this sport.

As with other contact sports, the incidence of concussion in Rugby Union may be poorly recorded due to athletes underreporting of symptoms (Bruce, Schut and Sutton, 1982; Gerberich, Priest, Boen, Struab, & Maxwell, 1983; Koh et al., 2003). The average reported number of concussions could therefore be a gross underestimation and not accurately reflect the true incidence. For example, in a study by Roux, Goedeke, Visser, Van Zyl, & Noakes (1987) investigating the incidence of rugby injuries amongst schoolboys during an 18-week season in South Africa, although the sample was limited to one geographical area, the incidence of concussion was underreported in 19 of the 26 schools. Twenty schools reported only 27 cases of concussion whereas six schools reported 34 concussive injuries. Moreover, amongst a professional Rugby Union team in South Africa, the Open rugby group reported substantially less symptoms than the control group made up of cricket players (Shuttleworth-Edwards et al., 2004). As with most contact sports, the underreporting of postconcussive symptoms amongst athletes is largely due to fears of being withdrawn from the team. Ignorance amongst athletes and coaches as to what really constitutes a concussion, as well as differences in definitions or diagnoses, also serve to promote the underreporting of concussion injuries amongst players (Wills & Leatham, 2001).

Research suggests that the incidence of match injuries is twice as high in Rugby Union players compared with soccer players, with most injuries taking place during the tackling phase of the game (Bathgate et al., 2002; Bird et al., 1998; Garraway & Macleod, 1995), and with males sustaining more concussions than females with ratios of 1.8 and 2.2 respectively (Bird et al., 1998). In a European study comparing the incidence of soccer injuries relative to Rugby Union, results indicated that Rugby Union was associated with a significantly higher rate of injury than soccer, this was followed by field hockey (Junge et al, 2004). The incidence of injury amongst Rugby League, Australian Football League and American football players is also substantially lower than that in Rugby Union (Garraway, Lee, Hutton, Russell, & Macleod, 2000; Petterson &

Skelton, 2000; Junge et al., 2004; Seward et al., 1993). Whilst these differences are meaningful, researchers agree that it is very difficult to compare the incidences of sports injuries between the various sports because different evaluation methods and definitions of injury have been used in the literature (Junge et al, 2004).

According to researchers the incidence of injury rises with age and level of competition (Jakoet & Noakes, 1998; Roux et al., 1987). Reasons for this would be that the play is faster, the players larger and fitter, tackling is harder, the ball also is in play for longer periods which has certainly been the case since the 1995 World Cup, and with the onset of professionalism, players are able to spend more time increasing their power and speed, and improving their skills (Bathgate et al., 2002). Roy (1974) in his study investigating the frequency and nature of Rugby Union injuries amongst 300 university Rugby Union players in South Africa found that 10% of the all injuries were concussions. Similarly, Nathan, Goedeke, & Noakes (1983) reported that the single most common injury accounting for 21.5% of all injuries in their schoolboy sample was concussion. In South Africa schoolboy injuries are more common during the early season and after the mid-year winter vacation, with more injuries occurring during matches than practices (Nathan et al., 1983; Roux et al., 1987). Using self-reports from athletes and based on their survey of three South African school's top teams, Shuttleworth-Edwards et al. (2004) recorded an incidence of 2.3 concussions per rugby playing schoolboy. This compares with an average incidence of 0.4 concussions for an equivalent group of field-hockey players.

## **6.2 OUTCOME FOLLOWING MTBI IN CONTACT SPORT**

The following section will review the effects of MTBI in respect of neurocognitive sequelae and postconcussive symptoms for the different contact sport studies of boxing, ice hockey, soccer, American football, Australian Football League, Rugby League and Rugby Union. The sport studies investigating these effects in athletes will be discussed separately for the different levels of play in terms of professional and elite athlete groups compared to the amateur, collegiate and high school athlete groups. It should be noted however, that in some cases there is blurring between these groupings as studies have combined both amateur and professional athletes into their samples.

### 6.2.1 Outcome following MTBI in boxing

(For summary of outcome studies see Table 6.2.1, pp. 207 - 211)

Boxers are subjected to both acute and chronic neurological injuries, although research suggests that permanent and irreversible neurological dysfunction rarely occurs amongst amateurs (Heilbronner & Ravdin, 2004). According to Porter & O'Brien (1996), amongst amateur boxers cerebral injuries, which occur almost exclusively in competition, are mainly mild concussions. It is important to differentiate between amateur and professional boxers because although the mechanism of injury remains the same for both groups, amateur boxers unlike professionals, have fewer and shorter fights, engage in less sparring and are protected by a more stringent set of rules (Brooks, Kupshik, et al., 1987; Lampert & Hardman, 1984). Whilst much of the research examining the neurological effects in boxing has focussed on professional boxers, only recently has the interest turned towards amateurs (Heilbronner & Ravdin, 2004). Researchers have delineated three major forms of brain damage amongst boxers (to be discussed below) as follows: (i) Acute neurologic injuries, (ii) Groggy states and the postconcussive syndrome and (iii) Boxer's chronic traumatic brain injury or what is commonly referred to as the 'punch-drunk syndrome' (Heilbronner & Ravdin, 2004).

(i) The most common acute neurological injury in boxing is the knockout which is defined as an impairment in neurological function secondary to mechanical forces that result in either unconsciousness or a groggy (dazed) state (Jordan et al., 1997). According to the Council on Scientific Affairs (1983) the incidence of knockouts is six per 100 bouts. A knockout is synonymous with a grade II or grade III concussion on the traditional grading scales and the unconsciousness produced by the knockout blow is unusual in that its onset is abrupt, it is of short duration and complete recovery occurs (Heilbronner & Ravdin, 2004). Author's caution that the true rate of concussive blows may be underestimated which means that it is not the concussive blows which are responsible for persistent cognitive deficit but rather the cumulative effects of sub-concussive blows (events similar to those which result in concussion, but which involve smaller forces) (Heilbronner & Ravdin, 2004).

(ii) The "groggy state" in boxing is typically referred to as the postconcussion syndrome and occurs between the knockout and the manifestation of multiple head injuries (Heilbronner &

Ravdin, 2004). With this condition the boxer experiences persistent postconcussive symptoms in the form of severe headaches, dizziness, imbalance, irritability, memory problems and dysarthria which may last days or weeks after a fight.

(iii) Chronic traumatic brain injury (cTBI) also referred to as “dementia pugilistica” (Millsbaugh, 1937), is the devastating result of repeated TBI over a period of time, as well as the fact that the resulting cumulative deficit can predispose individuals to the early onset of dementia (Heilbronner & Ravdin, 2004). Manifestations of cTBI have been observed in many studies on professional boxers suggesting that cTBI is positively correlated with the frequency and degree of boxing related injuries as measured by the number of bouts, sparring exposure and neuroimaging abnormalities (Casson et al., 1984, Jordan, Matser, et al, 1996; Ross et al., 1987). There is therefore a need to differentiate between research investigating the effects of MTBI amongst amateur and professional boxing because of the shorter duration of bouts and strict medical supervision of the former (Butler et al., 1993).

#### **6.2.1.1 Professional and former boxers**

Early investigations into former professional boxers suggest a high incidence of neurological and neurocognitive dysfunction (Casson et al., 1982; Jordan et al., 1992; Kaste et al., 1982). Kaste et al. (1982) evaluated 14 boxers who had been Finnish, Scandinavian and European champions, eight of which were amateurs and six were professionals, averaging 31 years of age. Tests included Computed Tomography (CT) scanning, electroencephalograms (EEGs), a neurological examination and neurocognitive tests including the Wechsler Adult Intelligence Scale (WAIS) subtests: Information, Similarities, Digit Span, Block Design and Object Assembly, the Wechsler Memory Scale (WMS) subtest- Associate Learning, the Wisconsin Card Sorting Test (WCST), the Trail Making Test, Purdue Pegboard, and the Benton Visual Retention Test. The results showed that only one of the 14 boxers examined (professional) had neurological deficits indicative of boxer’s encephalopathy. Whilst Computed Tomography (CT) imaging revealed brain atrophy in three of the six professional boxers and in one of the eight amateurs, and EEG abnormalities were presented in six of the 13 boxers, two of the professionals and four of the amateurs. Twelve of the boxers took a significantly longer time to complete the Trail Making Test (sustained attention, concentration and cognitive flexibility) and two of the professional

boxers performed below average on all of the neuropsychological measures as well as demonstrated subjective symptoms but which were not specified in the study.

Similarly, this level of impairment was demonstrated in a study by Casson et al. (1982), who investigated the chronic effects in 10 professional boxers between the ages of 20-31 years in the United States. All boxers underwent detailed neurological examinations and EEG and CT scans shortly after being knocked out. Although intracerebral and subdural haematomas were not present, CT abnormalities revealing cerebral atrophy were discovered in five of the 10 boxers. This finding was most common in boxers with the most bouts and since none of the boxers were knocked out more than twice in their careers, the authors concluded that the cumulative effects of sub-concussive blows to the head were most likely the cause of the abnormal findings. A further study supporting neurological deficit in professional boxers involved the CT imaging of 338 active professional boxers in the United States (Jordan et al., 1992). The results demonstrated that 25 (7%) boxers had abnormal scans, whilst 37 (49%) boxers had borderline CT scans. Seventeen (68%) boxers with abnormal scans reported a previous knockout compared with only 89 (37%) of the 238 boxers with normal CT scans. The most common CT abnormality was brain atrophy found in 22 cases.

Two years after their study in 1982, Casson and colleagues (1984) studied 18 active and former boxers between the ages of 25 to 60 years in the United States, who in addition to neurological examinations, EEG tracings and CT imaging, also underwent neurocognitive testing. The researchers found that 87% of all boxers exhibited abnormal findings on at least two of the four examinations. Eight boxers had abnormal CT scans and of those six experienced more than 20 professional bouts. Seven boxers had abnormal EEG readings and five had abnormal findings on the neurological examination. Each boxer scored in the impaired range on more than one neurocognitive measure which included the Trail Making Test, the WAIS subtest – the Digit Symbol Substitution Test, the WMS and recall of the Bender Visual-Motor Gestalt figures. They found that the percentage of neurocognitive test scores correlated significantly with the abnormal CT scans, age and the number of fights of the boxers, thus lending further support to the relationship between the length of exposure to the sport and the presence of brain damage.

Researchers Ross et al. (1987) examined the cognitive and neurological functioning of 38 professional boxers in the United States, between the ages of 21 to 74 years, on the neurocognitive tests measures of the Trail Making Test, the WAIS- Digit Symbol Substitution Test, the WMS and the Bender Gestalt Test. Fourteen of the 15 former and active professional boxers scored abnormally on the recall of the Bender-Gestalt Test. All 13 boxers scored in the abnormal range on verbal portion of the WMS and 11 of the 13 boxers scored in the abnormal range on the visual portion of the WMS. Ninety percent of the memory scores fell in the abnormal range and 50% of the non-memory scores fell in the abnormal range. Every boxer who had either an abnormal CT scan or EEG had at least one abnormal memory test score whereas this did not hold true for the non-memory tests. Ross et al. (1987) however, caution that no single neurocognitive test is a strong predictor of an abnormal CT scan or EEG result but it is the overall pattern of impairment that is strongly associated with the abnormal findings. Years later, Jordan, Matser et al. (1996) examined the relationship between the history of sparring and cognitive performance amongst 42 professional boxers with an average age of 25.6 years in the United States. Neurocognitive tests included the WAIS subtests (Similarities, Vocabulary and Digit Symbol Substitution Test), the Verbal Fluency test, the Figure Detection test, the PASAT, the WMS, the Stroop Color and Word Test, the Rey Complex Figure, the WCST, the Trail Making Test, Puncture and Bourdon. Seventeen of the boxers had CT scans that revealed borderline brain atrophy and deficits on the Rey Complex Figure (visual perception), the Stroop Color and Word Test (cognitive flexibility and executive functioning), Verbal Memory and Visual Reproduction subtests of the WMS which were related to indices of increased sparring exposure.

The correlation between increased sparring exposure and chronic deficit was further investigated by Jordan et al. (1997) the following year using a sample of 30 professional boxers between the ages of 23 to 76 years in the United States. Jordan and his colleagues investigated the relationship between the Apolipoprotein E e4 (APOE e4) genotype and measures of the Chronic Brain Injury (CBI) scale. The CBI scale is a rating scale measuring motor, cognitive and behavioural functioning on a 10-point scale ranging from normal to mild, to moderate, to severely impaired. Amongst the boxers, 11 were normal, 12 showed mild impairments, four were moderately impaired and three showed severe impairments. High exposure boxers had significantly higher CBI scores, moreover high exposure boxers with the APOE e4 allele had

significantly higher CBI scores than high exposure boxers without the APOE e4 allele. All boxers with severe impairment possessed at least one APOE e4 allele. The results suggest that the possession of the APOE e4 allele is associated with increased chronic neurological deficits amongst high-exposure boxers and provides support for the BRC theory (see Chapter 3, section 3.6) in that the additive effects of cumulative MTBI combined with a genetic risk factor (APOE e4) are predictive of a worse outcome.

Whilst the studies reviewed above all support chronic deficit in respect of neurological and neurocognitive sequelae amongst boxers, none of the studies made use of control groups. Researchers Drew, Templer, Schuyler, Newell, & Cannon (1986) in the first boxing study to include a matched control group, compared the cognitive functioning of 19 active professional boxers to 10 matched control athletes participating in baseball and basketball, in the United States. Tests included subtests of the Quick Neurological Screening Test, subtests of the Halstead-Reitan Neuropsychological Test Battery (which includes the Category Test, Tactual Performance Test, Seashore Rhythm Test, Speech Sound Perception Test and the Finger Tapping Test), and the Randt Memory Test. Their test performances showed that 15 of the 19 boxers scored in the impaired range on the Reitan Impairment Index (compared to two controls) and that the boxers were more impaired than controls on all tests except the Seashore Rhythm, Finger Tapping and Category Test.

In contrast to the above studies that provide compelling support for the chronic and residual deficits in respect of MTBI in professional boxers, some researchers have found minimal effects, although this appears to be the exception. Researchers Levin, Lippold, et al. (1987) investigated the neurobehavioural functioning and magnetic resonance imaging of 13 boxers (11 active professionals and two amateurs) compared with a matched control group of 13 sportsmen, with an average age of 20 years in the United States. All athletes ( $n = 26$ ) underwent initial neurocognitive testing and 10 athletes in each group were tested again six months later. Nine boxers underwent magnetic resonance imaging (MRI). The neurocognitive tests consisted of measures of attention, information processing speed (Paced Auditory Serial Addition Test, Continuous Performance Test and a microcomputer reaction time task), memory (Modified Selective Reminding Test, Digit Span test and the Benton Visual Retention Test), reasoning (Controlled Word Association Test, Design Fluency) and visuomotor coordination and speed of

processing (Digit Symbol Test, Trail Making Test and Purdue Pegboard). The results from the initial testing demonstrated more proficient verbal learning in the control group, whereas there were no differences between the groups on the delayed memory recall and other tests of memory. Reaction time was faster in the boxing group compared with the controls. Six months after the initial baseline testing, 10 subjects in both the boxing and control groups demonstrated improvements in their neurocognitive test performances compared with their initial baseline performance, indicating a practice effect, with no differences in scores or in the magnitude of improvement between the groups. The MRI findings were also normal in the nine boxers evaluated. Methodologically this study appears less robust in terms of evaluating professional boxing in that the sample was small and that two out of the eleven boxers were amateurs. This may account for its negative results.

In one of the only studies to investigate the acute effects of MTBI amongst professional boxers Ravdin et al. (2003) assessed the cognitive recovery following concussion in 18 active professional boxers between the ages of 20 and 40 years in the United States. Testing was performed before (baseline) and at three points (approximately 4 days, 10 days and 1 month) after a professional bout. Tests included the subtests of the WAIS-Revised including the Digit Span, Vocabulary and Block Design subtests, the Hopkins Verbal Learning Test, Grooved Pegboard, the Trail Making Test, the Controlled Oral Word Association Test (COWAT) and the Stroop Color Word Test. The repeated measures revealed significant within subject differences across testing of verbal fluency (COWAT) and information processing (Trail Making Test) between the high exposure and the low exposure groups in the direction of the high exposure group performing worse. Significant differences were noted between baseline and one-month post bout testing, with scores one month following a bout significantly improving. This finding is consistent with other sports research investigating the acute effects of concussion (e.g. Dicker & Maddocks, 1988; McCrea, Kelly, Randolph, Cisler, & Berger, 2002; Iverson et al., 2004b; Schretlen and Shapiro, 2003; Wilberger et al., 1991). Although memory scores did not change across testing, prior boxing exposure was associated with poorer memory performance.

In summary, in spite of many methodological issues associated with the above studies for example, the lack of control groups (Casson et al., 1984; Jordan, Matser et al., 1996; Jordan et al., 1997; Kaste et al., 1982; Ravdin et al., 2003; Ross et al., 1987) small and mixed sample

groups (Casson et al., 1984; Drew et al., 1986; Kaste et al., 1982; Levin, Lippold et al., 1987; Ravdin et al., 2003; Ross et al., 1987), as well as the lack of investigation into the self-reporting of postconcussive symptoms (Casson et al., 1984; Drew et al., 1986; Jordan, Matser et al., 1996; Kaste et al., 1982; Levin, Lippold et al., 1987; Ravdin et al., 2003; Ross et al., 1987), the research reviewed above provides substantial support for the chronic effects, in respect of neuropsychological and neurological sequelae, associated with the long-term exposure to boxing amongst professional athletes. Further, the research consistently suggests that the longer the length of exposure to boxing, together with an increase in the number of bouts, the greater the impairment.

### **6.2.1.2 Amateur boxers**

Although the above findings seem to be highly consistent in professional boxers, amongst amateur boxers conflicting results have been reported. Studies involving Swedish, British, American and Danish amateur boxers all reported no significant abnormalities on CT, EEG, auditory evoked potentials or neurological assessments, suggesting that amateur boxing does not produce observable changes in neurological functioning (Butler et al., 1993; Casson et al., 1984; Haglund & Bergstrand, 1990; Moriarity et al., 2004; Murelius & Haglund, 1991). Conversely, in Kaste et al's (1982) study investigating both amateur and professional boxers, even though the professional boxers sustained more impairment than the amateur boxers, the amateur boxers were already starting to show subtle deficits as evident on neurocognitive and neurological tests. Researchers report that this damage is related to the length of exposure to the sport (Pearn, 1998). Whilst most of the studies on professional boxers have focussed on the chronic effects of MTBI in respect of neurocognitive deficits in professional athletes, studies on amateur athletes have investigated both the acute and chronic effects of MTBI. Studies investigating the acute effects will be reviewed first followed by those investigating the chronic effects.

Amongst amateur boxers, studies investigating the acute effects in respect of neurocognitive sequelae and postconcussive symptoms are consistent with other contact sports studies in that the acute effects usually resolve within three months post-injury (e.g. Bleiberg et al., 2004; Dicker & Maddocks, 1988; McCrea et al., 2002; Iverson et al., 2004; Schretlen and Shapiro, 2003; Wilberger et al., 1991). Heilbronner et al. (1991) investigated the effects of boxing in 23 amateur

boxers between the ages of 16 to 30 years in the United States before and after a boxing match using the tests Digit Symbol Incidental Recall – Immediate and Delayed, Visual and Verbal recall of words and symbols, psychomotor speed (hand tapping), the Trail Making Test and Digit Span. Their findings demonstrated that compared with their pre-fight performances minor deficits in verbal learning and motor speed were detected in the boxers. Moreover, boxers with the greater number of years of exposure to boxing had slower finger tapping speeds as well as lowered scores on the Digit Symbol Incidental Recall – Immediate and Delayed.

Ramirez (1998) investigated attentional deficits in youth boxing (13 to 17 years) following repeated concussions in a group of 10 amateur boxers and 10 basketball players in the United States, one hour after completion of the tournament and again eight weeks later. Attentional disruptions were measured by four variables of the Test of Variable Attention (TOVA). The results indicated that the cumulative effects of the head blows during the season did not have a significant effect on the TOVA measures of inattention, impulsivity or information processing and motor response speed. Variability of response time was the only index by which the mean score of the boxers differed from the norm, and consistency of attention the only variable affected by the number of head blows. The boxers reaction time was also lower than the basketball players at the end of the season as well as eight weeks later. Similarly, Warden et al. (2001) examined the effects of concussion on reaction time in a group of 14 cadets concussed whilst boxing during physical education, with an average age of 18.55 years in the United States. The cadets were assessed one hour after injury and then again four days later using the computerized neurocognitive assessment battery ANAM (Automated Neuropsychological Assessment Metrics). The results showed significant slowing on their post-injury reaction time and continuous performance tests, even after the cadets experienced symptom resolution and were permitted to resume all activities.

Bleiberg et al. (2004) investigated the cognitive functioning in 64 cadets (mean age = 18.8 years) who sustained concussions and 18 controls (mean age = 19.5 years) in the United States. Using the computerized neurocognitive assessment test battery, the Automated Neuropsychological Assessment Metrics (ANAM), which consists of five tests of Simple Reaction Time, Sternberg Memory, Math and Spatial Processing and Matching to Sample, the athletes were assessed at pre-season and then again at post-injury intervals of 0 to 23 hours, one to two days, three to

seven days and at 8 to 14 days post-injury. The results indicated that only one out of the five subtests (Spatial Processing) yielded a significant group difference between 0 to 23 hours and one to two days post-injury, and another subtest (Mathematical Processing) on the 0 to 23 hours test interval. Recovery of cognitive performance amongst all boxers occurred at seven days post-injury.

Using another computerized neurocognitive test, CogSport, Moriarity et al. (2004) assessed 82 collegiate amateur boxers and a group of 30 matched controls in the United States, in order to determine whether participation in a seven-day amateur boxing tournament was associated with acute deterioration on cognitive testing. No age range was given for this sample. Tests of simple reaction time, choice reaction time, working memory and learning were administered. The 82 boxers fought 159 times and testing was performed after 142 of these bouts. Seven boxers whose bout was stopped by the referee showed significant slowing in simple and choice reaction time, although they were not clinically diagnosed with concussion. None of the others displayed any evidence of cognitive dysfunction in the immediate post-bout period.

Butler et al. (1993) investigated the acute and chronic effects of MTBI amongst 86 amateur boxers between the ages of 12 and 27 years, compared with 47 rugby union players and 31 water polo player controls in the United Kingdom. All athletes were evaluated on three occasions: pre bout, immediate post bout and then followed up within two years. The neurocognitive tests consisted of the British Ability Scales, the PASAT, and the immediate and delayed recall of words and visual stimuli. No evidence of neurocognitive dysfunction due to boxing was found, either following a bout or a series of bouts at follow up. Factors such as the number of previous contests, recovery from an earlier bout, number of head blows received during a bout and the number of bouts between initial assessment and follow up, were found to be related to changes in cognitive functioning.

With respect to investigations into the chronic effects of MTBI, McLatchie et al. (1987) detected some neurological and neurocognitive dysfunction in their group of 20 active amateur boxers between the ages of 18 and 49 years in Scotland, compared to an equal number of orthopaedic outpatients. Tests included EEG and CT investigations as well as neurocognitive tests such as the Digit Span subtest of the WAIS, the three paired word learning test of Inglis (1959), word

learning from the WMS, the Complex Figure Test, the PASAT and Simple and Four Choice Reaction Time test. Neurological abnormalities were seen in seven boxers, one had an abnormal CT scan and eight had abnormal EEG readings. Nine of the 15 boxers performed poorly on neuropsychological testing of verbal learning (three paired word learning test of Inglis) and visual memory (immediate recall of Complex Figure).

Brooks, Kupsik, et al. (1987), however found no evidence of impairment in their investigation of 29 amateur boxers and 19 controls matched for age (15 to 27 years of age), education and race (11 of whom were prospective amateur boxers in training) in Scotland, although their sample consisted of a younger age group. Neurocognitive tests included WMS-Logical Memory and Associate Learning, the Digit Symbol Test, the Rey Complex Figure copy and recall, PASAT, the Simple and Four Choice Reaction Time Test. The results concluded that there was no evidence of impairment amongst the amateur boxers compared with the controls but one could argue for the lack of evidence here on the basis of a contaminated control sample group which consisted mostly of boxers in training. Thus, whilst the controls level of exposure to the sport may have been less than the amateur boxers the number of concussive and sub-concussive blows experienced amongst both groups was unreported and hence could have contributed to an insignificant result between groups.

Similarly, Stewart et al. (1994) conducted a two-year prospective study on 484 active amateur boxers in the United States who were examined at baseline and then 81% of them again two years later. All athletes were between the ages of 13 to 21 years. Tests included a neurological examination, EEGs, auditory evoked potentials and neurocognitive tests consisting of the Grooved Pegboard, the Rey Complex Figure, the Rey Auditory Verbal Test (RAVLT), the Digit Span test, Ravens Matrices, Block Design, Word Fluency Test, the Symbol Digit Modalities Test, Alphabet Recital, the Trail Making Test, the Two-Choice Reaction Time Test and Vigilance Test. Although no significant changes were detected over the course of the study, significant trends between the number of bouts prior to baseline testing and changes in memory and visuomotor construction ability were found on the Block Design subtest of the WAIS.

Further studies investigating the chronic effects amongst amateur boxers have also produced inconsistent test results with respect to neurocognitive investigations, with some measures (e.g.

the Finger Tapping Tests) demonstrating greatest sensitivity over others. For example, Murelius & Haglund (1991) investigated the effects of boxing on 50 Swedish amateur boxers (25 athletes who fought in a high number of matches, age = 26 to 41 years and 25 who fought in relatively low numbers of matches, age = 26 to 43 years), using a control group of 25 soccer players (age = 25 to 44 years) and 25 track and field athletes (age 25 to 44 years). The neurocognitive battery comprised of sensory, motor, cognitive and memory tasks from the Synonyms Test (SRB) and Halstead-Reitan batteries. Although none of the boxers were found to have definite signs of intellectual impairment, boxers who had taken part in a large number of bouts had a slightly inferior finger tapping speed compared to the controls, although this was still within the normal range. Consistent with these findings, Porter and Fricker (1996) investigated the association between amateur boxing and chronic traumatic encephalopathy over a period of 15 to 18 months in a group of 20 active competing amateur boxers and 20 controls, in Australia. All subjects were between 16 to 25 years of age. The neurocognitive test battery included the Trail Making Test, the Digit Symbol Substitution test, Finger Tapping tests, modified versions of the Finger Tapping tests, the WMS Paired Associate test, the Rey-Osterrieth Complex Figure Test. At the end of the study the boxers performed significantly better than the controls on the Trail Making Test, whereas the control's scores for the Finger Tapping Tests were significantly higher than those of the boxers. There was no association between boxing exposure and the performances in any of the neuropsychological tests used, however.

In a follow-up study, Porter (2003) investigated the neurocognitive functioning in the same sample group reported on above (20 active boxers and 20 controls), using the same test measures (Trail Making Test, the Digit Symbol Substitution Test, Finger Tapping tests, modified versions of the Finger Tapping tests, the WMS Paired Associate test, and the Rey-Osterrieth Complex Figure Test). Consistent with the findings in the 1996 study, the boxers demonstrated superior performance on the Trail Making Test and an inferior Finger Tapping speed performance on all test points. The controls however, demonstrated a significant deterioration in their performance on the Rey-Osterrieth Complex Figure test. Generally, the study showed no deterioration in the neurocognitive test performances of the boxers over nine years.

In summary, the studies investigating the effects of MTBI amongst amateur boxers are inconsistent, with some researchers reporting deficits (e.g. Heilbronner et al., 1991; Kaste et al.,

1982; McLatchie et al. 1987; Moriarity et al., 2004), whilst others reporting minimal or no significant effects (e.g. Brooks, Kupshik, et al., 1987; Butler et al., 1993; Murelius & Haglund, 1991; Porter, 2003; Porter & Fricker, 1996; Ramirez, 1998; Stewart et al., 1994). Generally, in comparison to studies on professional boxers, more amateur studies are reporting deficits at the acute phase following MTBI than at the chronic phase. Moreover, more studies investigating the chronic effects amongst amateurs report minimal or no deficits compared with studies on professional boxers which almost exclusively reveal deficits. This is consistent with the limited length of exposure to the sport amongst amateurs in comparison to the professional boxer, where deficit can be robustly demonstrated in most cases.

Generally with respect to both professional and amateur boxers, the cognitive domains of sustained attention, processing speed, verbal fluency, verbal and visual memory and executive functioning appear to be the most sensitive to the effects of MTBI, in both the acute and chronic condition (e.g. Casson et al., 1984; Jordan, Matser, et al., 1996; Kaste et al., 1982; McLatchie et al., 1987; Moriarity et al., 2004; Ross et al., 1987). Whilst there appears to be much research about the neurological and neurocognitive effects of multiple sub-concussive blows and repeated bouts, as well as the length of exposure to boxing (e.g. Casson et al., 1984; Drew et al., 1986; Jordan, Matser, et al., 1996; Kaste et al., 1982; Ravdin et al., 2003; Ross et al., 1997), the issue of postconcussive symptomology has largely been unexplored amongst both professional and amateur boxers. According to Jordan and colleagues (1997), the true frequency of postconcussive symptoms amongst boxers following a bout remains unknown. This could mostly be attributed to the underreporting of symptoms amongst boxers and also holds true for athletes in other contact sports.

To the authors knowledge there are no studies investigating the outcome following MTBI amongst the martial arts in terms of neurocognitive or postconcussive symptom outcome.

### **6.2.2 Outcome following MTBI in ice hockey**

(For summary of outcome studies see Table 6.2.2, pp. 211 - 213)

There are few studies investigating the effects of concussion amongst ice hockey athletes, with most studies reporting on the chronic effects in respect of neurocognitive and postconcussive

sequelae. The lack of studies is concerning considering that researchers have reported that overall ice hockey has shown to have the highest incidence of concussion amongst the team sports (American football, rugby and soccer) for high school male athletes, whilst at the professional level ice hockey and rugby had similar incidence rates (Koh et al., 2003). In contrast to the sport of boxing, however, to the author's knowledge there are no studies investigating the effects of MTBI amongst professional ice hockey athletes. Although the NHL and the NHL Player's Association have completed a 5-year clinical research project, namely "The National Hockey League Neuropsychological Testing Programme", the results from that study are in process (Lovell, Echemendia & Burke, 2004).

Researchers Ferguson et al. (1999) investigated the role of expectations in the symptom reports of 209 postgraduate, collegiate and high school male athletes between the ages of 19 and 21 years in the United States. The athletes were divided into two groups depending on their MTBI history. Fifty athletes reported having sustained a sports-related concussion in the past six months and consisted of the following sports groups: ice hockey (56%), American football (16%), rugby football (14%), lacrosse (12%) and soccer (2%). The non-injured control group consisted of 159 athletes consisting of the following sports groups: ice hockey (49%), American football (23%), lacrosse (13%), soccer (9%) and rugby football (6%). All athletes completed a 30-item symptom checklist consisting of symptoms relating to concussion including memory difficulties, affective and somatic symptoms and difficulties with attention and concentration. The results showed that the MTBI athletes symptom rates did not differ from controls. The MTBI athletes did however, consistently underestimated the incidence of symptoms pre-injury. Athletes with no history of MTBI overestimated the degree of pre- to post-injury change in symptom reporting. The authors concluded that symptom expectation as a psychological variable has a significant effect on the perception of persistent PCS, which means that athletes behaved in a way consistent with expectations for increased symptoms following MTBI (Ferguson et al., 1999).

Researchers Gaetz et al. (2000) investigated the cumulative effects of concussion amongst 271 junior ice hockey players using event-related potential recordings and postconcussive symptom reporting in Canada. No age range was given for the group. All players were assessed at the beginning of the 1998 to 1999 season and were assigned to one of four groups based on their

concussion history: non-concussed athletes (0), athletes who had experienced one concussion (1), athletes who had experienced two concussions (2) and athletes who experienced more than three concussions (3+). The numbers in each group were not given. All athletes were assessed pre-injury and at least six months post-injury using a postconcussion symptom questionnaire and event related potential recordings which involves the random presentation of mixed target and non-target stimuli. The results showed that athletes who sustained three or more concussions took significantly longer to respond to the stimuli on the event related potentials. Moreover, significant differences in the symptoms of 'memory' and 'taking longer to think' were reported for this groups which correlated with their slow responses on the event-related potential recordings.

Hatfield et al. (2004) investigated the effects of concussion amongst a group of 44 amateur ice hockey athletes between the ages of 15 to 18 years in the United States. All athletes were interviewed about their concussion history, prior to baseline assessment. Fifteen athletes reported that they experienced a prior concussion, and of those five players reported experiencing two or more concussions. All athletes were evaluated at baseline and then re-evaluated at 24 to 72 hours after sustaining a concussion (one player was evaluated at nine days post-injury), and then at regular time intervals until their scores had approximated their baseline scores. The neuropsychological test battery consisted of 14 measures which included the Peabody Picture Vocabulary Test III, the Digit Span test, the Symbol Digit Modalities test, the Stroop test, the Trail Making Test, the Selective Reminding Test, Visual Spatial Learning Test, the Controlled Oral Word Association Test, Animal Naming, Grooved Pegboard, Finger Tapping Test, Grip Strength Test, Lafayette Multi-Choice Reaction Timer and the Symptom Check List-90-Revised. The results demonstrated that at baseline there were no significant results between those who had experienced a concussion and those who did not, with only Digits Backwards (attention) approaching significance in the direction of those athletes with prior concussions performing worse. Six athletes sustained concussions over the season and were subjected to repeat testing, with the time period for return to play ranging from 5 to 175 days (approximately 6 months). The results indicated that tests demonstrating declines from baseline levels varied amongst the concussed athletes, although tests of complex attention (Trail Making Test, Digit Span test, the Symbol Digit Modalities Test) and memory (Selective Reminding Test, Visual Spatial Learning

Test) appeared to be the most affected. Although a symptom checklist was employed in this study it was not reported on.

Killam et al. (2005) examined the residual neuropsychological effects of head trauma in 28 male and female college athletes with ages ranging from 21 to 22 years, in the United States. The athletes participated in the sports of ice hockey, field hockey, lacrosse and soccer and were assigned to one of three groups: non-athlete non-concussed ( $n = 8$ , male = 5, female = 3), athlete non-concussed ( $n = 9$ , male = 6, female = 3), athlete non-recent concussed ( $n = 6$ , male = 3 and female = 3) and athlete/recent concussed ( $n = 5$ , male = 3, female = 2). Non-recent concussion was defined as being more than two years since the last diagnosable concussion and recent concussion was defined as being two years or less since the last diagnosable concussion. All athletes completed the Repeatable Battery for the Assessment of Neuropsychological status (RBANS) which consists of five cognitive domains (immediate memory, visuoconstruction/spatial ability, language, attention and delayed memory), the Stroop test and a Postconcussion Syndrome Checklist.

The results demonstrated that no significant group effects were noted for the cognitive domains of visuoconstruction, language and attention domains of the RBANS, although significant effects were noted for the immediate memory scores and total RBANS scores. Relative to the non-athlete group, the athlete/non-concussed and athlete/recent concussed groups were impaired in the immediate and delayed memory domains. Further, the total RBANS score was significantly lower in the athlete non-concussed, athlete non-recent concussed and athlete/recent concussed groups. The worst impairment was shown in the athlete/recent concussed group for the immediate memory domain. No significant differences on the scores obtained from the Postconcussion Syndrome Checklist and on the Stroop task were detected between the groups. The Postconcussion Syndrome Checklist correlated negatively with the attention, delayed memory and total scores from the RBANS and the higher number of concussions correlated with processing speed on the Stroop task. The authors comment that what was surprising in the study were the impairments in memory in athletes who never reported having sustained a concussion. Whilst the results could reflect the underreporting of symptoms in athletes, the authors assert that mere participation in a contact sport produces a degree of 'subclinical neuropsychological vulnerability' in these athletes.

In summary, consistent with the boxing studies reviewed above, the studies on ice hockey predominantly investigated the chronic effects of MTBI, in respect of neurocognitive and postconcussive sequelae, although only high school and collegiate athletes were evaluated. In the one study (Hatfield et al., 2004) that reported on both the acute and chronic effects amongst high school athletes, the results did not altogether support other MTBI research reporting on the acute recovery of boxers (Bleiberg et al., 2004; Ravdin et al., 2003) where recovery took place between seven days and one month post-injury. Whilst some athletes did recover before three months, others took longer than three months to recover, with total recovery of all athletes taking place in just over six months. Although the sample size was very small ( $n = 6$ ), this finding could largely be due to the fact that some of these athletes had a history of prior concussions resulting in a delay in the recovery process. For the studies that reported on the chronic effects, the results support the inconsistent findings in boxing investigating these effects in amateur athletes, with two of the above studies reporting deficits (Gaetz et al., 2000; Killam et al., 2005) and the other two studies reporting none (Ferguson et al., 1999; Hatfield et al., 2004). In addition, consistent with the boxing studies reported above, the cognitive domains of attention, memory and information processing speed are the most sensitive to the effects of MTBI. Although, unlike the boxing studies reporting on amateur athletes where no postconcussion inventories were employed, all of the above studies on ice hockey reported using postconcussion inventories. One study reported on the individual symptoms of 'memory' and 'taking longer to think' as being significant (Gaetz et al., 2000), whilst two reported on the symptom rates between athlete which were not significant (Ferguson et al., 1999; Killam et al., 2005). For the other study although a postconcussive inventory was employed this was not reported on in the total sample group (Hatfield et al., 2004). Methodological issues such as lack of control groups (Gaetz et al., 2000; Hatfield et al., 2004) and small sample sizes (Killam et al., 2005) limit the generalizability of the studies.

### **6.2.3 Outcome following MTBI in soccer or football**

(For summary of outcome studies see Table 6.2.3, pp. 214 - 217)

Head trauma in soccer is likely to be due to either 'unintentional' head impacts which involve collisions with body parts of other players involving head to head, or head to elbow blows,

collisions with the playing surface, goal posts, or even the ball when it is moving at great speed, or as a result of sub-concussive head trauma predominantly caused from the intentional use of the head to play the ball, termed heading (Rutherford et al., 2005; Webbe & Ochs, 2003). It is very difficult to determine whether the effects that may occur over the course of a soccer player's career, are as a result of sub-concussive trauma seen in the form of heading or concussive injuries, and sub-concussive repetitive trauma that occur as a result of general play (Rutherford et al., 2003). For over a century heading was not considered to be a serious event that could result in head trauma, although professional soccer players have been observed to head the ball between 6 to 16 times during a match, which it has been suggested, translates to a substantial cumulative effect over a 10 to 15 year career (Bangsbo, Norregaard, & Thorsoe, 1991, Rutherford et al., 2005). Other researchers postulate that heading the ball is unlikely to be a significant mechanism for concussion (Boden et al., 1998; Kirkendall et al., 2001) and that concussions due to collisions between players may be more influential and produce more substantial effects than the sub-concussive effects of heading (Barnes et al., 2002). Whether a relationship exists between sub-concussive heading and chronic cognitive impairment, however remains unknown (Shewchenko, Withnall, Gittens, & Dvorak, 2005).

As in the boxing literature, generally researchers of soccer argue that the longer the exposure to the sport and the greater the intensity of play, the greater the level of deficit. For these reasons the studies will be divided into those which investigate professional, former and elite soccer players and those which examine amateur, collegiate and high school soccer players, although as previously mentioned for boxing, sometimes these divisions are not always clear-cut as some researchers combine both professional and amateur athletes in their sample groups. To the author's knowledge studies investigating the effects of MTBI in soccer, with respect to the neurocognitive and postconcussive sequelae have focussed predominantly on the chronic effects with few studies investigating the acute effects.

#### **6.2.3.1 Professional, former and elite soccer players**

Neurological investigations of former and professional soccer players have produced some consistent results. In a study by Sortland and Tysvaer (1989), thirty-three former soccer players between the ages of 39 and 68 years from Norway, were examined for cerebral atrophy using CT

imaging. Cortical atrophy was found in 18% of the players and the researchers concluded that the brain damage was as a result of sustaining multiple head trauma as a consequence of playing soccer for many years. Similarly, Tysvaer and Storli (1989) compared 69 Norwegian First Division League Club's soccer players to a control group of 69 men of differing occupations, between the ages of 15 and 34 years. Both groups underwent neurological and EEG examinations and an increased incidence of EEG abnormalities was found in the soccer players compared to the control group.

With regards to the investigation of the neurocognitive effects amongst former and professional soccer players the results have also been relatively consistent. Tysvaer and Lochen (1991) evaluated 37 former Norwegian national soccer players between the ages of 35 and 65 years and a control group consisting of 20 hospitalised patients between the ages of 31 and 65 years. Tests included the WAIS-Revised, the Trail Making Test and the Benton Visual Retention Test. Relative to the controls, the soccer players consistently showed higher proportions of impaired functioning especially on the WAIS-R in terms of the difference between the mean Verbal and Performance Scale scores (Verbal Scale IQ = 117 and the Performance Scale IQ = 109), and on some of the WAIS-R subtests, with the soccer players displaying greater difficulty with the Digit Span (attention and working memory) and Block Design (visual-spatial ability) subtests. The soccer players also displayed impaired functioning on the Trail Making Test relative to controls. All the effects were attributed to heading.

Further deficits in respect of neurocognitive effects were demonstrated by Matser and his colleagues (1998) who investigated the chronic effects of TBI in a group of 53 professional soccer players and 27 elite swimming and track athlete controls, with a mean age of 25 years in the Netherlands. Neurocognitive test measures included the Bourdon-Wiersma test, the PASAT, the Stroop Colour Word Test, the Trail Making Test, the Wisconsin Card Sorting Test (WCST), Digit Span test, the Facial Recognition Test, the Figure Detection test, the Complex Figure Test, Verbal Fluency, Puncture Test, Total Raven Score, the Digit Symbol Substitution Test, the WMS Associate Learning, Logical Memory and Visual Reproduction tests and the 15 Word Learning Test. The soccer players exhibited impaired performances on tests of memory, planning and visuoperception (the Bourdon-Wiersma test, the WCST, the Complex Figure Test, Total Raven Score, the WMS Associate Learning, Logical Memory and Visual Reproduction tests and the

Figure Detection test). When adjustments were made for potential confounding variables such as prior unrelated concussions, alcohol consumption, level of education and number of general anaesthetics to the groups, and comparisons were made again between the groups across all measures, significant decrements were seen on the Figure Detection test, the Complex Figure Test, the WMS Logical Memory and Visual Reproduction test scores amongst the soccer players. Further investigations into the playing position of the athletes as well as numbers of headers made in the previous season predicted poor performances on the Figure Detection test, the Complex Figure Test, the WMS Logical Memory and Visual Reproduction tests respectively.

Three years later, Matser et al. (2001) continued to examine the relationship between headers, concussions and cognitive impairment amongst professional football players, with a mean age of 24 years in the Netherlands. Eighty-four active professional soccer players were evaluated using the same test battery that the researchers used in previous studies with the exception of the Digit Span test (the Bourdon-Wiersma test, the PASAT, the Stroop Colour Word Test, the Trail Making Test, the Wisconsin Card Sorting Test (WCST), the Facial Recognition Test, the Figure Detection test, the Complex Figure Test, Verbal Fluency, Puncture Test, Total Raven Score, the Digit Symbol Substitution Test, the WMS Associate Learning, Logical Memory and Visual Reproduction tests and the 15 Word Learning Test). The results showed that the number of headers in one season was related to poorer results on tests measuring attention and visual or verbal memory (the Complex Figure Test, the Trail Making Test, and the 15 Word Learning Test), whereas the number of soccer related concussions was related to poorer performances on tests measuring sustained attention and visuo-perceptual processing (Complex Figure Test, the Facial Recognition Test, and the Bourdon-Wiersma test). Taken together the findings suggest that two mechanisms contribute separately to cognitive impairment, headers as well as concussions sustained as a result of general play.

Whilst the above studies have consistently revealed deleterious effects in respect of neurological, neurocognitive effects amongst professional or top level athletes, some studies have produced no evidence of these effects. For example, recently Straume-Naesheim et al. (2005) examined the association between previous concussions and heading exposure amongst 271 professional Norwegian soccer players, with an average age of 25 years, on two consecutive baseline

neuropsychological tests using the computer neurocognitive test programme, CogSport. The computerized neurocognitive testing revealed no evidence of neurocognitive impairment due to heading or to previous concussions. Compared with the wide-ranging test batteries used in the other studies, including verbal and visual tests and tests of executive function, CogSport may be considered limited, being purely visual and largely a measure of reaction times. This may serve to account for negative findings on this study as well as in their study on boxing cited earlier (Moriarty et al., 2004) when the wealth of literature supports the presence of deficits. Furthermore, a non-traditional measure from a neurocognitive perspective with a single stimulus of a pack of playing cards may produce cultural bias and confound the results.

Finally, although the above studies have investigated the effects of MTBI with respect to neurological and neurocognitive sequelae, very few studies have investigated the postconcussive effects in terms of symptom reporting by the professional athlete. Jordan, Green et al. (1996), however, investigated this phenomenon in a study on acute and chronic encephalopathy in active soccer players in the United States. Twenty national soccer team members with an average age of 24.9 years were compared with 20 age-matched controls consisting of elite track athletes. All participants completed a questionnaire on head injury symptoms (headache, attention deficit, dizziness, memory deficit, depression, irritability, lack of energy, sleep disturbance, hearing impairment and neck pain) and had magnetic resonance imaging of the brain. Whilst neither measure demonstrated significant differences between the groups, soccer players reporting of head injury symptoms correlated with prior TBI. Their findings suggested that encephalopathy amongst soccer players related more to the acute head injuries sustained than from repetitive head injury. Similarly, in a study by Barnes et al. (1998) sixty-two male and 65 female elite soccer players between the ages of 17 and 30 years of age in the United States were evaluated as a result of their concussion history. Of the symptoms reported, headache was reported more frequently (54 male, 55 female) with over half of the players (54%) reporting that they had experienced at least one headache after heading the ball, with no differences in incidences between males and female players. Other common symptoms reported include being dazed (31%) and dizziness (18.1%), with only 6.9% of players reporting long-term sequelae such as recurrent headaches and vertigo.

In summary, the research reported on former and professional soccer players above provides substantial support for the chronic effects of MTBI associated with the long-term exposure to concussive and sub-concussive events as a result of general play and frequency of heading the ball. These findings are consistent with studies investigating the chronic effects of MTBI, with respect to neurological and neurocognitive effects amongst professional boxers, and as with boxing there is little investigation into self-reported postconcussive effects amongst professional soccer athletes.

#### **6.2.3.2 Amateur, collegiate or high school soccer players**

Researchers investigating the effects of recent heading amongst amateur soccer players have produced results consistent with other studies reporting on the acute recovery of concussive and sub-concussive effects amongst athletes (e.g. Bleiberg et al., 2004; Dicker & Maddocks, 1988; McCrea et al., 2002; Iverson et al., 2004b; Schretlen and Shapiro, 2003; Wilberger et al., 1991).

Putukian et al. (2000) examined the acute effects of heading in soccer on cognitive functioning in 44 male and 56 female collegiate soccer teams in the United States. Comparison groups were based on data collected from 23 male active headers and 30 female active headers with a total of 21 controls from the men's team and 26 controls from the female team. The average age of the groups was not reported. The neuropsychological battery included the Alphabet Backwards, the Trail Making Test, the Stroop Colour Word test and a modified version of the VIGIL/W which is a computerized continuous performance test that assesses reaction time and sustained attention and concentration, as well as a symptom checklist which consisted of symptoms (not specified) most commonly reported following MTBI (Putukian et al., 2000). Tests were administered before and after two training sessions, with two days separating the two assessment times. The pre-test scores indicated no significant differences between the groups and on the post-test scores no differences between heading and non-heading groups was evident. A significant difference between the pre- and post-season measures scores, however, was evident irrespective of whether the subjects were heading or not which meant that most subjects did better on their post-season testing, suggestive of a practice effect. There was a significant gender effect on measures of attention and concentration (Alphabet Backwards test and the Stroop Colour Word Test) and no difference in symptom reporting between the active headers and the non-headers groups.

Similarly, Webbe & Ochs (2003) investigated the role of recent heading activity in determining neurocognitive performance in a sample of 64 high school and collegiate soccer players (four of which were goalkeepers whose neuropsychological data was not analyzed) and 20 sports controls (runners, cyclists, basketball and baseball players), between the ages of 16 to 34 years, in the United States. Results support the notion that recent heading of moderate to high frequency was related to weaker neurocognitive performance, although the study did not support a strong effect for lifetime heading on neuropsychological performance (Webbe & Ochs, 2003). Depressed scores were seen on tasks of verbal learning (CVLT), planning and attention (Trails-B), as well as on information processing speed and divided-attention ability (PASAT). Thus, the above study supports the role of heading as a factor which depresses neurocognitive functioning in some players, at least temporarily.

Studies investigating the chronic effects of concussive and sub-concussive events, in respect of neurocognitive and postconcussive symptoms, have produced varied results with most studies demonstrating evidence of residual effects whilst few showing minimal or no effects. In a study comparing 31 college soccer players between the ages of 18 to 24 years with an equal number of tennis player controls in the United States, no significant differences were observed between the groups on any of the neurocognitive measures (the PASAT, Perceptual Speed test, the Symbol Digit Modalities test, Ravens Matrices) (Abreau et al., 1990). There was however, a negative correlation between the PASAT score and the number of games played (those players with a longer history of participation in the sport had slower processing speeds), but not between the PASAT scores and the number of headers. Moreover, the soccer players reported more symptoms of concussion on the symptoms questionnaire than the tennis group, with symptoms of headache, blurred vision, and dizziness being reported.

Matser and his colleagues (1999), using the same control group as in their previous study (Matser et al., 1998), compared 33 amateur soccer players to 27 controls in the Netherlands. The average age of the soccer players was 24.9 years and 24.5 years for the controls. The soccer players had a minimum of five years and a maximum of 17 years of experience playing soccer. Using the same neurocognitive test battery as in their other two studies (Bourdon-Wiersma test, the PASAT, the Stroop Colour Word Test, the Trail Making Test, the WCST, Digit Span test, Facial Recognition Test, the Figure Detection test, the Complex Figure Test, Verbal Fluency,

Puncture Test, Total Raven Score, the Digit Symbol Substitution Test, the WMS Associate Learning, Logical Memory and Visual Reproduction tests and the 15 Word Learning Test), deficits in planning and memory (the WCST, the Complex Figure Test, Digit Span test, the WMS Logical Memory and Visual Reproduction tests) were reported in the soccer players compared with controls. After adjustments were made for confounding variables decrements in performances were seen on the Digit Span test, the WMS Associate Learning, Logical Memory and Visual Reproduction tests. Unlike the previous study, no neurocognitive analyses were made on the relationship between soccer exposure and neurocognitive performance. The number of concussions reported however, were inversely correlated with performances on the Digits Forwards, the Facial Recognition Task, the Complex Figure Test, the Digit Symbol Modalities Test and the WMS Logical Memory test.

Downs and Abwender (2002) compared the cognitive functioning of 32 collegiate soccer players with 29 swimmers in the United States. The soccer players consisted of 26 college soccer players (15 males and 11 females) with an average age of 19.8 years, and six current or former professional male soccer players with an average age of 41.5 years. The controls consisted of 22 younger swimmers (seven male and 15 female) with an average age of 19.5 years, and seven older swimmers with an average age of 42.9 years. Participants were administered a demographic questionnaire in addition to a neurocognitive test battery consisting of the PASAT, the Finger Tapping test, the Continuous Performance test, and the WCST. The results showed that soccer players performed worse than the swimmers on tests of conceptual thinking (WCST), with the older soccer group, in particular, performing worse on measures of conceptual thinking, reaction time and concentration (the WCST, Finger Tapping test and the Continuous Performance test). Although the length of participation in soccer was associated with poorer neurocognitive performance, the findings did not specifically implicate heading as the cause.

Further studies which have investigated the role of heading frequency amongst athletes include a study by Witol and Webbe (2003), which also included professional soccer players in the sample. Sixty male high school, amateur and professional soccer players were compared with 12 non-playing sports controls, in the United States. The soccer group was divided into groups according to current (low: n=17, moderate: n=24 and high: n=19) and lifetime (low: n=19, moderate: n=20 and high: n=21) heading frequency, with the average age range of this group

ranging from 19 to 24 years. The average age of the control group was 22.92 years. Test measures included the Shipley Institute of Living Scale, the Trail Making Test, the PASAT, the Facial Recognition Test, the Complex Figure test, the Rey Auditory Learning Test (RAVLT) and a demographic questionnaire which included historical, vocational, academic and sports history information. Results showed that players with the highest lifetime estimates of heading obtained lower scores on tests of attention, concentration, cognitive flexibility and general intellectual functioning (the Trail Making Test, the PASAT and the Shipley Institute of Living Scale), whilst player's current level of heading did not show the same level of significance with only the test of attention and concentration (Trail Making Test-Part A) being significant in the direction of the players with the most headers performing worse than the other three groups. Thus, players with the highest estimated frequency of heading produced more impaired scores than control participants or players who didn't head the ball as frequently.

Similarly, Rutherford et al. (2005) investigated the effects of general soccer play and soccer heading on neuropsychological test performances. Comparisons were made between 22 amateur soccer players, 17 rugby players and 24 non-contact sports players with an average age of 20 years in the United Kingdom. Neuropsychological measures included the Beck Depression Inventory, the National Adult Reading Test, the Rey Complex Figure Test, the Digit Symbol Substitution Test, the Digit Span test, the Trail Making Test, the Stroop test, the WMS Logical Memory and Paired Associates tests, Tests of Attention Performance (TAP) including Alertness, Divided Attention, Visual Search, Covert Attention shift, Flexibility and Working Memory, the WCST, Alternate Uses and the COWAT. Across the groups the results showed that the number of head injuries predicted poorer performances on tasks of cognitive flexibility (Trails B and TAP Divided Attention tests). After controlling for the number of head injuries sustained, soccer players obtained lower scores on the TAP Divided Attention task than both the rugby and non-contact sports groups. Moreover, those soccer players who were frequent headers achieved fewer categories on the WCST. Rutherford et al. (2005) caution that these findings should not be interpreted as evidence of a link between neuropsychological impairment and soccer play, due to the exploratory nature of the research.

In contrast to the above studies that support impairment even in the younger soccer athlete, there are a few studies on amateurs that have demonstrated minimal effects with respect to

neurocognitive and postconcussive symptoms. For example, Guskiewicz et al. (2002) investigated the evidence of impaired neurocognitive performance in a group of 91 collegiate soccer players (male = 58 and female = 33), in the United States compared with a group of 96 non-soccer athletes (woman's field hockey = 18, woman's lacrosse = 51, and men's baseball = 27), and 53 college student controls. The average age for all the comparison groups was between 19 and 20 years of age. All subjects completed a concussion history questionnaire and a neurocognitive test battery (Scholastic Aptitude Test, the Trail Making Test, the COWAT, the Stroop Color Word Test, the Hopkins Verbal Learning Test, the Symbol Digit Modalities Test and the Digit Span test). No significant relationship between soccer athletes and controls on neurocognitive testing and scholastic aptitude was found, despite a history of soccer related concussions and an average of 15.3 seasons of soccer exposure amongst the soccer players.

Further, Stephens et al. (2005), recently examined whether the cumulative incidence of MTBI or cumulative heading frequency were related to neurocognitive functioning amongst 23 adolescent soccer players, 23 rugby players and 20 non-contact sports controls. The ages of the groups ranged from 13 to 16 years of age. The cumulative MTBI incidence was estimated using self-reports and cumulative heading was estimated using a combination of both self-reports and reports based on observation. No athlete had sustained a head injury three months prior to testing. The neurocognitive tests included the WISC-R Vocabulary, the Childhood Depression Inventory Short Form, Eysenck Personality Questionnaire Junior, the State-Trait Anxiety Inventory, the Rey Complex Figure test, the Digit Symbol Substitution Test, the Trail Making Test, the Stroop test, the WMS Logical Memory, the Test of Attention Performance (TAP), the WCST and Alternate Uses. The results demonstrated that there was no relationship between reported head injury and neurocognitive test performance on any of the sports groups. Within the soccer group, reports of cumulative heading did not predict any of the neurocognitive test scores.

Generally, the studies reviewed above with respect to amateur soccer players have produced somewhat inconsistent results, with some studies reporting deficits whilst others reporting minimal or no deficits, in both the acute and chronic conditions. This is in contrast to the studies investigating the effects of MTBI in professional soccer athletes where most studies supported chronic deficits with respect to neurocognitive and postconcussive sequelae. In summary, whilst it is difficult to separate out the effects of concussive and sub-concussive trauma as a result of

general play, and the cumulative sub-concussive effects of heading, generally the findings reviewed above are consistent with the boxing studies reporting of acute and chronic deficit amongst professional and amateur athletes. Although some researchers report deficits as supported by neurocognitive and neurological evidence (e.g. Abreau et al., 1990; Downs & Abwender, 2002; Matser et al., 1998, 1999, 2001; Rutherford et al., 2005; Sortland & Tysvaer, 1989; Tysvaer & Storli, 1989; Webbe & Ochs, 2003; Witol & Webbe, 2003), others do not (e.g. Guskiewicz et al., 2002; Jordan, Green, et al., 1996; Straume-Naesheim et al., 2005). Moreover, some researchers claim that the number of headers were mainly responsible for the deficits exhibited on objective tests (e.g. Matser et al., 1998, 2001; Rutherford et al., 2005; Tysvaer & Lochen, 1991; Webbe & Ochs, 2003; Witol & Webbe, 2003), whilst others disagree (Jordan, Green et al., 1996; Stephens et al., 2005; Straume-Naesheim et al., 2005). Some researchers attribute the number of concussions to the residual cognitive deficit (Matser et al., 1998, 2001), whilst others the number of years exposed to the sport (Abreau et al., 1990; Downs & Abwender, 2002), as well as the position played (Matser et al., 1998).

Consistent with studies reporting on the neurocognitive deficit amongst boxers and ice hockey athletes, the cognitive domains of attention and working memory, visual and verbal memory, and information processing speed appear to be the most sensitive to the effects of MTBI amongst professional and amateur soccer players in both the acute and chronic condition (Matser et al., 1998, 1999; Putukian et al., 2000; Rutherford et al., 2005; Tysvaer & Lochen, 1991; Webbe & Ochs, 2003; Witol & Webbe, 2003). Only four studies investigated the incidence of postconcussive symptoms reported by soccer players (Abreau et al., 1990; Barnes, 1998; Jordan, Green et al., 1996; Putukian et al., 2000). In two of these studies, headache was the most frequently reported symptom (Abreau et al., 1990; Barnes, 1998). Although many of the studies are beset with methodological problems, for example the small sample sizes (Rutherford et al., 2005; Witol & Webbe, 2003), mixed sample groups (Webbe & Ochs, 2003; Witol & Webbe, 2003), weak control groups (Downs & Abwender, 2002; Guskiewicz et al., 2002; Jordan, Green et al., 1996; Tysvaer & Lochen, 1991) and questionable test batteries (Straume-Naesheim et al., 2005) which could in turn question the validity of the current findings, generally, in keeping with the research on boxers, the findings amongst soccer players support the length of exposure to the sport as a possible cause for the long-term deficits amongst athletes, which seems to increase as athletes progress to higher levels of play.

#### **6.2.4 Outcome following MTBI in American football**

(For summary of outcome studies see Table 6.2.4, pp. 217 - 224)

American football is a contact sport characterized by vigorous body contact, with an emphasis on speed which is why this sport is known as a collision sport. Tackling manoeuvres and blocking have been shown to be the leading causes of head injury, including concussions amongst American football athletes (Bailes & Cantu, 2001). It would appear that studies investigating the effects of MTBI on boxing, ice hockey and soccer have predominantly focussed on the chronic or long-term and cumulative effects of multiple MTBI as a result of long-term exposure to the sport. In comparison researchers investigating the effects of MTBI in American football have predominantly focussed on the acute effects of MTBI, in respect of neuropsychological and postconcussive symptoms amongst collegiate and high school athletes, with few studies investigating the effects of MTBI amongst professional or former athletes. Recently, however, researchers have begun to show an expressed interest in the chronic and cumulative effects of multiple concussions on cognitive functioning and postconcussive symptom reporting in both professional and amateur groups. In this section the studies will be divided amongst professional or elite athletes, collegiate athletes, high school athletes, and then studies investigating both collegiate and high school athletes, and professional and high school athletes reported on together. In each level of play the acute effects will be discussed first followed by the chronic effects in respect of the long-term and cumulative effects of exposure to multiple concussive and sub-concussive events.

##### **6.2.4.1 Professional or elite American football players**

In a continued effort to improve the overall care of professional football players, a neuropsychological programme was initiated by the National Football League (NFL) over a six year period from 1996 to 2001 to determine the circumstances, causes and outcomes of concussion (Pellman et al., 2004). All participants were NFL athletes between the ages of 20 to 44 years, who underwent baseline neurocognitive testing during the 1996 to 2001 seasons but only 143 of the 650 athletes who experienced concussions underwent post-injury neurocognitive evaluation due to sample attrition. The neuropsychological tests sensitive to the effects of

concussion were selected, which included tests of attention, visual scanning, information processing speed, visual and verbal memory, visual-motor coordination and verbal fluency (the Hopkins Verbal Learning Test, the Brief Visuospatial Test-Revised, the Controlled Word Association Test, the Digit Span test and the Symbol Digit Modalities Test). Post-injury testing took place 1.4 days after injury and approximately 531 days from baseline testing. Results showed that the injured athletes did not perform significantly more poorly on any of the tests. They did however perform significantly better post-injury on tests of attention and processing speed (Trails A, Digit Span Forward and Symbol Digit Modality test). These findings reflect the influence of practice effects on these tests which have also been reported in findings elsewhere in the literature (Barr, 2003; Collins, Grindel, et al., 1999). The study also demonstrated that on-field signs of cognitive impairment, in this case memory, are useful in determining injury severity. A subsample of players with on-field memory impairments demonstrated memory impairments on neurocognitive testing one to two days post-injury but went on to make full recoveries (Pellman et al., 2004). Players with three or more concussions did not demonstrate significantly different neurocognitive test scores. The sample has to be criticised for being too small and for being a biased sample group, as many athletes who sustained concussions did not choose to take part in the study and were therefore not included in the analysis.

In one of the few studies to investigate the chronic effects of MTBI amongst professional American football athletes, Kutner et al. (2000) examined whether the cognitive status amongst professional American football players varies as a function of age and the apolipoprotein E (APOE) genotype, in the United States. Fifty-three active players with a mean age of 27 years were enlisted into the study and were grouped according to high or low exposure to contact and the presence or absence of the epsilon 4 allele. All players completed the short form of the computerized neurocognitive test battery MicroCog which consists of individual subtests assessing memory, attention, spatial processing, reasoning and reaction time. The results showed that the older players possessing the APOE e4 exhibited significantly lower cognitive test scores than all other players studied, with measures of general cognitive functioning, information processing speed and accuracy, and attention related to poorer performances amongst this group. This study is consistent with Jordan et al.'s (1997) study investigating the relationship between the APOE e4 allele and the Chronic Brain Injury (CBI) rating scale measuring motor, cognitive and behavioural functioning in professional boxers. High exposure boxers who possessed the

APOE e4 also had higher scores on the CBI scale. Both studies provide further support for BRC theory (described earlier in Chapter 3, section 3.6), such that the additive effect of age, high exposure to MTBI and the possession of a genetic risk factor will produce a significantly worse outcome.

Similarly, Guskiewicz et al's (2005) study evaluating the association between multiple concussion and late-life cognitive impairment also provided substantial support for the BRC theory in a sample of 2552 retired football players, with an average age of 53.8 years and an average playing career of 6.6 years, in the United States. Using a general health questionnaire and a questionnaire focussing on memory issues, retired players with a history of three or more concussions had a threefold prevalence of reported significant memory problems compared with retired players with no concussion history.

In summary, from the investigation of the studies that have been reviewed so far in respect of professional boxing, ice hockey and soccer athletes, there have been very few studies examining the acute effects of MTBI amongst professional athletes, with only Ravdin et al's (2003) investigation of acute cognitive recovery in a group of active professional boxers reported on. In light of the above, the study reporting on the acute effects amongst professional athletes in American football does not provide support for Ravdin et al's study. In Ravdin et al's study the acute effects of MTBI were demonstrated on testing immediately following injury with significant recovery being demonstrated at one month post-injury. In contrast, Pellman et al's (2004) study showed no deficits on testing immediately following injury, with athletes performing significantly better post-injury, demonstrating a practice effect. Although the authors identify potential methodological weaknesses in their studies, more research is needed to confirm these findings amongst professional athletes in general. In comparison, the two latter studies investigating the chronic effects of MTBI amongst retired or professional athletes (Guskiewicz et al., 2005; Kutner et al., 2000) provide further support for prior research investigating these effects in older and elite athletes in the contact sports of boxing and soccer. Further research is also needed, however, to support these chronic effects amongst professional or former athletes in American football.

#### 6.2.4.2 Collegiate American football players

The first large-scale study of concussion amongst American football players was performed at the college level by Barth (1989) and his colleagues. The objectives of the study were to determine, (i) the incidence of football-induced head injuries and to determine the extent and nature of the neurocognitive and psychosocial deficits of injured players; (ii) to establish a recovery curve for players with concussion and to determine return-to-play guidelines; (iii) to identify possible personal and football related factors which may predispose players to the risk of head injury, and (iv) to evaluate the long-term neurocognitive and psychosocial consequences of sustaining more than one head injury (Barth et al., 1989). Data was collected on a total of 2,350 players between the ages of 18 to 26 years from ten different universities in the United States. Neuropsychological assessment took place at intervals of 24 hours, 5 and 10 days post-injury and again at post-season. The paper-and-pencil assessment procedures included the Trail Making test, the Halstead-Reitan Neuropsychological Test Battery, the Symbol Digit Modalities Test, and the PASAT. Their study showed that a single mild head injury in football produces cognitive deficits that can be documented on neuropsychological assessment and that rapid although not complete recovery may take place over a period of 5 to 10 days. Although the study did not reveal severe cognitive fall-off following concussion, subtle difficulties were found on tests sensitive to information processing speed (Symbol Digit Modalities Test).

Using participants from the same cohort as in Barth et al's (1989) study above, Macciocchi and colleagues (1996) investigated the neuropsychological functioning and recovery of 183 college athletes between the ages of 18 to 26 years of age. The control group consisted of 48 student athletes matched for gender, age and years of education. The same neurocognitive tests as in Barth et al's study were used and consisted of the PASAT, the Digit Symbol Substitution Test (DSST), and the Trail Making Test, as well as a symptom checklist. Players who sustained MTBIs were evaluated pre-season and then at intervals of 24 hours, 5 days, 10 days and 12 weeks post-injury. The results showed that players displayed impairment in both sustained auditory attention (PASAT) and visuomotor speed (DSST, Trail Making Test), but that the impairment was largely as a result of the players failing to show an improvement over time as opposed to impairment relative to baseline. Significant increases in symptoms, especially headache, dizziness, and memory problems, relative to baseline were reported at five days post-

injury with no difference from baseline at ten days post-injury. Generally the impairment relative to controls resolved within five days in most athletes, with significant improvements made between 24 hours and 5 days and between 5 and 10 days on both the neuropsychological tests and the symptom checklist. The twelve week follow-up results are not discussed in the study.

Guskiewicz et al. (1997) investigated the cognitive functioning and postural stability in 11 Division I college athletes who sustained concussion compared with 11 matched controls in the United States. The average age of the football athletes was 18.6 years and the controls was 20.2 years. The neurocognitive battery consisted of the Stroop Test, the Trail Making Test, the Digits Span test and the Hopkins Verbal Learning Test. Concussed athletes were tested at intervals of one, three, five and ten days post-injury. The results on all of the above tests indicated that the MTBI athletes did not display any significantly poorer performances at any interval when compared with the control group (the researcher suggested that these tests were not sensitive enough to the effects of MTBI). All groups progressively improved from day one through to day five illustrating a practice effect. The authors suggest that their results contradict other studies because of the younger age of their sample studied.

Six years later, however, Guskiewicz and colleagues (2003) investigated the cumulative effects associated with recurrent concussion in collegiate American football players in the United States. Two thousand nine hundred and five collegiate football athletes were enlisted in the study, with data collected during three football seasons (1999-2001). No age range was given for the sample. A Graded Symptom Checklist and extensive health questionnaire was used to evaluate the athletes pre-season and then following concussion. All players who sustained concussions were monitored until symptom resolution and observed for repeat concussions until the end of the 2001 football season. The results showed that during the follow-up 184 players sustained concussions and 12 of these players sustained repeat concussions within the same season. Players reporting a history of three or more previous concussions were three times more likely to sustain another concussion than players with no concussion history. Only the most common symptoms and signs reported by the athletes are presented in the study, these include headache, dizziness or balance problems, feeling 'slowed down', fatigue, memory and dizziness. Of these symptoms headache was the most commonly reported symptom. The mean symptom recovery was 82 hours with players with a history of three or more concussions recovering at a slower rate (30 % of

individuals who sustained three or more concussions took longer than a week to recover compared with 14.6% of those athletes who sustained one previous concussion). Of the 12 repeat concussions, 11 occurred within 10 days of the initial injury and nine occurred within seven days of the initial injury. The authors suggested that players with a prior history of concussion are vulnerable to further injury and that previous concussion is associated with slower recovery.

Generally, the above studies all report on the acute effects amongst collegiate athletes demonstrating recovery in respect of improvement on neurocognitive testing and postconcussive symptom reporting within five to ten days of sustaining a MTBI. The following studies report on both acute and chronic effects which includes an investigation of the effects of cumulative subconcussive and concussive events amongst collegiate athletes.

Between May 1997 and February 1999, a large multi-site college football study was undertaken involving 395 male football players from Michigan State University, the University of Florida, the University of Pittsburgh and the University of Utah (Collins, Grindel et al., 1999). Forty-six percent of the sample reported no prior concussion, 34% reported one prior concussion and 20% reported a history of two or more concussions. Thirteen percent of the sample had a diagnosed learning disability. All athletes were administered a neuropsychological battery at pre-season and those within the sample who sustained acute concussions ( $n = 16$ ) were evaluated the same battery within 24 hours, three days, five days and seven days post-injury, using matched control athletes from within the sample ( $n = 10$ ). The test battery consisted of the Hopkins Verbal Learning Test (HVLT), the Trail Making Test, the Digit Span test, the Symbol Digit Modalities Test (SDMT), Grooved Pegboard, the Controlled Oral Word Association Test (COWAT), as well as a Concussion Symptom Scale. Consistent with acute studies reported above, the recent concussed athletes showed a decline in performance relative to controls on the immediate and delayed memory task (HVLT) at 24 hours, with moderate differences remaining between the groups until five days post-injury. Symptoms resolved before the resolution of cognitive deficits which is in contrast to other studies investigating the acute effects of MTBI where cognitive deficits resolved before symptom resolution (Lovell et al., 2003). Amongst the total sample group there was a significant relationship between the length of exposure to football and the total number of concussions sustained ( $r = 0.15$ ;  $p \leq .02$ ). The results indicated that a history of concussion is significantly and independently associated with long term deficits in tests which

measure executive functioning (Trails B) and speed of processing (SDMT) as well as an increase in self-reported symptoms. Players who had a history of two or more concussions and a learning disability performed significantly worse on tests of executive functioning (Trails B) and speed of information processing (SDMT). Again in support of BRC theory (described in Chapter 3, section 3.6) those athletes with a prior history of MTBI combined with a risk factor such as a learning disability are more vulnerable to further deficit in respect of cognitive functioning.

Macciocchi and colleagues (2001) investigated the neurocognitive and neurobehavioural consequences of one versus two concussions amongst 24 college football players in the United States. This sample was taken from the larger cohort of 2300 athletes used in previous studies cited above (Barth et al., 1989; Macciocchi et al., 1996). Half of the athletes sustained one concussion based on contemporary guidelines and the other half ( $n = 12$ ) sustained two concussions. The mean age of the sample was 19.1 years and the average number of years of experience in competitive football was 9.1 years. The neurocognitive battery, as in the previous studies, consisted of the PASAT, the Trail Making Test, and the Symbol Digit Modalities Test. A self-reporting symptom checklist was also used. All players were assessed using the neurocognitive battery and symptom checklist at pre-season and then during the season players who sustained concussions and failed the mental status examination were re-evaluated at 24 hours, five days and ten days post-injury. Both neurocognitive test scores and self-reported symptoms of the players who sustained two concussions were compared to the test scores of those athletes who sustained one concussion. The neurocognitive findings reported that those athletes, who sustained two concussions, two weeks, apart did not result in significant cognitive impairment compared with those who sustained a single concussive injury. Moreover, those athletes who sustained two concussions performed as well or better than those athletes who sustained one concussion on all neurocognitive measures after their first and second concussion and formed better at post-season than at pre-season. Players who sustained two concussions, however, reported more symptoms after their first and second concussions than those who sustained a single concussion, although the increase in symptoms was not significant after the first concussion. Headache was the most commonly reported symptom in both groups. There are limitations to these findings however, such as the documented rate of multiple injuries in the sample which was low (6%) and is not an adequate representation of players who typically sustain multiple injuries (Macciocchi et al., 2001).

In summary, with respect to MTBI studies reporting on collegiate athletes, as with studies investigating the acute and chronic effects of MTBI amongst boxing, ice hockey and soccer athletes, the findings are inconsistent. Whilst some studies provide support for acute and chronic effects following MTBI, in respect of neurocognitive and postconcussive sequelae (e.g. Barth et al., 1989; Collins, Grindel et al., 1999; Guskiewicz et al., 2003; Macciocchi et al., 1996) others do not (e.g. Guskiewicz et al., 1997; Macciocchi et al., 2001).

#### **6.2.4.3 High school American football players**

With the intent to gather preliminary norms and test-retest indices, Barr (2003) administered a brief neurocognitive test battery to a group of 100 high school athletes (60 male and 40 females) in the United States. Athletes participated in football ( $n = 47$ ), field hockey ( $n = 27$ ) or soccer ( $n = 26$ ), with the mean age of the sample being 15.9 years. All athletes were administered a neurocognitive test battery consisting of the Digit Span test, the Digit Symbol and Symbol Search subtests, the Trail Making Test and the COWAT, and the Hopkins Verbal Learning Test (HVL). All athletes completed the baseline assessment and 48 athletes repeated the assessment 60 days later. A re-test sample consisting of 32 males and 16 females was also tested over a two-day period. The baseline analysis revealed no differences on test scores between athletes participating in the different sports groups and no differences between athletes with or without prior concussions and with or without learning disabilities. The female athletes did however outperform the males on the Digit Symbol Subtest, Trail Making Test-Part B and the COWAT, but not on measures of verbal learning. At the re-test interval of 60 days significant practice effects were seen on the WAIS III Processing Speed Index, Trail Making Test and the COWAT, with no differences between the test scores of the males and females.

Lovell and colleagues (2003) evaluated memory dysfunction and self-reporting of postconcussive symptoms in a group of 64 high school athletes, using 24 athlete controls in the United States. The concussion group consisted of 60 boys and four girls (40 football players, eight basketball players, seven soccer athletes and five athletes from other sports groups) and the control group consisted of 16 boys and eight girls (22 swimmers and two football players). No age range was given for this group. The athletes were evaluated using a computerized

neurocognitive test battery (IMPACT) and a Postconcussion Symptom Scale. Evaluation took place at pre-season and then again at intervals of 36 hours, four and seven days post-injury. High school athletes who sustained concussions showed significant declines in memory performance in comparison to the control group, with significant differences reported between pre- and post-injury, and at four and seven days. There was also a significant difference in the number of symptoms reported by the concussion group at pre-season and at 36 hours post-injury, with more symptoms being reported at 36 hours, although no significant differences in symptom reporting at four and seven days was made. Overall, symptoms resolved before performances on memory tests which is consistent with previous investigations where self-reporting symptoms appeared to resolve before cognitive deficits (Collins, Grindel, et al., 1999)

In the following year Lovell, Collins, Iverson, et al (2004), evaluated 43 high school athletes in the United States between the ages of 13 to 18 years of age, who sustained Grade I concussions according to American Academy of Neurology (ANN) guidelines. Thirty-five of the athletes were male and eight were female. Twenty-four of the athletes were injured whilst playing football, eight whilst playing soccer, five basketball, two ice hockey, two lacrosse, one baseball and one softball. The whole sample was evaluated using the ImpACT computerized neurocognitive testing programme. All athletes were evaluate at pre-season and then again at intervals of between 36 to 72 hours, and between 5 to 10 days. Athletes with a history of concussion did not differ on baseline memory scores from those with no concussion history. Deficits in memory and an increase in symptom reporting amongst the high school athletes occurred within the first few days of recovery with all athletes returning at or above their pre-season evaluation level at six days. Subtle but non-significant decreases on reaction time and processing speed tests occurred at 36 hours post-injury with a significant improvement on both these domains occurring at six days post-injury.

The relationship between individual postconcussive symptoms and neurocognitive test performance has also been investigated amongst high school athletes. For example, the relationship between postconcussion headache and neurocognitive performance in a group of 109 high school athletes from 20 different high schools who sustained concussions during the 2000 and 2001 season in the United States was evaluated (Collins, Field, et al., 2003). The majority of the athletes were football players ( $n = 70$ ), other sports included basketball ( $n = 14$ ),

soccer (n = 13), hockey (n = 4), lacrosse (n = 3), softball (n = 2), track (n = 1), volleyball (n = 1) and wrestling (n = 1). The mean age of the athletes was 15.8 years, and 93 of the athletes were male and 16 were female. The ImPACT neurocognitive test battery was used for the study which includes a Postconcussion Symptom Scale. Testing took place between 5 to 10 days (average 6.8 days) following a concussion. The athletes were divided into two groups, those who experienced headache and those who did not. The results indicated that athletes who suffered from posttraumatic headache performed significantly worse on the reaction time and memory composite scores of the ImPACT programme, and reported significantly more symptoms other than headache. Although the findings suggest that postconcussion headache in high school athletes at seven days post-injury could be associated with incomplete recovery, the exact time frame of symptom onset and symptom resolution was not known, nor if headache resolution was correlated with resolution of neurocognitive data on the ImPACT test.

Using the same cohort as in the Collins, Field, et al. (2003) study reported above, the relationship between self-reported “fogginess” and neurocognitive performance one-week following a concussion was investigated (Iverson et al., 2004b). The ImPACT programme was also used for the purpose of this study. As with the study above investigating the relationship between postconcussion headache and neuropsychological performance, the findings revealed that those athletes who suffered from persistent ‘fogginess’ reported significantly more symptoms, as well as demonstrated significantly reduced reaction times, memory performances and processing speeds. The effect sizes for the total symptom score, reaction time and memory were greater for this study involving ‘fogginess’ than in the previous study investigating the relationship between postconcussion headache and neurocognitive performance headache.

Studies reporting on the cumulative effects of multiple concussions amongst high school athletes have produced results consistent with other studies involving older athletes demonstrating residual deficits. Wilberger et al. (1991) evaluated 62 high school players with two concussions occurring within a single season in the United States. Neurocognitive tests involving the WMS, the Selective Reminding test, the PASAT, the Symbol Digit Modality test, the Stroop and the Trail Making Test, were performed within 24 hours, 30 days and 90 days of sustaining a concussion. In addition, MRI scans were obtained from 29 of the players. At 24 hours, 47 of the 62 players (75%) had abnormal test results. At one month post-injury 29 of 47 players (61%) still

showed deficits on the Stroop Test, the PASAT and the Symbol Digit Modality Test. At three months the same tests showed continued abnormalities in 16 of the remaining 29 players (55%). These 16 patients also reported symptoms such as headache, dizziness and poor concentration. Magnetic Resonance Imaging was abnormal in two players (Wilberger et al., 1991).

Similarly, Moser et al. (2005) investigated the prolonged effects of concussion amongst high school athletes in the United States between the years of 1999 and 2000. Athletes ranged in age between 13 to 19 years and participated in the following sports of ice hockey, football, soccer, field hockey and lacrosse. The number of participants in each group was not specified. All athletes were divided into different groups depending on their concussion history: athletes with no concussion history, ( $n = 82$ ), athletes with one concussion and no symptoms ( $n = 56$ ), athletes with two or more concussions not in the past six months with no symptoms ( $n = 45$ ) and those who experienced a concussion a week before testing ( $n = 40$ ). Neurocognitive tests employed consisted of the Repeatable Battery for the assessment of Neuropsychological Status and the Trail Making Test. A symptom checklist was also employed. The results showed that athletes who sustained two or more concussions were not distinguishable from those athletes who experienced a concussion within the past week suggesting that those athletes with a history of two or more concussions were experiencing prolonged neurocognitive effects.

In summary, studies investigating the effects of MTBI amongst high school American football athletes have mostly evaluated the acute effects following MTBI (Collins, Field, et al., 2003; Iverson et al., 2004b; Lovell et al., 2003; Lovell, Collins, Iverson, et al., 2004) with some of these studies investigating both acute and chronic effects (Lovell et al., 2003; Lovell, Collins, Iverson et al., 2004). Only two studies have investigated the chronic and cumulative effects with respect to multiple concussions in high school athletes only (Moser et al., 2005; Wilberger et al., 1991). Most acute investigations support other research evaluating the acute effects amongst contact sport players at different levels of play, where recovery takes place within one month post-injury (Bleiberg et al., 2004; Ravdin et al., 2003). The studies reported above investigating the cumulative and long-term effects amongst high school athletes also support some of the amateur studies of boxing, ice hockey and soccer, by demonstrating residual deficits in respect of neurocognitive and postconcussive sequelae, albeit there is more consistent evidence of residual deficits demonstrated in respect of the American football literature. This may be due to more

sophisticated test batteries for the American football studies compared with those in boxing where batteries were more limited, and due to the much higher incidence of concussion in American football than in soccer.

#### **6.2.4.4 Collegiate and high school American football players**

Guskiewicz et al. (2000) investigated the epidemiology of concussion amongst collegiate and high school football players in the United States. No age range was given for either group and the results were not stratified into collegiate and high school groups and are thus reported on together. Of the 17,549 football players, 888 sustained one concussion and 131 sustained another in the same season. In response to their concussion checklist report, of the 1003 total injuries sustained 86% of the players complained of headache, 67% dizziness, 59% confusion, 49% disorientation, and 37.3% blurred vision. Of those athletes reporting headache, 28% reported that it resolved in 24 hours, whilst 10% reported that it lasted for more than 24 hours. None of the other symptoms lasted more than 24 hours.

Field et al. (2003) also evaluated neurocognitive and symptom recovery in a group of high school and college athletes. The sample consisted of 371 college and 183 high school athletes (male American football athletes,  $n=161$ ; male soccer athletes,  $n=22$ ), all athletes underwent pre-season evaluation, in the United States. Fifty-four athletes who sustained concussions during the season underwent serial testing within 24 hours of injury and then again at three, five and seven days post-injury and were compared to a non-injured control group consisting of 38 athletes. The tests included the Hopkins Verbal Learning Test (HVLT), the Digit Span test, the Symbol Digit Modalities Test, the Trail Making Test, the COWAT and a Concussion Symptom Scale. High school athletes were also administered the Brief Visual Spatial Memory Test-Revised (BVMT-R). The results showed that the high school athletes took longer to recover in terms of prolonged memory dysfunction on memory tests (HVLT and BVMT-R) than the college athletes. Significant increases in self-reporting of postconcussive symptoms occurred amongst the high school group relative to controls at 24 hours, three and five days post-injury. At the college level significant differences relative to controls were reported at 24 hours and at day three post-injury only.

Following the above studies investigating the relationship between neurocognitive functioning and postconcussive symptom reporting (Collins, Field, et al, 2003; Iverson et al., 2004b), Mihalik et al. (2005), evaluated posttraumatic migraine characteristics in athletes after sustaining a sports-related concussion. A sample of 261 high-school and collegiate athletes with a mean age of 16.36 years in the United States were employed for the purpose of this study. Athletes who suffered concussion but who reported no headaches were compared with athletes with postconcussion headaches, as well as to those athletes with posttraumatic migraine characteristics. Athletes with migraine characteristics consisted of those reporting headaches without nausea, photophobia or phonophobia during their first concussion evaluation. The ImPACT programme which includes a Postconcussion Symptom Scale was also used for the purpose of this study. The results showed that significant differences existed between the groups on all outcome measures. The migraine group had significantly lower verbal memory, visual memory and processing speed scores than both the groups, with or without postconcussive headaches, although those athletes who reported headache post-injury had a significantly lower reaction time than those athletes who reported no headache post-injury. This is in keeping with prior research investigating the relationship between postconcussion headache and neurocognitive performance (Collins, Field, et al., 2003). Overall symptom reporting was greater amongst the migraine group than both the headache and non-headache groups, and greater amongst the headache group in comparison with the non-headache group. The migraine group also demonstrated the greatest departure from baseline scores.

McClincy et al. (2006) investigated the recovery from concussion in 104 concussed high school and collegiate athletes in the United States. The ImPACT computerized neurocognitive battery was used to evaluate athletes pre-season and at two days, seven days and at 14 days post-injury. Their results showed that differences were observed between pre-season scores and all ImPACT composite scores and in the total symptom scores, at two and seven days post-injury. At 14 days post-injury only the verbal scores differed significantly from pre-season scores. The results therefore indicate that deficits may persist for longer than two weeks in athletes who have sustained concussions.

Studies reporting on the cumulative effects of multiple concussions amongst collegiate and high school athletes groups collectively have produced somewhat inconsistent results in terms of

demonstrating residual deficits. Iverson et al. (2004a) evaluated the cumulative effects of concussion in a sample of 38 athletes, 19 athletes (11 high school and 8 college students) with a history of three or more prior concussions and 19 with no history of concussions. The average age of the sample was 17.8 years. The ImPACT battery and its Postconcussion Symptom Scale were used for the purpose of the study. The findings revealed that the group with a history of three or more prior concussions reported significantly more symptoms at pre-season than the other group with no history of concussion, which is consistent with Collins, Grindel et al. (1999) and Moser et al's (2005) studies above investigating the effects of two or more concussions amongst university and high school athletes. There was a trend towards lower memory scores at pre-season for the multiple concussion group as well as significantly lower memory scores during the post-season assessment.

Two years later, Iverson et al. (2006) demonstrated different findings in their investigation of the cumulative effects of one or two previous concussions in a sample of 867 male high school and university athletes (football 86.7%, ice hockey 9.6%, soccer 2.3%, and other sports 1.4%), between the ages of 13 to 22 years in the United States. All athletes were divided into three groups on the basis of their concussion history. Six hundred and sixty four athletes had no prior concussion, 149 athletes had one prior concussion and 54 had two previous concussions. Using the ImPACT computer-based neurocognitive battery to evaluate the athletes at pre-season only, no differences were observed between the concussion groups in terms of cognitive functioning and symptom reporting.

In summary, most of the studies reported above incorporating both collegiate and high school athlete samples have investigated the acute effects in respect of neurocognitive and postconcussive sequelae following MTBI (Field et al., 2003; Guskiewicz et al., 2000; McClincy et al., 2006; Mihalik et al., 2005), with only two studies investigating the chronic and cumulative effects of multiple concussive trauma (Iverson et al., 2004a; Iverson et al., 2006). All studies provide support for both acute and chronic deficits following MTBI except for Iverson et al's (2006) study. With reference to the other studies reporting on the collegiate and high school athlete groups individually above, most studies provide consistent support for deficits in respect of neurocognitive and postconcussive sequelae following MTBI, in both the acute and chronic conditions.

#### **6.2.4.5 Professional and high school American football players**

Researchers Pellman et al. (2006) investigated the acute recovery of MTBI amongst National Football League (NFL) (n = 68) and high school athletes (n = 125) in the United States. All neurocognitive testing took place during the 2002 to 2004 seasons. The average age range of the high school sample was 13 to 18 years and the NFL sample's average age range was 20 to 34 years. Using the computerized neurocognitive battery, ImpACT, the athletes were evaluated pre-season and then within days of sustaining a MTBI. Consistent with the results of the NFL players evaluated in their 2004 study during the 1996 and 2001 seasons (Pellman et al., 2004), and unlike other studies investigating the immediate effects of MTBI amongst professional athletes, the NFL athletes demonstrated a rapid recovery, returning to pre-season levels within a week of sustaining a concussion. In comparison, the younger high school athletes demonstrated a slower recovery when compared with the older athletes. Factors such as neuro-developmental issues and that the older group represented a highly select group with regards to skill, size and injury tolerance have been suggested as possible reasons for the differences between the groups. This result is consistent with Field et al's (2003) finding in their study comparing collegiate with high school athletes, where the high school athletes also took longer to recover in comparison to the collegiate athletes.

In summary, the studies reviewed above investigating the acute and chronic effects of MTBI in respect of neurocognitive and postconcussive sequelae amongst American football athletes have predominantly focussed on high school and collegiate American football athletes, with few studies investigating these effects amongst professional or former American football athletes. In contrast to the investigation of MTBI in amateur boxing and soccer athletes which have mainly focussed on the chronic effects following MTBI, investigations amongst amateur American football athletes have predominantly focussed on the acute effects employing both neurocognitive tests and postconcussive inventories. Most studies reported neurocognitive recovery and symptom resolution within 10 days post-injury (Barth et al., 1989; Guskiewicz et al., 2003; Lovell, Collins, Iverson, et al., 2004; Macciocchi et al., 1996) with one study still showing cognitive deficits at 14 days post-injury (McClincy et al., 2006).

Consistent with prior studies on boxing, ice hockey and soccer, the cognitive domains of processing speed, reaction time, memory and sustained attention appear to be the most sensitive

to the effects of MTBI amongst American football athletes (Barth et al., 1989; Collins et al., 2003; Field et al., 2003; Iverson et al., 2002; Lovell et al., 2003, 2004; Macciocchi et al., 1996), with headache being the most commonly reported postconcussive symptom in the studies, followed by confusion and dizziness (Collins et al., 1999, 2003; Guskiewicz et al., 2000, 2003; Macciocchi et al., 1996). Some researchers showed the relationship between postconcussive symptoms and neurocognitive performance with athletes who reported degrees of headache, foginess and migraine having significantly reduced memory, reaction time and processing speed scores, as well as an increased overall reporting of symptoms (Collins, Field, et al., 2003; Iverson et al., 2002; Mihalik et al., 2005). Whilst most studies reported that cognitive deficit resolved before symptom resolution, some studies reported symptoms resolving prior to cognitive deficits (Collins, Grindel et al., 1999; Lovell et al., 2003; McClincy et al., 2006), which could be the result of the underreporting of symptoms by athletes.

Whilst amongst the amateur boxing and soccer groups, support for chronic deficit in respect of residual neurocognitive and postconcussive sequelae is not as obvious as in the professional athlete, there are still some studies which provide subtle support for the chronic effects amongst these groups. Although studies reporting on the chronic and cumulative effects of concussion amongst collegiate and high school American football athletes are relatively few, at least five of these have suggested long term deficits as a result of multiple concussions (Guskiewicz et al., 2005; Collins, Grindel, et al., 1999; Iverson et al., 2002; Moser et al. 2005; Wilberger et al., 1991) with a lesser number of studies finding none (Iverson et al., 2006; Macciocchi et al., 2001). Thus, taken together these findings suggest that the subtle residual deficits as reported amongst these younger athlete groups are detectable in a substantial number of instances, albeit not all instances, and that this more tenuous evidence develops into more robust evidence for significant chronic effects at the professional level.

### **6.2.5 Outcome following MTBI in Australian Football League (Australian Rules Football), Rugby League and Rugby Union**

(For summary of outcome studies see Table 6.2.5 to 6.2.7, pp.225 - 229)

In comparison to the studies investigating the acute and chronic effects, in respect of neurocognitive and postconcussive sequelae, amongst the contact sports of boxing, soccer and American football, there are relatively few studies investigating these effects in Australian

Football League, Rugby League and Rugby Union, which is alarming considering the high risk of head injury associated with these sports (Garraway et al., 2000; Maddocks, Saling, et al., 1995; Petterson & Skelton, 2000; Junge et al., 2004; Seward et al., 1993).

All studies investigating the effects of MTBI on AFL and Rugby League players have used Australian athletes in their samples and have also focussed predominantly on the acute effects sustained amongst professional athletes, with minimal or no studies reporting on the long-term effects especially amongst the younger athlete. Published research on the effects of concussion amongst Rugby Union players also appears to be limited. To the knowledge of the researcher neuropsychological research in the area of Rugby Union has mainly been conducted in South Africa with a particular focus on chronic rather than acute effects. Two other studies investigating the acute and chronic effects of MTBI using Canadian and American Rugby Union athletes have been reviewed. Whilst some studies on boxers (Butler et al., 1993), ice hockey (Ferguson et al., 1999) and soccer athletes (Rutherford et al., 2005; Stephens et al., 2005) have incorporated Rugby Union players as a comparative sample group in their studies, these groups did not form part of the main analysis of the study and therefore contain no observations in these players that warrants being reported on. Studies investigating the acute effects of MTBI with respect to neurocognitive and postconcussive sequelae will be reported on first followed by studies investigating the effects of chronic and cumulative MTBI.

#### **6.2.5.1 Australian Football League (AFL) (see Table 6.2.5, pp. 225 - 226)**

Neuropsychological research in Australia began with Australian Football League (AFL). In a study by Dicker and Maddocks (1988) thirteen concussed AFL players were assessed pre- and post-injury using the Digit Symbol Substitution Test. When their performance was compared to their pre-season testing the concussed athletes completed significantly fewer items on the Digit Symbol Substitution test 24 hours post-injury although they returned to their pre-season performance at five days post-injury (Dicker & Maddocks, 1988). No control group was used in the study however, which makes it difficult to ascertain whether their performances were due to recovery of function or to practice effects.

Maddocks and Saling (1996) compared 10 AFL athletes to an age-matched control group of 10 AFL referees, although no age range was given for either group. Neuropsychological measures included using the Digit Symbol Substitution Test, the Four-Choice Reaction Time test and the PASAT. Significant differences on the Digit Symbol Substitution Test and the reaction time task was reported between the concussed players and their controls when reassessed at five days post-injury. Whilst concussed players' performances were comparable to their pre-season scores at five days post-injury, the controls demonstrated significant improvements from their pre-season performances. The fact that the control group demonstrated significant improvements in their pre-season performances whereas the AFL athletes simply returned to pre-season levels suggests a lack of practice on the part of the AFL athletes which may, as previously discussed, demonstrate a deficit in the learning process (Lezak et al., 2004). The results as they stand may therefore underestimate the true incidence of deficit amongst AFL athletes.

Similarly, McCrory et al. (2000) documented the nature and clinical symptoms of acute sports-related concussion amongst 303 elite AFL players (no age range was again given for this group). Twenty-three concussions were recorded over an AFL season. The most common symptom reported was headache, with 40% of the players reporting the headache symptom for more than 15 minutes. Ten players returned to play on the day of injury and the remaining 13 players returned to play two weeks later. Players with a history of three or more concussions or where symptoms lasted for more than 15 minutes were less likely to return to play on the day of injury. These findings were consistent with poor performances on the Digit Symbol Substitution Test.

Recently Makdissi and colleagues (2001) evaluated 240 AFL players from six different AFL teams (four elite, one semi-elite and the other amateur). Six concussed athletes between the ages of 17 to 26 years were observed over a period of nine weeks, with concussed athletes re-tested at 2 and 14 days post-injury. Athletes were administered two conventional neurocognitive tests (i.e. the Digit Symbol Substitution Test and the Trail Making Test) as well as a brief neurocognitive computerized test battery, CogSport. When compared to seven non-injured, age-matched control athletes, the concussed athletes displayed significant deterioration on measures of psychomotor speed and psychomotor variability included in the CogSport battery. On the conventional measures (the Digit Symbol Substitution Test and the Trail Making test) however, both control and concussed groups showed improved performances. This also supports the use of

computerized neuropsychological measures over more conventional ones as being sensitive measures in repeat assessments of the subtle effects of concussion.

Collie et al. (2006) evaluated the cognitive functioning of 615 male AFL players in Australia. All athletes were evaluated at pre-season and 61 athletes (25 symptomatic athletes and 36 asymptomatic athletes) were evaluated within 11 days of sustaining a MTBI. Eighty-four controls were also re-evaluated. The average age of the sample was 23 years. The three groups (controls, asymptomatic and symptomatic athletes) were compared using the computerized neurocognitive battery, CogSport, as well as other paper-and-pencil neurocognitive measures. In comparison to the asymptomatic and control group, the symptomatic group's performance on the CogSport tests of simple, choice and reaction time declined at the post-injury interval relative to their pre-injury performance, demonstrating deficits in motor function and attention although both memory and learning ability was preserved. The symptomatic group's performance on the paper-and-pencil tasks demonstrated no changes from their pre-injury performances whereas both the asymptomatic and control groups demonstrated large improvements from their pre-injury testing, which suggests significant practice effects.

There is very limited support or evidence for persistent complaints following concussion in AFL. In a study by Cremona-Meteyard & Geffen (1994), however, deficits were observed in nine concussed players (average age = 23.5 years) relative to a sample of 12 non-injured sportsmen using a cued reaction time task one year post-injury. Six of the nine players concussed had a history of previous concussions suggesting a cumulative effect of repeated head trauma. In contrast, a retrospective study by Maddocks, Saling, et al. (1995) revealed that of a sample of 198 professional AFL players, 79 athletes reported sustaining no concussions (average age = 21.3 years), 50 athletes reported that they sustained one concussion (average age = 21.9 years), 69 reported that they had sustained two or more concussions (mean age = 24.6 years), with no athlete sustaining a concussion in the six months prior to assessment. Performance on the Digit Symbol Substitution Test revealed no significant difference between the groups at six months post-injury.

In summary, when compared to other contact sports there have been relatively few studies investigating both the acute and chronic effects of MTBI amongst AFL players. Most studies

also fail to differentiate between professional or elite and amateur athletes, with some studies failing to report on the average age range of the sample groups, making it difficult to assess the athletes average length of exposure to the sport. Consistent with previous contact sports studies reporting on the acute deficits following MTBI however, acute recovery amongst AFL players took place between 5 to 14 days post-injury. In respect of the few studies which investigated the long-term and cumulative effects of MTBI amongst AFL players (Cremona-Meteyard & Geffen, 1994; Maddocks, Saling, et al., 1995), these effects have been present in one study and not in another, which does support muted results in previous research investigating the chronic and cumulative effects of MTBI amongst younger athletes in the sports of boxing, ice hockey, soccer and American football.

Consistent with previous research the cognitive domains of information processing speed and reaction time seem to be the most sensitive to the effects of MTBI amongst AFL athletes (Collie et al., 2006; Cremona-Meteyard & Geffen, 1994; Dicker & Maddocks, 1988; Maddocks & Saling, 1996; Makdissi et al., 2001; McCrory et al., 2000). Although no other studies have investigated the incidence of postconcussive symptoms amongst athletes other than McCrory et al's (2000) study, in line with other contact sports studies headache was also the most commonly reported symptom amongst AFL athletes. Whilst the studies reported above provide some evidence of the acute and chronic effects of MTBI amongst AFL athletes, most of the studies have significant methodological issues such as small sample sizes (Cremona-Meteyard & Geffen, 1994; Dicker & Maddocks, 1988, Maddocks & Saling, 1996; Makdissi et al., 2001), lack of or weak control groups (Dicker & Maddocks, 1988, McCrory et al., 2000 as well as having athletes at various levels of play included in their sample groups (Makdissi et al., 2001).

#### **6.2.5.2 Rugby League** (see Table 6.2.6, p. 227)

Researchers Hinton-Bayre et al. (1997) evaluated 54 professional Rugby League players between the ages of 17 to 26 years. The neurocognitive test battery included the Symbol Digit Modalities Test, the Digit Symbol Substitution Test and the Speed of Comprehension Test. Two studies were conducted. The aim of the first study was to examine the effect of practice and the second was to determine sensitivity of the test to cognitive impairment following head injury. The first study involved two pre-season testings with an interval of one to two weeks between testing. The

results demonstrated that performance on the Digit Symbol Substitution Test and the Speed of Comprehension Test improved with practice whereas performance on the Symbol Digit Modalities Test remained stable. The second study evaluated the cognitive functioning of the ten concussed players at 24 to 48 hours post-injury. The findings showed measurable changes in tests for Speed of Comprehension and Symbol Digit perception. Speed of Comprehension appeared to be more sensitive to post-injury impairment than both of the Symbol Digit tests, although researchers failed to take into account a prior history of concussion amongst the athletes.

In a follow-up study by Hinton-Bayre et al. (1999), twenty professional players ranging from 19 to 21 years were analysed following a MTBI. The results showed impaired performances on at least one of the three measures (Digit Symbol Substitution Test, Symbol Digit Modalities Test and Speed of Comprehension) in 16 players at one to three days post-injury. At one to two weeks post-injury, seven players were impaired on at least one of the three measures, returning to pre-season levels only three to five weeks later. Symptoms of headache, amnesia, unsteadiness, visual disturbances, dizziness, nausea and feeling dazed were experienced immediately after injury in only five of the athletes, with few complaining of ongoing symptoms. Consistent with prior studies on American football (Collins, Grindel et al., 1999; Lovell et al., 2003; McClincy et al., 2006), the symptom reporting amongst athletes resolved prior to the resolution of cognitive deficit, again suggesting the underreporting of symptoms by athletes.

The same authors recorded the incidence, signs and symptoms of concussion amongst 175 professional Rugby League players, although the age of the group was not specified (Hinton-Bayre et al., 2004). The most frequently reported symptoms after concussion were amnesia, headache and unsteadiness. Consistent with common signs and symptoms experienced in other Rugby League studies, the symptoms resolved on the same day of injury or within twelve hours of sustaining the injury (Hinton-Bayre et al., 1999).

According to researchers Hinton-Bayre and Geffen (2004), no studies to date have found strong evidence for chronic and cumulative effects from concussion in Rugby League since most studies have investigated the acute effects and no long-term effects have been reported or demonstrated on performance measures.

Although the AFL and Rugby League studies reviewed above are significantly less than those investigating the effects of concussion amongst boxers, soccer players and American football athletes, the findings are generally very similar. In line with the studies reported above, measures of information processing speed seem to be the most sensitive to the effects of MTBI (Dicker and Maddocks; Hinton-Bayre et al., 1997, 1999; Maddocks & Saling, 1996; Makdissi et al., 2001; McCrory et al., 2000) although the paper-and-pencil measures used to investigate such functions such as the Digit Substitution Test and the Symbol Digit Modalities Test are subject to practice effects (Hinton-Bayre & Geffen, 2004; Maddocks & Saling, 1996). The symptom headache is again reportedly to be the most commonly reported symptom amongst athletes (Hinton-Bayre et al., 2004) with some studies also reporting the resolution of symptoms before the resolution of cognitive deficit (Hinton-Bayre et al., 1999), in line with previous studies (Collins, Grindel et al, 1999; Lovell et al., 2003; McClincy et al., 2006). Consistent with American football studies investigating the acute and chronic effects of MTBI, the above research suggests that acute recovery following MTBI takes place within 5 to 10 days post-injury, with few AFL and Rugby League studies investigating the chronic and cumulative effects of MTBI.

#### **6.2.5.3 Rugby Union** (see Table 6.2.7, pp. 228 - 229)

The effects of concussion amongst a group of 60 South African university Rugby Union players was investigated on various measures of neurocognitive functioning known to be sensitive to the effects of concussion (Shuttleworth-Jordan et al., 1993). A control group consisting of 25 non-contact sports players (squash, hockey and tennis) was employed into the study for comparison. All athletes were between the ages of 18 and 25 years. Neurocognitive measures included tests of verbal new learning ability (Digit Supraspan), immediate short-term verbal memory (Digits Forwards), speed of information processing and working memory (Trail Making Tests – Part A and B and Digit Span Backwards) and hand motor dexterity (Denckla finger tapping and Purdue Pegboard tests). Subjects were tested at both pre- and post-season intervals and concussed players and matched controls were tested at intervals of one week, one month, two months & three months post-injury. The results at pre-season revealed that the controls performed significantly better on tests of working memory, verbal new learning ability and hand-motor dexterity (Trail Making Test, Digit Backwards and the Purdue Pegboard) in comparison to the

Rugby Union players. Results of the pre- and post-season comparison revealed a significant practice effect for the control group on the Trail Making Test, Digit Supraspan, Finger Tapping task and Purdue Pegboard. The rugby group also showed less capacity than the control group for practice effects between pre- and post-season testing, with the only improvement noted on the Finger Tapping Task.

Similarly, the repeated test measures, measuring the acute recovery curve of concussed players up to three months post-injury revealed deficits in attention, verbal new learning, working memory and hand motor dexterity at three days post injury, with substantial recovery up to the two month interval. By the three-month interval however, although significant recovery by the concussed group was made, the concussed group also showed less capacity for practice effects than the control group. Researchers suggest that one reason for this effect may be concussions sustained in prior seasons (Shuttleworth-Jordan et al., 1993), i.e. the presence of underlying chronic effects. Postconcussive symptoms reported amongst the concussed players mirrored the classic postconcussion syndrome as described in other studies, with symptoms significantly reduced at one month post-injury (headache, fatigue, attention, concentration and blurred vision) and the presence of only some symptoms at the two-month interval (headache, fatigue, attention, concentration and restlessness). Of concern is that the objective cognitive and hand motor tests indicated that recovery to baseline levels may not have occurred at three months post-injury, whilst subjects reported no symptoms at that stage (Shuttleworth-Jordan, et al., 1993). This is in line with previous sports studies where symptoms resolved before cognitive deficits (Collins, Grindel et al, 1999; Hinton-Bayre et al., 1999; Lovell et al., 2003; McClincy et al., 2006).

The above study suggested that the level of impairment on the repeated measures section of the study was highly comparable to the pattern of impairment amongst the rugby group in the pre- and post-season comparisons. Although the tests for this research were chosen for their expected sensitivity to the effects of concussion, the test battery was small and so too was the sample size. The sample was also not strictly controlled for age and education even though the sample consisted of university athletes between the ages of 18 to 15 years of age. A pre- and post-season repeated measures design was also not employed since random controls and rugby players were selected for the post-season analysis of the non-concussed rugby cohort. Despite the limitations

of the study, it provides further support for the chronic effects of concussion amongst contact sport players on tests known to be sensitive to diffuse brain injury.

A study was conducted in Canada using rugby players to study the effects of glucose on memory functioning (Pettersson & Skelton, 2000). Twenty-four male rugby athletes (12 previously concussed athletes and 13 controls) between the ages of 18 to 24 years were employed into the study. All participants were evaluated consecutively over three days. Both groups were given glucose-or saccharin sweetened beverages and then administered a neurocognitive test battery which consisted of the Wechsler Logical Memory Scale, Wechsler Paired Associates, Auditory consonant trigrams, Symbol Digits Modalities Test (SDMT), SDMT Incidental Recall, Listening Span Test and the Automated Neuropsychological Assessments Metrics (ANAM). Previously concussed individuals performed slightly worse than controls in the saccharin (placebo) condition on tests of memory and divided attention (SDMT Incidental Recall, Wechsler Logical Memory Scale, Auditory consonant trigrams and Listening Span Test), but performed better than controls when given the glucose beverage on tests of declarative memory (Logical Memory).

Ferrace et al. (2003) evaluated the long-term effects of sports-related concussions in a group of collegiate woman's rugby players. Fifty-four woman athletes between the ages of 17 to 22 years in the United States were cognitively evaluated using a brief neurocognitive test battery consisting of the Standardized Assessment of Concussion (SAC), the Trail Making Test, and the Paced Auditory Serial attention Test (PASAT). Twenty-nine athletes had previously experienced at least one concussion. The results demonstrated that no differences were found on either the PASAT or the SAC on athletes with or without a history of concussion. Athletes with a history of concussion made significantly more errors on the Trail Making Test than those athletes with no history of concussion.

Shuttleworth-Edwards et al. (2004), reported on a study conducted in collaboration with the South African Rugby Football Union (SARFU) and the MRC/UCT Exercise Research Unit in Cape Town. This study into the persistent effects of concussion in Rugby Union was conducted across three levels of play: professional level players, which included the top national open players, university players, which included national under-21 players and high school players. More specifically, the sample consisted of: (i) Twenty-six professional Rugby Union players

(mean age of 27.5 years) who were compared to a group of 21 national open non-contact cricket players (mean age of 27.1 years). (ii) Nineteen national under-21 Rugby Union players were (mean age of 19.7 years) compared to a group of 21 national open field hockey players (mean age of 23.3 years). (ii) Forty-seven school Rugby Union players (mean age of 17.3 years) were compared to a group of 34 school field hockey players (mean age of 17.0 years).

All participants were administered a self-reported postconcussive symptom questionnaire, in addition to the following neurocognitive test battery. *General intellectual functioning*: SAWAIS Picture Completion and Comprehension subtests (national open rugby, national open cricket, national under-21 rugby, national open field hockey); the SAWAIS- Picture Completion and Vocabulary subtests, National Adult Reading Test (NART), (School rugby and school field hockey). *Attention and concentration*: SAWAIS- Digit Span Test, (all groups). WAIS- III Letter-number Sequencing subtest (LNS) and the Stroop Test, (School rugby and field hockey). *Visuoperceptual scanning speed*: SAWAIS Digit-Symbol Substitution Test, the Trail Making Test, (all groups). *Memory*: WMS- Associate Learning subtests, the Digit-Symbol Incidental recall-Immediate and Delayed, the WMS-Visual Reproduction subtest, (all groups). *Verbal fluency*: Words-in-One-Minute Unstructured Verbal Fluency Test, Structured Verbal Fluency Test – ‘S’ Words, (all groups). *Fine-motor dexterity*: Sequential Finger Tapping Test, (all groups).

For both the national open team and the national under-21 team, deficits were reported in the cognitive domains of speed of information processing, memory, verbal fluency and hand motor dexterity, with only one result in the opposite direction where 10% of field hockey players exhibited deficit on one trial of the sequential Finger Tapping Test. In contrast to the two adult teams, only one test reported deficit at the school level, which was on one trial of the hand motor function. In spite of this the researchers reported that there were a number of subtle indicators of cognitive function in the memory domain. Of interest was a small subset of schoolboy rugby players with a learning disability (LD). Although the rugby group with a LD had a higher estimate IQ than the field hockey group with a LD, the percentage of cognitive deficit for the rugby group clustered around 67% compared to a close to zero indication of deficit for the field hockey group with a LD, which is commensurate with other research reporting increased cognitive deficit in football players with a LD (Collins, Grindel et al., 1999).

Both adult teams reported substantial postconcussive symptoms in each of the emotional, behavioural, somatic and cognitive symptom clusters when compared to controls. Although the researchers noted that there was a marked reversal of findings on a few symptoms of headaches, eyesight, attention and concentration, amongst the open rugby groups which could be attributed to the known underreporting of symptoms that takes place amongst top athletes. Amongst the schoolboy rugby sample higher percentages were reported in comparison to controls, although they weren't as prevalent as those reported by the adult groups. Symptoms reported included clumsy speech, sleep difficulties, clumsiness, easily angered and hearing problems (Shuttleworth-Edwards et al., 2004).

Thus, across the different levels of play cognitive deficits (particularly in the areas of information processing speed and memory) as well as postconcussive symptomatology (speech-related and aggressive/argumentative symptoms suggestive of frontal lobe involvement) were more robustly in evidence amongst the top professional players in comparison to the schoolboy rugby players (Shuttleworth-Edwards et al., 2004). These findings are in support of the findings amongst researchers that report that the incidence of injury rises with age and level of competition (Jakoet & Noakes, 1998; Roux et al., 1987). Thus it is hypothesized that top professional players, with more years of exposure to the game, are more likely to be at risk for sustaining multiple concussive and sub-concussive effects in comparison to the younger athlete.

In summary, the studies evaluating acute recovery in Rugby Union (up to three months post-injury) do not support the acute recovery in respect of the contact sports of American football, AFL and Rugby League where acute recovery took place, at most, 14 days post-injury (e.g. Barth et al., 1989; Guskiewicz et al., 1997; Lovell, Collins, Iverson, et al., 2004; Macciocchi et al., 1996; McCrory et al., 2000). The acute recovery amongst Rugby Union athletes does however support a study investigating the acute effects in ice hockey athletes (Hatfield et al., 2004) where athletes took longer than three months to recover, with total recovery of all athletes taking place in just over six months. In support of studies investigating the chronic effects of MTBI amongst professional athletes, research on Rugby Union athletes demonstrate that as the length of exposure to the sport increases so too does the risk of deficit, in respect of neurocognitive and postconcussive sequelae, which would explain the more consistent evidence

for significant findings amongst older athletes as opposed to the younger ones. Amongst school contact sports, however, the risks are still evident but it is suggested that these effects are less robust due to less number of years of being exposed to the sport. Consistent with prior studies the cognitive domains of visuoprocessing speed and memory are the most sensitive to the effects of MTBI and the postconcussive symptoms of headache and fatigue, the most commonly reported symptoms.

### **6.3 METHODOLOGICAL ISSUES IN MTBI IN SPORTS**

Research investigating MTBI in sport has been limited to quasi-experimental designs (Macciocchi & Barth, 2004). In an experimental design, control is exercised through the manipulation of the independent variable (presumed cause) or through randomization (random assignment or manipulation of participants to different experimental groups). Since neither experimental manipulation nor random assignment can be implemented with quasi-experimental designs one assumes that the independent variable has some logical effect on the dependent variable (the condition one is trying to explain) (Kerlinger, 1986). Hence, a type of “self-selection” occurs in which members can “select themselves” on the basis of characteristics extraneous to the research problem but which influence or are otherwise related to the variables of the research problem (Kerlinger, 1986). With reference to the sports arena the independent variable would be the contact sport or the multiple concussions sustained as a result of participating in a contact sport (presumed cause) and the dependent variable (presumed effect) would be the outcome in respect of neuropsychological and postconcussive sequelae. Hence athletes by virtue of their participation in a contact sport are at risk of sustaining MTBI and are more vulnerable to impairment with respect to neurocognitive and postconcussive sequelae.

Generally, the above contact sport studies of boxing, ice hockey, soccer, American football, Australian Rules Football, Rugby League and Rugby Union provide support for the acute and chronic effects of MTBI with respect to neurocognitive and postconcussive sequelae, although by virtue of their design, sports studies are subjected to many threats to internal validity.

Internal validity refers to the extent to which extraneous variables in the research design produce effects that would confound the researcher’s interpretation of the data (Campbell & Stanley,

1963, Cook & Campbell, 1979). Some sources of internal validity include: (i) **History:** History refers to pre-morbid factors which the athletes possess, which are not linked to the independent variable (MTBI) but which may affect or influence the test results (Cook & Campbell, 1979). These factors would include variables such as age, education, IQ, psychological or medical history. (ii) **Selection:** Because quasi-experimental designs do not assign subjects randomly to groups there is a risk of unintentional bias that may occur as a result of non-random selection. (iii) **History-selection interaction:** A history-selection interaction can occur when researchers select athletes which may have pre-injury features that account for the observed group differences and as a result an interaction of history and selection takes place. For example, athletes with prior histories of concussions not due to sport and who are selected in order to produce a desired effect by influencing the dependent variable (Cook & Campbell, 1979). (iv) **Instrumentation and testing:** Instrumentation and testing refer to measurement bias and error. (v) **Maturation:** Maturation involves changes that occur in the participants over time, such as aging or psychological changes (Cook & Campbell, 1979).

### 6.3.1 Experimental designs used in MTBI in sport

According to researchers threats to internal validity are related to the design employed by the researcher (Cook & Campbell, 1979). Some of the typical designs used in MTBI research include the post-test only design; the one group pre-test, post test design; time series design; the post-test only control group design, time series control design and the non-equivalent control group design (see Tables 6.3.1 to 6.3.6).

#### 6.3.1.1 Post-test only design

The post-test only design involves athletes being administered tests at some point in time following injury. The test results are then compared to normative data in order to determine if deficits in functioning are present (Campbell & Stanley, 1969). Studies reporting on the chronic effects of MTBI in boxing (e.g. Kaste et al., 1982; Casson et al., 1984; Heilbronner et al., 1991, Ross et al., 1987), ice hockey (Hatfield et al., 2004), soccer (e.g. Matser et al., 2001; Struame-Naesheim et al., 2005), American Football (e.g. Guskiewicz et al., 2000; Kutner et al., 2000; Moser et al., 2005), Australian Football League (e.g. Maddocks, Saling, et al., 1995; McCrory et

al., 2000), Rugby League (Hinton-Bayre et al., 2004) and Rugby Union (e.g. Ferrace et al., 2003) are examples of post-test only designs.

Post-test only designs are vulnerable to all threats of internal validity (see Tables 6.3.1 to 6.3.6). None of these studies reported on above have control groups and researchers justify this by the use of standardized tests, hence comparing their sample group to the general population. In order for researchers to determine the influence of participation in a contact sport, it is important to compare contact sport players with control groups that match those contact sport players on all aspects other than participation in a contact sport. Since it is only by comparing the contact sport players to a control group that any differences attributed to the contact sport can be observed (Kerlinger, 1986).

Many of these studies reported on above have no control for pre-morbid variables such as age, level of education or IQ and concussion history (e.g. Barnes et al., 1998; Barth et al., 1989; Bleiberg et al., 2004; Brooks, Kupsik, et al., 1987; Butler et al., 1993; Casson et al., 1984; Downs & Abwender et al., 2002; Drew et al., 1986; Hatfield et al., 2004; Gaetz et al., 2000; Guskiewicz et al., 2002; Heilbronner et al., 1991; Hinton-Baryre & Geffen et al., 1997; Iverson et al., 2004b; Jordan et al., 1997; Jordan, Green et al., 1996; Jordan, Matser et al., 1996; Kaste et al., 1982; Killam et al., 2005; Levin, Lippold et al., 1987; Makdissi et al., 2001; Matser et al., 1998, 1999, 2001; McClincy et al., 2006; McLatchie et al., 1987; Moriarity et al., 2004; Porter, 2003; Porter & Fricker et al. 1996; Putukian et al., 2000; Ramirez, 1998; Ravdin et al., 2003; Ross et al., 1987; Rutherford et al., 2005; Stewart et al., 1994; Straume-Naesheim et al, 2005; Tysvaer & Lochen, 1991; Warden et al., 2001, Wilberger et al., 1991; Witol & Webbe, 2003). This is problematic since it is uncertain whether the effects one is seeing are attributed to concussion or to low pre-morbid levels of education, IQ, age or length of exposure to the sport. With respect to age, many studies included athletes of different ages and at various levels of play in their careers without controlling for the length of exposure to the sport. For example in the boxing studies, researchers Casson et al. (1984) included both former and active boxers in his sample, with an age range of 25 to 60 years. Similarly, Ross et al. (1987) included former, professional and active boxers between the ages of 21 to 74 years in their sample. Amongst American football studies Guskiewicz et al. (2000) included a cohort of both collegiate and high school athletes in their sample, moreover no age range was given for either group. Similarly, in a

study on Australian Rules Football, McCrory et al. (2000) reported no age range for his sample of 303 elite athletes.

With respect to level of education and IQ, researchers Casson et al. (1984), Ross et al. (1987) and Kaste et al. (1982) reporting on boxing athletes, all had no control for either level of education or IQ in their samples. This is important considering that generally amongst the boxing community it is thought that these individuals have fewer years of formal education when compared with other athletic control groups. Although Casson et al. (1984) disputed this in their study where the boxers were reported to have secondary or college education, the different levels of education were not controlled for in the sample. It is also uncertain whether researchers Guskiewicz et al. (2000), McCrory et al. (2000) and Maddocks, Saling, et al. (1995) controlled for the level of education or IQ in their samples since this was not reported.

#### **6.3.1.2 Pre-test, Post-test design and Time series design**

In the single group pre-test, post-test design athletes are assessed prior to and following an injury but no control groups are used. Examples of studies using pre-test, post-test designs include boxing (e.g. Warden et al., 2001), ice hockey (e.g. Gaetz et al., 2000), American football (e.g. Pellman et al., 2004,2006), Australian Rules Football (e.g. Dicker & Maddocks, 1988) and Rugby League (e.g. Hinton-Bayre et al., 1997). To the author's knowledge no pre-test, post-test designs have been used in studies investigating the effects of MTBI in either soccer or Rugby Union.

A time series design collects data on the same variable at regular intervals such as days, weeks months or even years. Examples of studies using time series designs include boxing (e.g. Ravdin et al. (2003), ice hockey (e.g. Hatfield et al., 2004), American football (e.g. Barth et al., 1989; Guskiewicz et al., 2003; Macciocchi et al., 2001; McClincy et al., 2006; McCrea et al., 2002; Wilberger et al., 1991) and Rugby League (e.g. Hinton-Bayre et al., 1999). To the author's knowledge no time series designs have been used in soccer, Australian Rules Football and Rugby Union.

Both single group pre-test, post-test designs and time series designs are also vulnerable to all threats to internal validity mentioned above. Apart from the lack of control for pre-morbid characteristics of the athletes and selection bias, both designs are also subjected to threats of instrumentation and maturation. As mentioned previously the neuropsychological evaluation of athletes is different from normal testing situations in that testing takes place over brief intervals of days, weeks, months or years. Although most of the neuropsychological tests employed in testing are valid and reliable, many do not have multiple test forms and hence they can vary as a function of the inter-test intervals (Franzen et al., 2004). According to Lezak et al. (2004), tests with a speed component, which require an unfamiliar mode of response or which have an easy solution are more likely to show practice effects in the form of learning of the test material. For many tests the greatest practice effects seem to occur between the first and second administration (Lezak et al., 2004). Many researchers prevent this problem by administering two baseline tests before introducing an experimental condition and in that way the athlete's optimal level of performance is achieved (Lezak et al., 2004; Macciocchi & Barth, 2004). For example Straume-Naesheim et al. (2005) administered the computerized neurocognitive test battery, CogSport, twice at baseline to a group of professional soccer players in order to control for practice effects in their study although no control group was used. Another solution to control for practice effects is to use alternative versions of the test, but the effectiveness of this method too is questionable particularly with multiple administrations over short test intervals (Macciocchi & Barth, 2004). The recent use of computerized neurocognitive batteries have also provided solutions to assist with repeat-testing, thereby limiting practice effects, although these too are not immune to practice effects (Randolph et al., 2005).

Practice effects also seem to vary between studies and test administrations (Lezak et al., 2004). For example, researchers Macciocchi et al. (2001), in their time series design evaluated collegiate American football athletes at pre-season and then at intervals of 24 hours, 5 and 10 days post-injury. Practice effects were demonstrated on the neurocognitive measures of the PASAT, Symbol Digit Modalities Test and the Trail Making Test. Similarly, Pellman et al. (2004) in their investigation of NFL athletes, assessment took place pre-season and then at 1.4 days after injury (approximately 531 days from baseline testing). Although the time between the pre-season and post-injury evaluation was approximately a year and a half, the results showed that the injured athletes performed significantly better post-injury on tests of attention and

processing speed (Trails A, Digit Span Forward and the Symbol Digit Modalities Test). Whilst Hinton-Bayre et al. (1999) using the same tests of processing speed (e.g. Digit Symbol Substitution Test, Symbol Digit Modalities Test) in their study reported no practice effects with respect to learning amongst Rugby League players at one to three days and at two weeks post-injury (Hinton-Bayre et al., 1999).

### **6.3.1.3 Post-test only control group design**

The post-test only control group design involves athletes being administered tests at some point in time following injury but includes a comparison control group which helps to control for the effect of pre-injury factors (Campbell & Stanley, 1969). Examples of studies using post-test only control group designs are in boxing (e.g. Brooks, Kupsik, et al., 1987; Downs & Abwender, 2002; Drew et al., 1986; Guskiewicz et al., 2002; Porter & Fricker, 1996; McLatchie et al., 1987; Moriarity et al., 2004, Murelius & Haglund, 1991), ice hockey (e.g. Killam et al., 2005), soccer (Abreau et al., 1990; Barnes et al., 1998; Matser et al., 1998, 1999; Rutherford et al., 2005; Stephens et al., 2005; Tysvaer & Lochen, 1991; Webbe & Ochs, 2003; Witol & Webbe, 2003), American football (e.g. Collins, Field, et al., 2003; Iverson et al., 2004b, 2006; Mihalik et al., 2005) and Rugby Union (e.g. Shuttleworth-Edwards et al., 2004). To the author's knowledge no post-test only designs have been used in studies investigating the effects of MTBI in Australian Football League, or Rugby League.

Whilst this post-test only control group design is an improvement from the post-test only no control group design, it is still vulnerable to bias introduced by selection and the interaction of history and selection. For example in Matser et al's (1999) study involving amateur soccer players it was noted that over a quarter of the athletes had suffered a prior concussion not due to sports which was double the incidence of non-sports concussions amongst the controls. Hence, this history-selection interaction could significantly alter the results in respect of the amateur soccer players displaying significant deficits on testing as a result of multiple concussions sustained from both participation and non-participation in soccer. Similarly, in a study by Shuttleworth-Edwards et al. (2004) involving professional Rugby Union players and a control group which was made up of cricket players, some of the cricket players had a prior history of rugby playing which could have contaminated the results.

Another concern amongst post-test control group designs and other designs using control groups involves the matching of the control groups to the experimental groups. In particular, if neither group is matched for IQ or education it is uncertain whether the effects being shown are attributed to the condition being tested. For example, in a boxing study by Drew et al. (1986) nineteen active professional boxers were compared with 10 baseball and basketball athletes. Since neither group was matched for education or IQ it is uncertain whether the effects one is seeing are due to pre-existing low intellectual ability or educational attainment in addition to the multiple trauma sustained. Conversely, a control group with a low pre-existing intellectual impairment or low education would cause the experimental group to look unimpaired by comparison. Collins, Grindel et al. (1999) demonstrated the relationship between a pre-existing impairment such as a learning disability, in combination with multiple concussions in their study on university American football athletes. The concussion groups with two or more concussions and a learning disability performed significantly worse than those athletes with two or more concussions without the learning disability.

#### **6.3.1.4 Time series control group design**

A time series control group design collects data on the same variable at regular intervals such as days, weeks months or even years but includes a comparison control group. Examples of studies using a time series control group design are in boxing (e.g. Bleiberg et al., 2004; Butler et al., 1993; Porter, 2003), American football (e.g. Collins, Grindel, et al., 1999; Field et al., 2003; Guskiewicz et al., 1997; Iverson et al., 2004a; Lovell et al., 2003; Macciocchi et al., 1996), Australian Football League (e.g. Cremona-Meteyard & Geffen, 1994) and Rugby Union (e.g. Pettersen & Skelton, 2000; Shuttleworth-Jordan et al., 1993). To the author's knowledge no time series control group designs have been used in studies investigating the effects of MTBI in ice hockey, soccer or Rugby League. Although this type of design is an improvement on the time series no control group design it still has limitations with regards to instrumentation and maturation.

Whilst limitations with regards to instrumentation have been discussed above, repeated measures designs are particularly vulnerable to maturation effects. Changes that occur to subjects (aging or psychological factors) over time, other than practice, may influence the overall outcome of the

results. For example, Butler et al. (1993) investigated 86 amateur boxers pre-bout, immediately post-bout and then again two years later. Although improvements were evident amongst the groups at post-bout and were maintained at two years, it is uncertain whether the changes seen were attributed to practice effects or to changes as a result of the individuals cognitively maturing over time, since neither group were matched for education or IQ. Similarly, Porter (2003) followed-up a sample of 20 active amateur athletes over a nine year period. Whilst there was a general improvement or preservation of some of the neuropsychological skills relative to the controls these results could have also been due to a maturation effect.

#### **6.3.1.5 Non-equivalent control group design**

According to researchers, the non-equivalent control group design is a more powerful design in respect of providing the strongest inference about the relationship between concussions and neurocognitive and postconcussive sequelae (Macciocchi & Barth, 2004). It is a pre-test, post-test design which uses a matched control group for comparative purposes. Thus, concussed athletes are compared to controls with similar histories (Macciocchi & Barth, 2004). Examples of studies using non-equivalent control group designs in boxing (e.g. Levin, Lippold et al., 1987; Ramirez et al., 2003), soccer (e.g. Putukian et al., 2000), American football (e.g. Barr, 2003; Lovell et al., 2004), Australian Rules Football (e.g. Collie et al., 2006; Maddocks & Saling, 1996; Makdissi et al., 2001) and Rugby Union (e.g. Shuttleworth-Jordan et al., 1993; Shuttleworth-Edwards et al., 2004). To the author's knowledge no non-equivalent control group designs have been used in studies investigating the effects of MTBI ice hockey or Rugby League.

The only possible weakness suggested for the non-equivalent control group design is that of instrumentation, discussed above. In addition to practice effects associated with objective neuropsychological measures, there are many problems associated with the self-reporting postconcussion inventories employed in MTBI research. Whilst more studies are starting to employ postconcussion inventories in conjunction with neurocognitive testing, the validity of such inventories is questionable, owing to the mass underreporting of symptoms that occurs amongst athletes.

### 6.3.2 Statistical and other methodological problems

A further criticism of MTBI studies in sports involves the response rates amongst athletes, which makes one question how representative the samples are if one has to generalize those findings to other sports groups. Generally most MTBI studies do not report response rates. Rutherford et al. (2003) reviewed the response rates amongst soccer studies and reported that very few of the studies reviewed reported response rates. For example, Matser et al. (1999) reported a 100% response rate for their study, whilst Tysvaer & Lochen (1991) and Jordan et al. (1996), reported 86% and 80% response rates respectively (Rutherford et al., 2003). Further methodological issues relate to the use of a limited number of tests, i.e. one or two tests (e.g. Macciocchi et al., 2001; Putukian et al., 2000) thereby increasing the risk for Type II error (not findings significance when it does exist), the lack of consensus regarding the definition of concussion which questions the validity of the reports of concussion history, and the lack of baseline data in some studies.

Other statistical problems include the use of small sample sizes in MTBI research. Since statistical power increases with an increase in the sample size, the probability of finding significance also increases. For example, researchers Macciocchi et al. (2001) evaluating 24 college players found no significant evidence of impairment amongst athletes who sustained one or two concussions. In comparison Collins, Grindel et al. (1999) reporting on their study on 393 male university athletes found that athletes with a history of two or more concussions showed significant impairment with respect to neurocognitive functioning and symptom reporting. The sample size also influences the effect size which is dependent on many things, although a small effect suggests minimal influence of the independent variable (contact sport or multiple MTBI) and a large effect size suggests that the independent variable has considerably influenced the dependent variable (Macciocchi & Barth, 2004). It is interesting to note however, that in Collins, Grindel et al.'s (1999) study the effect size was small, despite the large number of athletes employed into the sample. Whilst researchers assert that in most MTBI studies the effects sizes are generally very small (Binder et al., 1997), given the number of tests used in Collins, Grindel et al.'s (1999) research, their results do have some meaning in terms of MTBI research (Macciocchi & Barth, 2004).

Further statistical weaknesses of these studies investigating MTBI in sport concerns the lack of control for Type I error (finding significance when it does not exist), especially when using multiple measures. Type I error can be controlled for by either increasing the stringency of the criterion for statistical significance or by implementing Bonferroni adjustments. Since the chance of finding significance increases with an increase in the number of measures and most MTBI studies include multiple dependent measures (neuropsychological tests), statistical corrections must be made. Unfortunately few studies evaluating the neuropsychological effects of MTBI in contact sport have made this correction, whilst some have provided very liberal control for Type I error which means that generally there is a good chance that only spurious effects are detected amongst the studies (Rutherford et al., 2003). In order to control for Type I error but also to minimize Type II errors (failing to find significance when it does exist), Rutherford et al. (2005) and Stephens et al. (2005) increased the stringency of the significance level to  $p = 0.01$  in their study, which they did in order to compensate for the use of 16 and 13 neuropsychological tests respectively, without any Bonferroni's adjustments.

Rutherford et al. (2003) further maintain that all studies carried out so far on the neuropsychological effects of concussion (with particular reference to soccer) should be considered as exploratory and not confirmatory research, since the effects reported as significant do not meet the accepted standards for statistical significance but they do indicate associations which should be investigated further in a confirmatory manner. In particular the studies on soccer of Matser and his colleagues (1998, 1999, 2001) have been strongly criticized for failing to provide statistical adjustments for the use of an exceedingly wide battery of tests in their studies (Bourdon-Wiersma test, PASAT, Stroop, Trail Making Test, Wisconsin Card Sorting Test (WCST), Digit Span, Facial Recognition Test, Figure Detection, Complex Figure Test, Verbal Fluency, Puncture Test, Total Raven Score, Digit Symbol Substitution Test, WMS Associate Learning, WMS Logical Memory and WMS Visual Reproduction, 15 Word Learning Test). In conclusion, given the above methodological evaluation of sports studies, a relatively strong study should comprise a non-equivalent control group design, where there is strict control for age, IQ and education between comparative groups and the issue of accepted standards for statistical relevance is addressed.

## 6.4 RATIONALE AND HYPOTHESES FOR THE PRESENT STUDY

### 6.4.1 Rationale for present study

From the above review it appears that most research investigating the long-term effects of MTBI with respect to neurocognitive and postconcussive sequelae have mainly focussed on professional or elite athletes and amateur, collegiate or university athletes (e.g. Farace et al., 2003; Hinton-Bayre & Geffen, 2004; Jordan et al., 1997; Kutner et al., 2000; Macciocchi et al., 1996; Matser et al., 1999; McCrory et al., 2000) with the exception of ice-hockey which has mainly focussed on younger athletes (Ferguson et al., 1999; Gaetz et al., 2000; Hatfield et al., 2004; Killam et al., 2005). The rationale behind this choice of subject group was that these athletes have experienced many years of contact play and hence have been more exposed to the effects of multiple concussive and sub-concussive events in comparison to the less exposed high school athlete. Moreover, the level of play amongst professional and elite groups is generally more intense which further supports the fact that as a whole this group is more vulnerable to residual and long-term concussive effects. The less experienced younger group, however, with relatively less years of exposure to a contact sport would not be expected to demonstrate the same residual and long-term effects as compared with the professional athlete. This has been demonstrated in the studies reported on above where most significant persistent deficits were in evidence for the higher levels of play, including professional, elite and even amateur and collegiate athletes relative to the high school athlete (e.g. Barnes et al., 1998; Casson et al., 1984; Collins, Grindel et al., 1999; Downs & Abwender, 2002; Drew et al., 1986; Guskiewicz et al., 2005; Heilbronner et al., 1991; Jordan et al., 1997; Jordan, Matser et al., 1996; Kaste et al., 1982; Kutner et al., 2000; Levin et al., 1987; Maddocks & Saling, 1996; Matser et al., 1998, 1999, 2001; McLatchie et al., 1987; Murelius & Haglund, 1991; Ross et al., 1987; Rutherford et al., 2005; Tysvaer & Lochen, 1991; Shuttleworth-Edwards et al., 2004).

Recently, however there has been an increase in the number of studies investigating the long-term effects of concussion amongst high school athletes (13 to 18 years), particularly within the last three years (e.g. Barr, 2003; Collins, Field, et al., 2003; Field et al., 2003; Lovell et al., 2003, 2004; Iverson et al., 2004a, 2004b, 2006; McClincy et al., 2006; Mihalik et al., 2005; Moser et al., 2005; Pellman et al., 2006; Ramirez, 1998; Stephens et al., 2005; Webbe & Ochs, 2003;

Witol & Webbe, 2003). Whilst there are no studies investigating the effects of concussion amongst high school athletes in respect of the contact sports of Australian Football League and Rugby League. Few studies have investigated these effects amongst high school athletes in the contact sports of boxing (Ramirez, 1998), ice hockey (Ferguson et al., 1999), soccer (Stephen et al., 2005; Webbe & Ochs, 2003; Witol & Webbe, 2003) and Rugby Union (Shuttleworth-Edwards et al., 2004). The majority of studies, (n=15) investigating the effects of concussion amongst high school athletes have employed American football athletes (Barr, 2003; Collins, Field, et al., 2003; Field et al., 2003; Guskiewicz et al., 2000; Iverson et al., 2002, 2004a, 2004b, 2006; Lovell et al., 2003; Lovell, Collins, Iverson, et al., 2004; McClincy et al., 2006; Mihalik et al., 2005; Moser et al., 2005; Pellman et al., 2006; Wilberger et al., 1991), although eight of these studies have also included collegiate or university and professional athletes in their samples (Field et al., 2003; Guskiewicz et al., 2000; Iverson et al., 2002, 2004a, 2006; McClincy et al., 2006; Mihalik et al., 2005; Pellman et al., 2006). Moreover, in the one ice hockey study reported on above (Ferguson et al., 2004), both high school and collegiate athletes were investigated together in one sample. This was also the case with two of the three soccer studies where high school athletes were incorporated together with collegiate athletes in one study (Webbe & Ochs, 2003) and high school athletes incorporated with amateur and professional athletes in another study (Witol & Webbe, 2003). Although the Rugby Union study investigated the effects of concussion at three different levels of play (high school, university and professional), all these analyses were conducted separately for each level of play (Shuttleworth-Edwards et al., 2004).

In total, there appear to be only seven studies which have investigated the chronic effects of concussion employing high school athletes only, and these include one soccer study (Stephens et al., 2005), five American football studies (Barr, 2003; Lovell et al., 2003; Lovell, Collins, Iverson, et al., 2004; Moser et al., 2005; Wilberger et al., 1991) and one Rugby Union study (Shuttleworth-Edwards et al., 2004). Of these seven studies on chronic effects, one American football study (Barr, 2003) investigated neurocognitive sequelae following concussion only and employed paper-and-pencil measures; two studies, both involving American football athletes (Lovell et al., 2003; Lovell, Collins, Iverson, et al., 2004), employed a computerized neurocognitive battery, (ImPACT); and three studies employed paper-and-pencil measures to investigate both neurocognitive and postconcussion sequelae following concussion (Moser et al., 2005; Shuttleworth-Edwards et al., 2004; Wilberger et al., 1991). Only one soccer study

employed a combination of both paper-and-pencil and computerized neurocognitive tests, although no postconcussion inventory was used (Stephens et al., 2005). None of the studies used a combination of both paper-and-pencil and computerized neurocognitive and postconcussion symptom inventories in their studies investigating high school athletes.

Further, from a methodological perspective six of the seven studies (5 = American football high school athletes and 1 = soccer high school athletes) reported on above can be seen to have sampling limitations. Two studies have no control groups (Lovell, Collins, Iverson, et al., 2004; Wilberger et al., 1991), three studies have no control for gender (Barr, 2003; Lovell et al., 2003; Lovell, Collins, Iverson, et al., 2004), four studies have no control for IQ (Barr, 2003; Lovell et al., 2003; Lovell, Collins, Iverson, et al., 2004; Moser et al., 2005), two studies have no control for age (Lovell et al., 2003; Moser et al., 2005) and three studies have no control for education (Barr, 2003; Moser et al., 2005; Stephens et al., 2005). The only study examining the chronic effects of high school athletes and where strict control for age, IQ and education between comparative groups was made was conducted on Rugby Union high school athletes (Shuttleworth-Edwards et al., 2004). However, this last study was limited to the use of paper-and-pencil tests only, and comprised only a single assessment at pre-season.

Thus in summary, few studies up to now have focussed on the long-term neuropsychological effects of concussion amongst high school athletes, comprising only seven and (as reported above) only one of these studies demonstrated strict control for age, IQ and education (Shuttleworth-Edwards et al., 2004). None of these studies have used a combination of both paper-and-pencil and computerized neurocognitive and postconcussion symptom inventories in their investigations of concussive and sub-concussive effects in this age group.

In light of the above, it was decided to conduct an extensive investigation into the long-term consequences of male high school participation in a contact sport. For this particular study, the main contact sport played within the school targeted for the research was Rugby Union, although a small proportion of the boys were involved in other contact sports such as soccer and the martial arts. The current study followed modern developments in concussion assessment, by utilizing an internationally recognised and widely used computerized test for this purpose, viz. the ImPACT programme. This test appears to be the most comprehensive and extensively

researched computerized neuropsychological tool used in the assessment of MTBI in sport (Podell, 2004). For comparative purposes in terms of test sensitivity to concussion sequelae, the ImPACT test was used in conjunction with a prototypically sensitive paper-and-pencil measure, the Digit Symbol Substitution Test which taps visuo-perceptual processing at speed, and has the added advantage of providing access to an adjunctive Digit Symbol Substitution Incidental Paired Associate Recall task which was incorporated for the purposes of this study in both immediate and delayed form. In addition to the above cognitive measures, two postconcussion symptom measures were employed in the research for the purpose of evaluating the postconcussive symptoms experienced by the high school athletes. One formed part of the ImPACT programme and the other, a paper-and pencil questionnaire, based on a questionnaire used in prior research in South Africa on Rugby Union athletes (Shuttleworth-Edwards et al., 2004) contained four additional symptoms over the ImPACT scale. Two of the additional symptoms (aggression and speech problems) were shown to be sensitive to the effects of diffuse closed MTBI (Shuttleworth-Edwards et al., 2004).

In conclusion, in lieu of the above methodological concerns and evaluation of sports studies in general, the present study comprised a non-equivalent control group design, where strict control for age, IQ and education between comparative groups was made and where the issue of accepted standards for statistical relevance are addressed (see Methodology, Chapter seven).

#### **6.4.2 Theoretical Hypotheses**

Based on the rationale proposed for the present study, the following formal theoretical hypotheses were posed:

(i) It was hypothesized that in comparison with the Non-Contact group, the Contact group would demonstrate deleterious neuropsychological effects in respect of cognitive deficits made evident on objective neurocognitive testing and self-reported postconcussive symptoms established on the basis of two postconcussion inventories, due to postulated exposure to repetitive concussion (MTBI), including both concussive and sub-concussive events, during high school participation in contact as opposed to non-contact sport at both pre- and post-season occasions.

(ii) Further it was hypothesized that in comparison with the 0 concussion group, the 2+ concussion group would demonstrate deleterious neuropsychological effects in respect of cognitive deficits made evident on objective neurocognitive testing and self-reported postconcussive symptoms established on the basis of two postconcussion inventories, due to postulated exposure to repetitive concussion (MTBI) at both pre- and post-season occasions.

(iii) It was hypothesized that *within* the Contact group, the 2+ concussion group would demonstrate deleterious neuropsychological effects made evident on objective neurocognitive testing and self-reported postconcussive symptoms established on the basis of two postconcussion inventories, due to exposure to repetitive concussion during high school participation in a contact sport.

(iv) It was hypothesized that *either*

- in comparison to the Non-Contact group, the Contact group would demonstrate deleterious neuropsychological effects in respect of cognitive deficits made evident on objective neurocognitive testing and self-reported postconcussive symptoms established on the basis of two postconcussion inventories at post-season compared to pre-season, due to the effects of unreported concussive and sub-concussive events, sustained during participation in a contact sport over a six month winter sports season;

*or*

- in comparison with the Non-Contact group the Contact group would demonstrate no significant neurocognitive impairment due to practice effects at the post-season compared to the pre-season interval, due to the inability to benefit from practice, being cerebrally vulnerable as a consequence of participating in a contact as opposed to a non-contact sport.

(v) Finally, in support of the BRC theory, it was hypothesized that these effects would be less in evidence amongst this sample of high school athletes compared with adult professional level players owing to the lower level of play (less intense and less years of exposure to contact sport as opposed to a non-contact sport).

**Table 6.2.1**  
**Summary of studies investigating the cognitive and postconcussive symptom outcome following MTBI in boxing ('C' refers to studies investigating chronic effects, 'A' refers to studies investigating acute effects<sup>1</sup>)**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Kaste et al. (1982)	Cross-sectional (C)	14 boxers: 8 amateurs, 6 professionals (reported on separately)	WAIS-Information, Similarities, Digit Span, Block Design and Object Assembly, WMS-Associate learning, WCST, Trail Making test, Purdue pegboard, Benton Visual Retention Test	Not measured	12 professional boxers took longer on the Trail Making Test. Two professionals showed objective signs of brain injury being below normal on all tests and showed other subjective symptoms (specific symptoms not reported in article).	-Small sample size in each comparative group (professionals: n=6; amateurs: n=6) -No control group -No control for age (range = 19-54yrs) -No control for education and weak control for IQ (mean IQ=112, higher than the largest group in the sample consisting of Finnish champions, average IQ = 100)
Casson et al. (1984)	Cross-sectional (C)	18 former and active boxers (13 former, 5 active professional boxers, reported on separately)	Trail Making test, Digit Symbol Test, WMS and recall of the Bender Visual-Motor Gestalt figures	Not measured	All boxers scored in the impaired range on more than one neuropsychological measure	-Small sample size (N=18) -No control group -No control for age (range=18-60 yrs), education or IQ
Drew et al. (1986)	Cross-sectional (C)	19 active professional boxers and ten matched control athletes (baseball and basketball)	Halstead-Reitan Neuropsychological Test Battery and Randt Memory Test	Not measured	Fifteen of the 19 boxers scored in the impaired range on the Reitan impairment Index (compared to two controls) boxers were more impaired than controls on all tests except the Seashore Rhythm, Finger Tapping and Category Test.	-Small sample size in each comparative group (boxers: n=19; controls: n=10)
Brooks, Kupsik, et al. (1987)	Cross-sectional (C)	29 amateur boxers and 19 controls	WMS-Logical Memory, Digit Symbol and Associate Learning, Rey Complex Figure copy and recall, PASAT, Simple and four choice reaction time	Not measured	Concluded no evidence of impairment	-Small sample size in each comparative group (boxers: n=29; controls: n=19) -Weak control group (11 of 19 controls were prospective boxers in training) -No control for education or IQ (boxers IQ=98.8 and control IQ=84)
Levin, Lippold et al. (1987)	Prospective: Time one testing, & 6 months later (C)	11 active professional, 2 active amateur and 13 sports men controls	PASAT, Continuous Performance Test, microcomputer reaction time task, Modified Selective Reminding Test, Digit Span test, the Benton Visual Retention Test, Controlled Word Association Test, Design Fluency, Digit Symbol Test, Trail-Making Test and Purdue Pegboard	Not measured	The results from the initial testing demonstrated more proficient verbal learning in the control group but no differences between the groups on the delayed memory recall and other tests of memory. Reaction time was faster in the boxing group compared with the controls. Six months after the initial baseline testing, 10 subjects in both the boxing and control groups demonstrated improvements in their neurocognitive performances compared with their initial baseline performance, indicating a practice effect, with no differences in scores or in the magnitude of improvement between the groups.	-Small sample size in each comparative group (boxers: n=13; controls: n=13) -No control for level of play

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size =  $n \leq 30$

Tables 6.2.1 cont.<sup>1</sup>  
Outcome following MTBI in boxing

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
McLatchie et al. (1987)	Cross-sectional (C)	20 active amateur boxers (only 15 of the 20 boxers underwent neuropsychological evaluation) and 20 orthopaedic patient controls	Digit Span subtest of the WAIS, the three paired word learning test of Inglis (1959), word learning from the WMS, Complex Figure Test, the PASAT and simple and Four Choice ReactionTimeTest	Not measured	Nine of the 15 boxers performed poorly on neurocognitive testing of verbal learning (three paired word learning test of Inglis) and visual memory (immediate recall of Complex Figure).	-Small sample size in each comparative group (boxers: n=20; controls: n=20) -No control for age (range =18-49yrs), education or IQ
Ross et al. (1987)	Cross-sectional (C)	38 professional boxers (only 15 of the 38 boxers underwent neuropsychological evaluation)	Trail Making test, WAIS- Digit Symbol Substitution test, Bender-Gestalt Test and WMS	Not measured	Fourteen of the 15 active professional boxers scored abnormally on the recall of the Bender-Gestalt Test. All 13 boxers scored in the abnormal range on verbal portion of Wechsler Memory Scale (WMS) and 11 of the 13 scored in the abnormal range on the visual portion of the WMS. Ninety percent of the memory scores fell in the abnormal range and 50% of the non-memory scores fell in the abnormal range.	-Small sample size (N=15) -No control group -No control for age (range=21-74 yrs), education or IQ
Heilbronner et al. (1991)	Cross-sectional and Prospective: Pre- and post-boxing match (A)	23 amateur boxers	Digit Symbol Incidental Recall – Immediate and Delayed, Visual and Verbal recall of words and symbols, psychomotor speed (hand tapping), Trail Making Test, Digit Span	Not measured	Their findings demonstrated that compared with their pre-fight performances minor deficits in verbal learning and motor speed were detected in the boxers. Boxers with the greater number of years of exposure to boxing had slower finger tapping speeds as well as lowered scores on the Digit Symbol Incidental Recall – Immediate and Delayed.	-Sample size small (N=23) -No control group -No control for age (range=16 to 30 yrs), education or IQ
Murelius & Haglund 1991	Cross-sectional (C)	50 amateur boxers 25 soccer players and 25 track and field athletes	Synonyms Test (SRB) and Halstead-Reitan batteries	Not measured	No definite signs of intellectual impairment amongst boxers, although slightly inferior finger tapping speeds seen in boxers who had taken part in a large number of bouts although this was still within the normal range.	-Weak control for education or IQ (74% of boxers had a low level of education, whilst 66% of the soccer and track and field athletes had a high degree of education)
Butler et al. (1993)	Prospective: Pre-bout, immediately post-bout and again 2 years post-injury (A & C)	86 amateur boxers, 47 rugby players and 31 waterpolo players	British Ability Scales, a serial addition task, immediate and delayed recall of words and visual stimuli	Not measured	No evidence of neuropsychological dysfunction due to boxing was found, either following a bout or a series of bouts. Significant improvements amongst all the groups were seen at the immediate post-bout testing and were maintained at two years. The boxers showed the greatest improvements on the timed tasks in relation to the other groups	-No control for age (range=12 to 27 yrs), education or IQ

Note.<sup>1</sup> For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

Note.<sup>2</sup> Small sample size =  $n \leq 30$

Table 6.2.1 cont. <sup>1</sup>  
Outcome following MTBI in boxing

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Stewart et al. (1994)	Cross-sectional & Prospective: Pre-season and 2yrs later (C)	484 amateur boxers evaluated pre-season and of those 393 evaluated two years later	Grooved Pegboard, Rey Complex Figure, RAVLT, Digit Span test, Ravens Matrices, Block Design, Word Fluency Test, Symbol Digit Modalities Test, Alphabet Recital, Trail Making Test, Two-Choice Reaction Time Test and Vigilance Test	Not measured	No significant changes were detected over the course of the study, but significant trends between the number of bouts prior to baseline testing and changes in memory and visuomotor construction ability were found on the Block Design subtest of the WAIS.	-No control group -No control for age (range=13-21 yrs), education or IQ
Porter & Fricker (1996)	Cross-sectional & Prospective: (15-18month) (C)	20 active amateurs and 20 controls	Trail Making Test – Part A and B and Finger Tapping tests, Rey Complex Figure, Digit Symbol, the Digit Span test and the WMS–Paired Associate Learning test	Not measured	The boxers performed significantly better on the TMT-A and B than the controls but the controls demonstrated significantly higher finger tapping speeds on the Finger Tapping test. There was no association between boxing exposure and the performances on any of the neuropsychological tests used.	-Small sample size in each comparative group (boxers: n=20; controls:n=20) -No control for age (range=16-25 yrs), education or IQ
Jordan, Matser et al. (1996)	Cross-sectional (C)	42 professional boxers	WAIS subtests, Similarities, Vocabulary and Digit Symbol Substitution Test, Verbal Fluency test, Figure Detection, PASAT, WMS, Stroop Color and Word test, Complex Figure, WCST, Trail Making Test, Puncture and Bourdon	Not measured	Seventeen of the boxers had CT scans that revealed borderline brain atrophy and deficits on the Rey Complex Figure, Stroop Color and Word Test, Verbal Memory and Visual Reproduction subtests of the WMS, which were related to indices of increased sparring exposure.	-No control group -No control for age (range = 19- 31 yrs), education or IQ (although estimated IQ was evaluated the results were not reported on)
Jordan et al. (1997)	Cross-sectional (C)	30 professional boxers	Chronic Brain Injury (CBI) Scale	Some symptoms included in CBI scale such as agitation or aggression, delusions, hallucinations, dysphoria, anxiety, euphoria, apathy, disinhibition, irritability or lability	Amongst the boxers, 11 were normal, 12 showed mild impairments, four were moderately impaired and three showed severe impairments. High exposure boxers had significantly higher CBI scores. Moreover, the high exposure boxers with the APOE e4 allele had significantly higher CBI scores than high exposure boxers without the APOE e4 allele. All boxers with severe impairment possessed at least one APOE e4 allele. The results suggest that the possession of the APOE e4 allele is associated with increased chronic neurological deficits amongst high-exposure boxers.	-No control group -No control for age (range= 23 to 76 yrs), education or IQ

*Note.*<sup>1</sup> For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.*<sup>2</sup> Small sample size =  $n \leq 30$

**Table 6.2.1 cont. <sup>1</sup>**  
**Outcome following MTBI in boxing**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Ramirez (1998)	Cross-sectional & Prospective: One hour and 8 weeks postconcussion (A)	10 amateur boxers and 10 basketball players	Test of Variable Attention	Not measured	The cumulative effects of the head blows during the season did not have a significant effect on the TOVA measures of inattention, impulsivity or information processing and motor response speed. Variability of response time was the only index by which the mean score of the boxers differed from the norm and consistency of attention the only variable affected by the number of head blows. The boxers' reaction time was also lower than the basketball players at the end of the season as well as eight weeks later.	-Small sample size for each comparative group (boxers: n=10; controls: n=10) -No control for education or IQ
Warden et al. (2001)	Cross-sectional & Prospective: One hour, 4 days postconcussion (A)	14 military cadets involved in physical education boxing	Automated Neuropsychological Assessment Metrics (ANAM) computerized neurocognitive test battery	Not measured	The results showed significant slowing on their post-injury reaction time and continuous performance tests, even after the cadets experienced symptom resolution	-Small sample size (N=14) -No control group -No control for education or IQ
Porter (2003)	Prospective: 9 year study (C)	20 male active amateurs and 20 age matched controls	Trail Making Test, The Digit Symbol Substitution Test, Finger Tapping Test, WMS-Paired Associate Learning, Digit Span, Rey Complex Figure Test,	Not measured	No deterioration in neuropsychological test performance was observed over the 9-year period amongst the amateur boxers	-Small sample size for each comparative group (boxers: n=20; controls: n=20) -No control for age (range=16-25 yrs), education or IQ
Ravdin et al. (2003)	Prospective: Pre-injury and at 3 time points postconcussion (i.e. mean of 4days, 10 days and one month post-bout) (A)	18 active professional boxers (boxers divided into high exposure boxers: >12 professional bouts and low exposure, 12 professional bouts)	WAIS-R- Digit Span, Block Design and Vocabulary subtests, Hopkins Verbal Learning Test, Grooved Pegboard, Trail Making Test, Controlled Oral Word Association Test and Stroop Color Word Test	Not measured	Repeated measures revealed significant within subject differences across testing of verbal fluency (COWAT) and information processing (Trail Making Test) between the high exposure and the low exposure groups in the direction of the high exposure group performing worse. Significant differences were noted between baseline and one-month post bout testing, with scores one month following a bout significantly improving. Although memory scores did not change across testing, prior boxing exposure was associated with poorer memory performance.	-Small sample size (N=18) -No control group -No control for age (range=20-40yrs) and particularly between high exposure (mean=33.3 yrs) and low exposure (29.8 yrs) groups

*Note.*<sup>1</sup> For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.*<sup>2</sup> Small sample size =  $n \leq 30$

**Table 6.2.1 cont. <sup>1</sup>**  
**Outcome following MTBI in boxing**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Bleiberg et al. (2004)	Prospective: Pre-season and at intervals of 0 to 23 hours, 1 to 2 days, 3 to 7 days and at 8 to 14 days postconcussion (A)	64 cadets who sustained boxing related concussions and 18 controls	ANAM computerized neurocognitive test battery	Not measured	Only one out of the 6 subtests (Spatial Processing) yielded a significant group difference between 0 to 23 hours and 1 to 2 days post-injury, and another subtest (Mathematical Processing) on the 0 to 23 hours test interval. Full recovery took place 7 days post-injury.	-Small sample size for control group (n=18)
Moriarty et al. (2004)	Prospective: Baseline and within 7 days post-bout (A)	82 amateur boxers and 30 matched controls	Computerized test battery, CogSport which includes tests of simple reaction time, choice reaction time, working memory and learning	Not measured	Seven boxers not diagnosed with a concussion but whose bout was stopped by the referee, showed significant slowing in simple and choice reaction time. None of the others displayed any evidence of cognitive dysfunction in the immediate post-bout period.	-No control for age, education or IQ

**Table 6.2.2**  
**Summary of studies investigating the cognitive and postconcussive symptom outcome following MTBI in ice hockey ('C' refers to studies investigating chronic effects, 'A' refers to studies investigating acute effects)**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations
Ferguson et al. (1999)	Cross-sectional (C)	209 male collegiate and high school athletes: MTBI group, n = 50 athletes (56% ice hockey, 16% American football, 14% rugby football, 12% lacrosse, 2% soccer) sustained. Non-injured control group, n = 159 (49% ice-hockey, 23% American football, 13% lacrosse, 9% soccer, 6% rugby football)	None	30-item symptom checklist includes symptoms of memory, e.g. loses car keys, forgets names of new acquaintances, groceries, yesterday's breakfast, appt dates, why entered a room, daily conversation, where car was parked, who they saw yesterday, directions, faces on new acquaintances, recent phone conversations, etc, concentration difficulties, anxiety, sensitivity to light, irritability, depression, fatigue, trouble thinking, dizziness, blurry / double vision, headache	MTBI athletes symptom rates did not differ from controls. The head injured athletes did however, consistently underestimated the incidence of symptoms pre-injury. Athletes with no history of MTBI overestimated the degree of pre- to post-injury change in symptom reporting. The authors concluded that symptom expectation as a psychological variable has a significant effect on the perception of persistent PCS	-No control for level of play

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size =  $n \leq 30$

**Table 6.2.2 cont. <sup>1</sup>**  
**Outcome following MTBI in ice hockey**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Gaetz et al. (2000)	Cross-sectional  Pre-season and 6 months postconcussion (C)	271 junior ice hockey athletes: All athletes were assigned to one of four groups based on their concussion history: non-concussed athletes (0), athletes who had one concussion (1), athletes who had two concussions (2) and athletes who had more than three concussions (3+).	None Neurological measure (Event-related potentials)	Postconcussion questionnaire includes symptoms of headache, irritability, anxiety, depression, memory, concentration and taking longer to think	The results showed that athletes who sustained three or more concussions took significantly longer to respond to the stimuli on the event related potentials. Moreover, significant differences in the symptoms of 'memory' and 'taking longer to think' were reported for this groups which correlated with their slow responses on the event-related potential recordings.	-No control group -No control for age, education or IQ
Hatfield et al. (2004)	Cross-sectional  (i) Pre-season  (ii) 24-72 hours postconcussion and at different time points until symptom resolution  (A&C)	44 amateur athletes	Peabody Picture Vocabulary Test III, the Digit Span test, the Symbol Digit Modalities test, the Stroop test, the Trail Making Test, the Selective Reminding Test, Visual Spatial Learning Test, the Controlled Oral Word Association Test, Animal Naming, Grooved Pegboard, Finger Tapping Test, Grip Strength Test, and the Lafayette Multi-Choice Reaction Timer	Symptom Check List-90-Revised (individual symptom unknown)	The results at baseline demonstrated no significant results between those who had experienced a concussion and those who didn't, with only Digits Backwards (attention) approaching significance in the direction of those athletes with prior concussions performing worse. Six athletes sustained concussions over the season and were subjected to repeat testing, with the time period for return to play ranging from 5 to 175 days (approximately 6 months). The results indicated that tests demonstrating declines from baseline levels varied amongst the concussed athletes, although tests of complex attention (Trail Making Test, Digit Span test, the Symbol Digit Modalities Test) and memory (Selective Reminding Test, Visual Spatial Learning Test) appeared to be the most affected. Although a symptom checklist was employed in this study it was not reported on.	-No control group -No control for age

*Note.*<sup>1</sup> For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.*<sup>2</sup> Small sample size =  $n \leq 30$

**Table 6.2.2 cont. <sup>1</sup>**  
**Outcome following MTBI in ice hockey**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Killam et al. (2005)	Cross-sectional (C)	28 collegiate athletes (ice-hockey, field hockey, lacrosse and soccer)  a) non-athlete non-concussed (n = 8, male = 5, female = 3), b) athlete non-concussed (n = 9, male = 6, female = 3), c) athlete non-recent concussed (n = 6, male = 3 and female = 3) and d) athlete/recent concussed (n = 5, male = 3, female = 2).	Repeatable Battery for the Assessment of Neuropsychological Status (RBANS) and the Stroop test	Postconcussion Syndrome Checklist: forgetting, anxiety, depression, sensitivity to light, fatigue, trouble thinking, dizziness, blurry or double vision, and headaches	The results demonstrated that no significant group effects were noted for the cognitive domains of visuoconstructional, language and attention domains of the RBANS, although significant effects were noted for the immediate memory scores and total RBANS scores. Relative to the non-athlete group, the athlete/non-concussed and athlete/recent concussed groups were impaired in the immediate and delayed memory domains. Further, the total RBANS score was significantly lower in the athlete non-concussed, athlete non-recent concussed and athlete/recent concussed groups. The worst impairment was shown in the athlete/recent concussed group for the immediate memory domain. No significant differences on the scores obtained from the Postconcussion Syndrome Checklist and on the Stroop task were detected between the groups. The Postconcussion Syndrome Checklist correlated negatively with the attention, delayed memory and total scores from the RBANS and the higher number of concussions correlated with processing speed on the Stroop task.	-Small sample size in each comparative group (a) n=8; (b) n=9; (c) n=6; (d) n=5 -No control for IQ, gender or level of play

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size =  $n \leq 30$

Table 6.2.3

Summary of studies investigating the cognitive and postconcussive symptom outcome following MTBI in soccer/football ('C' refers to studies investigating chronic effects, 'A' refers to studies investigating acute effects<sup>1</sup>)

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Abreau et al. (1990)	Cross sectional (C)	31 college soccer players with 31 tennis player controls	PASAT, Perceptual Speed Test, Symbol Digit Modalities test, Ravens Matrices	Symptom Questionnaire included symptoms of headache, blurred vision, dizziness and knocked out	No significant differences observed between the groups on any of the neuropsychological measures but a negative correlation evident in soccer players between the PASAT score and the number of games played. Soccer players also reported more symptoms on the questionnaire (headache, blurred vision, and dizziness)	-No control for level of play
Tysvaer and Lochen (1991)	Cross sectional (C)	37 former Norwegian soccer players and 20 patient controls	WAIS-R, Trail-Making Test, Benton Visual Retention Test	Not measured	The soccer players consistently showed higher proportions of impaired functioning especially on the WAIS-R and the Trail Making Test relative to controls.	-Small sample size for control group (n=20) -Weak control group (non-athletes)
Jordan, Green et al. (1996)	Cross sectional (C)	20 National soccer team players and 20 age matched controls consisting of track athletes	Not measured	10-symptom questionnaire includes symptoms of headache, attention deficit, dizziness, memory deficit, depression, irritability, lack of energy, sleep disturbance, hearing impairment and neck pain	Evidence of encephalopathy relates more to acute head injuries than to the effects of heading. No significant differences in symptom reporting between the groups. But the number of prior head injuries correlated significantly with the head injury cognitive and somatic symptoms	-Small sample size in each comparative group (soccer players: n=20; controls: n=20) -Weak control group (8 of the 20 controls experienced prior head injuries) -No control for education or IQ
Barnes et al. (1998)	Cross-sectional (C)	62 male and 65 female elite soccer players	Not measured	Postconcussion symptom questionnaire included symptoms of headache, dazed, dizziness, poor concentration, blurred vision, loss of consciousness, numbness/tingling, amnesia	Headache was the symptom that was more often reported in both groups (54 male, 55 female). Men reported more symptoms of blurred vision and numbness and tingling whereas females reported more symptoms of feeling dazed and dizzy.	-No control group -No control for age (17 to 30 yrs), education or IQ between groups
Matser et al. (1998)	Cross-sectional (C)	53 professional Dutch soccer players and 27 elite non-contact sports athletes (track and swimming)	Bourdon-Wiersma test, PASAT, Stroop, Trail Making Test, Wisconsin Card Sorting Test (WCST), Digit Span, Facial Recognition Test, Figure Detection, Complex Figure Test, Verbal Fluency, Puncture Test, Total Raven Score, Digit Symbol Substitution Test, WMS Associate Learning, WMS Logical Memory and WMS Visual Reproduction, 15 Word Learning Test	Not measured	Soccer players demonstrated impaired performances on the Bourdon-Wiersma test, the WCST, the CFT, the Total Raven Score, the Associate Learning, Logical Memory and Visual Reproduction test scores and the Figure Detection test. Once confounding variables had been controlled for significant decrements were found on the Figure Detection test, CF Test, Logical Memory and Visual Reproduction test scores. Performances varied according to field position, number of concussions and frequency of heading	-No control for IQ

Note.1 For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

Note.2 Small sample size =  $n \leq 30$

**Table 6.2.3 cont. <sup>1</sup>**  
**Outcome following MTBI in soccer/football**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Matser et al. (1999)	Cross-sectional (C)	33 amateur soccer players to 27 controls	Bourdon-Wiersma test, PASAT, Stroop Colour Word Test, the Trail Making Test, Wisconsin Card Sorting Test (WCST), Digit Span, Facial Recognition Test, Figure Detection test, Complex Figure Test (CFT), Verbal Fluency, Puncture Test, Total Raven Score, Digit Symbol Substitution Test, WMS Associate Learning, WMS Logical Memory and WMS Visual Reproduction and 15 Word Learning Test	Not measured	Deficits in planning and memory (WCST, CFT, Digit Span Test, Logical Memory and Visual Reproduction tests) were reported in the soccer players compared with controls. Once confounding variables were controlled for decrements in performances were seen on the Digit Span test, Assoc. Learning, Logical Memory and Visual Reproduction tests. Unlike the previous study (1998) no neuropsychological analyses were made on the relationship between soccer exposure and neuropsychological performance. The number of concussions reported however, were inversely correlated with performances on the Digits Forwards, Facial Recognition Task, CFT, DSST and Logical Memory.	-Small sample size for control group (n=27) -No control for IQ
Putukian et al. (2000)	Cross-sectional & Prospective: Pre-injury and after two training sessions, 2 days separating assessment times (A&C)	44 males and 56 female college soccer players. Comparison groups were based on data collected from 23 male active headers and 30 female active headers with a total of 21 controls from the men's team and 26 controls from the female	Alphabet backwards, Trail Making Test, Stroop Colour Word Test, and modified version of the VIGIL/W (computerized continuous performance test)	Symptom checklist (symptoms not specified) including symptoms most commonly reported after MTBI	The pre- and post-test scores indicated no significant differences between the groups. A significant difference between the pre and post-season measures scores was evident, irrespective of whether the subjects were heading or not which meant that most subjects did better on their postseason testing, suggestive of a practice effect. There was a significant gender effect on measures of attention and concentration Where females performed significantly better on the Stroop Test whilst males performed better on the Alphabet Backwards Test. No difference in symptom reporting between the active headers and the non-headers	-Small sample size in each comparative group (male headers: n=23; male controls: n=21; female headers: n=30; female controls : n=26) -Weak control groups
Matser et al. (2001)	Cross-sectional (C)	84 active professional soccer players	Bourdon-Wiersma test, PASAT, Stroop, Trail Making Test, WCST, Facial Recognition Test, Figure Detection, CFT, Verbal Fluency, Puncture Test, Total Raven Score, DI Substitution Test, WMS Associate Learning, WMS Logical Memory and WMS Visual Reproduction, 15 Word Learning Test	Not measured	Number of headers in one season was related to poorer results on tests measuring attention & visual or verbal memory (CFT, Trails, & 15 Word Learning Test) & the number of soccer related concussions related to poorer performances on tests of attention & visuo-perceptual processing (CFT, Facial Recog. Test, & Bourdon-Wiersma test).	-No control group -No control for IQ

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size =  $n \leq 30$

**Table 6.2.3 cont. <sup>1</sup>**  
**Outcome following MTBI in soccer/football**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Downs & Abwender (2002)	Cross-sectional (C)	26 college soccer players (15 males, 11 females) and 6 male current or former professional soccer players. 22 young swimmers (7 males and 15 females) and 7 older male swimmers	PASAT, Finger Tapping test, Continuous Performance test, and the WCST	Not measured	The results showed that soccer players performed worse than the swimmers on tests of conceptual thinking (WCST), with the older soccer group, in particular, performing worse on measures of conceptual thinking, reaction time and concentration (WCST, Finger Tapping test, Continuous Performance test). Although the length of participation in soccer was associated with poorer neuropsychological performance, the findings did not specifically implicate heading as the cause	-Small sample size in each comparative group (soccer players: n=26; controls: n=22; former soccer players: n=6 and older controls: n=7) -No control for IQ or gender
Guskiewicz et al. (2002)	Cross-sectional (C)	91 college soccer players, 96 athletes (woman's field hockey n= 18, woman's lacrosse, n= 51 and men's baseball n= 27) and 53 student controls	Scholastic Aptitude test, Trail Making test, COWAT, Stroop Color Word Test, Hopkins Verbal Learning Test, Symbol Digit Modalities Test, and Digit Span test.	Not measured	No significant relationship between soccer athletes and controls on neuropsychological testing and scholastic aptitude was found, despite a history of soccer related concussions and an average of 15.3 seasons of soccer exposure amongst the soccer players.	-No control for IQ or gender
Webbe & Ochs (2003)	Cross-sectional Individuals evaluated after recent heading (A)	64 high school and collegiate soccer players and 20 sports controls	California Verbal Learning test (CVLT), Complex Figure test, Trail Making test, Benton Facial Recognition test, PASAT and Shipley Institute of Living Scale	Not measured	Depressed scores were seen on tasks of verbal learning (CVLT), planning and attention (Trails-B), as well as on information processing speed and divided-attention ability (PASAT)	-Small sample size for control group (n=20) -No control for level of play (high school and collegiate athletes mixed)
Witol & Webbe (2003)	Cross-sectional: (C)	60 male high school, adult amateur and professional soccer players divided into low, moderate and high heading groups according to current and lifetime heading frequency and 12 non-soccer controls	The Shipley Institute of Living Scale, Trail Making Test, PASAT, Facial Recognition Test, Complex Figure test, Rey Auditory Leaning Test (RAVLT)	Not measured	Players with the highest estimated frequency of heading produced more impaired scores (Trail Making Test, PASAT and the Shipley Institute of Living Scale) than did control participants or player who didn't head the ball as frequently	(i) Current frequency analysis: -Small sample size for each comparative group (control: n=12, low: n=17, moderate: n=24 and high: n=19) -No control for IQ or level of play  (ii)Lifetime heading analysis -Small sample size for each comparative group (control: n=12, low: n=19, moderate: n=20 and high: n=21) -Weak control for education (moderate heading group had 11.85 years of education in comparison to the control and high frequency heading groups who had education levels of 14.92 and 14.19 respectively) -No control for IQ or level of play

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size =  $n \leq 30$

**Table 6.2.3 cont. <sup>1</sup>**  
**Outcome following MTBI in soccer/football**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Rutherford et al. (2005)	Cross-sectional (C)	22 amateur soccer players, 17 rugby players and 24 non-contact sportsmen	Beck Depression Inventory, National Adult Reading Test, Rey Complex Figure Test, Digit Symbol Substitution Test, Digit Span Test, Trail Making Test, Stroop, Logical Memory, Paired Associates, Tests of Attention Performance (TAP) including Alertness, Divided Attention, Visual Search, Covert Attention shift, Flexibility and Working Memory, WCST, Alternate Uses and COWAT	Not measured	Across the groups the results showed that the number of head injuries predicted poorer performances on asks of cognitive flexibility (Trails B and TAP Divided Attention tests). After controlling for the number of head injuries sustained soccer players obtained lower scores on the TAP Divided Attention task than both the rugby and non-contact sports groups. Moreover, those soccer players who were frequent headers achieved fewer categories on the WCST.	-Small sample size for each comparative group (soccer players: n=22; rugby players: n=17; controls: n=24)
Stephens et al. (2005)	Cross-sectional (C)	23 high school soccer, 23 rugby and 20 non-contact sports players (badminton, basketball, swimming, athletics, climbing)	WISC-R Vocabulary, Childhood Depression Inventory Short Form, Eysenck Personality Questionnaire Junior, State-Trait Anxiety Inventory, RCF, Digit Symbol, Trail Making Test, Stroop, WMS Logical Memory, the Test of Attention Performance (TAP), WCST and Alternate Uses	Not measured	There was no relationship between head injury and neuropsychological performance amongst the sports groups. Amongst the soccer players, heading did not predict any of the neuropsychological test scores	-Small sample size for each comparative group (soccer players: n=23; rugby players: n=23; controls: n=20) -Weak control for education
Straume-Naesheim et al. (2005)	Cross-sectional (C)	271 professional soccer players	Computerized test battery, CogSport which includes tests of simple reaction time, choice reaction time, working memory and learning	Not measured	No evidence of neuropsychological impairment due to heading or previous concussions on the computerized test battery, CogSport	-No control group -No control for IQ

**Table 6.2.4**  
**Summary of studies investigating the cognitive and postconcussive symptom outcome following MTBI in American football ('C' refers to studies investigating chronic effects, 'A' refers to studies investigating acute effects)**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations
Barth et al. (1989)	Prospective: Pre-season & at intervals at 24 hours, 5 days, 10 days and 12 weeks postconcussion (A)	2,350 College American Football players	Trail Making test -Part A and B, the Halstead-Reitan Neuropsychological Test Battery, the Symbol Digit Test, and the Paced Auditory Serial Addition Task (PASAT)	Not measured	Rapid although not complete recovery took place over a period of five to ten days. Although the study did not reveal severe cognitive fall-off following concussion, subtle difficulties were found on the tests sensitive to information processing speed (Symbol Digit Modalities Test).	-No control group -No control for IQ

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size =  $n \leq 30$

**Table 6.2.4 cont. <sup>1</sup>**  
**Outcome following MTBI in American football**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Wilberger, Haag & Maroon (1991)	Prospective: Pre-season and at 24 hours, 30 days and 90 days, post-concussion  (A&C)	62 high school athletes who sustained 2+ concussions	Wechsler Memory Scale, Selective Reminding test, PASAT, Symbol Digit Modality Test, Stroop and Trail Making Test	Symptom Checklist included symptoms of tiredness, difficulty concentrating, memory disturbances, dizziness, irritability, anxiety, light sensitivity, blurred vision, headaches, speech problems and difficulty sleeping	At 24 hours 47 of the 62 players (75%) had abnormal test results. At one month post-injury 29 of 47 players (61%) still showed deficits on the Stroop Test, the PASAT and Symbol Digit Modality Test. At three months the same tests showed continued abnormalities in 16 of the remaining 29 players (55%). These 16 patients also reported symptoms such as headache, dizziness and poor concentration. MRI scans were abnormal in two players	-No control group
Macciocchi et al. (1996)	Cross-sectional & Prospective: Pre-season and at intervals of 24 hours, 5 days, 10 days and 12 weeks postconcussion (A&C)	183 athletes from cohort of 2300 College athletes who sustained a single MTBI and 48 student athlete controls	PASAT, Digit Symbol Substitution Test (DSST), Trail Making Test	Symptom checklist (symptoms not specified)	The results showed that players displayed impairment in both sustained auditory attention (PASAT) and visuomotor speed (DSST, Trail Making Test), but that the impairment was largely as a result of the players failing to show an improvement over time relative to controls as opposed to impairment relative to baseline. Significant increases in symptoms, especially headache, dizziness, and memory problems, relative to baseline were reported at five days post-injury with no difference from baseline at 10 days post-injury. Generally the impairment relative to controls resolved within 5 days in most athletes, with significant improvements made between 24 hours and five days and between 5 and 10 days on both the neuropsychological tests and the symptom checklist	-No control for IQ
Guskiewicz et al. (1997)	Prospective: evaluated day 1, 3, 5 and 10 postconcussion (A)	11 Division I collegiate athletes and 11 matched controls	Stroop Test, Trail Making Test, Digit Span and Hopkins Verbal Learning Test	Not measured	The MTBI athletes did not display any significantly poorer performances on any of the tests and at any interval when compared with the control group. All groups progressively improved from day 1 to day 5 illustrating a practice effect.	-Small sample size (football athletes: n=11 and controls: n=11) -No control for age, IQ or education

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size = n ≤ 30

**Table 6.2.4 cont. <sup>1</sup>**  
**Outcome following MTBI in American football**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Collins, Grindel et al. (1999)	Cross-sectional Prospective: Pre-injury and at intervals of 24 hours, 3 days, 5 days & 7 days postconcussion (A&C)	393 male university athletes evaluated at pre-season. Sixteen athletes evaluated post-injury using ten matched controls from within the sample	Hopkins Verbal Learning Test, the Trail Making Test, Digit Span Test, Symbol Modalities Test, Grooved Pegboard, the Controlled Oral Word Association Test (COWAT), as well as a Concussion Symptom Scale	Concussion Symptom Scale include symptoms of headache, nausea, vomiting, balance problems, dizziness, fatigue, trouble falling asleep, sleeping more than usual, sleeping less than usual, drowsiness, sensitivity to light, sensitivity to noise, irritability, sadness, nervousness, feeling more emotional, numbness or tingling, feeling slowed down, feeling mentally 'foggy', difficulty concentrating, difficulty remembering.	The results demonstrated that history of concussion is significantly and independently associated with long term deficits in tests which measure executive functioning (Trails B) and speed of processing (Symbol Digit Modalities Test) as well as an increase in self-reported symptoms. Moreover, players who had a history of two or more concussions and a learning disability also performed significantly worse on tests of executive functioning (Trails B) and speed of information processing (SDMT). Consistent with acute studies reported above, the recent concussed athletes showed a decline in performance relative to controls on the immediate and delayed memory task (HVLT) at 24 hours, with moderate differences remaining between the groups until 5 days post-injury. Self-reporting symptoms also appeared to resolve before cognitive deficits	-Small sample size in each comparative group (football athletes: n=16 and controls n=10) -No control for age or IQ
Guskiewicz et al. (2000)	Cross-sectional Athletes asked to report on symptoms for recent post-concussion (A)	Study reported on a cohort of 17,549 collegiate and high school football players, with 888 sustaining concussions	Not measured	Symptom checklist include symptoms of LOC, confusion, disorientation, memory loss, neck pain, headache, vomiting/nausea, photophobia, dizziness, fatigue, sleepiness, irritability, disequilibrium & blurred vision	Of the 1003 total injuries sustained 86% of the players complained of headache, 67% dizziness, 59% confusion, 49% disorientation, and 37.3% blurred vision. Of those athletes reporting headache, 28% reported that it resolved in 24 hours, whilst 10% reported that it lasted for more than 24 hours. None of the other symptoms lasted more than 24 hours.	-No control group -No control for age, education, IQ or level of play
Kutner et al. (2000)	Cross-sectional (C)	53 active professional American football players	MicroCog computerized neuropsychological test battery.	Not measured	The results showed that the older players possessing the APOE e4 exhibited significantly lower cognitive test scores than all other players studied, with measures of general cognitive functioning, information processing speed and accuracy, and attention related to poorer performances amongst this group.	-No control group -No control for age (range= 18 to 34 years) or IQ

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size =  $n \leq 30$

**Table 6.2.4 cont. <sup>1</sup>**  
**Outcome following MTBI in American football**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Macciocchi et al. (2001)	Prospective: Pre-season and at intervals of 24 hours, five days and ten days post-concussion (A & C)	24 college football players	PASAT Symbol digit Modalities Test, Trail Making Test	Symptom checklist (symptoms not specified)	No significant cognitive impairment in athletes with two concussions compared with those who sustained a single concussive injury. Players who sustained two concussions, however, reported more symptoms after their first and second concussions than those who sustained a single concussion, although the increase in symptoms was not significant after the first concussion. Headache was the most commonly reported symptom in both groups	-Small sample size (N=24) -No control group
Iverson et al. (2002)	Prospective: Pre-injury and within 5 days postconcussion (average = 1.7 days) (A&C)	Total sample= 38 athletes: 19 athletes with a history of 3+ concussions (11 high school and 8 college students) and 19 with no history of concussion	Immediate Postconcussion Assessment and Cognitive Testing (ImPACT), computerized assessment battery	Postconcussion Symptom Scale included in the ImPACT programme (symptoms include the same as those in Collins, Grindel et al's 1999 study, as well as the symptom 'visual problems')	The findings revealed that the group with a history of 3+ concussions reported significantly more symptoms at pre-season than the other group with no history of concussion. There was a trend towards lower memory scores at pre-season for the multiple concussion group as well as significantly lower memory scores during the post-season assessment	-Small sample size in each comparative group (football athletes: n=19, controls: n=19) -Weak control group (controls =football players with no concussion history) -No control for level of play
McCrea et al. (2002)	Prospective: re-season, & at intervals of 15 min, 48 hrs & 90 days postconcussion (A)	Total of 2385 athletes evaluated and 91 players who sustained concussion	Standard Assessment of Concussion (SAC), brief neurocognitive screening battery	Not measured	Changes were observed on the SAC immediately after sustaining a concussion, but no significant impairment was noted at 48 hours or at 90 days post-injury.	-No control group -No control for age, education, IQ or level of play
Barr (2003)	Cross-sectional & Prospective: pre-injury and at 60 days post-concussion (A&C)	60 male and 40 female high school athletes (47 football, 27 field hockey, 26 soccer)	WAIS-III- Digit Span, Digit Symbol and Symbol Search, Trail Making Test, COWAT, HVLTD-delay + recognition	Not measured	The baseline analysis revealed no differences on test scores between athletes participating in the different sports groups and no differences between athletes with or without prior concussions and with or without learning disabilities. The female athletes did however outperform the males on the DSST, Trail Making Test-Part B and the COWAT, but not on measures of verbal learning. At the re-test interval of 60 days significant practice effects were seen on the WAIS III Processing Speed Index, Trail Making Test and the COWAT, with no differences between the test scores of the males and females.	-No control for education , IQ or gender

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size =  $n \leq 30$

**Table 6.2.4 cont. <sup>1</sup>**  
**Outcome following MTBI in American football**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Collins, Field, et al. (2003)	Prospective Testing 5 & 10 days post-injury  (A)	109 high school athletes consisting of football players (n = 70), basketball (n = 14), soccer (n = 13), hockey (n = 4), lacrosse (n = 3), softball (n = 2), track (n = 1), volleyball (n = 1) and wrestling (n = 1)	ImPACT computerized test battery	ImPACT Symptom Scale (as reported above)	Athletes who suffered from posttraumatic headache performed significantly worse on the reaction time and memory composite scores of the ImPACT programme, and reported significantly more symptoms other than headache. The findings suggest that postconcussion headache in high school athletes at 7 days post-injury could be associated with incomplete recovery. Exact time frame of symptom onset and resolution not known, nor if headache resolution was correlated with resolution of neurocognitive data	-No control group -No control for IQ or gender
Field et al. (2003)	Prospective: Pre-season and then at 24 hours, 3 days, 5 and 7 days postconcussion  (A)	371 college American football athletes and 161 high school American football and 22 soccer athletes. Fifty-four athletes sustained concussions within total sample group and were compared to 38 non-injured controls	Hopkins Verbal Learning Test (HVLt), Digit Span Test, Symbol Digit Modalities Test, Trail Making Test, the COWAT and a Concussion Symptom Scale. High school athletes administered BVMt-R in addition to the above tests.	Postconcussion Symptom Scale (symptoms the same as those in Collins, Grindel et al's 1999 study reported on above)	High school athletes took longer to recover in terms of prolonged memory dysfunction on memory tests (HVLt and BVMt-R) than college athletes. Significant increases in self-reporting of postconcussive symptoms occurred amongst the high school group relative to controls at 24 hours, 3 and 5 days post-injury. At the college level significant differences relative to controls were reported at 24 hours & 3 days post-injury only.	
Guskiewicz et al. (2003)	Prospective: Pre-season and concussed athletes followed up at intervals of 3 hours, 1, 2, 3, 5 and 7 + days post-injury  (A)	2905 collegiate football athletes evaluated over three seasons (1991-2001).	Not measured	Grade Symptom Checklist and health questionnaire (symptoms included not specified)	Overall 184 players sustained concussions and 12 of these players sustained repeat concussions within the same season. Players reporting a history of 3+ previous concussions were 3 times more likely to sustain another concussion than players with no concussion history. Symptoms reported included headache, dizziness or balance problems, feeling 'slowed down', fatigue, memory and dizziness. With headache being the most commonly reported. The mean symptom recovery was 82 hours with players with a history of 3+concussions recovering at a slower rate. Of the 12 repeat concussions, 11 occurred within 10 days of the initial injury and 9 occurred within 7 days of the initial injury. Authors suggested that players with a prior history of concussion are vulnerable to further injury & previous concussion is associated with slower recovery.	-No control group -No control for age, education or IQ

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size = n ≤ 30

**Table 6.2.4 cont. <sup>1</sup>**  
**Outcome following MTBI in American football**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Lovell et al. (2003)	Prospective: Pre-season and at 36 hours, four and seven days postconcussion  (A&C)	64 high school athletes, 60 males and 4 females (40 football players, eight basketball players, seven soccer athletes and five athletes from other sports groups) and 24 control athletes, 16 males and 8 females (22 swimmers and two football players)	ImPACT computerized test battery	ImPACT Symptom Scale included in the ImPACT programme (symptoms included as in Iverson et al's 2002, study)	High school athletes who sustained concussions showed significant declines in memory performance in comparison to the control group, with significant differences reported between pre and post-injury, and at four and seven days. There was also a significant difference in the number of symptoms reported by the concussion group at pre-season and at 36 hours post-injury, with more symptoms being reported at 36 hours, although no significant differences in symptom reporting at four and seven days. Overall, symptoms resolved before test performances on memory tests.	-Small sample size for control group (n=24) -No control for age, IQ or gender
McCrea et al. (2003)	Prospective: Pre-season and at 3 hours, 1,2,3,5,7, and 90 days postconcussion  (A&C)	1631 college athletes underwent pre-season testing. 94 concussed players were followed up with 56 non-injured controls	Standardized Assessment of concussion, Trail Making Test-Part B, Stroop Color Word Test, COWAT, Symbol Digit Modalities Test, and Hopkins Verbal Learning Test.	Graded Symptom Checklist	Concussed athletes exhibited more severe symptoms and cognitive impairment immediately after concussion. Generally symptoms resolved by day 7 post-injury and cognitive functioning improved to baseline levels within 5 to 7 days post-injury. No significant differences between the concussed and controls groups were evident 90 days post-injury.	-No control for IQ
Iverson et al. (2004a)	Prospective: pre-season, and within 5 days postconcussion  (A&C)	Athletes with no prior concussions n = 19 (11 high school and 8 college), 15 football, 3 soccer, 1 baseball and athletes with 3+ concussions n = 19 (12 high school and 7 college), 14 football, 2 soccer, 2 basketball and 1 lacrosse	ImPACT computerized test battery	ImPACT Symptom Scale (as reported above)	At baseline athletes with 3+ concussions reported more symptoms and demonstrated a trend towards lower memory scores than those with no history of concussion. At 2 days post-injury athletes with multiple concussions scored lower on memory tasks than those athletes with a history of one concussion, with athletes with a history of multiple concussions 7.7 times more likely to have a decrease in memory than athletes with no history of concussion	-Small sample size in each comparative group (no concussions: n=19; 3+ concussions: n=19) -No control for level of play
Iverson et al. (2004b)	Prospective: Testing 5 & 10 days post-injury  (A)	Same cohort as in Collins, Field, et al's (2003) study, i.e. 110 high school athletes	ImPACT computerized test battery	ImPACT Symptom Scale (as reported above)	Athletes who suffered from self-reported foginess performed significantly worse on the reaction time, memory and processing speed composite scores of the ImPACT programme, and reported significantly more symptoms other than foginess. Exact time frame of symptom onset and resolution not known.	-No control group -No control for IQ or gender

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size =  $n \leq 30$

**Table 6.2.4 cont. <sup>1</sup>**  
**Outcome following MTBI in American football**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations
Lovell, Collins, Iverson, et al. (2004)	Prospective: pre-season and then between 36 to 72 hours, and between 5 to 10 days, postconcussion. (A)	43 high school concussed athletes. 24 football players, 8 soccer, 5 basketball, 2 ice hockey, 2 lacrosse, one baseball and one softball. Thirty-five of the athletes were male and eight were female	ImPACT computerized test battery	ImPACT Symptom Scale (as reported above)	Deficits in memory and an increase in symptom reporting amongst the high school athletes occurred within the first few days of recovery with all athletes returning at or above their pre-season evaluation level at six days. Subtle but non-significant decreases on reaction time and processing speed tests occurred at 36 hours post-injury with a significant improvement on both these domains occurring at 6 days post-injury.	-No control group -No control for IQ or gender
Pellman et al (2004)	Prospective: pre-injury and then at an average of 24 to 48 hours postconcussion. Athletes with a history of 3+ concussions also evaluated (A & C)	650 NFL athletes tested at pre-season and of those 143 tested post-injury	Hopkins Verbal Learning Test (HVLT), Brief Visuospatial Test-Revised (BVMT-R), Controlled Word Association Test (COWAT), Digit Span and Symbol Digit Modalities Test	Not measured	The injured athletes did not perform significantly more poorly on any of the tests. They did however, perform significantly better post-injury on tests of attention and processing speed (i.e. Trails A, Digit Span Forward and Symbol Digit Modality test). A sub-sample of players with on-field memory impairments demonstrated memory impairments on neuropsychological testing one to two days post-injury but went on to make full recoveries. Players with a history of 3+ concussions did not demonstrate significantly different neuro-psychological test scores	-No control group -No control for age (range=21-35yrs), education or IQ
Guskiewicz et al. (2005)	Cross-sectional (C)	2552 retired professional football players	Not measured	General health questionnaire and a questionnaire focussing on memory issues	Retired players with a history of three or more concussions had a threefold prevalence of reported significant memory problems compared with retired players with no concussion history.	-No control group -No control for age, IQ or education
Mihalik et al. (2005)	Prospective: Pre-season and (3 to 5 days postconcussion) (A)	261 high school and collegiate athletes who sustained concussions were divided into three groups: those with posttraumatic migraine, post-injury headache and no headache post-injury	ImPACT computerized test battery	ImPACT Symptom Scale (as reported above)	The migraine group had significantly lower verbal memory, visual memory and processing speed scores than both the groups, with or without postconcussion headaches, although those athletes who reported headache post-injury had a significantly lower reaction time than those athletes who reported no headache post-injury. Overall symptom reporting was greater in the migraine group than both the headache and non-headache groups, and greater in the headache group in comparison with the non-headache group. The migraine group also demonstrated the greatest departure from baseline scores.	-No control group -No control for gender, education, IQ or level of play

*Note.<sup>1</sup>* For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.<sup>2</sup>* Small sample size =  $n \leq 30$

**Table 6.2.4 cont. <sup>1</sup>**  
**Outcome following MTBI in American football**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Moser et al. (2005)	Cross-sectional within sport (C)	223 high school athletes: Athletes were divided into different concussion history groups: athletes with no concussion history, (n = 82), athletes with one concussion and no symptoms (n = 56), athletes with 2+ concussions not in the past 6 months with no symptoms (n = 45) and those who experienced a concussion a week before testing (n = 40).	Repeatable Battery for the assessment of Neuropsychological Status and the Trail Making Test	Symptom Checklist: headache, nausea, dizziness, 'blacking out, loss of consciousness, having your 'bell rung' or 'seeing stars', visual changes, slower thinking or reactions, feeling 'different' than before experiencing the hit.	The results showed that athletes who sustained two or more concussions were not distinguishable from those athletes who experienced a concussion within the past week suggesting that those athletes with a history of two or more concussions were experiencing prolonged neuropsychological effects.	-No control for age (range =13 to 19yrs), education or IQ
Iverson et al. (2006)	Cross-sectional Within sport (C)	867 male high school and university athletes (football 86.7%, ice hockey 9.6%, soccer 2.3%, and other sports 1.4%). Athletes divided into 3 groups based on concussion history (i.e. no concussion (n=664), 1 prior concussion (n=149) and 2 prior concussions (n=54)	ImPACT computerized test battery	ImPACT Symptom Scale (as reported above)	No differences were observed between those individuals with zero, one and two prior concussions in terms of cognitive functioning and symptom reporting at pre-season	-No control for age (range=13 to 22yrs), education, IQ or level of play
McClincy et al. (2006)	Prospective: pre-season and at 2,7, and at 14 days post-concussion. (A&C)	104 concussed American football players	ImPACT computerized test battery	ImPACT Symptom Scale (as reported above)	Differences were observed between pre-season scores and all ImPACT composite scores and in the total symptom scores, at two and seven days post-injury. At 14 days post-injury only the verbal scores differed significantly from pre-season scores. The results therefore indicate that deficits may persist for longer than two weeks in athletes who have sustained concussions.	-No control group
Pellman et al. (2006)	Prospective: pre-season and then 1-2 days post-concussion (A)	68 NFL and 125 high school athletes	ImPACT computerized test battery	ImPACT Symptom Scale (as reported above)	The results showed that the older NFL players demonstrated a rapid recovery, returning to pre-season levels within a week of sustaining a concussion, whereas the younger high school athletes demonstrated a slower recovery when compared with the older athletes.	-No control group -No control for age (high school range = 13 to 18 & professional range = 20 to 34 yrs), education or IQ

*Note.*<sup>1</sup> For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.*<sup>2</sup> Small sample size =  $n \leq 30$

Table 6.2.5

Summary of studies investigating the cognitive and postconcussive symptom outcome following MTBI in Australian Football League ('C' refers to studies investigating chronic effects, 'A' refers to studies investigating acute effects<sup>1</sup>)

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Dicker & Maddocks (1988)	Cross-sectional & prospective: Pre-season & 24 hours post-Concussion (A)	13 AFL athletes	Digit Symbol Substitution Test	Not measured	Athletes completed significantly fewer items on the Digit Symbol Substitution Test 24 hours post-injury although they returned to pre-season performance at five days post-injury	-Small sample size (N=13) -No control group
Cremona-McEyard & Geffen (1994)	Prospective: Athletes evaluated 2 weeks post-injury & 1yr post-injury  (A&C)	Nine concussed AFL athletes (Exp. 1) and 8 concussed AFL (Exp. 2) and 12 non-injured controls	Cued Reaction time task	Not measured	In Experiment 1, nine AFL (7 of which had sustained prior concussions) were tested within 2 weeks of sustaining a MTBI and then again one year later. Their performance on the reaction time task was the same as controls with regards to their response to targets in the unexpected visual field. However, their response to targets in expected visual fields, following valid cues, was slightly slower compared with controls. At one-year post-injury the athletes overall reaction time performance had improved but their pattern of response was the same as in their prior testing. Experiment 2 consisted of 8 AFL athletes, with an average age of 23.5 years (6 of the 8 athletes had sustained multiple MTBIs). The findings at 1 year post-injury replicated the findings in the AFL players in Experiment 1. It was suggested that a persistent finding amongst athletes who have sustained MTBIs may be the inability to take fast action in response to expected events.	-Small sample size in each comparative group (Exp 1. concussed athletes: n=9 and control: n=12 ; Exp 2. concussed athletes: n=8 and control: n =12)
Maddocks, Saling, et al. (1995)	Cross-sectional within sport (C)	198 professional AFL athletes. 79 sustained no concussions, 50 sustained one concussions, 69 sustained 2+ concussions	Digit Symbol Substitution Test	Not measured	No significant difference between the groups on DSST	
Maddocks & Saling (1996)	Prospective Pre-season and 5 days post-injury (A)	10 AFL athletes 10 AFL referee controls	Digit Symbol Substitution Test (DSST), Four-Choice Reaction Time test and the PASAT	Not measured	Significant differences on the Digit Symbol Substitution Test and the reaction time task was reported between the concussed players and their controls at five days post-injury. Whilst concussed players' performances were comparable to their pre-season scores at five days post-injury, the controls demonstrated significant improvements from their pre-season performances.	-Small sample size in each comparative group (AFL athletes: n=10; controls: n=10)

*Note.*<sup>1</sup> For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.*<sup>2</sup> Small sample size =  $n \leq 30$

**Table 6.2.5 cont. <sup>1</sup>**  
**Outcome following MTBI in Australian Football League**

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
McCrory et al. (2000)	Prospective: Athletes evaluated over a season, within 24 hours & 2 weeks (A)	303 elite AFL athletes	Digit Symbol Substitution Test	Assessment form consisting of self-reported symptoms	Most common symptom reported was headache. Ten players returned to play on the day of injury and the remaining thirteen players returned to play two weeks later. Players with a history of 3+ concussions or where symptoms lasted for more than 15 minutes were less likely to return to play on the day of injury. These findings were consistent with poor performances on the DSST	-No control group -No control for age, education or IQ
Makdissi et al. (2001)	Prospective: Players observed over nine weeks. Concussed athletes tested at 2 and 14 days post-injury (A)	Total of 240 athletes evaluated at pre-season. Six AFL athletes who sustained concussions compared with 7 age matched non-injured controls	Digit Symbol Substitution Test, Trail Making Test and computerized neurocognitive battery, CogSport	Not measured	Concussed athletes displayed significant deterioration on the computerized neurocognitive battery, CogSport. On the conventional measures (Digit Symbol Substitution test and the Trail Making test) however, both control and concussed groups showed improved performances.	-Small sample size (N=13) -No control for age (range=17-26yrs), education, IQ or level of play
Collie et al. (2006)	Prospective: pre-season and within 11 days post-injury (A)	615 male AFL evaluated pre-season & 61 concussed athletes (25 = symptomatic and 36 = asymptomatic) evaluated within 11 days post-injury as well as 84 controls	CogSport computerized neurocognitive test battery and Trail Making Test and Digit Substitution Test	Not measured	In comparison to the asymptomatic and control group, the symptomatic group's performance on the CogSport tests of simple, choice and reaction time declined at the post-injury interval relative to their pre-injury performance, demonstrating deficits in motor function and attention although both memory and learning ability was preserved. The symptomatic group's performance on the paper-and-pencil tasks demonstrated no changes from their pre-injury performances whereas both the asymptomatic and control groups demonstrated large improvements from their pre-injury testing, which suggests significant practice effects for the asymptomatic and control groups but not for the symptomatic players.	

*Note.*<sup>1</sup> For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.*<sup>2</sup> Small sample size =  $n \leq 30$

Table 6.2.6

Summary of studies investigating the cognitive and postconcussive symptom outcome following MTBI in Rugby League ('C' refers to studies investigating chronic effects, 'A' refers to studies investigating acute effects<sup>1</sup>)

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Hinton-Bayre, Geffen, & McFarland (1997)	Prospective: Study 1: Two pre-season testings one to 2 weeks apart  Study 2: Concussed athletes evaluated at 24 to 48 hours post-concussion (A)	54 professional Rugby League players. Ten concussed players were evaluated separately	Digit Symbol Substitution Test, Symbol Digit Modalities Test and the Speed of Comprehension Test	Not measured	Performance on the Digit Symbol Substitution Test and the Speed of Comprehension Test improved with practice whereas performance on the Symbol Digit Modalities Test remained stable. The cognitive functioning of the 10 players who were concussed, and the findings showed measurable changes in tests for Speed of Comprehension and Symbol Digit perception. Speed of Comprehension appeared to be more sensitive to post-injury impairment than both of Symbol Digit tests.	-No control group -No control for age (range=17-26yrs), education or IQ
Hinton-Bayre et al. (1999)	Prospective:  Pre-season 1-3 days, 1-2 weeks and 3-5 weeks post-concussion (A)	20 Rugby League players who sustained concussions	Digit Symbol Substitution Test, Symbol Digit Modalities Test and the Speed of Comprehension Test	Symptom checklist included symptoms of headache, amnesia, unsteadiness, visual disturbances, dizziness, nausea and feeling dazed	16 players reported impaired performances on at least one of the three measures (Digit Symbol Substitution Test, Symbol Digit Modalities Test and Speed of Comprehension) at one to three days post-injury. At one to two weeks post-injury, seven players were impaired on at least one of the three measures, returning to pre-season levels only three to five weeks later. Symptoms of headache, amnesia, unsteadiness, visual disturbances, dizziness, nausea and feeling dazed were experienced immediately after injury in only five of the athletes, with few complaining of ongoing symptoms	-Small sample size (N=20) -No control group
Hinton-Bayre & Geffen (2004)	Cross-sectional (C)	119 Rugby League players	Digit Symbol Substitution Test, Digit Symbol Modalities test and Speed of Comprehension	Not measured	No significant differences seen between groups of players with zero concussion history and one or two or more concussions	-No control for age, education or IQ

*Note.*<sup>1</sup> For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

*Note.*<sup>2</sup> Small sample size =  $n \leq 30$

Table 6.2.7

Summary of studies investigating the cognitive and postconcussive symptom outcome following MTBI in Rugby Union ('C' refers to studies investigating chronic effects, 'A' refers to studies investigating acute effects<sup>1</sup>)

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Shuttleworth-Jordan et al. (1993)	Cross-sectional and Prospective i) Pre- and post-season testing  ii) Repeat testing of concussed athletes at 3 days, one month, 2 and 3 months post-injury (A&C)	60 Rugby Union players and 25 non-contact controls (squash, hockey and tennis)	Digit Supraspan, Trail Making Test, Digit Span Backwards, Denckla finger tapping and Purdue Pegboard tests	Postconcussion symptom Questionnaire included the following questions: Do you suffer from headaches, poor eyesight, difficulty hearing, experience weakness in your limbs, are you clumsy, do you have fits or seizures, do you become dizzy, do you tire easily, are you sensitive to noise, have you every felt that you were seeing, hearing or feeling unusual things, are you experiencing sexual problems, do you have problems with your speech, stumble over your words when you speak, do you stutter or stammer, do you slur your words, do you have memory difficulties, do you have problems with memory and concentration, does your attention wander, are you impatient, are you irritable, do you become easily angry or hurt, do you feel sad, or depressed, do you enjoy seeing your friends and having social contact, do you suffer from restlessness, do you have problems sleeping, problems with your appetite, do you feel nervous or anxious, worried or on edge, are you argumentative, short-tempered, do you become aggressive for no reason?	The results at pre-season revealed that the controls performed significantly better on tests of working memory, verbal new learning ability and hand-motor dexterity (Trail Making Test, Digit Backwards and the Purdue Pegboard) in comparison to the Rugby Union players. Results of the pre- and post-season comparison revealed a significant practice effect for the control group on the Trail Making Test, Digit Supraspan, Finger Tapping task and Purdue Pegboard. The rugby group showed less capacity for practice effects between pre and post-season testing, with the only improvement noted on the Finger Tapping Task. The repeated test measures, up to three months post-injury revealed deficits in attention, verbal new learning, working memory and hand motor dexterity at three days post injury, with substantial recovery up to the two month interval. Acute symptoms of headache, fatigue, attention and concentration and blurred vision were reported at one-month post-injury, with no symptoms reported at three months post-injury.	-Small sample size for control group (n=25) -Weak control for age (range 18-25yrs) and education
Pettersen & Skelton (2000)	Cross-sectional individuals assessed before treatment and at intervals of 1, 2 and 3 days with and without glucose beverage (A&C)	12 previously concussed athletes and 12 controls	Wechsler Logical Memory Scale, Wechsler Paired Associates, Auditory consonant trigrams, Symbol Digits Modalities Test (SDMT), SDMT Incidental Recall, Listening Span Test and the Automated Neuropsychological Assessments Metrics (ANAM).	Not measured	Previously concussed individuals performed slightly worse than controls in the saccharin (placebo) condition on tests of memory and divided attention (SDMT Incidental Recall, Wechsler Logical Memory Scale, Auditory consonant trigrams and Listening Span Test), but performed better than controls when given the glucose beverage on tests of declarative memory (LM).	-Small sample size in each comparative group (concussed: n=12 ;controls: n=12) -No control for age (18-24yrs) and education

Note.<sup>1</sup> For the purpose of this study the term 'acute' denotes studies investigating the short-term effects following reportable concussion and the term 'chronic' refers to studies where it is assumed that there are residual or permanent effects

Note.<sup>2</sup> Small sample size =  $n \leq 30$

Table 6.2.7 cont. <sup>1</sup>  
Outcome following MTBI in Rugby Union

Authors	Type of study	Sample	Cognitive Measures	Postconcussive Symptom Measures	Findings	Limitations <sup>2</sup>
Farace et al. (2003)	Cross-sectional Within sport (C)	54 female collegiate rugby players	Standardized Assessment of Concussion (SAC), the Trail Making Test, and the Paced Auditory Serial attention Test (PASAT).	Not measured	The results demonstrated that no differences were found on either the PASAT or the SAC on athletes with or without a history of concussion. Athletes with a history of concussion made significantly more errors on the Trail Making Test than those athletes with no history of concussion.	-No control group -No control for age (range =17-22 yrs) and education
Shuttleworth-Edwards et al. (2004)	Cross-sectional across different levels of play (C)	(i) 26 professional Rugby Union players (mean age of 27.5 years) who were compared to a group of 21 national open non-contact cricket players (mean age of 27.1 years). (ii) 19 national under-21 Rugby Union players were (mean age of 19.7 years) compared to a group of 21 national open field hockey players (mean age of 23.3 years). (ii) 47 school Rugby Union players (mean age of 17.3 years) compared to a group of 34 school field hockey players (mean age of 17.0 years).	<i>General intellectual functioning:</i> SAWAIS –Picture Completion and Comprehension subtests (national open rugby, national open cricket, national under-21 rugby, national open field hockey); the SAWAIS-Picture Completion and Vocabulary subtests, National Adult Reading Test (NART), (School rugby and school field hockey). <i>Attention and concentration:</i> SAWAIS- Digit Span Test, (all groups). WAIS- III Letter-number Sequencing subtest (LNS) and Stroop, (School rugby and field hockey). <i>Visuoperceptual scanning speed:</i> SAWAIS Digit-Symbol Substitution Test, Trail Making Test, (all groups). <i>Memory:</i> WMS-Associate Learning subtests, Digit-Symbol Incidental recall-Immediate and Delayed, WMS-Visual Reproduction subtest, (all groups). <i>Verbal fluency:</i> Words-in-One-Minute Unstructured Verbal Fluency Test, Structured Verbal Fluency Test – ‘S’ Words, (all groups). <i>Fine-motor dexterity:</i> Sequential Finger Tapping Test, (all groups).	Postconcussion Symptom Questionnaire (reported above as in Shuttleworth-Jordan et al. .1993)	For both the national open team and the national under-21 team, deficits were reported in the cognitive domains of speed of information processing, memory, verbal fluency and hand motor dexterity, with only one result in the opposite direction where 10% of field hockey players exhibited deficit on one trial of the sequential Finger Tapping Test. In contrast to the two adult teams, only one test reported deficit at the school level, which was on one trial of the hand motor function. In spite of this the researchers reported that there were a number of subtle indicators of cognitive dysfunction in the memory domain. Both adult teams reported substantial postconcussive symptoms in each of the emotional, behavioural, somatic and cognitive symptom clusters when compared to controls. The researchers noted that there was a marked reversal of findings on a few symptoms of headaches, eyesight, attention and concentration, amongst the open rugby groups which could be attributed to the known underreporting of symptoms that takes place amongst top athletes. Amongst the schoolboy rugby sample higher percentages were reported in comparison to controls, although they weren't as prevalent as those reported by the adult groups. Symptoms reported included clumsy speech, sleep difficulties, clumsiness, easily angered and hearing problems	-Small sample size for older teams (professional athletes: n=26 and controls: n=21; university ; n=19 and controls: n=21) -Weak control for professional players (some cricket athletes from the control group were former rugby players) -Weak control for age, education and IQ in the university athletes

Note.<sup>1</sup> For the purpose of this study the term ‘acute’ denotes studies investigating the short-term effects following reportable concussion and the term ‘chronic’ refers to studies where it is assumed that there are residual or permanent effects

Note.<sup>2</sup> Small sample size = n ≤ 30



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**VOLUME TWO  
RESEARCH STUDY**

**BY**

**VICTORIA JANE WHITEFIELD**

**Thesis presented for the Degree of  
DOCTOR OF SCIENCE  
in the Department of Science  
RHODES UNIVERSITY**

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## CHAPTER SEVEN

### RESEARCH DESIGN AND METHODOLOGY

This chapter reports on the research design and methodological procedures of the present study. It begins by reviewing the participants involved in respect of the subject selection criteria and comparison groups selected for the purpose of the analysis. This is followed by a description of the demographic data including age, education level, IQ and concussion history for each comparative group. The research design, testing procedures, and materials used for the study in respect of demographic questionnaires, neurocognitive measures and postconcussion inventories will be reviewed next. Finally, the processing and analysis of data will be addressed which is followed by the statistical hypotheses for the study.

#### 7.1 PARTICIPANTS

##### 7.1.1 Selection Criteria

All final year, sports playing Grade 12 high school boys, from an English medium school in the Western Cape Province, in South Africa were targeted for participation in the study. The study took place over two years and involved Grade 12 learners from both the years of 2003 and 2004. The total number of learners participating in the study in each year was collated to create a combined sample of 297 boys (2003, N = 138 and 2004, N = 159), ranging from 17 to 19 years of age. Forty-one boys were eliminated from the study due to the fact that none of them were actively involved in any sport during the time of the research, leaving a pool of 256 athletes.

Following this, a further 67 athletes were excluded on the basis of having any of the nine exclusion criteria. As identified on the basis of the ImPACT computer based and paper-and-pencil sport and health history questionnaires, these comprised a reported history of substance abuse, any previous diagnosis of a learning disorder which included a diagnosis of Attention Deficit Disorder, any grades repeated, any remedial instruction (specialized teaching for children with learning difficulties), and any neurological disorder (which included meningitis or epilepsy). In addition, it was considered that a WAIS-III Vocabulary subtest scaled score of less than nine points might indicate a low average to borderline score. Three individuals in the total sample (Contact, n = 2 and Non-Contact, n = 1)

obtained a Vocabulary scaled score of less than nine points and were thus excluded from the sample group. Once these exclusions had been made, a total of 189 athletes remained out of the original 297. Any discrepancy in sport and health history information reported on the ImPACT and paper-and-pencil questionnaires were followed up by contacting the athletes directly. In the event that an athlete failed to complete the information required on the paper-and-pencil questionnaire the relevant information was taken from the ImPACT sport and health history questionnaire.

**Table 7.1.1.1**  
**Exclusions amongst the sports groups**

Exclusion Categories <sup>1</sup>	Contact (n = 38)	Non-Contact (n = 29)
Remedial	16	10
Grades Repeated	11	11
ADD	9	2
LD	2	1
Epilepsy	2	1
Meningitis	3	3
Psychiatric Disorder	1	1
Substance Abuse	3	1
Vocabulary subtest	2	1

*Note.* <sup>1</sup> The numbers that make up the exclusions in the different groups do not match the totals of the categories owing to the fact that some participants had more than one exclusion criteria

Participation in the study was voluntary and as a result a further reduction to the sample took place at the post-season test interval, in that 23 athletes that did not arrive for testing which coincided with preliminary Grade 12 examinations. In addition, contact sports players who reported to have sustained concussions during the season (n = 12) were excluded from the post-season analysis. Factors such as fatigue due to lack of sleep as well as poor motivation were considered to be extraneous variables which could potentially influence the test performance of the athlete. The ImPACT programme, used in this study, controls for this by including relevant questions (e.g. hours of sleep last night) in the sport and health history questionnaire which appears prior to the ImPACT Symptom Scale which reports on the athlete's current symptoms and conditions. No exclusions were deemed necessary on the basis of such extraneous variables.

**Table 7.1.1.2**  
**Exclusions amongst the concussion history groups (all sports)**

Exclusion Categories <sup>1</sup>	2+ Concussion (n = 19)	0 Concussion (n = 31)
Remedial	10	13
Grades Repeated	6	12
Attention Deficit Disorder	6	3
Learning Disability	1	1
Epilepsy	0	2
Meningitis	2	2
Substance Abuse	3	1
Psychiatric Disorder	1	2
Vocabulary subtest	1	2

*Note.* <sup>1</sup> The numbers that make up the exclusions in the different groups do not match the totals of the categories owing to the fact that some participants had more than one exclusion criteria

The final pre-season sample comprised 189 athletes and the final post-season sample comprised 154 athletes. These remaining athletes were divided into the six subgroups (see p. 234) for comparative analysis. The distribution of athletes taking part in the contact sports yielded predominantly rugby players (90%) and the remaining 10% comprised soccer players and a single Jeet Kune Do (a form of Chinese martial arts) participant (pre-season only). The small number of athletes from a contact sport, other than rugby, did not warrant analysis as a separate subgroup. In order not to lose data they were incorporated into a combined contact sports group, as is frequently done in other sports studies (see earlier review in Chapter six). All contact sports athletes included in the study had to be exposed to that sport at least for the duration of their high school careers (i.e. four years). Conversely, for inclusion, non-contact sports participants were not to have participated in a contact sport for at least the duration of their high school careers. Generally in South Africa, and definitely at the target school of the present research, it is mandatory for all high school athletes to make the choice between participation in a contact sport versus a non-contact sport in their first year of high school. However, it is likely that most of the rugby playing contact sports athletes included in the study would have participated for an average of eight years by the time they reached their final high school year, as many would have started at the junior school level.

In respect of the concussion history groups, the researcher instructed the athletes to report on each prior concussion event that was formally diagnosed by a doctor or first aid personnel on both the ImPACT and paper-and-pencil sport and health history questionnaire. For the purpose of this study the definition of concussion was based on the American Academy of Neurology's terminology where concussion is defined as a "traumatically induced alteration in mental status that may or may not be accompanied by a loss of consciousness" (Kelly & Rosenburg, 1997, p.577).

**Table 7.1.1.3**  
**Exclusions amongst the concussion history groups (Contact sports) <sup>2</sup>**

<b>Exclusion Categories<sup>1</sup></b>	<b>Contact 2+ Concussion (n = 16)</b>	<b>Contact 0 Concussion (n = 15)</b>
Remedial	6	8
Grades Repeated	5	3
ADD	5	2
LD	1	0
Epilepsy	0	1
Meningitis	2	0
Substance Abuse	3	1
Psychiatric Disorder	1	1
Vocabulary subtest	1	1

*Note.* <sup>1</sup> The numbers that make up the exclusions in the different groups do not match the totals of the categories owing to the fact that some participants had more than one exclusion criteria.

*Note.* <sup>2</sup> The numbers in these two groups are reduced in that those individuals with only one concussion did not form part of the group.

Given the confusion around the allocation of grades of sports concussion, especially in a retrospective self-report history of sports concussive episodes (see discussion in Chapter two), no differentiation was made in terms of severity of the sports-related concussions reported by those included in the study, and nor was any differentiation made within the concussion groups in terms of the presence or absence of loss of consciousness, confusion, PTA, or retrograde amnesia reported in association with the concussions. Duration of symptoms was not elicited on the questionnaires, but it is assumed that the concussions would mostly if not entirely have been in the milder spectrum of TBI as is generally the case in sports TBI, with most concussions not involving a loss of consciousness (Lovell et al., 1999). In the total sample  $n = 91$  sports-related concussions were reported (rugby,  $n = 75$ ; soccer,  $n = 4$ ; martial arts,  $n = 1$ ; hockey,  $n = 7$ ; basketball,  $n = 3$  and cycling,  $n = 1$ ), and of these only  $n = 31$

involved LOC, of which  $n = 29$  were amongst contact sports players (rugby,  $n = 28$ ; martial arts,  $n = 1$ ) and only  $n = 2$  were amongst non-contact sports players (hockey,  $n = 2$ ). All non-contact sports players with a prior history of concussion were excluded from the study ( $n = 11$ ).

### 7.1.2 Pre-season independent sample comparative groups (N = 189)

(i) *Contact* group  $n = 115$ , versus *Non-Contact* group  $n = 74$

(ii) *2+ Concussion* group  $n = 43$ , versus *0 Concussion* group  $n = 108$

(iii) *Within Contact 2+ Concussion* group  $n = 40$ , versus *within Contact 0 Concussion* group  $n = 45$

(i) At pre-season the *Contact* group ( $n = 115$ ) was made up of individuals participating in rugby ( $n = 105$ ), soccer ( $n = 9$ ) and Jeet Kune Do, ( $n = 1$ ).

The *Non-Contact* group ( $n = 74$ ) was made up of individuals participating in field hockey ( $n = 29$ ), basketball ( $n = 11$ ), waterpolo ( $n = 10$ ), athletics ( $n = 5$ ), tennis ( $n = 3$ ), squash ( $n = 3$ ); swimming ( $n = 3$ ), cycling ( $n = 3$ ), cricket ( $n = 2$ ), rowing ( $n = 2$ ), golf ( $n = 2$ ), gymnastics ( $n = 1$ ).

(ii) The *2+ Concussion* group ( $n = 43$ ) was made up of individuals participating in rugby ( $n = 39$ ), field hockey ( $n = 2$ ), soccer ( $n = 1$ ) and Jeet Kune Do ( $n = 1$ ).

The *0 Concussion* group ( $n = 108$ ) was made up of individuals participating in rugby ( $n = 38$ ), field hockey (21), basketball ( $n = 9$ ), soccer ( $n = 7$ ), athletics ( $n = 5$ ), waterpolo ( $n = 9$ ), tennis ( $n = 3$ ), squash ( $n = 3$ ), swimming ( $n = 3$ ), cycling ( $n = 3$ ), golf ( $n = 2$ ), rowing ( $n = 2$ ), cricket ( $n = 2$ ), gymnastics ( $n = 1$ ).

(iii) The *within Contact 2+ Concussion* group ( $n = 40$ ) was made up of individuals participating in a contact sport and who had sustained two or more concussions. These individuals participated in rugby ( $n = 38$ ), soccer ( $n = 1$ ) and Jeet Kune Do ( $n = 1$ ).

The *within Contact 0 Concussion* group ( $n = 45$ ) was made up of individuals participating in a contact sport and who had sustained zero concussions. These individuals participated in rugby ( $n = 38$ ) and soccer ( $n = 7$ ).

### 7.1.3 Post-season independent sample comparative groups (N = 154)

(i) *Contact* group n = 90, versus *Non-Contact* group n = 64

(ii) *2+ Concussion* group n = 32, versus *0 Concussion* group n = 91

(iii) *Within Contact 2+ Concussion* group n = 29, versus *within Contact 0 Concussion* group n = 38

(i) At post-season the *Contact* group (n = 90) was made up of individuals participating in rugby (n = 81) and soccer (n = 9).

The *Non-Contact* group (n = 64) was made up of individuals participating in field hockey (n = 22), basketball (n = 9), waterpolo (n = 10), athletics (n = 4), tennis (n = 3), squash (n = 3), swimming (n = 3), cycling (n = 3), cricket (n = 2), rowing (n = 2), golf (n = 2), gymnastics (n = 1).

(ii) The *2+ concussion* group (n = 32) was made up of individuals participating in rugby (n = 29), field hockey (n = 2), soccer (n = 1).

The *0 concussion* group (n = 91) was made up of individuals participating in rugby (n = 32), field hockey (21), basketball (n = 7), soccer (n = 6), waterpolo (n = 6), athletics (n = 3), tennis (n = 3), squash (n = 1), swimming (n = 2), cycling (n = 3), golf (n = 2), rowing (n = 2), cricket (n = 2), gymnastics (n = 1).

(iii) The *within Contact 2+ Concussion* group (n = 29) was made up of individuals participating in a contact sport and who had sustained two or more concussions. These individuals participated in rugby (n = 28) and soccer (n = 1).

The *within Contact 0 Concussion* group (n = 38) was made up of individuals participating in a contact sport and who had sustained zero concussions. These individuals participated in rugby (n = 32) and soccer (n = 6).

#### 7.1.4 Pre-season versus post-season dependent sample comparative groups (N = 150)

(i) The *Contact* group (n = 88) in this pre-season versus post-season analysis was made up of individuals participating in rugby (n = 80) and soccer (n = 8).

(ii) The *Non-Contact* group (n = 62) in this pre-season versus post-season analysis was made up of individuals participating in field hockey (n = 21), basketball (n = 9), waterpolo (n = 9), athletics (n = 4), tennis (n = 3), squash (n = 3); swimming (n = 3), cycling (n = 3), cricket (n = 2), rowing (n = 2), golf (n = 2), gymnastics (n = 1).

## 7.2 DEMOGRAPHIC DATA

### 7.2.1 Sports Groups (see Table 7.2.1)

Since variables such as age, educational level, IQ and history of MTBI are known to have an obvious effect on cognitive test performances (Lezak et al., 2004) group mean comparisons were calculated for these variables, in order to establish between-group homogeneity. Independent two-sampled t-tests were used for the Contact versus Non-Contact group comparison. All participants in the study were at the same educational level, which was calculated according to the number of years of education completed (final school year, Grade 12), so this was not included in the comparison.

No significant differences between the means for any of the comparative sports groups was apparent for the variables of age (pre-season,  $p = 0.968$ ; post-season,  $p = 0.995$ ) and the Vocabulary scaled score (pre-season,  $p = 0.063$ , post-season,  $p = 0.096$ ), suggesting that all comparative groups were well matched and therefore equivalent for both age and estimated IQ (see Table 7.2.1). The age range was 16 to 18 years for all groups and the Vocabulary scaled score ranged from 9 to 18.

The group mean comparisons of reported incidences of concussions were analysed amongst the Contact and Non-Contact groups. Independent two-sampled t-tests were used for the Contact versus Non-Contact group comparison. The group mean comparisons of the reported head injuries are presented in Table 7.2.1. Highly significant differences at both the pre- and post-season occasions in

the number of reported concussions was evident between the Contact and Non-Contact sports groups ( $p = 0.000$ ), in the direction of the Contact group sustaining more concussions. Perusal of Table 7.2.1 revealed that on average the Contact group reported in excess of one concussion, with standard deviations around 1.5, implying that a substantial number of individuals had in excess of two concussions in comparison with the Non-Contact group who reported averages closer to zero concussions with standard deviations all less than one, implying that minimal if any individuals had more than one concussion.

**Table 7.2.1**  
**Demographic data of Contact versus Non-Contact groups**

Pre-season	Contact (n = 115) Mean (SD)	Non-Contact (n = 74) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Age	17.08 (0.33)	17.08 (0.46)	-0.040	0.968
Vocabulary scaled score <sup>†</sup>	12.26 (1.90)	12.83 (2.23)	-1.873	0.063
No of concussions	1.28 (1.41)	0.27 (0.81)	6.265	0.000 <sup>†</sup>
Post-season	Contact (n = 90) Mean (SD)	Non-Contact (n = 64) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Age	17.08 (0.34)	17.08 (0.47)	-0.006	0.995
Vocabulary scaled score <sup>†</sup>	12.27 (1.96)	12.80 (2.24)	-1.674	0.096
No of concussions	1.24 (1.43)	0.28 (0.83)	5.670	0.000 <sup>†</sup>

Note. <sup>†</sup>Control for estimated IQ established on the basis of WAIS-III Vocabulary Scaled Scores.

<sup>\*</sup> $p < .01$ , one-tailed.

## 7.2.2 Concussion history groups (see Tables 7.2.2.1 and 7.2.2.2)

Independent two-sampled t-tests were used for the 2+ Concussion group versus 0 Concussion group comparison, and the *within* Contact group comparison between the 2+ Concussion group and the 0 Concussion group.

No significant differences between the means for any of the comparative concussion groups was apparent for the variables of age (pre-season,  $p = 0.290$ ; post-season,  $p = 0.330$ ) and the Vocabulary Scaled Score (pre-season,  $p = 0.901$ ; post-season,  $p = 0.686$ ) suggesting that all comparative groups

were well matched and therefore equivalent for both age and estimated IQ (see Table 7.2.2.1). The age range was 16 to 18 years for both groups and the Vocabulary scaled score ranged from 9 to 18. No significant differences between the means for any of the comparative concussion groups *within* the Contact group was apparent for the variables of age (pre-season,  $p = 0.503$ ; post-season,  $p = 0.946$ ) and the Vocabulary Scaled Score (pre-season,  $p = 0.205$ ; post-season,  $p = 0.170$ ) suggesting that all comparative groups were well matched and therefore equivalent for both age and estimated IQ (see Table 7.2.2.2). The age range was 16 to 18 years for both groups and the Vocabulary scaled score ranged from 9 to 18.

**Table 7.2.2.1**  
Demographic data of 2+ Concussion versus 0 Concussion

Pre-season	2+ Concussion (n = 43) Mean (SD)	0 Concussions (n = 108) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Age	17.05 (0.30)	17.12 (0.43)	1.061	0.290
Vocabulary scaled score <sup>1</sup>	12.52 (2.06)	12.48 (2.06)	0.122	0.901
Post-season	2+ Concussion (n = 32) Mean (SD)	0 Concussions (n = 91) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Age	17.05 (0.32)	17.13 (0.44)	0.977	0.330
Vocabulary scaled score <sup>1</sup>	12.62 (2.12)	12.46 (2.08)	0.404	0.686

Note. <sup>1</sup>Control for estimated IQ established on the basis of WAIS-III Vocabulary Scaled Scores.

**Table 7.2.2.2**  
Demographic data *within* the Contact group between 2+ Concussion versus 0 Concussion

Pre-season	2+ Concussion (n = 40) Mean (SD)	0 Concussions (n = 45) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Age	17.54 (0.51)	17.61 (0.49)	0.673	0.503
Vocabulary scaled score <sup>1</sup>	12.15 (2.07)	11.61 (1.86)	-1.278	0.205
Post-season	No Concussion (n = 29) Mean (SD)	2 + Concussions (n = 38) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Age	17.58 (0.50)	17.59 (0.50)	0.068	0.946
Vocabulary scaled score <sup>1</sup>	12.64 (2.07)	12.02 (1.90)	-1.386	0.170

Note. <sup>1</sup>Control for estimated IQ established on the WAIS-III Vocabulary Scaled Scores.

### 7.3 CONSENT (see Appendix A)

Written consent to conduct the research at the school and within school hours was obtained from the Western Cape Department of Education as well as from the school headmaster. All Grade 12 parents were sent letters informing them of the nature and purpose of the research and all were given the option of withdrawing their child from the study by signing an attached waiver form. No parents withdrew their children from the study. Written consent was received from all participants, prior to testing, who agreed to participate in the research.

### 7.4 RESEARCH DESIGN

A cross-sectional quasi-experimental design was employed in respect of the different sports and concussion history groups at the pre- and post-season intervals (see Chapter Six). For the comparison of the different sports groups (Contact versus Non-Contact) a non-equivalent control group design was used in respect of the different sports groups for both pre- and post-season test measurement. Further, when comparing the different concussion history groups (2+ Concussion versus 0 Concussion) a non-equivalent control group design was used in respect of the distribution of participants across sports types within the two comparison groups, for both pre- and post-season test measurement. In order to improve on the non-equivalence of this comparison in respect of sports types, a further analysis was performed on equivalent groups in respect of sport (equivalent control group design) comparing individuals who sustained 2+ Concussion versus 0 Concussion *within* the Contact group. A prospective quasi-experimental design was employed for the comparison of the different sports groups at pre- versus post-season.

### 7.5 PROCEDURE

In South African schools, the winter sports season begins at the beginning of April each year, and therefore pre-season (baseline) testing took place between February and April before any contact training began. Post-season testing was performed at the end of the season which is at the end of September. The research team consisted of the researcher and a research assistant who had an Honours degree in Psychology. Before testing took place, the research assistant was trained in the

administration and scoring of the tests to be employed in the research. Standardized test instructions were given to all participants during testing.

Participants were tested in groups of approximately 25 boys in the school computer laboratory, without any noise or distractions. Testing took place during school hours and 45 minutes was allocated to testing, which corresponded to one lesson period. Prior to testing, all participants were provided with an explanation and purpose of the assessment, and confidentiality of all test results was emphasized. The computer laboratory was monitored by members of staff in addition to the researcher and the research assistant to control for any possible environmental distractors. Participants appeared motivated during the testing situations. At the end of the ImPACT programme the participant was given the opportunity to report on any factors that could have negatively effected their performances. These factors related to the clarity of the instructions given on the ImPACT programme, technical or computer problems, and environmental problems which included possible distractors such as environmental noise. As indicated above, no participants reported difficulties on any of these dimensions that suggested impaired validity of their test taking.

## **7.6 MATERIALS**

According to researchers, a test battery for MTBI in sports should be based on test sensitivity, must be of brief duration and include tests that have face validity (Kozora & Gerber, 2004). Neuropsychological tests commonly used for the assessment of concussion were selected for neuropsychological assessment. These tests assess the domains of attention, concentration, learning, short-term memory, speed of processing speed and reaction time and have demonstrated their effectiveness in identifying subtle changes associated with concussion in athletes (see Chapter 4).

Materials for the study consisted of a demographic questionnaire, the WAIS-III Vocabulary subtest as a measure of estimated pre-morbid IQ functioning, a paper-and pencil postconcussion symptom questionnaire, the ImPACT computer based screening test (including the ImPACT Postconcussion Symptom Scale and four cognitive modalities) and traditional paper-and-pencil measures, including the Digit Symbol Substitution test and two adjunctive measures, the Digit Symbol Incidental Recall-Immediate and Delayed. All neurocognitive tests and both postconcussion measures were administered at the pre- and post-season intervals. Two neuropsychological tests were used as

interference tasks preceding the administration of Digit Symbol Incidental Paired Associate Recall-Delayed, but were not analysed for the purpose of this study. They were the California Computerized Assessment Package (CalCAP) (Miller, 1987-1999) which was used in 2003, and the Controlled Oral Word Association test (COWAT) (Benton & Hamsher, 1989; Spreen & Straus, 1998) which was used in 2004. The CalCAP test took about 15 minutes to complete. Although the COWAT consists of three, one minute trials, due to the group administration procedure the test took around ten minutes to administer in that each letter was administered separately and protocols were collected after each trial. Hence it was considered that each of these tests would offer 10 to 15 minutes of interference.

Technical difficulties precluded the use of the CalCAP test as an interference task in the second year of testing. However, it was considered that each of these tests (CalCAP and COWAT) would operate as suitable interference prior to the delayed recall of symbols, precluding rehearsal of symbols. Furthermore all contact and non-contact sports participants were exposed to the same dispensation within each year. Hence, it is not considered that the change in interference task would confound outcome in respect of the delayed recall of symbols.

### **7.6.1 Pre-season testing**

#### **7.6.1.1 Demographic questionnaire (see Appendix B)**

The pre-season demographic questionnaire was administered to all participants before the pre-season testing. It was designed to provide the researcher with i) biographical information such as age, date of birth, first language, etc. ii) sporting history including the sport which would be played during the winter season, iii) head injury or concussion history and iv) information relating to the various exclusion criteria (history of substance abuse, any current psychiatric or psychological disorder, any previous learning disorder, Attention Deficit Disorder, any grades repeated, any remedial instruction, and any neurological disorder).

#### **7.6.1.2 Pre-morbid IQ estimate**

Estimates of pre-morbid levels of intellectual functioning were made in order to ensure that the groups were equivalent in terms of IQ level. There is sufficient research to suggest that intellectual functioning affects neuropsychological performances (Lezak et al., 2004) and that by merely

controlling for level of education does not control for the variability in intellectual capacity (Macchiocci & Barth, 2004). Thus, when athletes with low pre-morbid intelligence are assigned to control groups this may obscure the overall test results between the different groups, with the experimental group appearing unimpaired by comparison.

The Wechsler Adult Intelligence Scale (WAIS) – III Vocabulary subtest, which measures verbal abilities, was used to measure the estimated IQ level. Due to time constraints only one subtest was selected. This test was selected due to the fact that it is considered to be a good indicator of pre-morbid ability based on the assumption that these cognitive skills hold in the face of diffuse brain damage (Lezak et al., 2004), that is, the Vocabulary subtest of the WAIS seems to be very sensitive to education and relatively resistant to brain damage (Reitan and Wolfson, 1997).

The Vocabulary subtest is used to evaluate an individual's semantic knowledge, language development and verbal ability and consists of 33 words arranged in order of increasing difficulty. In accordance with the instruction manual, the participant is instructed to state the meaning of each word. Testing time is approximately 15 to 20 minutes. Although this test is administered orally for individual assessments, in order to facilitate group assessment, participants were instructed to write their definitions in the booklets which were provided. Moreover, the group assessment would not allow for the discontinued rule which applies when the subject has made six consecutive errors. A deviation from the standardized administration was considered legitimate in that the purpose was not to achieve an estimate of IQ in itself, but rather to control for equivalence between comparative subgroups on this variable that were all exposed to the same administrative procedure. The Vocabulary subtest is scored according to the WAIS-III manual where scores of either 2 or 1 or 0 points are awarded to the subject's definition of the given word.

#### **7.6.1.3 Neuropsychological measures**

##### *(i) Postconcussion symptom measures (Appendix B)*

Two postconcussive symptom measures were administered at pre-season. One formed part of the ImPACT computerized testing programme (ImPACT Postconcussion Symptom Scale) and the other comprised a paper-and-pencil symptom checklist (Postconcussion Symptom Questionnaire) which

was attached to the demographic questionnaire at pre-season and administered separately at post-season.

*(a) ImPACT Postconcussion Symptom Scale (see Chapter five)*

The ImPACT Postconcussion Symptom Scale is a self-report inventory included within the ImPACT programme and has been shown to be sensitive to the effects of MTBI (e.g. Lovell et al., 2003; Lovell & Collins, 1998). It consists of a 22-item scale, which requires the athletes to subjectively report on their current symptom status. This is achieved by rating the symptom on a 7-point Likert scale which ranges from 0 = no complaint to 6 = severe symptom. It includes the 22 symptoms of headache, nausea, vomiting, balance problems, dizziness, fatigue, trouble falling asleep, sleeping more than usual, sleeping less than usual, drowsiness, sensitivity to light, sensitivity to noise, irritability, sadness, nervousness, feeling more emotional, numbness or tingling, feeling slowed down, feeling mentally foggy, difficulty concentrating, difficulty remembering and visual problems (Lovell & Collins, 2002).

*(b) Postconcussion Symptom Questionnaire (see Chapter five)*

The paper-and-pencil symptom questionnaire was devised from a paper-and-pencil questionnaire developed for use in prior research in South Africa on Rugby Union athletes (Shuttleworth-Edwards et al., 2004). The original questionnaire consisted of 31 items on a six point Likert scale, which, for the purposes of the present study was reduced to 26 items by removing what were considered to be overlapping and therefore redundant items, whilst retaining the six point Likert scale. The end result was a questionnaire with the same 22 items included in the ImPACT Postconcussion Scale, plus four additional symptoms from the original Shuttleworth-Edwards et al. questionnaire that do not form part of the ImPACT Postconcussion Scale (poor appetite, difficulty hearing, speech problems and aggression). Of these four symptoms, two had demonstrated their sensitivity in the prior research, where professional and high school Rugby Union players reported significantly more speech problems and aggression than non-contact sports controls (Shuttleworth-Edwards et al., 2004). Although there are no validity studies on this revised postconcussion inventory designed especially for the present study, the inventory contains all the items in the ImPACT Symptom Scale which has established validity (Lovell et al., in press), and as indicated above, two of the four additional items

served to discriminate Rugby Union players from controls in the research of Shuttleworth-Edwards et al. (2004).

*(ii) Neurocognitive tests*

*(A) Immediate post-concussion assessment and cognitive testing (ImPACT)*

ImPACT is a computerized neuropsychological screening test specifically designed for the evaluation of sports MTBI and has shown to be both sensitive and specific for the determination of concussion (Schatz et al., 2006). There are four versions of the ImPACT test (see Chapter four for earlier description of the ImPACT test battery)

ImPACT is a windows-based programme which was put onto the school's network in order to facilitate multiple testing. Testing time took approximately 20 to 25 minutes. There are four versions of the ImPACT test. For this study the second version, ImPACT 2.0, was used. The ImPACT test consists of three parts: (a) Sport and health history, (b) Postconcussion Symptom Scale and (c) Neurocognitive test battery

*(a) Sport and health history*

The first part of the ImPACT programme requires the individual to supply basic demographic and descriptive information through a series of easy to follow instructional screens. The individual performs this task via the keyboard and utilizes an external mouse to select responses on the screen. This section asks the individual to answer questions regarding height, weight, sport, position, concussion history, history of learning disabilities and other important descriptive information. In order to ensure confidentiality, the individual is asked to supply their social security number, which in this case was their personal identity number. This provides unique identification of each athlete within the user's database. Once the demographic information is recorded during the first evaluation, subsequent evaluations do not require entry of this information as the computer automatically retrieves the original record.

*(b) Postconcussion Symptom Scale*

As previously discussed, this section of ImPACT requires the individual to rate the current severity of 22 concussive symptoms, via a 7-point Likert scale. This is accomplished by using the external

mouse that is utilized to complete other sections of ImpACT. Individual scores are provided as well as a graphic representation of the symptom total score as part of the computer generated report which can be retrieved at the end of the test.

*(c) Neurocognitive test battery*

ImpACT 2.0 consists of six modules (see Table 7.6.1.1) which measure aspects of cognitive function that have been shown to be sensitive to the effects of concussion in prior research (e.g. Collins, Field, et al., 2003; Lovell & Collins, 2002; Schatz et al., 2006). These include attention span, sustained and selective attention, dimensions of memory, reaction time to one-hundredth of a second across individual test modules and processing speed.

A description of each module and its application is cited from the Clinical Interpretation and Software User's Manual for ImpACT 2.0. (2002)

**Table 7.6.1.1**  
**ImpACT test modules**

Test Module	Ability areas
Word Memory	Immediate and delayed memory for words
Design Memory	Immediate and delayed memory for designs
X's and O's	Attention, concentration, working memory and reaction time
Symbol Match	Visual processing speed, learning and memory
Colour Match	Focused attention, response inhibition, reaction time
Three Letters	Attention, concentration, working memory, visual-motor speed
Symptom Scale	Rating of individual self-reported symptoms

(Iverson, Lovell, & Collins (2002), ImpACT Version 2.0 Manual)

*Module 1: Word Memory*

This module evaluates attention processes or verbal recognition memory and utilizes a word discrimination paradigm. Twelve target words from a word list are presented 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 individual is tested for recall via the presentation of the 24-word list that includes 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 individual 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 individual 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 attention processes and visual recognition memory and utilizes a design discrimination paradigm. Twelve target designs are presented on the computer screen. This sequence is presented twice to facilitate learning. At the end of the second presentation of the list, the individual is tested for recognition via the presentation of 24-designs consisting of 12 target designs and 12 non-target designs (target designs that have been rotated in space). Similar to the word recognition task, the individual 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 individual 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 individual 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 individual 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 individual 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 individual is asked to remember the position 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 individual is asked to click on the previously illuminated X's and O's. Scores are provided for correct identification of the X's and O's (memory), reaction time for the distractor task, and number of errors on the distractor task. For each administration of ImPACT, the individual completes four trials.

#### *Module 4: Symbol Matching*

This module evaluates visual processing speed, learning and memory. Initially, the individual 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 individual is required to click the matching number as quickly as possible and to remember the symbol/number pairings. Correct performance is illustrated through the highlighting of a correctly clicked number in green. Incorrect performance highlights 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 individual is asked to recall the correct symbol or number pairing by clicking the appropriate number button. This module provides an average reaction time score and a score for memory.

#### *Module 5: Colour Match*

This module represents a choice reaction time task and also measures impulse control or response inhibition. First, the individual 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 coloured ink (**GREEN** or **BLUE**). The individual 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 individual is allowed to practice with 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 backwards starting with 25 and ending with 1. Once the individual has completed this initial practice task, he or she is presented with three consonant letters that are displayed on the screen. Immediately following the display of the three letters, the numbered grid re-appears and the individual is instructed to click the numbered buttons backwards (25 to 1) as quickly as possible. After a period of 18 seconds, the numbered grid disappears and the individual 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 distractor test. Five trials of this task are presented for each administration of the test.

From these six modules five composite scores are created: Verbal memory, Visual memory, Visual Motor Speed, Reaction time and Impulse Control (see Table 7.6.1.2 below).

**Table 7.6.1.2**  
**Computations of the composite scores**

Composite Scores	Contributing scores
Verbal Memory	<ul style="list-style-type: none"> <li>• Word Memory (learning and delayed),</li> <li>• Symbol Match memory score</li> </ul>
Visual Memory	<ul style="list-style-type: none"> <li>• Design Memory (learning and delayed)</li> <li>• X's and O's percent correct</li> </ul>
Reaction Time	<ul style="list-style-type: none"> <li>• X's and O's (average counted correct reaction time),</li> <li>• Symbol Match (average weighted reaction time for correct responses)</li> <li>• Colour Match (average reaction time for correct responses)</li> </ul>
Visual Motor Speed	<ul style="list-style-type: none"> <li>• X's and O's (average correct distracters),</li> <li>• Symbol Match (average correct responses)</li> <li>• Three letters (number of correct numbers correctly counted)</li> </ul>
Impulse Control <sup>1</sup>	<ul style="list-style-type: none"> <li>• X's and O's (number of incorrect distracters)</li> <li>• Colour Match (number of errors)</li> </ul>

(Iverson, et al. (2002b), ImpACT Version 2.0 Manual)

*Note.* <sup>1</sup> The Impulse Control composite score was developed to measure profile validity rather than actual postconcussion performance and was therefore not used in the data analysis

As the battery was designed to be used repeatedly over short intervals, the test consists of near infinite number of random forms by alternating the stimulus array with each module, thereby minimizing practice effects (Maroon et al, 2002).

*(B) Digit Symbol Substitution Test*

The Digit Symbol Substitution Test is a test of visuo-perceptual processing speed which has shown to be sensitive to the subtle effects of cognitive impairment and is therefore a good measure of diffuse brain injury following concussion (see earlier review in Chapter four). The South African Wechsler Adult Intelligence Scale (SAWAIS) - Digit Symbol Substitution test (1969) was used for the purpose of this study rather than the longer more contemporary WAIS-III version (1997) as the former is shorter and more economical for research purposes. Moreover, the administration and scoring manual of the WAIS-III version does not provide normative data for this test (Lezak et al., 2004). The South African WAIS version consists of three rows containing blank squares which are each paired with a number which is randomly assigned from one to nine. Above these rows is a key that pairs a number with a symbol. The participant is therefore instructed to pair the corresponding symbol with the number and to write the symbol in the blank squares provided. After completing a practice trial consisting of eight squares, the participant is instructed to complete the first three rows of the pairs of the SAWAIS form. The time limit for the SAWAIS version of this test is 90 seconds and the total score is the sum total of the number of correctly completed squares within this limit.

*(C) Digit Symbol Incidental Paired Associate Recall- Immediate*

Incidental learning is a method that looks at the learning process as it naturally occurs (Lezak et al., 2004). There are three versions of this test but for the purposes of this research the South African Wechsler Adult Intelligence Scale form was employed (Shuttleworth-Edwards, 2002). After noting how many blocks the subject completed within the 90 second period of the Digit Symbol Substitution test, the subjects were then asked to continue with the task until the end of the end of the third row. Following this the subjects were instructed to put their completed Digit Symbol Substitution test into a buff envelope provided and were given a sheet of paper consisting of the numbers one to nine, paired with blank squares. Subjects were asked to recall the nine symbols from the Digit Symbol Substitution test by pairing them with the corresponding numbers in the blank squares provided.

*(D) Digit Symbol Incidental Paired Associate Recall- Delayed*

This test followed the interference task, which translated into a 20 minute delay. An identical sheet of paper to the Digit Symbol Incidental Paired Associate Recall – Immediate test was administered to the participants who were asked to recall the same nine symbols by pairing them with the corresponding numbers in the blank squares provided. It is expected for most individuals to recall as many or almost as many digit-symbol pairs on the delayed trial, although in cases of diffuse brain injury recall may be much less due to the fact that delayed memory is more sensitive to effects of MTBI than immediate memory recall (Lezak et al., 2004).

### **7.6.2 Post-season testing (Appendix B)**

The post-season assessment mirrored the pre-season assessment except for the post-season paper-and-pencil Postconcussion Symptom Questionnaire. Due to time constraints an abbreviated version of this questionnaire was used which consisted of nine symptoms, including the four additional symptoms not included as part of the ImPACT Postconcussion Scale (poor appetite, difficulty hearing, speech problems and aggression) and five symptoms common to both the ImPACT Postconcussion Scale and the Postconcussion Symptom Questionnaire (headache, mentally foggy, difficulty concentrating, difficulty remembering, feeling slowed down) and which have been frequently reported in previous research (e.g. Barnes et al., 1998; Collins, Field, et al., 2003; Collins, Grindel, et al., 1999; Field et al., 2003; Iverson et al., 2002, 2004a, 2004b; Lovell et al., in press), thus demonstrating their sensitivity. To improve the response set, the four additional symptoms were embedded within the five symptoms from the larger questionnaires so that they would not be tested in isolation leading possibly to a skewed response effect. The post-season paper-and-pencil Postconcussion Symptom Questionnaire was labelled the ‘Post-Season Questionnaire’ so as not to create confusion amongst the respondents especially amongst those athletes who had never sustained a concussion and who would therefore have been inclined not to complete the questionnaire. In this way the questionnaire did not discriminate between athletes of the different sports and concussion history groups and therefore all athletes were encouraged to report on whether they were experiencing the current symptoms as outlined in the abbreviated questionnaire.

## 7.7 DATA PROCESSING

Every paper-and-pencil test protocol was scored twice, once by the researcher and once by the research assistant to ensure inter-rater reliability. The WAIS–III Vocabulary subtest, the Digit Symbol Substitution Test and the Digit Symbol Incidental Paired Associate Recall- Immediate and Delayed tests were scored as per the relevant manuals. Due to the fact that ImPACT is a computerized test battery with automated scoring, all data were collated and stored in a standardized manner.

## 7.8 DATA ANALYSIS

Cross-sectional independent t-test analyses involved direct comparisons of group means between the different sports groups as well as between the concussion history groups, on all cognitive test measures and on individual symptoms from both postconcussion symptom questionnaires, at both the pre- and post-season intervals. The prospective dependent t-test analyses on the sports groups at pre-versus post-season were not conducted on the concussion history groups in that sampling attrition at the post-season interval would have resulted in an unacceptably small number of participants. Furthermore it was considered that this would be a less effective mode of identifying the effects than based on sports affiliation due to the fact that concussion history was based on self-reports by the athletes themselves and may have been subjected to considerable bias, with athletes not reporting incidences of concussion history either on purpose or due to their inability to recognise or remember the signs and symptoms associated with concussion or as a result of varying definitions of what constitutes a concussion. In terms of the two aims of the study as delineated in Chapter One, the following statistical analyses took place.

### (i) Independent sample analyses

Independent t-test analyses were conducted on the data derived from the whole sample of sports playing final year high school male athletes at the pre- and post-season intervals to investigate differences in neuropsychological effects in respect of four ImPACT cognitive composite scores (Verbal Memory, Visual Memory, Reaction time and Visual Motor Speed), the Digit Symbol Substitution Test, the Digit Symbol Incidental Paired Associate Recall-Immediate and Delayed, and

the presence of self-reported postconcussive symptoms established on the basis of postconcussion measures between the Contact and Non-Contact sports groups, as well as between the two or more concussion and zero concussion groups within the whole sample.

In addition, independent t-test analyses were conducted on the data derived from the Contact sports subgroup of final year high school male athletes at the pre- and post-season intervals to investigate differences in neuropsychological effects in respect of four ImPACT cognitive composite scores (Verbal Memory, Visual Memory, Reaction time and Visual Motor Speed), the Digit Symbol Substitution Test, the Digit Symbol Incidental Paired Associate Recall-Immediate and Delayed, and the presence of self-reported postconcussive symptoms established on the basis of postconcussion measures between the two or more concussion and zero concussion groups *within* the Contact sports subgroup.

#### (ii) Dependent sample analysis

Dependent t-test analyses were conducted on the data derived from the whole sample of sports playing final year high school male athletes at the pre- and post-season intervals to investigate differences in neuropsychological effects in respect of four ImPACT cognitive composite scores (Verbal Memory, Visual Memory, Reaction time and Visual Motor Speed), the Digit Symbol Substitution Test, the Digit Symbol Paired Associate Incidental Recall-Immediate and Delayed, and the presence of self-reported postconcussive symptoms established on the basis of postconcussion measures between the pre- and post-season intervals for the Contact and Non-Contact sports groups.

### 7.8.1 Significance level

The significance level or *p*-value is the criterion used for rejecting the null hypothesis; it is the probability of a Type I error, that is concluding that a difference exists between means when it does not exist (Howell, 1989). Most researchers use the 5% level of significance ( $p = 0.05$ ) as a cut-off point, that is there is only a 5% probability that any observed differences in mean scores could have occurred by chance, others prefer the 1% level ( $p = 0.01$ ). Whilst some researchers consider the 5% level to be too lenient others maintain that a test at the 1% level is more likely to have a Type II error

(failing to find significance when it does exist) rather than a test at the 5% level. For the purpose of this research both 5% and 1% levels were used for discussion purposes.

Based on prior research (Shuttleworth-Edwards et al., 2004), a directional hypothesis (one-tailed test) was assumed for the comparison between Contact versus Non-Contact groups as well as between the 2+ Concussion and 0 Concussion groups since it was expected that the Contact as well as the 2+ Concussion groups would perform more poorly on tests and report more symptoms than the Non-Contact and 0 Concussion groups. Hence, a prediction was made for the direction in which the groups would differ from the mean, and one-tailed tests were employed which permits the division of the  $p$ -value by two.

In respect of dependent comparisons it was uncertain a) whether there would be improvements on cognitive testing or improvements in overall symptom reporting would be noted, b) that individuals reporting symptoms would be vulnerable to psychosocial or environmental influences and as such for extraneous reasons, individuals would be more stressed and anxious at pre- than at post-season, c) whether poorer performances would occur due to sub-concussive effects sustained during the season or because of an overlay on persistent effects, and d) that there would be no change because a single season is not long enough to see effects. Therefore, where no differences could be assumed between the groups, the  $p$ -value was retained and a two-tailed test (non-directional test) was used. In all tables significance is represented by:  $^{\dagger} p < 0.05$ , one-tailed,  $^{\dagger\dagger} p < 0.01$ , one-tailed,  $* p < 0.05$ , two-tailed,  $** p < 0.01$ , two tailed.

### 7.8.2 Bonferroni's adjustments

The Bonferroni correction is a statistical adjustment for multiple comparisons made simultaneously on the same data set (Hsu, 1996). This provides a more stringent level of statistical significance according to the number of times an analysis takes place on the same data set. In the present study this occurred in respect of the Contact group that was further analysed a second time in respect of 2+ Concussion versus 0 Concussion groups.

Bonferroni's correction may also help to avoid committing a Type I error (finding significance when it does not exist) when multiple measures are used, for example as discussed in Chapter six. Criticisms in the literature have been levelled at researchers such as Matser et al. (1998; 1999) for the use of multiple measures and using the same control group in different studies whilst not controlling for significance by making Bonferroni's adjustments. Rutherford and colleagues (2005), in respect of professional soccer players used a wide range of neuropsychological measures (16 altogether) and in an attempt to dispel such criticisms changed the significance level to  $p = 0.01$  as a measure of increased stringency in his analysis. Further they incorporated different tests into broad cognitive domains, for example, the cognitive domain of memory included the following nine neuropsychological tests: Rey Complex Figure, Logical Memory Immediate and Delayed Recall, Verbal Paired Associate tests Immediate and Delayed, Digit Span Forwards, Digit Span Backwards, Computerized test of Attention Performance Work Memory Latency and the Computerized test of Attention Performance Work Memory Accuracy. Clearly these researchers are attempting to provide stringency without creating an absurdly stringent adjustment that would with certainty incur a Type II error (failing to find significance when it does exist).

Typically in the application of a neuropsychological assessment in a clinical setting, a wide range of tests are employed so as not to miss differential or subtle presentations of cognitive dysfunction, thereby improving the overall sensitivity of the assessment. However, in the research context, if an overly stringent statistical adjustment is made to compensate for such use of multiple measures, ironically there may be an inappropriate loss of statistical sensitivity and the danger of Type II error. In other words, making an appropriate statistical adjustment means getting the correct balance of statistical power by neither under or over-correction that would thereby result in committing Type I or Type II error, respectively (Johnson & Wichern, 2002). In the sports MTBI literature there appears to have been a drive to eliminate Type 1 error (exemplified by the discussions raised in Rutherford et al. in response to criticisms of the soccer studies of Matser et al.), with little concern raised about the inverse situation of thereby enhancing the chance of Type II error. However, the issue of Type II error is a pressing concern that is increasingly being raised in the most current reviews of MTBI research and neuropsychological research on clinical populations in general (Demakis, 2006; Frencham et al., 2005; Reitan & Wolfson, 1999; Ruff, 2005; Woods et al., 2006). These authors provide a powerful body of counterbalancing caution by pointing out the potential for

lack of statistical power in group MTBI research that can result in over-looking subtle, albeit clinically meaningful effects, i.e. Type II error . Therefore it is important to evaluate a particular study for its susceptibility to Type II *as well as* Type I error when conducting research involving neuropsychological screening for MTBI. On the basis of such an evaluation, it would appear that the present research is particularly at risk for Type II error for the following three reasons:

(i) The research employed a focussed battery of relatively few measures (five cognitive measures and two postconcussion symptom inventories) rather than a wide ranging battery of tests such as used by researchers Matser et al. (1998, 1999, & 2001) and Rutherford et al. (2003, 2005).

(ii) Only a proportion of contact sports players will sustain multiple concussions over their sports playing careers and hence significant individual effects are likely to be diluted in the analyses of group effects.

(iii) Results at school level are likely to be much less pronounced than at the professional level due to significantly less years of participation in games and less exposure to acute and sub-concussive effects. Accordingly, it would appear that the present research is at greater risk for failing to identify significance when it is present (Type II error) than Type I error (finding significance when this is not present), and Bonferroni's adjustments were not made on account of the several measurement parameters employed (far less than in the typical full neuropsychological assessment, or similar research in this field). Furthermore it was considered that the risk of committing Type I error in this instance would be counterproductive in terms of heuristic possibilities for future research on the basis of any subtle indications of problems due to cumulative MTBI that might emerge from the present research.

## **7.9 STATISTICAL HYPOTHESES FOR STUDY**

Based on the theoretical hypothesis proposed in Chapter six and outlined above, the following formal statistical hypotheses were posed:

(i) It was hypothesized that *on the basis of independent t-test analyses* there would be significant differences in the mean scores in respect of (a) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and pencil tests), and (b) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories, between the Contact and the Non-Contact groups at both pre- and post-season intervals, in support of deleterious neuropsychological sequelae for the Contact group relative to the Non-Contact group, due to postulated exposure to repetitive concussion, including concussive and sub-concussive events, during high school participation in a contact as opposed to a non-contact sport.

(ii) It was hypothesized that *on the basis of independent t-test analyses* there would be significant differences in the mean scores in respect of (a) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and pencil tests), and (b) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories, between the 2+ Concussion and the 0 Concussion groups at both pre- and post-season intervals, in support of deleterious neuropsychological sequelae for the 2+ Concussion group relative to the 0 Concussion group, due to cumulative exposure of repetitive concussion during high school participation in a contact sport.

(iii) It was hypothesized that *within the Contact group on the basis of independent t-test analyses* there would be significant differences in the mean scores in respect of (a) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and pencil tests), and (b) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories, between the 2+ Concussion group compared with the 0 Concussion group at both pre- and post-season intervals, in support of deleterious neuropsychological sequelae for the 2+ Concussion group relative to the 0 concussion group due to postulated exposure to repetitive concussion during high school participation in a contact sport.

(iv) It was hypothesized *on the basis of dependent t-test analyses* that *either* there would be significant differences in the mean scores in respect of (a) cognitive deficits established on objective

neurocognitive testing (using both computerized and paper and pencil tests), and (b) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories for the Contact group at post-season compared to pre-season, in support of deleterious neuropsychological effects of unreported concussive and sub-concussive events, sustained during participation in a contact sport over a six month winter sports season, *or* that there would be no significant improvement in the mean scores for the Contact group at post-season compared to the pre-season interval on objective neurocognitive testing, in contrast to significant improvement for the Non-Contact group.

(v) Finally, in terms of BRC theory it was hypothesized that *descriptively* compared with deleterious neuropsychological effects that have been reported in the literature for adult high level professional athletes who have a long history of participation in contact sport, in this high school cohort there would only be relatively subtle indications of (a) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and pencil tests), and (b) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories due to shorter exposure to the sport and lower, less intensive level of play.

## CHAPTER EIGHT

### RESULTS OF PRESENT STUDY

In this chapter, the results of the group mean comparisons across all cognitive and postconcussion symptom (PCS) measures (for both the pre- and post-season intervals) will be presented as follows: Significant results for each analysis will be discussed first, followed by the overall trends. A synthesis of the findings for the comparisons between all comparative groups analysed will be made at the end of each section for both the pre and post-season intervals. Reference will be made to specific tables throughout which provide the means, standard deviations, t-statistics (or F-ratios) and significant differences (p-values) for each comparison. A number of figures will delineate selected aspects of the results for emphasis. Tables and figures appear at the end of the first appropriate paragraph or page break following their initial mention in the text.

#### 8.1 PRE- AND POST-SEASON COMPARISONS OF ALL COGNITIVE MEASURES

This subsection reports on the comparisons across all cognitive measures including the composite scores on ImPACT and the paper-and pencil tests.

##### 8.1.1 Pre- and post-season comparisons of all cognitive measures between the Contact versus Non-Contact groups (see Table 8.1.1, p. 259)

In the comparison between the Contact versus Non-Contact groups on the cognitive measures at *pre-season* there was one result that was significant (Reaction Time,  $p = 0.014$ ) in the direction of the Contact group performing worse than the Non-Contact group. Overall there was a marginally predominant trend in the same direction, with the Contact group performing worse than the Non-Contact group (four out of the seven measures, with the exception of the Visual Motor Speed and Digit Symbol Substitution Incidental Recall-Immediate and Delayed).

In the comparison between the Contact versus Non-Contact groups on the cognitive measures at *post-season* there were no significant results. Overall there was a predominant trend in the direction of the Contact group performing worse than the Non-Contact group (six out of the seven measures, with the exception of the Digit Symbol Substitution Incidental Recall-

Immediate). The result that most closely approached significance was Reaction Time ( $p = 0.052$ ), in the direction of the Contact group performing worse than the Non-Contact group.

**Table 8.1.1**  
Pre- and Post-season comparisons of all cognitive measures between the Contact and Non-Contact groups

Pre-season Cognitive Measures	Contact Mean (SD) (n=115)	Non-Contact Mean (SD) (n=74)	<i>t-value</i>	<i>p-value</i>
Verbal Memory	85.41 (8.68)	87.20 (7.80)	1.436	0.076
Visual Memory	77.77 (11.93)	79.04 (10.84)	0.738	0.230
VMS	37.79 (6.60)	37.61 (7.23)	0.181	0.427
RXT	0.60 (7.17)	0.58 (5.43)	2.232	0.014 <sup>†</sup>
DSS	50.72 (11.41)	53.14 (9.99)	1.489	0.069
DSSIR-Immed	7.82 (1.53)	7.70 (1.65)	0.487	0.313
DSSIR-Delay	7.38 (1.92)	7.15 (1.98)	0.808	0.210
Post-season Cognitive Measures	Contact Mean (SD) (n=90)	Non-Contact Mean (SD) (n= 64)	<i>t-value</i>	<i>p-value</i>
Verbal Memory	86.96 (10.05)	87.41 (9.63)	0.888	0.389
Visual Memory	79.37 (12.16)	80.83 (12.41)	0.724	0.235
VMS	37.85 (6.26)	39.05 (6.87)	0.127	0.130
RXT	0.59 (7.57)	0.58 (5.46)	1.631	0.052
DSS	58.02 (10.54)	58.14 (9.74)	0.007	0.472
DSSIR-Immed	8.21 (1.27)	8.09 (1.73)	0.494	0.310
DSSIR-Delay	7.38 (1.93)	7.58 (1.95)	0.616	0.269

Note. <sup>†</sup>  $p < .05$ , one-tailed.

An overview of the pre- and post-season analyses between the Contact versus Non-Contact groups on all cognitive measures (Table 8.1.1), reveals that the results at post-season are broadly similar to the results at pre-season in that there were no significant differences between the Contact and Non-Contact groups on any of the tests with the exception of reaction time which revealed significant, or very close to significantly poorer performance for the Contact group at both the pre- and post-season occasions, respectively. On the trends analyses, there was a

predominant trend in the direction of the Contact group performing worse than the Non-Contact group at both the pre- and post-season intervals, although this was more pronounced at the post-season occasion than at the pre-season occasion (six out of seven versus four out of seven measures, respectively).

### **8.1.2 Pre- and post-season comparisons of all cognitive measures between the 2+ Concussion versus 0 Concussion groups (see Table 8.1.2, p. 261)**

In the comparison between the 2+ Concussion versus 0 Concussion groups on the cognitive measures at *pre-season*, there were no significant results. Overall there was a predominant trend in the direction of the 2+ Concussion group performing worse than the 0 Concussion group (five out of the seven measures, with the exception of the Digit Symbol Substitution Incidental Recall-Immediate and Delayed).

In the comparison between the 2+ Concussion versus 0 Concussion groups on the cognitive measures at *post-season* there were no significant results. Overall there was a strongly predominant trend in the direction of the 2+ Concussion group performing worse than the 0 Concussion group (seven out of the seven measures).

An overview of the pre- and post-season analyses between the 2+ Concussion versus 0 Concussion groups on all cognitive measures (Table 8.1.2), reveals that the results at post-season replicate the results at pre-season in that there were no significant differences between the 2+ Concussion and 0 Concussion groups on any of the tests. On the trends analyses, there was a predominant trend in the direction of the 2+ Concussion group performing worse than the 0 Concussion group at both the pre- and post-season intervals, although this was more pronounced at the post-season occasion than at the pre-season occasion (seven out of seven versus five out of seven measures, respectively).

On the present analysis of number of concussions, the findings for the cognitive test measures are broadly similar to the findings for the different sports groups, with a predominant absence of significant effects across any of the measures on both sets of analyses, with the exception of significant or very close to significantly poorer outcome on Reaction Time at both the pre- and post-season intervals for the sports groups. On the overall trends analyses, the findings on the cognitive tests, in the direction of the 2+ Concussion group performing worse than the 0

Concussion group on the majority of measures at the pre- and particularly at the post-season intervals, are strongly reflective of the findings on the trends analyses reported above (Table 8.1.1) in respect of sports groups, where the Contact group also performed worse than the Non-Contact group on the majority of measures at the pre- and particularly at the post-season intervals.

**Table 8.1.2**  
Pre- and post-season comparisons of all measures between the 2+ Concussion versus 0 Concussion groups

Pre-season Cognitive Measures	2+ Concussion Mean (SD) (n=43)	0 Concussion Mean (SD) (n=108)	<i>t-value</i>	<i>p-value</i>
Verbal Memory	85.88 (8.51)	86.93 (8.25)	0.699	0.242
Visual Memory	77.70 (12.87)	78.44 (11.06)	0.353	0.362
VMS	37.46 (6.04)	37.82 (7.08)	0.294	0.384
RXT	0.59 (7.24)	0.59 (6.91)	0.187	0.426
DSS	49.65 (11.44)	52.10 (10.70)	1.245	0.107
DSSIR-Immed	8.07 (1.14)	7.61 (1.74)	1.595	0.056
DSSIR-Delay	7.40 (1.93)	7.19 (1.96)	0.597	0.275
Post-season Cognitive Measures	2+ Concussion Mean (SD) (n=32)	0 Concussion Mean (SD) (n=91)	<i>t-value</i>	<i>p-value</i>
Verbal Memory	84.88 (11.77)	87.81 (9.87)	1.375	0.085
Visual Memory	78.72 (9.88)	80.63 (12.29)	0.792	0.215
VMS	38.06 (7.56)	38.79 (6.20)	0.537	0.296
RXT	0.60 (8.41)	0.58 (5.71)	0.988	0.162
DSS	57.39 (9.67)	58.26 (10.07)	0.423	0.336
DSSIR-Immed	8.06 (1.55)	8.27 (1.42)	0.694	0.244
DSSIR-Delay	7.26 (2.34)	7.62 (1.75)	0.897	0.185

### **8.1.3 Pre- and post-season *within* Contact group comparisons of all cognitive measures between the 2+ Concussion versus 0 Concussion groups (see Table 8.1.3, p. 263)**

In the *within* Contact group comparison between the 2+ Concussion versus 0 concussion groups on all cognitive measures at *pre-season* there were no significant results. Overall there was a predominant trend in the direction of the 0 Concussion group performing worse than the 2+ Concussion group (five out of the seven measures, with the exception of Visual Motor Speed and the Digit Symbol Substitution test).

In the *within* group comparison between the 2+ Concussion versus 0 Concussion groups on all cognitive measures at *post-season* there were no significant results. Overall, however, there was a more predominant trend in the direction of the 2+ Concussion group performing worse than the 0 Concussion group (six out of the seven measures with the exception of Visual Motor Speed).

An overview of the pre- and post-season analyses for the *within* Contact group comparison between the 2+ Concussion versus 0 Concussion groups on all cognitive measures (Table 8.1.3) replicate each other in that there were no significant results on either occasion. However, the direction of trends at post-season do not replicate the direction of trends at pre-season, in that at pre-season there was an opposite overall trend in the direction of the 0 Concussion group performing worse than the 2+ Concussion group, although this was not repeated at post-season where there was a more predominant trend in the direction of the 2+ Concussion group performing worse than the 0 Concussion group (six out of seven versus five out of seven measures, respectively).

### **8.1.4 Synthesis of findings for pre and post-season comparisons of all cognitive measures**

For both the between and within groups analyses of the 2+ Concussion versus 0 Concussion groups there were no significant effects, in respect of any of the cognitive measures, at either pre or post-season. This is in contrast to the significant, or approaching significant results on Reaction Time for the Contact versus Non-Contact groups at the pre and post-season intervals, respectively. In respect of overall trends, the findings for all between and within groups analyses of the sports groups and the concussion groups, at both the pre- and post-season intervals, with only one exception, were in the direction of predominantly more cognitive measures being worse for the Contact versus Non-Contact groups, and for the 2+ Concussion versus 0 Concussion

groups (at least greater than 50% of measures at pre-season, and at least 90% at post-season, being more for the Contact versus Non-Contact groups and for the 2+ versus 0 Concussion groups). The only exception was for the *within* groups pre-season analysis, where an opposite overall trend of five out of the seven measures being in the direction of the 0 Concussion group performing worse than the 2+ Concussion group occurred.

**Table 8.1.3**  
**Pre- and post-season *within* Contact group comparisons of all cognitive measures**  
**Between the 2+ Concussion versus 0 Concussion groups**

Pre-season Cognitive Measures	2+ Concussion Mean (SD) (n=40)	0 Concussion Mean (SD) (n=45)	<i>t-value</i>	<i>p-value</i>
Verbal Memory	85.95 (8.25)	85.09 (9.37)	- 0.447	0.328
Visual Memory	77.88 (13.18)	77.64 (11.05)	- 0.085	0.466
VMS	37.48 (6.00)	37.68 (7.34)	0.139	0.445
RXT	0.59 (7.45)	0.61 (7.76)	1.673	0.049
DSS	49.75 (11.82)	50.46 (11.43)	0.281	0.390
Immediate	8.03 (1.17)	7.63 (1.76)	- 1.242	0.109
Delayed	7.45 (1.77)	7.20 (2.03)	- 0.615	0.270
Post-season Cognitive Measures	2+ Concussion Mean (SD) (n=29)	0 Concussion Mean (SD) (n=38)	<i>t-value</i>	<i>p-value</i>
Verbal Memory	85.34 (11.99)	87.87 (10.03)	0.937	0.176
Visual Memory	78.03 (9.94)	80.24 (12.06)	0.798	0.214
VMS	38.18 (6.56)	37.82 (6.06)	- 0.230	0.410
RXT	0.60 (8.45)	0.59 (6.14)	- 0.589	0.279
DSS	57.07 (9.96)	58.50 (10.38)	0.562	0.288
Immediate	8.21 (1.10)	8.29 (1.18)	0.263	0.397
Delayed	7.32 (2.20)	7.45 (1.69)	0.264	0.397

*Note.* No '*p*' values reached significance ( $p < .05$ ) with Bonferroni's adjustment

## 8.2 PRE- AND POST-SEASON COMPARISONS OF TOTAL AND INDIVIDUAL SYMPTOMS

This section reports on all the analyses for the Contact versus Non-Contact groups and for the 2+ Concussion versus 0 Concussion groups, in respect of the comparisons of individual symptoms including the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire. As noted in the methodology, invariably the numbers are lower within each inventory at post-season compared with pre-season due to sample attrition. Furthermore, at both pre- and post-season intervals the numbers are lower for the Postconcussion Symptom Questionnaire compared with the ImPACT Postconcussion Symptom Scale, due to the fact that the ImPACT Postconcussion Symptom Scale was administered as part of the ImPACT programme whereas the Postconcussion Symptom Questionnaire was taken home for completion and there was a fall-off in the rate of the number of questionnaires returned.

### 8.2.1 Pre- and post-season comparisons of total and individual symptoms between the Contact versus Non-Contact groups on the ImPACT Postconcussion Symptom Scale (see Tables 8.2.1.1 and 8.2.1.2, pp. 265-266)

In the comparison between the Contact versus Non-Contact groups on the ImPACT Postconcussion Symptom Scale at *pre-season* (Table 8.2.1.1), there were five significant results. These were for the total symptom score ( $p = 0.024$ ) and for the symptoms of fatigue,  $p = 0.010$ ; sleeping less,  $p = 0.018$ , nervousness,  $p = 0.003$  and visual problems,  $p = 0.039$ , all in the direction of the Contact group reportedly being worse than the Non-Contact group. Overall there was a predominant trend in the same direction, with the Contact group reportedly being worse in respect of more symptoms than the Non-Contact group (19 out of the 22 symptoms, with the exception of sensitivity to light, numbness and slowed down).

In the comparison between the Contact versus Non-Contact groups on the ImPACT Symptom Scale at *post-season* (Table 8.2.1.2), the total symptom score approached significance ( $p = 0.056$ ), and there were seven significant results for the symptoms of balance problems,  $p = 0.013$ ; dizziness,  $p = 0.019$ ; fatigue,  $p = 0.041$ ; sleeping less,  $p = 0.019$ ; sadness,  $p = 0.038$ ; nervousness,  $p = 0.007$  and emotional,  $p = 0.004$ , all in the direction of the Contact group reportedly being worse than the Non-Contact group. Overall there was a predominant trend in the same direction with the Contact group reportedly being worse in respect of more symptoms

than the Non-Contact group (19 out of the 22 symptoms, with the exception of sleeping more, sensitivity to light and sensitivity to noise).

**Table 8.2.1.1**  
Pre-season comparisons of total and individual symptoms between the Contact versus Non-Contact groups on the ImPACT Postconcussion Symptom Scale

Symptoms	Contact (n = 115) Mean (SD)	Non-Contact (n = 74) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Total Symptom	16.32 (14.55)	12.50 (11.60)	1.995	0.024 <sup>†</sup>
Headache	0.67 (1.05)	0.50 (0.96)	1.094	0.138
Nausea	0.25 (0.70)	0.15 (0.48)	1.218	0.113
Vomiting	0.11 (0.50)	0.04 (0.19)	1.231	0.110
Balance problems	0.18 (0.50)	0.15 (0.48)	0.476	0.317
Dizziness	0.48 (0.85)	0.31 (0.68)	1.525	0.065
Fatigue	1.62 (1.49)	1.12 (1.31)	2.359	0.010 <sup>†</sup>
Difficult falling asleep	1.18 (1.44)	0.85 (1.36)	1.535	0.063
Sleeping more	0.72 (1.33)	0.45 (1.04)	1.566	0.060
Sleeping less	1.50 (1.68)	1.03 (1.38)	2.110	0.018 <sup>†</sup>
Drowsiness	0.86 (1.32)	0.81 (1.13)	0.262	0.400
Sensitivity to light	0.45 (0.99)	0.55 (1.06)	-0.699	0.243
Sensitivity to noise	0.33 (0.82)	0.19 (0.63)	1.357	0.088
Irritability	1.37 (1.38)	1.04 (1.43)	1.571	0.059
Sadness	0.58 (1.07)	0.55 (1.06)	0.156	0.438
Nervousness	0.81 (1.29)	0.38 (0.75)	2.864	0.003 <sup>††</sup>
Emotional	0.89 (1.23)	0.73 (1.28)	0.886	0.189
Numbness	0.18 (0.60)	0.19 (0.59)	-0.056	0.478
Slowed down	0.71 (1.06)	0.76 (1.02)	-0.296	0.384
Mentally foggy	0.73 (1.09)	0.61 (1.02)	0.756	0.226
Concentration	1.44 (1.50)	1.09 (1.32)	1.603	0.056
Remembering	0.88 (1.30)	0.78 (1.14)	0.504	0.308
Visual Problems	0.39 (0.80)	0.22 (0.50)	1.779	0.039 <sup>†</sup>

Note. <sup>†</sup> $p < .05$ , one-tailed. <sup>††</sup> $p < .01$ , one-tailed.

**Table 8.2.1.2**  
**Post-season comparisons of total and individual symptoms between the Contact**  
**Versus Non-Contact groups on the ImPACT Postconcussion Symptom Scale**

Symptoms	Contact (n = 90) Mean (SD)	Non-contact (n = 64) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Total Symptom	18.71 (17.00)	14.59 (13.62)	1.601	0.056
Headache	0.84 (1.36)	0.64 (1.10)	0.981	0.164
Nausea	0.45 (0.81)	0.30 (0.66)	1.238	0.109
Vomiting	0.17 (0.53)	0.14 (0.50)	0.330	0.371
Balance problems	0.38 (0.76)	0.16 (0.48)	2.247	0.013 <sup>†</sup>
Dizziness	0.64 (0.93)	0.34 (0.80)	2.109	0.019 <sup>†</sup>
Fatigue	1.80 (1.66)	1.36 (1.33)	1.749	0.041 <sup>†</sup>
Difficult falling asleep	1.09 (1.47)	1.08 (1.48)	0.049	0.481
Sleeping more	0.74 (1.23)	0.78 (1.36)	- 0.188	0.426
Sleeping less	1.43 (1.81)	0.88 (1.43)	2.105	0.019 <sup>†</sup>
Drowsiness	1.16 (1.95)	0.88 (1.24)	1.017	0.156
Sensitivity to light	0.62 (1.20)	0.75 (1.27)	- 0.654	0.257
Sensitivity to noise	0.44 (0.94)	0.61 (0.97)	- 1.096	0.138
Irritability	1.34 (1.51)	1.11 (1.38)	0.963	0.169
Sadness	0.69 (1.15)	0.41 (0.77)	1.793	0.038 <sup>†</sup>
Nervousness	0.65 (0.97)	0.31 (0.71)	2.503	0.007 <sup>††</sup>
Emotional	1.12 (1.53)	0.55 (1.14)	2.673	0.004 <sup>††</sup>
Numbness	0.30 (0.93)	0.23 (0.68)	0.502	0.309
Slowed down	1.13 (1.36)	0.86 (1.14)	1.322	0.094
Mentally foggy	1.06 (1.36)	1.00 (1.32)	0.255	0.400
Concentration	1.42 (1.53)	1.17 (1.38)	1.015	0.156
Remembering	0.84 (1.32)	0.72 (1.11)	0.612	0.271
Visual Problems	0.40 (1.05)	0.33 (0.80)	0.488	0.313

Note. <sup>†</sup>  $p < .05$ , one-tailed. <sup>††</sup>  $p < .01$ , one-tailed.

An overview of the pre- and post-season analyses between the Contact versus Non-Contact groups on the ImPACT Postconcussion Symptom Scale (Tables 8.2.1.1 and 8.2.1.2) at both the pre- and post-season intervals, reveals that the total symptom score was significant at pre-season ( $p = 0.024$ ) in the direction of the Contact group reportedly being worse than the Non-Contact

group, and approaching significance in the same direction at post-season ( $p = 0.056$ ). At pre-season, there were only four symptoms that were significant (fatigue, sleeping less, nervousness and visual problems) in the direction of the Contact group reportedly being worse than the Non-Contact group, whereas at post-season this number increased to seven symptoms being significantly worse for the Contact group, including the new symptoms of balance problems, dizziness, sadness and emotional. The results at post-season replicate the results at pre-season in respect of the symptoms of fatigue, sleeping less, and nervousness, being significant at both intervals in the direction of the Contact group reportedly being worse than the Non-Contact group. This occurred in combination with a predominant trend in the same direction at both the pre- and post-season intervals, for the Contact group to be worse in respect of more symptoms than the Non-Contact group (19 out of 22 symptoms at both pre- and post-season intervals).

### **8.2.2 Pre- and post-season comparisons of total and individual symptoms between the Contact versus Non-Contact groups on the Postconcussion Symptom Questionnaire (see Tables 8.2.2.1 and 8.2.2.2, pp. 269-270)**

(As indicated in the Methodology, the Postconcussion Symptom Questionnaire comprised 26 symptoms at pre-season and was reduced to nine symptoms at the post-season interval).

In the comparison between the Contact versus Non-Contact groups on the Postconcussion Symptom Questionnaire at *pre-season* (Table 8.2.2.1), there were nine significant results. These were for the total symptom score ( $p = 0.050$ ) and the symptoms of headache,  $p = 0.001$ ; fatigue,  $p = 0.038$ ; difficulty falling asleep,  $p = 0.047$ ; sleeping less,  $p = 0.028$ ; difficulty hearing,  $p = 0.018$ ; aggression,  $p = 0.019$ ; nervousness,  $p = 0.001$  and emotional,  $p = 0.009$ , all in the direction of the Contact group reportedly being worse than the Non-Contact group. Overall, there was a predominant trend in the same direction, with the Contact group reportedly being worse in respect of more symptoms than the Non-Contact group (18 out of the 26 symptoms, with the exception of nausea, balance problems, sleeping more, sensitivity to light, sensitivity to noise, mentally foggy, remembering and speech problems).

In the comparison between the Contact versus Non-Contact groups on the Postconcussion Symptom Questionnaire at *post-season* (Table 8.2.2.2) there were nine significant results. These were for the total symptom score ( $p = 0.000$ ) and the symptoms of headache,  $p = 0.040$ ; poor appetite,  $p = 0.030$ ; mentally foggy,  $p = 0.022$ ; concentration,  $p = 0.002$ ; remembering,  $p =$

0.004; speech problems,  $p = 0.006$ ; aggression,  $p = 0.001$  and slowed down,  $p = 0.001$ , all in the direction of the Contact group reportedly being worse than the Non-Contact group. Overall, there was a strongly predominant trend in the same direction, with the Contact group reportedly being worse in respect of more symptoms than the Non-Contact group (9 out of the 9 symptoms).

An overview of the pre- and post-season analyses between the Contact versus Non-Contact groups on the Postconcussion Symptom Questionnaire (Tables 8.2.2.1 and 8.2.2.2), reveals that at both pre- and post-season intervals the total symptom scores were significant, and that in both cases there were eight symptoms that were significant, all in the direction of the Contact group reportedly being worse than the Non-Contact group. At pre-season, of the symptoms that were significant in the direction of the Contact group reportedly being worse than the Non-Contact group, five symptoms (fatigue, difficulty falling asleep, sleeping less, nervousness and emotional) did not form part of the post-season questionnaire and the sixth symptom (difficulty hearing) showed a comparative trend for the Contact group to be worse than the Non-Contact group at the post-season interval ( $p = 0.078$ ). The results at post-season replicate the results at pre-season in respect of the symptoms of headache and aggression, being significant at both intervals in the direction of the Contact group reportedly being worse than the Non-Contact group. Generally there was a highly consistent trend for the Contact group to be worse in respect of more symptoms than the Non-Contact group at both the pre- and post-season occasions (18 out of 26 versus nine out of nine symptoms, respectively).

### **8.2.3 Synthesis of findings for pre- and post-season comparisons of total and individual symptoms between the Contact versus Non-Contact groups**

The findings on the pre- and post-season comparisons between the Contact versus Non-Contact groups on both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire consistently reveal significantly higher reporting of symptoms for the Contact versus Non-Contact groups at both the pre- and post-season intervals, on both inventories. None of the significant results were in the opposite direction (i.e. the Non-Contact group reportedly being worse than the Contact group). In all instances the total symptom score is either significantly lower for the Contact versus Non-Contact group, or approaching significance ( $p = 0.024$ ;  $p = 0.056$ ;  $p = 0.050$  and  $p = 0.000$ ). Finally, the trends analyses also reflect a consistently predominant trend for the Contact group to be worse in respect of more symptoms than the Non-Contact group, across all analyses.

**Table 8.2.2.1**  
**Pre-season comparisons of total and individual symptoms between the Contact versus Non-Contact groups on the Postconcussion Symptom Questionnaire**

Symptoms	Contact (n = 102) Mean (SD)	Non-contact (n = 50) Mean (SD)	<i>t</i> -value	<i>p</i> -value
Total Symptom	15.22 (12.31)	11.74 (11.78)	1.659	0.050 <sup>†</sup>
Headache	0.73 (1.10)	0.36 (0.77)	2.373	0.001 <sup>††</sup>
Nausea	0.17 (0.51)	0.20 (0.57)	- 0.364	0.358
Vomiting	0.06 (0.31)	0.02 (0.14)	0.845	0.200
Poor appetite	0.27 (0.68)	0.24 (0.66)	0.298	0.383
Balance problems	0.11 (0.42)	0.14 (0.50)	- 0.412	0.339
Dizziness	0.39 (0.82)	0.22 (0.58)	1.326	0.094
Fatigue	1.81 (1.43)	1.38 (1.34)	1.791	0.038 <sup>†</sup>
Difficult falling asleep	1.17 (1.47)	0.78 (1.25)	1.689	0.047 <sup>†</sup>
Sleeping more	0.51 (1.10)	0.56 (1.22)	- 0.256	0.395
Sleeping less	1.06 (1.43)	0.66 (1.06)	1.935	0.028 <sup>†</sup>
Drowsiness	0.64 (1.06)	0.52 (0.95)	0.662	0.255
Sensitivity to light	0.27 (0.79)	0.50 (0.91)	- 0.501	0.069
Sensitivity to noise	0.20 (0.63)	0.26 (0.75)	- 0.551	0.291
Difficulty hearing	0.30 (0.87)	0.10 (0.30)	2.129	0.018 <sup>†</sup>
Irritability	1.13 (1.26)	0.86 (1.11)	1.335	0.093
Aggression	0.97 (1.32)	0.58 (0.93)	2.106	0.019 <sup>†</sup>
Sadness	0.48 (0.94)	0.34 (0.90)	0.878	0.191
Nervousness	0.66 (1.14)	0.22 (0.55)	3.198	0.001 <sup>††</sup>
Emotional	0.83 (1.30)	0.40 (0.88)	2.421	0.009 <sup>††</sup>
Numbness	0.10 (0.36)	0.10 (0.36)	- 0.031	0.488
Slowed down	0.55 (0.95)	0.54 (0.91)	0.050	0.478
Mentally foggy	0.54 (0.93)	0.60 (1.00)	0.371	0.356
Concentration	1.15 (1.21)	0.94 (1.19)	- 1.000	0.160
Remembering	0.69 (0.98)	0.80 (1.07)	- 0.654	0.257
Visual problems	0.31 (0.84)	0.28 (0.73)	0.242	0.405
Speech problems	0.13 (0.46)	0.14 (0.41)	- 0.164	0.435

Note. <sup>†</sup>  $p \leq .05$ , one-tailed. <sup>††</sup>  $p \leq .01$ , one-tailed.

**Table 8.2.2.2**  
**Post-season comparisons of total and individual symptoms between the Contact versus Non-Contact groups on the Postconcussion Symptom Questionnaire**

Symptoms	Contact (n = 84) Mean (SD)	Non-Contact (n = 61) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Total Symptom	8.23 (7.85)	4.46 (4.98)	3.529	0.000 <sup>††</sup>
Headache	0.70 (1.10)	0.43 (0.78)	1.769	0.040 <sup>†</sup>
Poor appetite	0.73 (1.18)	0.41 (0.82)	1.905	0.030 <sup>†</sup>
Mentally foggy	1.11 (1.28)	0.70 (1.02)	2.028	0.022 <sup>†</sup>
Difficulty hearing	0.52 (0.89)	0.34 (0.63)	1.428	0.078
Concentration	1.44 (1.41)	0.83 (1.04)	2.970	0.002 <sup>††</sup>
Remembering	1.04 (1.19)	0.57 (0.85)	2.737	0.004 <sup>††</sup>
Speech problems	0.45 (0.84)	0.18 (0.43)	2.543	0.006 <sup>††</sup>
Aggression	0.98 (1.34)	0.36 (0.75)	3.523	0.001 <sup>††</sup>
Slowed down	1.26 (1.38)	0.64 (0.90)	3.289	0.001 <sup>††</sup>

Note. <sup>†</sup> $p < .05$ , one-tailed, <sup>††</sup> $p < .01$ , one-tailed.

#### 8.2.4 Pre- and Post-season comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussion groups on the ImPACT Postconcussion Symptom Scale (see Tables 8.2.4.1 and 8.2.4.2, pp. 272-273)

In the comparison between the 2+ Concussion versus 0 Concussion groups on the ImPACT Symptom Scale at *pre-season* there were eight results that were significant. These were for the total symptom score ( $p = 0.020$ ) and for the symptoms of balance problems,  $p = 0.037$ ; dizziness,  $p = 0.046$ ; fatigue,  $p = 0.006$ ; drowsiness,  $p = 0.035$ ; irritability,  $p = 0.001$ ; slowed down,  $p = 0.034$  and concentration,  $p = 0.025$ , all in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group. Overall, there was a predominant trend in the same direction, for the 2+ Concussion group to be worse in respect of more symptoms than the 0 Concussion group (20 out of the 22 symptoms, with the exception of sadness and remembering).

In the comparison between the 2+ Concussion versus 0 Concussion groups on the ImPACT Symptom Scale at *post-season* (Table 8.2.4.2) the total symptom score was not significant ( $p = 0.116$ ), and there were only two significant results. These were for the symptoms of dizziness,  $p$

= 0.031 and emotional,  $p = 0.042$ , both in the direction of the 2+ Concussion group reportedly being worse than the Concussion group. Overall, there was a predominant trend in the same direction, for the 2+ Concussion group to be worse in respect of more symptoms than the 0 Concussion group (the total symptom score, and 17 out of the 22 symptoms, with the exception of sleeping less, sensitivity to light, mentally foggy, concentration and remembering).

An overview of the pre and post-season analyses reveals that for the comparison between the 2+ Concussion versus 0 Concussion groups on the ImPACT Postconcussion Symptom Scale (Tables 8.2.4.1 and 8.2.4.2) at both pre and post-season intervals, the total symptom score was significant at pre-season only ( $p = 0.020$ ), in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group. The total symptom score was not significant at the post-season interval ( $p = 0.116$ ), but the trend presented in the same direction with the 2+ Concussion group reportedly being worse than the 0 Concussion group. At pre-season there were seven symptoms that were significant (balance problems, dizziness, fatigue, drowsiness, irritability, slowed down and concentration), all in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group, whereas at post-season this number decreased to two symptoms (dizziness and emotional), in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group. The results at post-season replicate the results at pre-season in respect of the symptoms of dizziness and emotional, being significant at both intervals in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group. At the pre-season interval there were five symptoms that reached significance but not at the post-season interval. Four of these symptoms at post-season showed a comparative trend for the 2+ Concussion group to be worse than the 0 Concussion group (balance problems, fatigue, irritability and slowed down), but there was an opposite trend for one symptom (concentration), which was worse for the 0 Concussion group than the 2+ Concussion group at the post-season interval. Overall, there was a highly consistent trend for the 2+ Concussion group to be worse in respect of more symptoms than the 0 Concussion group, at both the pre and post-season occasions (20 out of 22 and 17 out of 22 symptoms, respectively).

**Table 8.2.4.1**  
**Pre-season comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussions groups on the ImPACT Postconcussion Symptom Scale**

Symptoms	2+ Concussion (n = 43) Mean (SD)	0 Concussion (n = 108) Mean (SD)	<i>t</i> -value	<i>p</i> -value
Total Symptom	18.86 (14.19)	13.67 (13.64)	- 2.081	0.020 <sup>†</sup>
Headache	0.58 (0.93)	0.55 (1.06)	- 0.162	0.436
Nausea	0.37 (0.85)	0.17 (0.57)	- 1.452	0.076
Vomiting	0.19 (0.66)	0.07 (0.34)	- 1.132	0.132
Balance problems	0.30 (0.67)	0.10 (0.38)	- 1.825	0.037 <sup>†</sup>
Dizziness	0.60 (0.93)	0.34 (0.68)	- 1.714	0.046 <sup>†</sup>
Fatigue	1.86 (1.52)	1.20 (1.41)	0.747	0.006 <sup>††</sup>
Difficult falling asleep	1.26 (1.38)	0.93 (1.40)	- 1.274	0.103
Sleeping more	0.79 (1.44)	0.46 (1.01)	- 1.384	0.086
Sleeping less	1.47 (1.81)	1.35 (1.55)	- 0.406	0.343
Drowsiness	1.19 (1.53)	0.71 (1.13)	- 1.844	0.035 <sup>†</sup>
Sensitivity to light	0.53 (1.05)	0.50 (1.07)	- 0.157	0.438
Sensitivity to noise	0.35 (0.87)	0.30 (0.78)	- 0.342	0.367
Irritability	1.84 (1.36)	1.07 (1.37)	- 3.085	0.001 <sup>††</sup>
Sadness	0.51 (0.98)	0.64 (1.18)	0.606	0.273
Nervousness	0.86 (1.20)	0.54 (1.01)	1.528	0.066
Emotional	1.07 (1.33)	0.83 (1.28)	- 1.019	0.155
Numbness	0.26 (0.82)	0.15 (0.45)	- 0.803	0.213
Slowed down	1.07 (1.30)	0.66 (0.95)	- 1.861	0.034 <sup>†</sup>
Mentally foggy	0.86 (1.25)	0.62 (1.02)	- 1.243	0.108
Concentration	1.81 (1.68)	1.28 (1.42)	- 1.973	0.025 <sup>†</sup>
Remembering	0.77 (1.31)	0.92 (1.30)	0.630	0.265
Visual Problems	0.33 (0.68)	0.29 (0.70)	- 0.386	0.388

Note. <sup>†</sup>  $p < .05$ , one-tailed. <sup>††</sup>  $p < .01$ , one-tailed.

**Table 8.2.4.2**  
**Post-season comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussion groups on the ImPACT Postconcussion Symptom Scale**

Symptoms	2+ Concussion (n = 32) Mean (SD)	0 Concussion (n = 91) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Total Symptom	20.55 (19.24)	16.51 (14.97)	- 1.204	0.116
Headache	0.90 (1.33)	0.66 (1.20)	- 0.955	0.171
Nausea	0.68 (1.05)	0.34 (0.67)	- 1.680	0.051
Vomiting	0.35 (0.80)	0.12 (0.44)	- 1.553	0.065
Balance problems	0.45 (0.77)	0.22 (0.61)	- 1.525	0.067
Dizziness	0.87 (1.12)	0.45 (0.82)	- 1.925	0.031 <sup>†</sup>
Fatigue	1.97 (1.58)	1.49 (1.52)	- 1.480	0.071
Difficult falling asleep	1.19 (1.28)	1.02 (1.48)	- 0.575	0.283
Sleeping more	0.97 (1.45)	0.64 (1.16)	- 1.283	0.101
Sleeping less	1.23 (1.56)	1.29 (1.73)	0.170	0.433
Drowsiness	1.65 (2.67)	0.92 (1.34)	- 1.448	0.078
Sensitivity to light	0.68 (1.28)	0.69 (1.24)	0.057	0.478
Sensitivity to noise	0.61 (1.05)	0.53 (0.97)	0.414	0.340
Irritability	1.42 (1.57)	1.25 (1.38)	- 0.561	0.288
Sadness	0.71 (1.07)	0.60 (1.11)	- 0.459	0.323
Nervousness	0.58 (0.81)	0.52 (0.94)	- 0.341	0.367
Emotional	1.23 (1.59)	0.71 (1.34)	- 1.747	0.042 <sup>†</sup>
Numbness	0.42 (0.89)	0.26 (0.89)	- 0.840	0.202
Slowed down	1.23 (1.48)	1.08 (1.28)	- 0.536	0.297
Mentally foggy	1.03 (1.47)	1.04 (1.29)	0.042	0.484
Concentration	1.29 (1.70)	1.40 (1.40)	0.343	0.367
Remembering	0.71 (1.13)	0.88 (1.32)	0.641	0.262
Visual Problems	0.39 (0.92)	0.38 (0.95)	- 0.013	0.495

Note. <sup>†</sup>*p* < .05, one-tailed.

### **8.2.5 Pre- and post-season comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussion groups on the Postconcussion Symptom Questionnaire (see Tables 8.2.5.1 and 8.2.5.2, pp. 275-276)**

(As indicated in the Methodology, the Postconcussion Symptom Questionnaire comprised 26 symptoms at pre-season and was reduced to nine symptoms at the post-season interval)

In the comparison between the 2+ Concussion versus 0 Concussion groups on the Postconcussion Symptom Questionnaire at *pre-season* (Table 8.2.5.1) there were twelve significant results. These were for the total symptom score ( $p = 0.000$ ) and for the symptoms of dizziness,  $p = 0.031$ ; fatigue,  $p = 0.001$ ; difficulty falling asleep,  $p = 0.014$ ; sleeping less,  $p = 0.011$ ; drowsiness,  $p = 0.005$ ; sensitivity to noise,  $p = 0.029$ ; irritability,  $p = 0.000$ ; aggression,  $p = 0.016$ ; nervousness,  $p = 0.002$ ; emotional,  $p = 0.014$  and concentration,  $p = 0.009$ , all in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group. Overall, there was a predominant trend in the same direction, with the 2+ Concussion group reportedly being worse in respect of more symptoms than the 0 Concussion group (24 out of the 26 symptoms, with the exception of remembering and speech problems).

In the comparison between the 2+ Concussion versus 0 Concussion groups on the PCS Questionnaire at *post-season* (Table 8.2.5.2) there were seven significant results. These were for the total symptom score ( $p = 0.006$ ) and for the symptoms of mentally foggy,  $p = 0.027$ ; concentration,  $p = 0.015$ ; remembering,  $p = 0.011$ ; speech problems,  $p = 0.015$ ; aggression,  $p = 0.001$  and slowed down,  $p = 0.040$ , all in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group. Overall, there was a strongly predominant trend in the same direction, with the 2+ Concussion group reportedly being worse in respect of more symptoms than the 0 Concussion group (nine out of the nine symptoms).

An overview of the pre and post-season analyses for the comparison between the 2+ Concussion versus 0 Concussion groups on the Postconcussion Symptom Questionnaire (Tables 8.2.5.1 and 8.2.5.2), at both pre and post-season intervals, reveals that the total symptom scores were significant ( $p = 0.000$  and  $p = 0.006$ ) in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group. At pre-season there were eleven symptoms that were significant and at post-season six symptoms that were significant, all in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group.

**Table 8.2.5.1**  
**Pre-season comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussion groups on the Postconcussion Symptom Questionnaire**

Symptoms	2+ Concussions (n = 40) Mean (SD)	0 Concussions (n = 79) Mean (SD)	<i>t</i> -value	<i>p</i> -value
Total Symptom	20.05 (11.86)	11.77 (11.78)	- 3.613	0.000 <sup>††</sup>
Headache	0.70 (1.20)	0.56 (1.01)	- 0.684	0.248
Nausea	0.28 (0.68)	0.14 (0.47)	- 1.133	0.131
Vomiting	0.13 (0.46)	0.01 (0.11)	- 1.511	0.069
Poor appetite	0.48 (0.91)	0.23 (0.60)	- 1.562	0.062
Balance problems	0.13 (0.46)	0.11 (0.43)	- 0.125	0.451
Dizziness	0.55 (0.93)	0.24 (0.60)	- 1.907	0.031 <sup>†</sup>
Fatigue	2.35 (1.53)	1.15 (1.37)	- 3.194	0.001 <sup>††</sup>
Difficult falling asleep	1.35 (1.58)	0.72 (1.11)	- 2.253	0.014 <sup>†</sup>
Sleeping more	0.55 (1.20)	0.53 (1.15)	0.908	0.468
Sleeping less	1.43 (1.57)	0.76 (1.22)	- 2.349	0.011 <sup>†</sup>
Drowsiness	0.95 (1.28)	0.35 (0.73)	- 2.724	0.005 <sup>††</sup>
Sensitivity to light	0.35 (0.89)	0.33 (0.76)	- 0.133	0.447
Sensitivity to noise	0.45 (0.97)	0.13 (0.52)	- 1.945	0.029 <sup>†</sup>
Difficulty hearing	0.43 (1.13)	0.16 (0.49)	- 1.393	0.085
Irritability	1.75 (1.26)	0.82 (1.08)	- 4.178	0.000 <sup>††</sup>
Aggression	1.23 (1.51)	0.65 (0.99)	- 2.200	0.016 <sup>†</sup>
Sadness	0.58 (1.08)	0.39 (0.93)	- 0.959	0.170
Nervousness	0.95 (1.24)	0.28 (0.77)	- 3.136	0.002 <sup>††</sup>
Emotional	1.18 (1.41)	0.59 (1.16)	- 2.243	0.014 <sup>†</sup>
Numbness	0.15 (0.43)	0.09 (0.33)	- 0.870	0.193
Slowed down	0.63 (0.95)	0.47 (0.92)	- 0.869	0.194
Mentally foggy	0.68 (0.92)	0.49 (0.97)	- 0.979	0.165
Concentration	1.60 (1.32)	1.03 (1.18)	- 2.418	0.009 <sup>††</sup>
Remembering	0.75 (1.01)	0.81 (1.05)	0.299	0.383
Visual problems	0.40 (0.90)	0.22 (0.61)	- 1.168	0.124
Speech problems	0.08 (0.27)	0.19 (0.56)	1.522	0.066

Note. <sup>†</sup>  $p < .05$ , one-tailed. <sup>††</sup>  $p < .01$ , one-tailed.

**Table 8.2.5.2**

**Post-season comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussion groups on the Postconcussion Symptom Questionnaire**

Symptoms	2+ Concussions (n = 30)	0 Concussions (n = 86)	<i>t-value</i>	<i>p-value</i>
Total Symptom	10.00 (8.57)	5.55 (6.14)	- 2.622	0.006 <sup>††</sup>
Headache	0.70 (1.15)	0.53 (0.93)	- 0.787	0.217
Poor appetite	0.87 (1.36)	0.47 (0.89)	- 1.510	0.070
Mentally foggy	1.27 (1.26)	0.79 (1.12)	- 1.943	0.027 <sup>†</sup>
Difficulty hearing	0.67 (1.06)	0.40 (0.72)	- 1.299	0.101
Concentration	1.76 (1.55)	1.05 (1.21)	- 2.254	0.015 <sup>†</sup>
Remembering	1.30 (1.24)	0.71 (0.97)	- 2.376	0.011 <sup>†</sup>
Speech problems	0.67 (1.06)	0.21 (0.49)	- 2.278	0.015 <sup>†</sup>
Aggression	1.43 (1.41)	0.52 (1.05)	- 3.244	0.001 <sup>††</sup>
Slowed down	1.40 (1.45)	0.87 (1.20)	- 1.790	0.040 <sup>†</sup>

Note. <sup>†</sup>  $p < .05$ , one-tailed. <sup>††</sup>  $p < .01$ , one-tailed.

Of the symptoms that were significant at pre-season in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group, nine symptoms (dizziness, fatigue, difficulty falling asleep, sleeping less, drowsiness, sensitivity to noise, irritability, nervousness and emotional) did not form part of the post-season questionnaire. There were four symptoms (slowed down, mentally foggy, remembering and speech problems) that were significant only at the post-season interval, all in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group. Two of these symptoms (slowed down and mentally foggy) at pre-season showed a comparative trend for the 2+ Concussion group to be worse than the 0 Concussion group, whereas the other two symptoms (remembering and speech problems) showed an opposite trend for the 0 Concussion group to be worse than the 2+ Concussion group, at pre-season. The results at post-season replicate the results at pre-season in respect of the symptoms of concentration and aggression, being significant at both intervals in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group. This occurred in combination with a highly consistent trend in the same direction, for the 2+ concussion group to

be worse in respect of more symptoms than the 0 concussion group, at both pre- and post-season occasions (24 out of 26 versus nine out of nine symptoms, respectively).

### **8.2.6 Synthesis of findings for pre- and post-season comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussion groups**

The findings on the pre- and post-season comparisons between the 2+ and 0 Concussion groups on both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire consistently reveal instances of significantly higher reporting of symptoms for the 2+ Concussion group at both the pre and post-season intervals, on both inventories. None of the significant results were in the opposite direction (i.e. the 0 Concussion group reportedly being worse than the 2+ Concussion group). In all instances the total symptom score is either significant or shows a strong trend in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group ( $p = 0.020$ ;  $p = 0.116$ ;  $p = 0.000$  and  $p = 0.006$ ). Finally, the trends analyses also reflect a consistently predominant trend in the direction of the 2+ Concussion group reportedly being worse in respect of more symptoms than the 0 Concussion group, across all analyses.

Overall, across both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire at both pre and post season intervals there was a predominant trend in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group, revealing more deleterious effects for the 2+ Concussion group with respect to symptom presentation. None of the symptoms on either the ImPACT Postconcussion Symptom Scale or the Postconcussion Questionnaire at both pre and post-season occasions reached significance in the direction of the 0 Concussion group reportedly being worse than the 2+ Concussion group.

### **8.2.7 Pre- and Post-season *within* Contact group comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussion groups on the ImPACT Postconcussion Symptom Scale (see Tables 8.2.7.1 and 8.2.7.2, pp. 278-279)**

In the comparison of symptoms *within* the Contact group, between the 2+ Concussion versus 0 Concussion groups on the ImPACT Postconcussion Symptom Scale at *pre-season* (Table 8.2.7.1), there were no significant results. Overall, however, there was a strong trend in the direction of the 2+ Concussion group reportedly being worse in respect of more symptoms than

the 0 Concussion group (16 out of the 22 symptoms, with the exception of headache, sleeping less, sensitivity to noise, sadness, remembering and visual problems).

**Table 8.2.7.1**  
Pre-season *within* Contact group comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussion groups on the ImPACT Postconcussion Symptom Scale

Symptoms	2+ Concussions (n = 40) Mean (SD)	0 Concussions (n = 45) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Total Symptom	19.05 (14.45)	16.13 (15.99)	- 0.878	0.191
Headache	0.58 (0.93)	0.71 (1.22)	0.573	0.284
Nausea	0.38 (0.87)	0.18 (0.65)	- 1.174	0.122
Vomiting	0.20 (0.68)	0.09 (0.47)	- 0.880	0.191
Balance problems	0.33 (0.69)	0.09 (0.29)	- 2.004	0.025
Dizziness	0.63 (0.95)	0.44 (0.84)	- 0.928	0.178
Fatigue	1.93 (1.51)	1.40 (1.57)	- 1.566	0.061
Difficult falling asleep	1.30 (1.40)	1.00 (1.40)	- 0.987	0.164
Sleeping more	0.85 (1.48)	0.51 (0.97)	- 1.234	0.111
Sleeping less	1.55 (1.85)	1.80 (1.67)	0.654	0.258
Drowsiness	1.20 (1.54)	0.76 (1.33)	- 1.426	0.079
Sensitivity to light	0.48 (1.04)	0.44 (1.01)	- 0.137	0.446
Sensitivity to noise	0.35 (0.89)	0.42 (0.89)	0.372	0.355
Irritability	1.75 (1.37)	1.31 (1.41)	- 1.449	0.076
Sadness	0.45 (0.93)	0.80 (1.33)	1.392	0.084
Nervousness	0.90 (1.24)	0.78 (1.28)	- 0.447	0.328
Emotional	1.10 (1.36)	1.02 (1.29)	- 0.271	0.394
Numbness	0.28 (0.85)	0.13 (0.34)	- 0.988	0.164
Slowed down	1.00 (1.24)	0.60 (0.92)	- 1.704	0.046
Mentally foggy	0.88 (1.27)	0.67 (1.00)	- 0.847	0.200
Concentration	1.90 (1.69)	1.47 (1.50)	- 1.251	0.107
Remembering	0.73 (1.30)	1.13 (1.49)	1.340	0.092
Visual Problems	0.33 (0.69)	0.38 (0.89)	0.303	0.382

Note. No '*p*' values reached significance ( $p < .05$ ) with Bonferroni's adjustment

In the comparison of individual symptoms *within* the Contact group, between the 2+ Concussion versus 0 Concussion groups on the ImPACT Postconcussion Symptom Scale at *post-season* (Table 8.2.7.2), there were no significant results.

**Table 8.2.7.2**  
**Post-season *within* Contact group comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussion groups on the ImPACT Postconcussion Symptom Scale**

Symptoms	2+ Concussions (n = 29) Mean (SD)	0 Concussions (n = 38) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Total Symptom	20.96 (19.60)	20.16 (16.78)	- 0.180	0.429
Headache	0.93 (1.36)	0.68 (1.25)	- 0.755	0.227
Nausea	0.71 (1.08)	0.39 (0.68)	- 1.374	0.089
Vomiting	0.39 (0.83)	0.08 (0.27)	- 1.922	0.032
Balance problems	0.50 (0.79)	0.37 (0.85)	- 1.638	0.263
Dizziness	0.93 (1.15)	0.66 (0.88)	- 1.083	0.142
Fatigue	1.96 (1.64)	1.79 (1.74)	- 0.413	0.341
Difficult falling asleep	1.21 (1.26)	1.03 (1.55)	- 0.526	0.301
Sleeping more	1.04 (1.50)	0.53 (0.92)	- 1.587	0.060
Sleeping less	1.32 (1.61)	1.84 (2.05)	1.156	0.126
Drowsiness	1.68 (2.78)	1.03 (1.55)	- 1.216	0.115
Sensitivity to light	0.68 (1.34)	0.68 (1.21)	0.018	0.493
Sensitivity to noise	0.57 (1.07)	0.53 (1.03)	- 0.173	0.432
Irritability	1.46 (1.62)	1.53 (1.43)	0.165	0.435
Sadness	0.75 (1.11)	0.89 (1.41)	0.450	0.327
Nervousness	0.61 (0.83)	0.82 (1.16)	0.810	0.211
Emotional	1.25 (1.60)	0.13 (1.63)	- 0.294	0.385
Numbness	0.43 (0.92)	0.29 (1.09)	- 0.547	0.293
Slowed down	1.21 (1.50)	1.34 (1.44)	0.350	0.364
Mentally foggy	1.00 (1.49)	1.24 (1.38)	0.665	0.254
Concentration	1.25 (1.74)	1.71 (1.37)	1.204	0.117
Remembering	0.68 (1.16)	1.11 (1.52)	1.242	0.110
Visual Problems	0.39 (0.96)	0.50 (1.23)	0.384	0.351

Note. No 'p' values reached significance ( $p < .05$ ) with Bonferroni's adjustment

Overall, there was a marginally predominant trend in the direction of the 2+ Concussion group reportedly being worse in respect of more symptoms than the 0 Concussion group (13 out of the 22 symptoms, with the exception of sleeping less, irritability, sadness, nervousness, slowed down, mentally foggy, concentration, remembering and visual problems).

An overview of the pre- and post-season analyses for the *within* Contact group comparison of symptoms, between the 2+ Concussion versus 0 Concussion groups on the ImPACT Postconcussion Symptom Scale (Tables 8.2.7.1 and 8.2.7.2), reveals that the results at *post-season* replicate the results at *pre-season* in respect of the absence of any significant effects. In addition, there is a trend in the direction of the 2+ Concussion group reportedly being worse in respect of more symptoms than the 0 Concussion group, which is more predominant at the pre-season than at the post-season occasion (16 out of 22 versus 13 out of 22 symptoms, respectively).

#### **8.2.8 Pre- and post-season *within* Contact group comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussion groups on the Postconcussion Symptom Questionnaire (see Tables 8.2.8.1 and 8.2.8.2, pp. 281-282)**

(As indicated in the Methodology, the Postconcussion Symptom Questionnaire comprised 26 symptoms at pre-season and was reduced to nine symptoms at the post-season interval)

In the comparison of symptoms *within* the Contact sports group, between the 2+ Concussion versus 0 Concussion groups on the PCS Questionnaire at *pre-season* (Table 8.2.8.1), there were seven significant results. These were for the total symptom score ( $p = 0.002$ ) and the individual symptoms of fatigue,  $p = 0.003$ ; difficulty falling asleep,  $p = 0.009$ ; drowsiness,  $p = 0.003$ ; sensitivity to noise,  $p = 0.015$ ; irritability,  $p = 0.001$  and nervousness,  $p = 0.019$ , all in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group. Overall, there was a predominant trend in the direction of 2+ Concussion group reportedly being worse in respect of more symptoms than the 0 Concussion group (23 out of the 26 symptoms, with the exception of headache, balance problems and speech problems).

**Table 8.2.8.1**  
**Pre-season *within* Contact group comparisons of total and individual symptoms between the 2+ Concussion versus 0 Concussion groups on the Postconcussion Symptom Questionnaire**

Symptoms	2+ Concussions (n = 38) Mean (SD)	0 Concussions (n = 36) Mean (SD)	<i>t</i> -value	<i>p</i> -value
Total Symptom	20.03 (12.13)	11.61 (11.41)	- 3.070	0.002 <sup>††</sup>
Headache	0.71 (1.23)	0.78 (1.17)	0.241	0.406
Nausea	0.26 (0.69)	0.06 (0.23)	- 1.764	0.042
Vomiting	0.13 (0.48)	0.00 (0.00)	- 1.708	0.048
Poor appetite	0.50 (0.92)	0.17 (0.45)	- 1.993	0.026
Balance problems	0.13 (0.48)	0.80 (0.36)	- 0.486	0.314
Dizziness	0.55 (0.95)	0.25 (0.60)	- 1.644	0.053
Fatigue	2.42 (1.52)	1.44 (1.38)	2.889	0.003 <sup>††</sup>
Difficult falling asleep	1.37 (1.60)	0.64 (0.90)	- 2.433	0.009 <sup>†</sup>
Sleeping more	0.58 (1.22)	0.42 (0.97)	- 0.631	0.265
Sleeping less	1.47 (1.59)	0.86 (1.36)	- 1.787	0.039
Drowsiness	0.95 (1.29)	0.28 (0.62)	- 2.868	0.003 <sup>††</sup>
Sensitivity to light	0.24 (0.75)	0.17 (0.61)	- 0.440	0.331
Sensitivity to noise	0.37 (0.91)	0.03 (0.17)	- 2.261	0.015 <sup>†</sup>
Difficulty hearing	0.45 (1.16)	0.28 (0.66)	- 0.770	0.222
Irritability	1.71 (1.27)	0.78 (1.07)	- 3.402	0.001 <sup>††</sup>
Aggression	1.81 (1.52)	0.72 (1.09)	- 1.509	0.068
Sadness	0.61 (1.10)	0.39 (0.90)	- 0.920	0.181
Nervousness	0.95 (1.25)	0.39 (0.99)	- 2.132	0.019 <sup>†</sup>
Emotional	1.24 (1.42)	0.78 (1.38)	- 1.410	0.082
Numbness	0.16 (0.44)	0.06 (0.23)	- 1.286	0.105
Slowed down	0.63 (0.97)	0.33 (0.86)	- 1.395	0.084
Mentally foggy	0.66 (0.91)	0.42 (0.10)	- 1.089	0.140
Concentration	1.63 (1.32)	1.11 (1.17)	- 1.791	0.039
Remembering	0.76 (1.03)	0.69 (0.98)	- 0.294	0.385
Visual problems	0.32 (0.81)	0.25 (0.65)	- 0.385	0.351
Speech problems	0.05 (0.23)	0.25 (0.69)	1.631	0.055

Note. <sup>†</sup>*p* < .05, one-tailed. <sup>††</sup>*p* < .01, one-tailed, with Bonferroni's adjustments



In the comparison of individual symptoms *within* the Contact group, between the 2+ Concussions versus 0 Concussion groups on the PCS Questionnaire at *post-season* (see Table 8.2.8.2), there was one significant result (aggression,  $p = 0.019$ ), in the direction of the 2+ Concussion group reportedly being worse than the 0 Concussion group. Overall, there was a strongly predominant trend in the same direction, for the 2+ Concussion group to be worse in respect of more symptoms than the 0 Concussion group (nine out of the nine symptoms).

An overview of the pre- and post-season analyses for the *within* Contact group comparison of symptoms between the 2+ Concussion versus 0 Concussion groups on the PCS Questionnaire (Tables 8.2.8.1 and 8.2.8.2), reveals that the results at *post-season* do replicate the results at *pre-season* in respect of at least some significance attained at both intervals, in the direction of the 2+ Concussion group being worse than the 0 Concussion group. This occurred in combination with a highly consistent trend in the same direction, for the 2+ Concussion group reportedly being worse in respect of more symptoms than the 0 Concussion group, at both pre- and post-season occasions (23 out of 26 versus 9 out of nine symptoms, respectively).

**Table 8.2.8.2**  
**Post-season *within* Contact group comparisons of total and individual symptoms between 2+ Concussion versus 0 Concussion groups on the Postconcussion Symptom Questionnaire**

Symptoms	2+ Concussions (n = 27) Mean (SD)	0 Concussions (n = 36) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Total Symptom	10.44 (8.81)	7.58 (7.40)	- 1.400	0.084
Headache	0.74 (1.20)	0.64 (1.05)	- 0.360	0.360
Poor appetite	0.85 (1.35)	0.69 (1.09)	- 0.512	0.306
Mentally foggy	1.33 (1.30)	1.00 (1.29)	- 1.013	0.156
Difficulty hearing	0.74 (1.10)	0.47 (0.85)	- 1.099	0.138
Concentration	1.74 (1.61)	1.47 (1.38)	- 0.711	0.240
Remembering	1.30 (1.24)	1.03 (1.16)	- 0.885	0.190
Speech problems	0.74 (1.10)	0.28 (0.62)	- 1.976	0.028
Aggression	1.52 (1.42)	0.78 (1.33)	- 2.119	0.019 <sup>†</sup>
Slowed down	1.48 (1.50)	1.22 (1.48)	- 1.685	0.248

Note. <sup>†</sup>  $p < .05$ , one-tailed, with Bonferroni's adjustment

### **8.2.9 Synthesis of findings for pre- and post-season *within* Contact group comparisons of symptoms between the 2+ Concussion versus 0 Concussion groups**

The findings on the pre- and post-season comparisons *within* the Contact group, between the 2+ and 0 Concussion groups on the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire reveal instances of significantly higher reporting of symptoms for the 2+ Concussion group at pre-season and at post-season, exclusively on the Postconcussion Symptom Questionnaire (i.e. there were no significant effects revealed on the ImPACT Postconcussion Symptom Scale). There were no significant results in the opposite direction (i.e. the 0 Concussion group reportedly being worse than the 2+ Concussion group). Finally, in respect of both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire the trends analyses reflect a consistent trend in the direction of the 2+ Concussion group reportedly being worse in respect of more symptoms than the 0 Concussion group, across all analyses.

### **8.2.10 Comparison of significant symptom outcome on all test occasions and across the two postconcussion inventories (see Tables 8.2.10.1, 8.2.10.2 and 8.2.10.3, pp. 285-287 and Figures 1, 2 and 3, pp. 288-289)**

On the between groups analyses (see Tables 8.2.10.1 and 8.2.10.2) of the Contact versus Non-Contact groups and the 2+ Concussion versus 0 Concussion groups, it is apparent that the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire provide relatively strong validation for each other in that in all analyses at both pre- and post-season there is significantly worse symptom outcome revealed for the Concussion and 2+ Concussion groups and no significantly worse outcome revealed for the Non-Concussion and 0 Concussion groups. In terms of the Contact versus Non-Contact analyses, there were four overlapping significant results at pre-season in respect of both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire in terms of the total symptom score and the symptoms of fatigue, sleeping less, and nervousness, with no overlapping significant results occurring at post-season. In terms of the 2+ Concussion versus the 0 Concussion analyses there were six overlapping significant results at pre-season in respect of both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire in terms of the total symptom score and the symptoms of dizziness, fatigue,

drowsiness, irritability, and concentration, with no overlapping significant results occurring at post-season.

Compared with the between groups analyses (Tables 8.2.10.1 and 8.2.10.2), where there appears to be a relatively clear overall replication of findings for both inventories (i.e. higher symptom reporting for the Contact and 2+ Concussion groups and nothing for the Non-Contact and 0 Concussion groups), on the *within* Contact analyses (Table 8.2.10.3), this is not apparent. In this case (Table 8.2.10.3) only the Postconcussion Symptom Questionnaire revealed any significant effects mainly at the pre-season interval (i.e. total symptom score, fatigue, difficulty falling asleep, drowsiness, sensitivity to noise, irritability and nervousness) and only one significant symptom (aggression) at post-season.

Perusal of Tables 8.2.10.1, 5.2.10.2, and 8.2.10.3 (pp. 285-287) and Figures 1, 2, and 3 (pp. 288-289) illustrate that the Postconcussion Symptom Questionnaire appears to have been substantially more sensitive to raised symptoms amongst the Contact and 2+ Concussion groups than the ImPACT Symptom Scale. Furthermore, the Postconcussion Symptom Questionnaire which was reduced from 26 symptoms at the pre-season interval to only nine symptoms at the post-season interval appears to have been particularly sensitive at the post-season interval in the two between groups analyses (Table 5.2.10.1 and 5.2.10.2, and Figure 1 and 2), where eight out of nine and six out of nine symptoms, respectively reached significance.

Finally, perusal of Figures 1, 2 and 3 (pp. 288-289), clearly demonstrates that all significant results for the between groups analyses in respect of both sports groups (Contact versus Non-Contact) and concussion groups (2+ versus 0 Concussion), at both the pre- and post-season intervals, were in the direction of the Contact group reporting more symptoms than the Non-Contact group and the 2+ Concussion group reporting more symptoms than the 0 Concussion group. There were no instances in the opposite direction of the Non-Contact and 0 Concussion groups reporting more symptoms than the Contact and 2+ Concussion groups.

Table 8.2.10.1

Overall comparison of significantly worse symptom outcome for the Contact versus Non-Contact groups on the ImPACT Symptom Scale and the Postconcussion Symptom Questionnaire at both pre and post-season

Symptoms	PRE-SEASON				POST-SEASON			
	CONTACT		NON		CONTACT		NON	
	IPSS	PCQ	IPSS	PCQ	IPSS	PCQ	IPSS	PCQ
Total Symptom	†	†				††		
Headache		††				†		
Nausea								
Vomiting								
Balance problems					†			
Dizziness					†			
Fatigue	†	†			†			
Diff. falling asleep		†						
Sleeping more								
Sleeping less	†	†			†			
Drowsiness								
Sensitivity to light								
Sensitivity to noise								
Irritability								
Sadness					†			
Nervousness	††	††			††			
Emotional		††			††			
Numbness								
Slowed down						††		
Mentally foggy						†		
Concentration						††		
Remembering						††		
Visual Problems	†							
Speech problems <sup>1</sup>						††		
Poor appetite <sup>1</sup>						†		
Diff. hearing <sup>1</sup>		†						
Aggression <sup>1</sup>		†				††		

Note. <sup>1</sup> Symptoms included on the Postconcussion Symptom Questionnaire

Note. <sup>2</sup> † Symptoms that consistently appeared across both inventories and across both seasons

Note. † p < .05, one-tailed. †† p < .01, one-tailed

Table 8.2.10.2

Overall comparison of significantly worse symptom outcome for the 2+ versus 0 Concussion groups on the ImPACT Symptom Scale and the Postconcussion Symptom Questionnaire at both pre and post-season

Symptoms	PRE-SEASON				POST-SEASON			
	2+ CONC		0 CONC		2+ CONC		0 CONC	
	IPSS	PCQ	IPSS	PCQ	IPSS	PCQ	IPSS	PCQ
Total Symptom	†	††				††		
Headache								
Nausea								
Vomiting								
Balance problems	†							
Dizziness	†	†			†			
Fatigue	††	††						
Diff. falling asleep		†						
Sleeping more								
Sleeping less		†						
Drowsiness	†	††						
Sensitivity to light								
Sensitivity to noise		†						
Irritability	††	††						
Sadness								
Nervousness		††						
Emotional		†			†			
Numbness								
Slowed down	†					†		
Mentally foggy						†		
Concentration	†	††				†		
Remembering						†		
Visual Problems								
Speech problems <sup>1</sup>						†		
Poor appetite <sup>1</sup>								
Diff. hearing <sup>1</sup>								
Aggression <sup>1</sup>		†				††		

Note. <sup>1</sup> Symptoms included on the Postconcussion Symptom Questionnaire

Note.<sup>2</sup> † Symptoms that consistently appeared across both inventories and across both seasons

Note. †  $p < .05$ , one-tailed. ††  $p < .01$ , one-tailed

Table 8.2.10.3

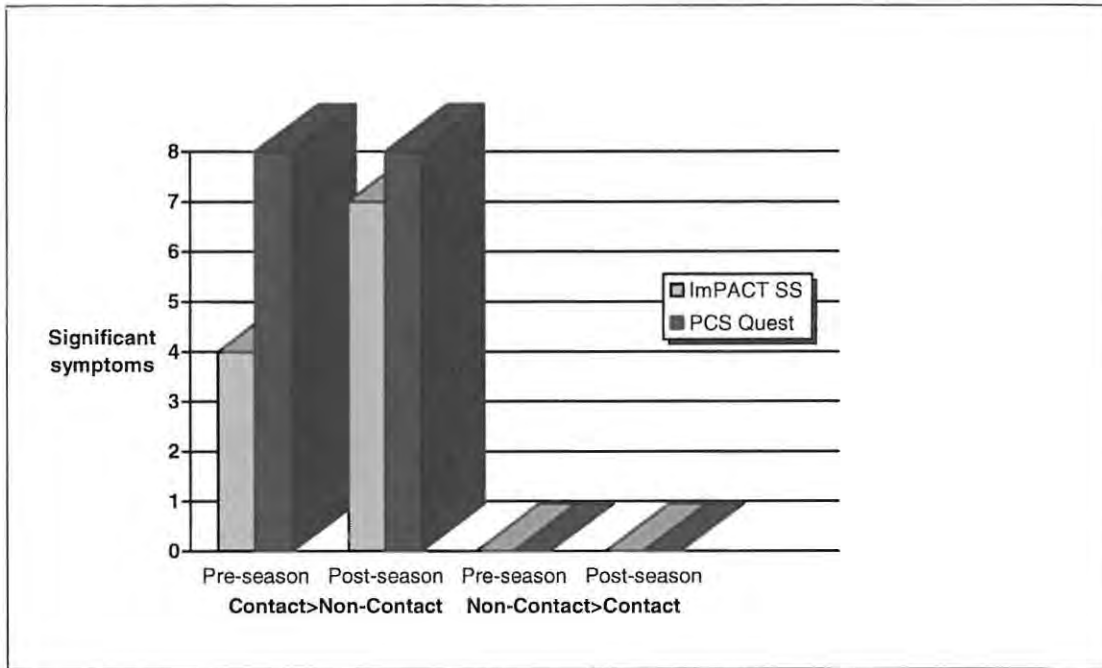
Overall comparison of significantly worse symptom outcome for the 2+ versus 0 Concussion groups *within* the Contact group on the ImPACT Symptom Scale and the Postconcussion Symptom Questionnaire at both pre and post-season

Symptoms	PRE-SEASON				POST-SEASON			
	2+ CONC		0 CONC		2+ CONC		0 CONC	
	IPSS	PCQ	IPSS	PCQ	IPSS	PCQ	IPSS	PCQ
Total Symptom		††						
Headache								
Nausea								
Vomiting								
Balance problems								
Dizziness								
Fatigue		††						
Diff. falling asleep		†						
Sleeping more								
Sleeping less								
Drowsiness		††						
Sensitivity to light								
Sensitivity to noise		†						
Irritability		††						
Sadness								
Nervousness		†						
Emotional								
Numbness								
Slowed down								
Mentally foggy								
Concentration								
Remembering								
Visual Problems								
Speech problems <sup>1</sup>								
Poor appetite <sup>1</sup>								
Diff. hearing <sup>1</sup>								
Aggression <sup>1</sup>						†		

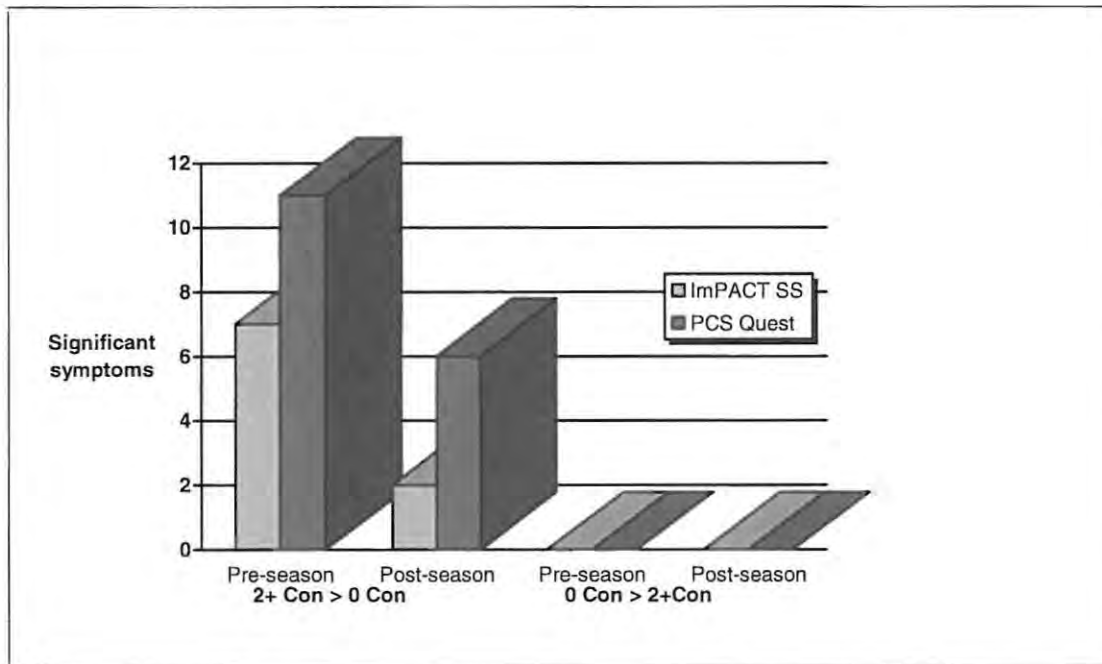
Note. <sup>1</sup> Symptoms included on the Postconcussion Symptom Questionnaire

Note. †  $p < .05$ , one-tailed. ††  $p < .01$ , one-tailed

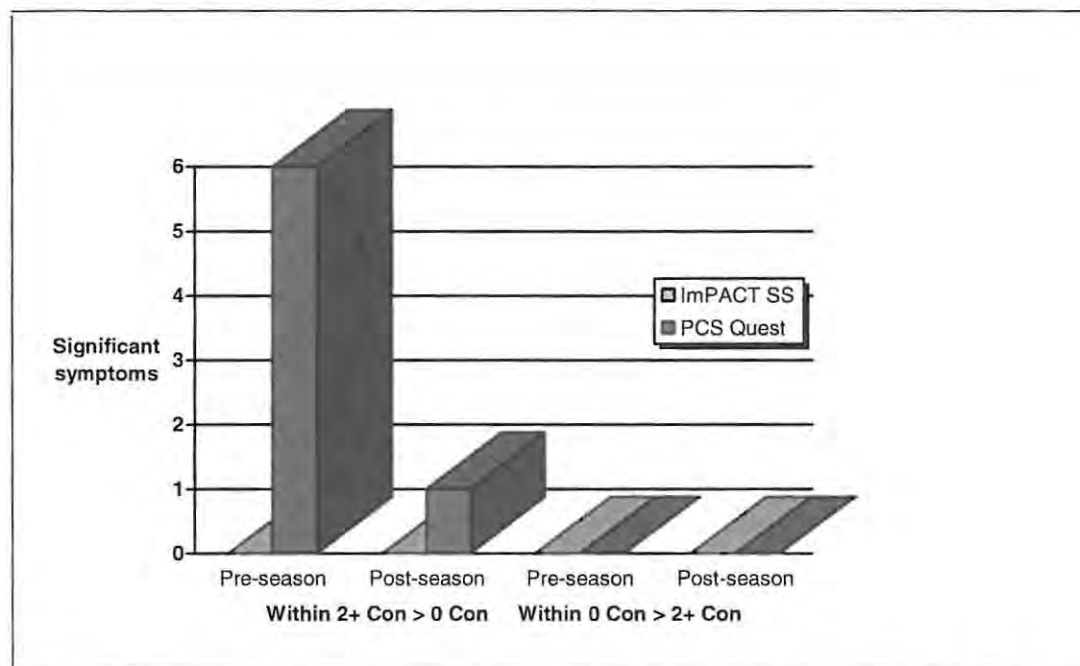
**Figure 1.** All significant results on the ImPACT Symptom Scale and the Postconcussion Symptom Questionnaire at pre and post-season indicating Contact > Non-Contact or Non-Contact > Contact groups



**Figure 2.** All significant results on the ImPACT Symptom Scale and the Postconcussion Symptom Questionnaire at pre and post-season indicating 2+ Concussion > 0 Concussion or 0 Concussion > 2+ Concussion groups



**Figure 3.** All significant results on the ImPACT Symptom Scale and the Postconcussion Symptom Questionnaire at pre and post-season *within* the Contact group indicating 2+ Concussion > 0 Concussion or 0 Concussion > 2+ Concussion groups



### 8.3 DEPENDENT T-TEST ANALYSES BETWEEN CONTACT GROUP PRE VERSUS POST-SEASON AND NON-CONTACT GROUP PRE VERSUS POST-SEASON

This subsection reports on all dependent t-test analyses between Contact groups pre-season versus post-season and Non-Contact group pre-season versus post-season for all measures, including all cognitive scores on ImPACT and the paper and pencil tests as well as for the individual symptoms on both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire.

#### 8.3.1 Dependent t-test analyses of all measures between the Contact group pre versus post-season and the Non-Contact group pre- versus post-season (see Table 8.3.1, p.290)

In the comparison of the Contact group between pre- and post-season intervals (Table 8.3.1), there were two results that were significant (Digit Symbol Substitution test,  $p = 0.000$  and Digit Symbol Substitution Incidental Recall- Immediate,  $p = 0.025$ ), in the direction of the Contact

group performing better at post-season than at the pre-season interval. Overall, there was a predominant trend in the same direction with the Contact group performing better at post-season than at pre-season (five out of the seven measures, with the exception of Visual Motor Speed and the Digit Symbol Substitution Incidental Recall-Delayed).

**Table 8.3.1**  
Dependent t-test analyses of all cognitive measures between the Contact group pre-versus post-season and Non-Contact group pre versus post-season

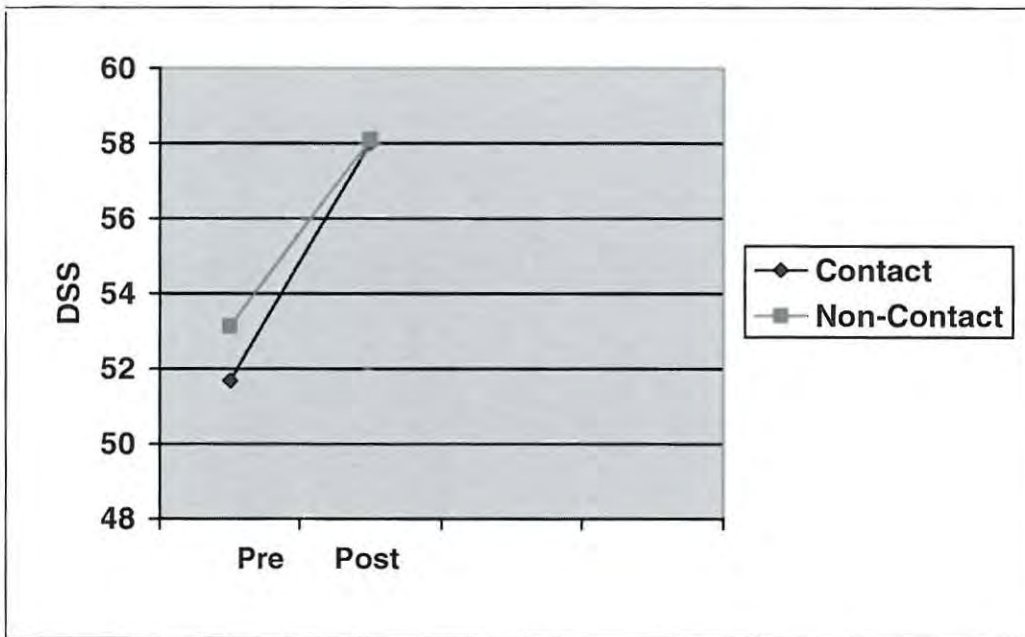
Cognitive Measures	Contact (n = 88) Mean (SD)	<i>t-value</i>	<i>p-value</i>	Non-Contact (n = 62) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Pre Verbal Memory	85.45 (8.89)	- 1.542	0.127	88.05 (8.01)	0.600	0.551
Post Verbal memory	87.06 (10.12)			87.31 (9.67)		
Pre Visual Memory	77.66 (12.01)	- 1.285	0.202	79.47 (11.26)	-0.708	0.481
Post Visual Memory	79.39 (12.24)			80.63 (12.41)		
Pre VMS	38.02 (6.74)	0.435	0.664	37.27 (7.62)	-2.625	0.011*
Post VMS	37.77 (6.23)			39.03 (6.92)		
Pre RXT	0.60 (7.24)	0.157	0.875	0.58 (5.59)	0.334	0.739
Post RXT	0.59 (7.60)			0.58 (5.51)		
Pre DSS	51.68 (11.23)	- 4.593	0.000**	53.14 (10.00)	-4.976	0.000**
Post DSS	58.01 (10.60)			58.11 (9.81)		
Pre DSSIR-Immed	7.81 (1.47)	- 2.286	0.025*	7.67 (1.69)	-1.590	0.117
Post DSSIR-Immed	8.22 (1.27)			8.08 (1.74)		
Pre DSSIR-Del	7.41 (1.84)	0.153	0.879	7.02 (2.03)	-2.318	0.024*
Post DSSIR- Del	7.38 (1.94)			7.56 (1.96)		

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

In the comparison of the Non-Contact group between pre- and post-season intervals (Table 8.3.1), there were three results that were significant (Visual Motor Speed,  $p = 0.011$ ; Digit Symbol Substitution test,  $p = 0.000$  and Digit Symbol Substitution Incidental Recall-Delayed,  $p = 0.024$ ), all in the direction of the Non-Contact group performing better at post-season than at pre-season. Overall there was a predominant trend in the same direction, with the Contact group performing better at post-season than at pre-season (six out of the seven measures, with the exception of Verbal Memory).

An overview of the analyses for the dependent t-test comparison of all measures between the Contact group pre-season versus post-season and the Non-Contact group pre-season versus post-season (Table 8.3.1), reveals that the results for the Contact group replicate the results for the Non-Contact group, in respect of a strong trend in the direction of both Contact and Non-Contact groups performing better at post-season than at pre-season, albeit only two tests reached significance for the Contact group (Digit Symbol Substitution test and the Digit Symbol Substitution Incidental Recall-Immediate) versus three tests for the Non-Contact group (Visual Motor Speed, Digit Symbol Substitution test and the Digit Symbol Substitution Incidental Recall- Delayed). Reference to Figures 4 to 7 highlights the following: There is significant improvement at post-season versus pre-season for both the Contact and Non-Contact groups on the Digit Symbol Substitution test (Figure 4), and similarly the significantly improved scores evident for the Contact group are replicated as a strong trend for the Non-Contact group on the Digit Symbol Substitution Incidental Recall-Immediate ( $p = 0.117$ ) (Figure 5). However, the significantly improved scores for the Visual Motor Speed and the Digit Symbol Substitution Incidental Recall- Delayed, evident for the Non-Contact group are entirely absent for the Contact group where the opposite trend of worsening scores at post-season versus pre-season is revealed (Figures 6 and 7 respectively).

*Figure 4.* Contact (pre- vs post-season) versus Controls (pre- vs post-season) for the Digit Symbol Substitution test



*Figure 5.* Contact (pre- vs post-season) versus Controls (pre- vs post-season) for the Digit Symbol Substitution Incidental Recall- Immediate

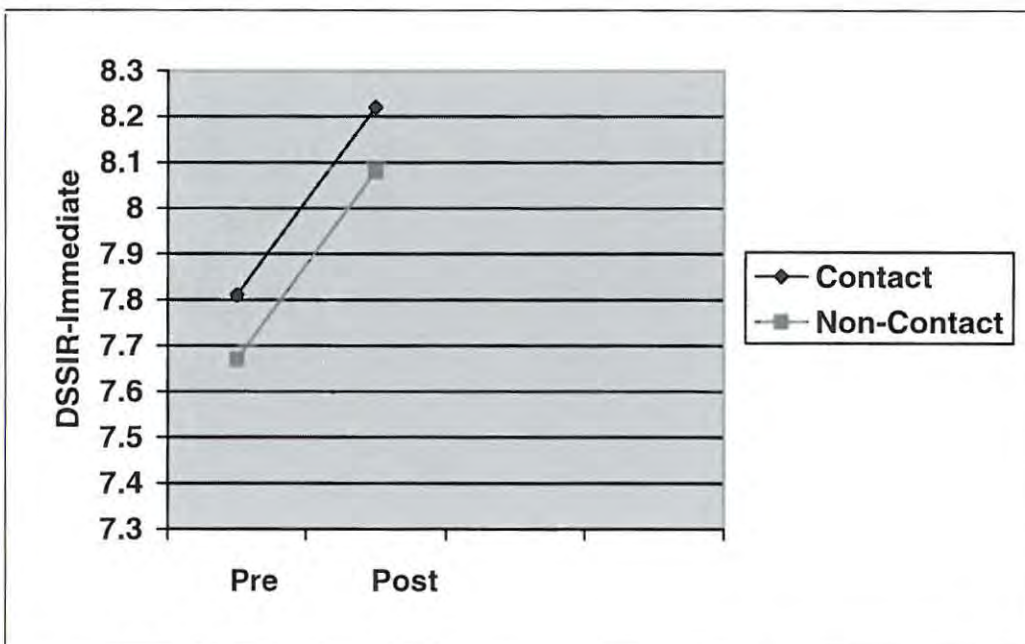


Figure 6. Contact (pre- vs post-season) versus Controls (pre- vs post-season) for Visual Motor Speed

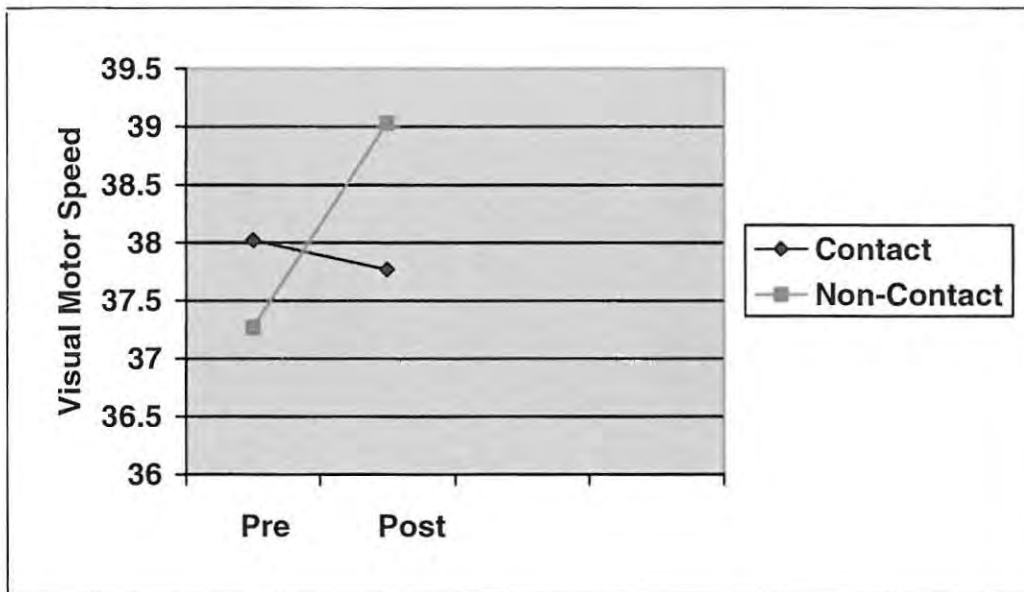
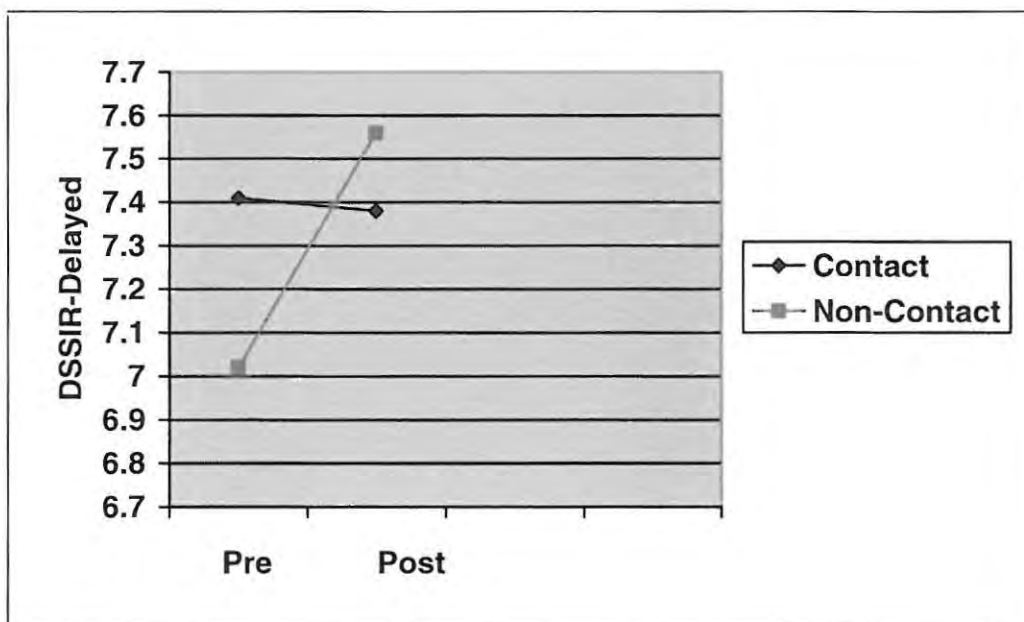


Figure 7. Contact (pre- vs post-season) versus Controls (pre- vs post-season) for the Digit Symbol Substitution Incidental Recall- Delayed



### **8.3.2 Dependent t-test analyses of individual symptoms between the Contact group pre-versus post-season and the Non-Contact group pre- versus post-season on the ImPACT Postconcussion Symptom Scale (see Table 8.3.2, p. 295)**

In the comparison of the Contact group between pre- and post-season intervals (Table 8.3.2) on the ImPACT Postconcussion Symptom Scale, the total symptom scores were not significantly different and for individual symptoms there were four results that were significant (nausea,  $p = 0.030$ ; balance problems,  $p = 0.015$  and slowed down,  $p = 0.002$  and mentally foggy,  $p = 0.030$ ), in the direction of the Contact group reportedly being worse at post-season than at pre-season. Overall, there was a marginally predominant trend in the same direction, with the Contact group reportedly being worse in respect of reporting more symptoms at post-season than at pre-season (14 out of the 22 symptoms, with the exception of difficulty falling asleep, sleeping more, sleeping less, irritability, nervousness, concentration, remembering and visual problems).

In the comparison of the Non-Contact group between pre- and post-season intervals (Table 8.3.2) on the ImPACT Postconcussion Symptom Scale, the total symptom scores were not significantly different and for individual symptoms there were four results that were significant (nausea,  $p = 0.032$ ; sleeping more,  $p = 0.006$ ; sensitivity to noise,  $p = 0.000$  and mentally foggy,  $p = 0.049$ ), in the direction of the Non-Contact group reportedly being worse at post-season than at pre-season. Overall, there was a predominant trend in the same direction, with the Non-Contact group reportedly being worse in respect of reporting more symptoms at post-season than at pre-season (17 out of the 22 symptoms, with the exception of sleeping less, sadness, nervousness, emotional and remembering).

An overview of the analyses for the dependent t-test comparison of individual symptoms between the Contact group pre-season versus post-season and the Non-Contact group pre-season versus post-season on the ImPACT Postconcussion Symptom Scale (Table 8.3.2), reveals that the results for the Contact group are similar to the results for the Non-Contact group, in that each yielded four significant effects in the direction of more symptoms being presented at post-season than at pre-season, two of these in each case being for the same symptoms (nausea and mentally foggy). The predominant trend also was for symptoms to get worse at post-season, especially in the Non-Contact group.

**Table 8.3.2**  
**Dependent t-test analyses of individual symptoms between Contact group**  
**Pre- versus post-season and Non-contact sports group pre- versus post-season**  
**on the ImPACT Postconcussion Symptom Scale**

ImPACT Symptom Scale	Contact (n = 88) Mean (SD)	<i>t</i> -value	<i>p</i> -value	Non-Contact (n = 62) Mean (SD)	<i>t</i> -value	<i>p</i> -value
Pre Total Symptom	16.61 (14.69)	- 1.342	0.183	13.08 (11.78)	- 1.095	0.278
Post Total Symptom	18.74 (17.15)			14.79 (13.64)		
Pre Headache	0.72 (1.11)	- 0.716	0.476	0.57 (1.03)	- 0.482	0.631
Post Headache	0.84 (1.36)			0.63 (1.11)		
Pre Nausea	0.24 (0.66)	- 2.213	0.030*	0.14 (0.50)	- 2.196	0.032*
Post Nausea	0.46 (0.82)			0.30 (0.66)		
Pre Vomiting	0.13 (0.57)	- 0.615	0.540	0.05 (0.22)	- 1.350	0.182
Post Vomiting	0.17 (0.53)			0.14 (0.50)		
Pre Balance problems	0.18 (0.50)	- 2.475	0.015*	0.10 (0.35)	- 1.000	0.321
Post Balance problems	0.39 (0.77)			0.16 (0.48)		
Pre Dizziness	0.47 (0.82)	- 1.725	0.088	0.33 (0.72)	- 0.168	0.867
Post Dizziness	0.66 (0.94)			0.35 (0.81)		
Pre Fatigue	1.70 (1.44)	- 0.424	0.672	1.16 (1.31)	- 1.331	0.188
Post Fatigue	1.77 (1.66)			1.38 (1.33)		
Pre Diff. falling asleep	1.21 (1.45)	0.739	0.462	0.94 (1.38)	- 0.875	0.385
Post Diff. falling asleep	1.09 (1.48)			1.10 (1.49)		
Pre Sleeping more	0.82 (1.36)	0.364	0.717	0.38 (0.96)	- 2.827	0.006**
Post Sleeping more	0.76 (1.24)			0.79 (1.37)		
Pre Sleeping less	1.55 (1.66)	0.964	0.338	1.05 (1.40)	0.778	0.439
Post Sleeping less	1.36 (1.77)			0.89 (1.44)		
Pre Drowsiness	0.90 (1.37)	- 1.133	0.260	0.89 (1.18)	0.000	1.000
Post Drowsiness	1.15 (1.96)			0.89 (1.25)		
Pre Sensitivity to light	0.44 (0.96)	- 1.286	0.202	0.60 (1.13)	- 1.182	0.242
Post Sensitivity to light	0.62 (1.21)			0.76 (1.28)		
Pre Sensitivity to noise	0.34 (0.80)	- 1.118	0.267	0.21 (0.68)	- 3.833	0.000**
Post Sensitivity to noise	0.45 (0.95)			0.62 (0.97)		
Pre Irritability	1.39 (1.36)	0.418	0.677	1.06 (1.44)	- 0.241	0.811
Post Irritability	1.32 (1.49)			1.11 (1.36)		
Pre Sadness	0.54 (1.04)	- 1.096	0.276	0.57 (1.07)	1.000	0.321
Post Sadness	0.69 (1.16)			0.41 (0.78)		
Pre Nervousness	0.85 (1.35)	1.359	0.178	0.41 (0.78)	0.785	0.435
Post Nervousness	0.67 (0.97)			0.32 (0.71)		
Pre Emotional	0.80 (1.12)	- 1.696	0.093	0.70 (1.25)	0.845	0.402
Post Emotional	1.10 (1.51)			0.56 (1.15)		
Pre Numbness	0.15 (0.47)	- 1.540	0.127	0.21 (0.63)	- 0.293	0.771
Post Numbness	0.31 (0.94)			0.24 (0.69)		
Pre Slowed down	0.68 (1.01)	- 3.168	0.002**	0.84 (1.05)	- 0.212	0.833
Post Slowed down	1.16 (1.36)			0.87 (1.14)		
Pre Mentally foggy	0.74 (1.06)	- 2.213	0.030*	0.68 (1.08)	- 2.005	0.049*
Post Mentally foggy	1.08 (1.37)			1.02 (1.33)		
Pre Concentration	1.47 (1.51)	0.263	0.793	1.13 (1.31)	- 0.369	0.714
Post Concentration	1.43 (1.54)			1.19 (1.38)		
Pre Remembering	0.85 (1.36)	0.000	1.000	0.81 (1.16)	0.582	0.563
Post Remembering	0.85 (1.33)			0.73 (1.11)		
Pre Visual Problems	0.44 (0.86)	0.203	0.840	0.25 (0.54)	- 0.743	0.460
Post Visual Problems	0.41 (1.06)			0.33 (0.80)		

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

### **8.3.3 Dependent t-test analyses of individual symptoms between the Contact group pre-versus post-season and the Non-Contact group pre- versus post-season on the Postconcussion Symptom Questionnaire (see Table 8.3.3, p. 297)**

(Since only nine symptoms formed part of the post-season Postconcussion Symptom Questionnaire, the pre-post analysis was restricted to these nine symptoms)

In the comparison of the Contact group between pre- and post-season intervals (Table 8.3.3) on the Postconcussion Symptom Questionnaire, the total symptom scores were significantly different ( $p = 0.006$ ) and for the individual symptoms there were five results that were significant (appetite,  $p = 0.002$ ; slowed down,  $p = 0.000$ ; mentally foggy,  $p = 0.004$  remembering,  $p = 0.010$  and speech problems,  $p = 0.025$ ), in the direction of the Contact group reportedly being worse at post-season than at pre-season. Overall there was a predominant trend in the same direction with the Contact group reportedly being worse in respect of reporting more symptoms at post-season than at pre-season (seven out of the nine symptoms, with the exception of headache and aggression).

In the comparison of the Non-Contact group between pre- and post-season intervals (Table 8.3.3) on the Postconcussion Symptom Questionnaire, the total symptom scores were not significantly different ( $p = 0.221$ ) and for the individual symptoms there was only one result that was significant (difficulty hearing,  $p = 0.003$ ), in the direction of the Non-Contact group reportedly being worse at post-season than at pre-season. Overall, there was a predominant trend in the same direction, with the Non-Contact group reportedly being worse in respect of reporting more symptoms at post-season than at pre-season (seven out of the nine symptoms, with the exception of remembering and aggression).

An overview of the analyses for the dependent t-test comparison of symptoms between the Contact group pre- versus post-season and the Non-Contact group pre- versus post-season on the Postconcussion Symptom Questionnaire (Table 8.3.3), reveals that the results for the Contact group are similar to the results for the Non-Contact group, in respect of a predominant trend in the direction of both Contact and Non-Contact groups presenting with more symptoms at post-season than at pre-season. However, the total symptom score for the Contact group reveals a highly significant incidence of symptoms at the post-season interval that is not present for the Non-Contact group, including significantly worsening for five symptoms for the Contact group

at post-season, compared with worsening for only one symptom at post-season for the Non-Contact group.

**Table 8.3.3**

**Dependent t-test analyses of individual symptoms between Contact group pre- versus post-season and Non-Contact group pre- versus post-season on the Postconcussion Symptom Questionnaire**

PCS Questionnaire	Contact (n = 73) Mean (SD)	<i>t-value</i>	<i>p-value</i>	Non-Contact (n = 40) Mean (SD)	<i>t-value</i>	<i>p-value</i>
Pre Total Symptom	5.53 (4.87)	- 2.859	0.006**	4.33 (4.15)	- 1.243	0.221
Post Total Symptom	7.84 (7.48)			5.23 (5.01)		
Pre Headache	0.84 (1.20)	1.022	0.310	0.38 (0.74)	- 0.650	0.520
Post Headache	0.67 (1.12)			0.45 (0.78)		
Pre poor Appetite	0.27 (0.67)	- 3.152	0.002**	0.30 (0.72)	- 1.347	0.186
Post poor Appetite	0.73 (1.21)			0.50 (0.88)		
Pre Diff. Hearing	0.32 (0.81)	- 1.344	0.183	0.13 (0.34)	- 3.134	0.003**
Post Diff. Hearing	0.45 (0.83)			0.45 (0.71)		
Pre Slowed down	0.52 (0.90)	- 4.045	0.000**	0.48 (0.85)	- 1.955	0.058
Post Slowed down	1.23 (1.37)			0.73 (0.88)		
Pre Aggression	1.16 (1.40)	1.513	0.135	0.45 (0.68)	0.172	0.864
Post Aggression	0.92 (1.23)			0.43 (0.78)		
Pre Mentally foggy	0.58 (0.91)	- 3.018	0.004**	0.73 (1.06)	- 0.539	0.593
Post Mentally foggy	1.08 (1.28)			0.83 (1.04)		
Pre Concentration	1.15 (1.20)	- 1.520	0.133	0.93 (1.07)	- 0.260	0.797
Post Concentration	1.41 (1.37)			0.98 (1.03)		
Pre Remembering	0.55 (0.91)	- 2.636	0.010*	0.78 (1.03)	0.453	0.653
Post Remembering	0.93 (1.07)			0.70 (0.85)		
Pre Speech problems	0.15 (0.52)	- 2.287	0.025*	0.18 (0.45)	0.000	1.000
Post Speech problems	0.41 (0.83)			0.18 (0.39)		

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

#### **8.3.4 Synthesis of findings for individual symptoms between the Contact group pre- versus post-season and the Non-Contact group pre- versus post-season on the ImPACT Postconcussion Symptom Scale and Postconcussion Symptom Questionnaire**

The findings on the individual symptoms between the Contact group pre- versus post-season and the Non-Contact group pre- versus post-season on both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire reveal instances of significantly higher reporting of symptoms for both Contact and Non-Contact groups at the post-season interval than at the pre-season interval. Perusal of Tables 8.3.2 and 8.3.3 clearly illustrates the absence of any significant results in the opposite direction (i.e. the Contact and Non-Contact groups reportedly being worse at pre-season than at post-season). The trends analyses also reflect a consistent trend in the direction of both the Contact and Non-Contact groups reportedly being worse in respect of more symptoms at post-season than at pre-season, across all analyses. Finally, reference to Table 8.3.4.1 and Figure 8 reveals that the 22-item ImPACT Symptom Scale elicits an equal number of significant symptoms being worse at post-season for the Contact and Non-Contact groups (four and four symptoms, respectively). On the other hand the analysis using the brief nine item Postconcussion Symptom Questionnaire elicits a powerful indication of worsening for the Contact group at post-season compared with minimal worsening for the non-contact group at post-season (total symptom score and five out of the nine symptoms for the Contact group, versus only one symptom for the Non-Contact group).

Table 8.3.4.1

Overall comparison of significant symptom outcome for the Contact group, pre versus post-season, and the Non-Contact group pre versus post-season on the ImPACT Symptom Scale and the Postconcussion Questionnaire

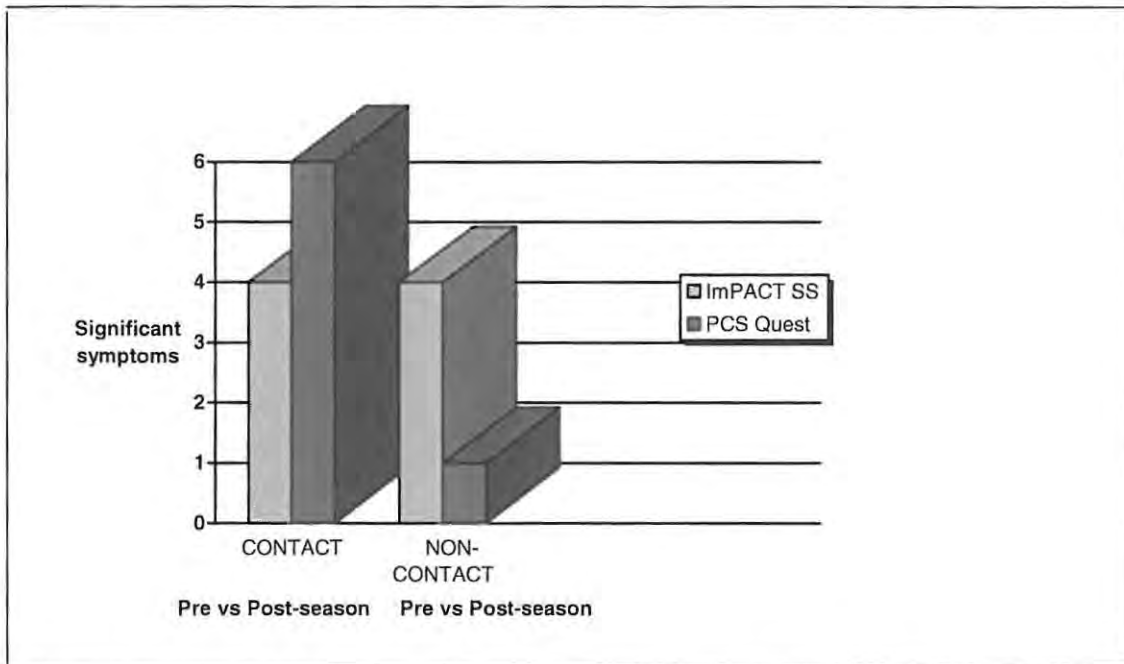
Symptoms	CONTACT Pre versus Post-season		NON-CONTACT Pre versus Post-season	
	IMPACT	PCQ <sup>1</sup>	IMPACT	PCQ
Total Symptom		††		
Headache				
Nausea	†		†	
Vomiting				
Balance problems	†			
Dizziness				
Fatigue				
Diff. falling asleep				
Sleeping more			††	
Sleeping less				
Drowsiness				
Sensitivity to light				
Sensitivity to noise			††	
Irritability				
Sadness				
Nervousness				
Emotional				
Numbness				
Slowed down	††	††		
Mentally foggy	†	††	†	
Concentration				
Remembering		†		
Speech problems <sup>2</sup>		†		
Poor appetite <sup>2</sup>		††		
Diff. hearing <sup>2</sup>				††
Aggression <sup>2</sup>				

Note. <sup>1</sup> This inventory comprised only nine symptoms (headache, poor appetite, difficulty hearing, slowed down, aggression, mentally foggy, concentration, remembering and aggression)

Note. <sup>2</sup> Symptoms exclusive to the Postconcussion Symptom Questionnaire

Note. <sup>3</sup> All significant values in the table were in the direction of more symptoms being reported at the post compared with the pre-season interval

**Figure 8.** All significant results on the ImPACT Symptom Scale and the Postconcussion Symptom Questionnaire indicating Contact > Non-Contact group at post- versus pre-season or Non-Contact > Contact group at post versus pre-season



## CHAPTER NINE

### DISCUSSION OF FINDINGS

#### 9.1 AIMS OF THE STUDY AND HYPOTHESIS

This research study sought to investigate the long-term neuropsychological effects of repetitive mild traumatic brain injury sustained as a consequence of participation in a contact sport amongst South African final year high school learners. For the younger athlete the potential risks associated with cumulative and repetitive concussions appears to be largely ignored in the literature since research investigating the effects of MTBI with respect to neurocognitive and postconcussive sequelae have mainly focussed on professional or elite athletes and amateur, collegiate or university athletes (e.g. Farace et al., 2003; Hinton-Bayre & Geffen, 2004; Jordan et al., 1997; Kutner et al., 2000; Macciocchi et al., 1996; Matser et al., 1999; McCrory et al., 2000). The rationale behind this choice of subject group by researchers was that these older athletes have experienced many years of contact play and hence have been more exposed to the effects of multiple concussive and sub-concussive events than the younger, less exposed high school athlete. Moreover, the level of play amongst professional and elite groups is generally more intense which further supports the fact that as a whole this group is more vulnerable to residual and cumulative concussive effects. This has been demonstrated in the studies reviewed earlier (see Chapter 6, pp. 138-191) where most significant persistent deficits were in evidence for the higher levels of play, including professional, elite and even amateur and collegiate athletes relative to the high school athlete (e.g. Barnes et al., 1998; Casson et al., 1984; Collins, Grindel et al., 1999; Downs & Abwender, 2002; Drew et al., 1986; Guskiewicz et al., 2005; Heilbronner et al., 1991; Jordan et al., 1997; Jordan, Matser et al., 1996; Kaste et al., 1982; Kutner et al., 2000; Levin, Lippold, et al., 1987; Maddocks & Saling, 1996; Matser et al., 1998, 1999, 2001; McLatchie et al., 1987; Murelius & Haglund, 1991; Ross et al., 1987; Rutherford et al., 2005; Tysvaer & Lochen, 1991; Shuttleworth-Edwards et al., 2004).

Recently, there has been an increase in the number of studies investigating the effects of concussion amongst the high school athletes (age range 13 to 18 years), particularly within the last three years (e.g. Barr, 2003; Collins, Field, et al., 2003; Field et al., 2003; Lovell et al., 2003, 2004; Iverson et al., 2004a, 2004b, 2006; McClincy et al., 2006; Mihalik et al., 2005; Moser et

al., 2005; Pellman et al., 2006; Stephens et al., 2005; Webbe & Ochs, 2003; Witol & Webbe, 2003). To the author's knowledge, there are no studies investigating the effects of concussion amongst high school athletes in respect of the contact sports of Australian Football League and Rugby League. However, there are a few studies that have investigated such effects on this adolescent sports population in the contact sports of boxing, ice hockey, soccer and Rugby Union, where one study has been reported in boxing (Ramirez, 1998) and one in ice hockey (Ferguson et al., 1999), three in soccer (Stephen et al., 2005; Webbe & Ochs, 2003; Witol & Webbe, 2003) and one in Rugby Union (Shuttleworth-Edwards et al., 2004). The majority of studies (comprising a total of 15 studies) investigating the effects of concussion amongst high school athletes have employed American football athletes (Barr, 2003; Collins, Field, et al., 2003; Field et al., 2003; Guskiewicz et al., 2000; Iverson et al., 2002, 2004a, 2004b, 2006; Lovell et al., 2003, Lovell, Collins, Iverson, et al., 2004; McClincy et al., 2006; Mihalik et al., 2005; Moser et al., 2005; Pellman et al., 2006; Wilberger et al., 1991), although eight of these studies have also included collegiate or university and professional athletes in their samples (Field et al., 2003; Guskiewicz et al., 2000; Iverson et al., 2002, 2004a, 2006; McClincy et al., 2006; Mihalik et al., 2005; Pellman et al., 2006). Moreover, in the one ice hockey study reported on above (Ferguson et al., 2004), both high school and collegiate athletes were investigated together in one sample. This was also the case with two of the three soccer studies where high school athletes were incorporated together with collegiate athletes in one study (Webbe & Ochs, 2003) and high school athletes incorporated with amateur and professional athletes in another study (Witol & Webbe, 2003). Although the Rugby Union study investigated the effects of concussion at three different levels of play (high school, university and professional), all these analyses were conducted separately for each level of play (Shuttleworth-Edwards et al., 2004).

Of all the studies employing high school athletes, five studies investigated the chronic effects following MTBI only, consisting of one ice hockey study (Ferguson et al., 1999), one soccer study (Stephens et al., 2005), two American football studies (Moser et al., 2005; Iverson et al., 2006), and one Rugby Union study (Shuttleworth-Edwards et al., 2004). Eight studies investigated the acute effects following MTBI, consisting of one boxing study (Ramirez, 1998), one soccer study (Webbe & Ochs, 2003) and six American football studies (Collins, Field, et al., 2003; Field et al., 2003; Guskiewicz et al., 2000, Iverson et al., 2004b, Mihalik et al., 2005; Pellman et al., 2006). Seven studies investigated both the acute and chronic effects (Barr, 2003;

Iverson et al., 2002, 2004a; Lovell et al., 2003; Lovell, Collins, Iverson et al., 2004; McClincy et al., 2006; Wilberger et al., 1991) all in respect of American football athletes. In total there appear to be only seven studies which have investigated the chronic effects of concussion employing high school athletes only, and these include one soccer study (Stephens et al., 2005), five American football studies (Barr, 2003; Lovell et al., 2003, Lovell, Collins, Iverson et al., 2004; Moser et al., 2005; Wilberger et al., 1991) and one Rugby Union study (Shuttleworth-Edwards et al., 2004). Of these seven studies on chronic effects, one American football study (Barr, 2003), investigated neurocognitive sequelae following concussion only and employed paper-and-pencil measures; two studies, both involving American football athletes (Lovell et al., 2003; Lovell, Collins, Iverson et al., 2004), employed a computerized neurocognitive battery, (ImPACT); and three studies employed paper-and-pencil measures to investigate both neurocognitive and postconcussion sequelae following concussion (Moser et al., 2005; Shuttleworth-Edwards et al., 2004; Wilberger et al., 1991). Only one soccer study employed a combination of both paper-and-pencil and computerized neurocognitive tests, although no postconcussion inventory was used (Stephens et al., 2005). None of the studies used a combination of both paper-and-pencil and computerized neurocognitive and postconcussion symptom inventories in their studies investigating high school athletes.

Further, from a methodological perspective, six of the seven studies reported on above can be seen to have sampling limitations. Two studies have no control group (Lovell et al., 2004; Wilberger et al., 1991), three studies demonstrated no control for gender (Barr, 2003; Lovell et al., 2003; Lovell, Collins, Iverson et al., 2004), four studies demonstrated no control for IQ (Barr, 2003; Lovell et al., 2003, 2004; Moser et al., 2005), two studies demonstrated no control for age (Lovell et al., 2003; Moser et al., 2005) and three studies demonstrated no control for education (Barr, 2003; Moser et al., 2005; Stephens et al., 2005). The only study examining the chronic effects of high school athletes which incorporated strict control for age, IQ and education between comparative groups was conducted on Rugby Union high school athletes (Shuttleworth-Edwards et al., 2004). However, this last study was limited to the use of paper-and-pencil testing only, and comprised only a single assessment at pre-season.

Thus, in light of the methodological limitations and paucity of studies available to investigate the effects of cumulative MTBI in sport specifically at the high school level, it was decided to

conduct such a study on a South African school boy population. Over a period of two years, all final year, sports playing Grade 12 high school boys, from an English medium high school in the Western Cape Province in South Africa were targeted for participation in the study. The average age of the sample was 17 years, the level of education was eleven years and the average WAIS-III Vocabulary subtest scaled score (used as a control for IQ level) was 12 (upper average range). Potentially confounding variables that might result in deleterious neuropsychological effects thereby influencing the findings such as any reported history of substance abuse, any previous diagnosis of a learning disorder which included a diagnosis of Attention Deficit Disorder, any grades repeated, any remedial instruction, any neurological disorder (which included meningitis or epilepsy), and a low WAIS-III Vocabulary subtest scaled score (i.e. < 9 points) were controlled for by eliminating these individuals from the total sample group.

Statistical comparisons revealed that all the comparative sports and concussion groups were equivalent for age, level of education and estimated IQ ( $p = > 0.05$  in all instances, see Tables 7.2.1, 7.2.2.1 & 7.2.2.2, pp. 237-238) suggesting that any deleterious neuropsychological effects in evidence on the cognitive and postconcussion symptom measures were unlikely to be due to the influence of these three factors, or due to the influence of the other extraneous variables on the basis of which individuals were excluded from the sample. Deleterious outcome in respect of contact sports groups, therefore, could be attributed with a greater degree of certainty to the high school athletes being exposed to cumulative and sub-concussive effects as a result of participating in a contact sport, rather than any of these extraneous variables. This is supported by the fact that there were highly significant differences for numbers of reported concussions between groups at pre- and post-season ( $p = 0.000$ ;  $p = 0.000$ , see Table 7.2.1) in the direction of enhanced injury for the contact group. Further, researchers contend from their data that in addition to concussive effects, subclinical microtrauma probably occur resulting in cumulative deleterious effects in association with participation in a contact sport (Killam et al., 2005; Rutherford et al., 2005).

Recently researchers (e.g. Van Kampen, Lovell, Pardini, Collins, & Fu, 2006) as well as the Concussion in Sport Group (Aubry et al., 2002; McCrory, Johnston, et al., 2005) have suggested utilizing *both* neurocognitive and postconcussion symptom measures as means of assessment in the evaluation of MTBI as both of these effects are evident in the clinical presentation of MTBI.

Accordingly, it was suggested that the use of both measures would increase the 'diagnostic yield' in terms of increased sensitivity to identifying these effects (Van Kampen et al., 2006). Following these current recommendations for concussion assessment, the present study utilized a widely used and renowned neurocognitive computerized test for this purpose, viz. the ImPACT programme in conjunction with a prototypically sensitive paper-and-pencil measure, the Digit Symbol Substitution Test which has been frequently used to identify neurocognitive deficits in association with concussion across most sports studies, either in isolation (e.g. Maddocks, Saling et al., 1995) or as part of a wider test battery (e.g. Abreau et al., 1990; Matser et al., 1998, 1999; Shuttleworth-Edwards et al., 2004). In addition an adjunctive Digit Symbol Substitution Paired Associate Incidental Recall task in both immediate and delayed form was incorporated for the purposes of this study and is also reputed to be sensitive to mild presentations of diffuse brain injury (Shuttleworth-Edwards, 2002). In addition to the above cognitive measures, two postconcussion symptom inventories were employed in the research for the purpose of evaluating the self-reported postconcussive symptoms experienced by the high school athletes. One formed part of the ImPACT programme and the other, a paper-and pencil questionnaire, was developed from a paper-and-pencil symptom scale used for past research on Rugby Union (Shuttleworth-Edwards et al., 2004) and consisted of four additional symptoms over the ImPACT scale. Two of the additional symptoms (aggression and speech problems) were shown to be sensitive to the effects of diffuse closed MTBI in prior research on Rugby Union athletes (Shuttleworth-Edwards et al., 2004).

For the purposes of the present study, the total sample of high school athletes was divided into two different sports groups comprising (i) the Contact sports group including participants of Rugby Union, soccer and martial arts, and (ii) the Non-Contact sports group including participants of field hockey, basketball, waterpolo, athletics, tennis, squash, swimming, cycling, cricket, rowing, golf and gymnastics, in order to investigate whether deleterious effects on testing were in evidence in respect of athletes with involvement in a contact sport when compared with athletes involved in a non-contact sport. In addition to the division of sports samples in terms of participation in contact versus non-contact sport, researchers have gone a step further and divided sports samples into concussion history groups in respect of one, one or two, two or more and three or more concussion groups. For example, researchers investigating the effects of one or two and two or more concussions include, Collins, Grindel et al., 1999;

Hinton-Bayre et al., 2004; Iverson et al., 2006; Macciocchi et al., 2001; Maddocks, Saling, et al., 1995; Moser et al., 2005 and Wilberger et al., 1991, and researchers investigating the effects of three or more concussions include, Gaetz et al., 2000; Guskiewicz et al., 2005; Iverson et al., 2002, 2004a; and McCrory et al., 2000. In the present study the sample group was further divided into groups on the basis of reported concussion history in order to compare those athletes who reported no concussions with those who had sustained two or more concussions, in order to assess whether deleterious effects were in evidence for athletes with cumulative (2+) MTBI.

Data were collected on all participants at the pre-season interval (February to April) in order to establish whether there were indications of permanent (residual) neuropsychological sequelae in respect of deficits reported on cognitive and postconcussion symptom measures on the assumption that participants would not have been involved in contact sport for the preceding four months since the termination of the previous winter season (October to January). Typically the literature suggests that recovery from MTBI takes place within three months, and that sequelae that persist longer than that can be considered chronic (e.g. Frencham, Fox, & Maybery, 2005; Schretlen & Shapiro, 2003). Further data were collected at the post-season interval in order to establish whether there were indications of deterioration in neuropsychological functioning in respect of deficits reported on cognitive and postconcussion symptom measures, sustained during participation in a contact sport over a six month winter sports season.

A cross-sectional, quasi-experimental design was employed for the purpose of this study. For the comparison of the different sports groups (Contact versus Non-Contact) a non-equivalent control group design was used in respect of the different sports groups for both pre- and post-season assessment. Further, when comparing the different concussion history groups (2+ Concussion versus 0 Concussion) a non-equivalent control group design was used in respect of the distribution of participants across sports types within the two comparison groups, for both pre- and post-season assessment. In order to improve on the non-equivalence of this comparison in respect of sports types, a further analysis was performed on equivalent groups in respect of sport (equivalent control group design) comparing the 2+ Concussion versus 0 Concussions *within* the Contact group. Finally a prospective, non-equivalent control group design was used in respect of the different sports groups for the comparison between the Contact groups pre- versus post-season and Non-Contact groups pre- versus post-season.

The concept of cognitive reserve (Stern, 2003; 2006), alternatively designated Brain Reserve Capacity Theory-BRC (Satz, 1993) was adopted as an interpretive framework for the present study. The concept has been widely applied in brain injury research, being a powerful mode for interpreting individual variability in the mediation of cognitive processes in the brain. Specifically the theory postulates that differential variation in terms of pre-existing cognitive reserve (i.e. brain size, intellectual capacity and genetic brain disposition) exists in individuals prior to neuronal attrition or neurological disease (Satz, 1993). This inherent inter-individual variation can be assumed to account for some differences in test scores amongst younger individuals such as the participants in the present study. Further, in line with the BRC theory it is postulated that the additive effect of cumulative MTBI (as a result of being exposed to a contact sport over a high school playing career) taken together with variations in pre-existing cognitive reserve, in addition to a sufficiently challenging task, would result in pushing some individuals over the functional threshold thereby producing clinically detectable deleterious neuropsychological effects. Amongst high school athletes one would propose that these effects would be diluted relative to the adult and elite professional level players, owing to the shorter length of exposure and less intensive exposure to the sport. This would imply that the number of high school athletes that would be pushed over the BRC threshold through cumulative MTBI would be variable due to pre-existing differences in cognitive reserve, and minimal compared with an older more exposed group. However, in terms of BRC theory the implication is that a process of cumulative MTBI sustained whilst playing contact sport will have been initiated at the high school level and particularly in the final grade 12 year of high school after being exposed to the contact sport for a minimum of eight years.

More specifically, from BRC theory it is expected that those with greater pre-existing vulnerability (lower BRC) in conjunction with two or more concussions would be at risk for showing signs of residual impairment given sufficient task challenge, whereas those with higher BRC and fewer concussive or sub-concussive episodes may not show signs of deficit at this stage of participation in the sport regardless of level of task challenge. Accordingly, in light of these suppositions in respect of BRC theory, for the purposes of this study a number of hypotheses were posed, as follows.

### 9.1.1 Theoretical hypotheses

(i) It was hypothesized that in comparison with the Non-Contact group, the Contact group would demonstrate deleterious neuropsychological effects in respect of cognitive deficits made evident on objective neurocognitive testing and self-reported postconcussive symptoms established on the basis of two postconcussion inventories, due to postulated exposure to repetitive concussion (MTBI), including both concussive and sub-concussive events, during high school participation in contact as opposed to non-contact sport at both pre- and post-season occasions.

(ii) Further it was hypothesized that in comparison with the 0 concussion group, the 2+ concussion group would demonstrate deleterious neuropsychological effects in respect of cognitive deficits made evident on objective neurocognitive testing and self-reported postconcussive symptoms established on the basis of two postconcussion inventories, due to postulated exposure to repetitive concussion (MTBI) at both pre- and post-season occasions.

(iii) It was hypothesized that *within* the Contact group, the 2+ concussion group would demonstrate deleterious neuropsychological effects made evident on objective neurocognitive testing and self-reported postconcussive symptoms established on the basis of two postconcussion inventories, due to exposure to repetitive concussion during high school participation in a contact sport.

(iv) It was hypothesized that *either*

- in comparison to the Non-Contact group, the Contact group would demonstrate deleterious neuropsychological effects in respect of cognitive deficits made evident on objective neurocognitive testing and self-reported postconcussive symptoms established on the basis of two postconcussion inventories at post-season compared to pre-season, due to the effects of unreported concussive and sub-concussive events, sustained during participation in a contact sport over a six month winter sports season

*or*

- in comparison with the Non-Contact group the Contact group would demonstrate no significant neurocognitive impairment due to practice effects on objective at the post-season compared to the pre-season interval, due to the inability to benefit from practice,

being cerebrally vulnerable as a consequence of participation in a contact as opposed to a non-contact sport.

(v) Finally, in support of the BRC theory, it was hypothesized that these effects would be less in evidence amongst this sample of high school athletes compared with adult professional level players owing to the lower level of play (less intense and less years of exposure to contact sport as opposed to a non-contact sport).

### 9.1.2 Statistical hypotheses

In light of the above theoretical hypotheses, the following more specific statistical hypotheses were posed as follows:

(i) It was hypothesized that *on the basis of independent t-test analyses* there would be significant differences in the mean scores in respect of (a) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and pencil tests), and (b) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories, between the Contact and the Non-Contact groups at both pre- and post-season intervals, in support of deleterious neuropsychological sequelae for the Contact group relative to the Non-Contact group, due to postulated exposure to repetitive concussion, including concussive and sub-concussive events, during high school participation in a contact as opposed to a non-contact sport.

(ii) It was hypothesized that *on the basis of independent t-test analyses* there would be significant differences in the mean scores in respect of (a) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and pencil tests), and (b) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories, between the 2+ Concussion and the 0 Concussion groups at both pre- and post-season intervals, in support of deleterious neuropsychological sequelae for the 2+ Concussion group relative to the 0 Concussion group, due to cumulative exposure of repetitive concussion during high school participation in a contact sport.

(iii) It was hypothesized that *within* the Contact group *on the basis of independent t-test analyses* there would be significant differences in the mean scores in respect of (a) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and pencil tests), and (b) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories, between the 2+ Concussion group compared with the 0 Concussion group at both pre- and post-season intervals, in support of deleterious neuropsychological sequelae for the 2+ Concussion group relative to the 0 concussion group due to postulated exposure to repetitive concussion during high school participation in a contact sport.

(iv) It was hypothesized *on the basis of dependent t-test analyses* that *either* there would be significant differences in the mean scores in respect of (a) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and pencil tests), and (b) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories for the Contact group at post-season compared to pre-season, in support of deleterious neuropsychological effects of unreported concussive and sub-concussive events, sustained during participation in a contact sport over a six month winter sports season, *or* that there would be no significant improvement in the mean scores for the Contact group at post-season compared to the pre-season interval on objective neurocognitive testing, in contrast to significant improvement for the Non-Contact group.

(v) Finally, in terms of BRC theory it was hypothesized that *descriptively* compared with deleterious neuropsychological effects that have been reported in the literature for adult high level professional athletes who have a long history of participation in contact sport, in this high school cohort there would only be relatively subtle indications of (a) cognitive deficits established on objective neurocognitive testing (using both computerized and paper and pencil tests), and (b) self-reported somatic, cognitive, emotional and behavioural postconcussive symptoms established on the basis of two postconcussion inventories due to shorter exposure to the sport and lower, less intensive level of play.

## 9.2 EVALUATION OF FINDINGS ON THE COGNITIVE MEASURES

This section reports on the neurocognitive findings for the independent, between and within groups analyses for the Contact versus Non-Contact groups and 2+ Concussion versus 0 Concussion groups at both pre- and post-season occasions. This is followed by the neurocognitive findings for the dependent analyses for the Contact group pre-season versus post-season and the Non-Contact group pre-season versus post-season.

### 9.2.1 Independent t-test analyses

In respect of the Contact versus Non-Contact groups and the between and within groups analyses for the 2+ Concussion versus 0 Concussion groups there were no significant results on any of the cognitive measures at both the pre- and post-season occasions. The only exception was between the Contact versus the Non-Contact group analysis at pre-season where the cognitive measure, Reaction Time reached significance ( $p = 0.014$ ) in the direction of the Contact group performing worse than the Non-Contact group.

In respect of the general trends for the between and within groups analyses, all were in the direction of the Contact and 2+ Concussion groups performing worse than the Non-Contact and 0 Concussion groups at both pre- and post-season occasions (at least greater than 50% of measures at pre-season, and at least 90% at post-season, being more for the Contact versus Non-Contact groups and for the 2+ versus 0 Concussion groups). The only exception was for the *within* Contact group, pre-season analysis where an overall opposite trend occurred in respect of the 0 Concussion group performing worse than the 2+ Concussion group (five out of the seven measures).

The overall lack of significant findings across all groups with the exception of the Reaction Time measure for the Contact versus Non-Contact group analysis at pre-season do not support the hypotheses in terms of the expectation of significant differences between the means in respect of the Contact and 2+ Concussion groups performing worse than the Non-Contact and 0 Concussion groups, on both the between and within groups analyses and at both pre- and post-season occasions. In terms of the general trends, however these do support the hypotheses in that

all trends (with the exception of the *within* Contact group analysis at pre-season) were in the direction of the Contact and 2+ Concussion group performing worse than the Non-Contact and 0 Concussion groups.

Generally the literature supports the fact that amongst older contact sports athletes the neurocognitive effects tend to be more in evidence due to the postulated long-term exposure to cumulative concussive events (see sports reviews, e.g. Baroff, 1998; Rutherford et al., 2003). Of the studies that have reported on the neurocognitive effects of concussion amongst high school athletes, the findings appear to be consistent with the present study in that no relationship between MTBI and cognitive deficit was reported (e.g. Barr, 2003; Lovell et al., 2003, 2004; Stephens et al., 2005). The isolated finding in respect of the Reaction Time measure being significant for the Contact group at pre-season is consistent with an isolated finding reported in respect of Rugby Union high school athletes, where Rugby Union players reported deficits on one trial of a hand motor function test (Shuttleworth-Edwards et al., 2004).

With reference to the findings amongst the concussion history groups there were no significant results in respect of individuals who reported a prior history of two or more concussions and those who reported no prior history of concussion across the different sports groups and *within* the Contact group. The trends however, were predominantly in the direction of the two or more concussion history group performing worse than the zero concussion group. Of the studies reviewed investigating the effects of concussion history amongst high school athletes, only two studies demonstrated deficits on neurocognitive testing where American football high school athletes with a history of two or more concussions seemed to be experiencing prolonged effects on tests of information processing speed and selective and divided attention (Moser et al., 2005; Wilberger et al., 1991). More detailed examination of these studies however, from a methodological point of view, serves to explain the absence of significant findings on the present study relative to their findings, and accords with the cautionary comments referred to in the methodology, on the potential for Type II error in MTBI research and neuropsychological research involving clinical populations in general (Demakis, 2006; Frencham et al., 2005; Reitan & Wolfson, 1999; Ruff, 2005; Woods et al., 2006).

Wilberger et al. (1991) are reporting on case-based rather than group research, in which he and his colleagues evaluated 62 athletes who reported to have sustained two or more concussions out of a cohort of 500 athletes. Of these 62 athletes, 16 demonstrated abnormalities at three months post-injury on the PASAT, the Stroop, the Trail Making Test and the Symbol Digit Modalities Test (i.e. only 3% of the cohort). Hence, the effects being reported by Wilberger et al. are the individual effects of a percentage of athletes who sustained two or more concussions whose deleterious outcome would likely be concealed in a group analysis. These deleterious effects would thus be averaged out in a group analysis and would appear as subtle trends, such as seen in the present study.

In Moser et al's study (2005), perusal of Table 9.2.1.1 reveals that significant differences in their study were evident on the neurocognitive tests RBANS ( $p = 0.012$ ) between those athletes who had recently sustained a concussion and those who sustained either none or one concussion and on the Trail Making Test-Part B ( $p = 0.006$ ) between those individuals who had recently sustained a concussion and those who hadn't sustained a concussion. The results however revealed *no* significant differences between the means in respect of those athletes who had recently sustained a concussion and those who sustained two or more concussions. Thus, on the basis of this Moser and colleagues suggest that those athletes who sustained two or more concussions are performing similarly to the recently concussed group, but worse than the no concussion group. Accordingly, although on the statistical analyses significant differences between the no concussion and two or more concussion groups were not specifically demonstrated (as in the present study), these authors make the extrapolation from their other significant findings that there is a substantive difference between these two groups. Perusal of the trends in respect of their data (see Table 9.2.1.1) reveal that they generally appear to be in the direction of those individuals with no concussions performing better than those who sustained two or more concussions on most of the cognitive measures, such as similarly occurred in the present study.

**Table 9.2.1.1**  
**Analysis of effects of concussion history on cognitive functioning performance <sup>a</sup>**

Variable	Range	Concussion history group <sup>b</sup>				F	Significance F
		None	1 previous	≥ 2 previous	Recent		
Attention (RBANS)	46-105	71.0 (10.7)	71.8 (10.5)	68.0 (10.0)	65.7 (8.3)	3.71	0.012 <sup>c</sup>
Immediate Mem (RBANS)	37-64	50.6 (4.8)	52.0 (4.2)	50.1 (5.7)	52.3 (4.8)	2.32	0.08
Delayed Memory (RBANS)	37-62	53.3 (4.7)	53.9 (3.8)	53.5 (4.7)	53.6 (4.0)	0.28	0.82
Language (RBANS)	18-43	30.9 (4.4)	31.2 (5.1)	29.4 (5.2)	39.8 (4.1)	1.63	0.18
Visuoconstructional ability (RBANS)	20-40	34.9 (4.4)	36.3 (3.3)	35.7 (3.6)	35.6 (3.8)	1.60	0.19
Trail Making Test Part A (by level)	0-2	0.40 (0.6)	0.38 (0.6)	0.38 (0.6)	0.41 (0.6)	0.09	0.99
Trail Making Test Part B (by level)	0-3	0.10 (0.3)	0.18 (0.5)	0.22 (0.6)	0.44 (0.7)	4.21	0.006 <sup>d</sup>

<sup>a</sup> RBANS, Repeatable Battery for the Assessment of Neuropsychological Status

<sup>b</sup> Means +/- standard deviation.

<sup>c</sup> Recent concussion group < No concussion and 1 previous concussion groups.

<sup>d</sup> Recent concussion group > No concussion group (higher scores relate to greater impairment).

(Moser et al., 2005)

The Moser et al. (2005) neurocognitive findings therefore, are highly commensurate with the findings in the present research, where although there were no significant results for the comparative zero versus two or more concussion history groups, generally the overall trends were in the direction of those athletes with a history of two or more concussions performing worse than those with no concussions. Accordingly, in conclusion of this methodological analysis, it can be seen that in line with the present study, the two studies that have investigated differential concussion history amongst high school athletes (Moser et al., 2005; Wilberger et al., 1991) have demonstrated relatively subtle effects when taking the whole cohort into account, and it is evident that these effects have been more visible on the individual analysis and less in evidence on the group analysis. This provides support for the warnings of recent reviewers on the dangers of Type II error (Demakis, 2006; Frencham et al., 2005; Reitan & Woolfson, 1999; Ruff, 2005; Woods et al., 2006), and points to the relevance of not ignoring consistently reported subtle effects in group MTBI research such occurred in the present study, where effects are expected to be relatively subtle.

Of the studies reviewed in Chapter six which have investigated the cumulative neurocognitive effects of one or two and two or more concussions amongst older contact sport athletes, these studies have produced varied results. Whilst some studies reported no significant results, with no relationship between the number of reported concussions and cognitive functioning on testing (Hinton-Bayre & Geffen, 2004; Iverson et al., 2006; Macciocchi et al., 2001; Maddocks et al., 1995), others reported an association between a history of concussion and long-term, persistent effects (Collins, Grindel, et al., 1999; Guskiewicz et al., 2003, 2005; Iverson et al., 2002). Thus, in light of the above with respect to both younger and older athletes, it would appear that dividing experimental groups on the basis of sports affiliation (i.e. contact versus non-contact) rather than on the basis of concussion history may be a more reliable way of capturing effects due to the known underreporting of concussive events by the athletes themselves. Research by Killam et al., (2005) is commensurate with this hypothesis, in that their 'non-concussed' group of athletes performed significantly worse than their 'non-concussed'/non-athletes, and these authors suggest that this may be due to the cumulative consequences of multiple mild undiagnosable concussive events amongst the athletes. In the present study, however the findings on the independent t-test analyses for the concussion groups did appear to be relatively similar to the findings for the sports groups, with the overall trends all providing consistent support in the hypothesized direction.

With regards to the only significant result at pre-season, in respect of the Reaction Time composite score on the ImPACT programme, this finding supports other MTBI research in that reaction time represents one of the cognitive domains that has consistently shown to be sensitive to the effects of MTBI (e.g. Levin, Lippold, et al., 1987; O'Connor & Burns, 2003). According to researchers, mental slowing shows up in slowed reaction times and in overall slow performance times in the absence of a specific motor disability which is the most common characteristic of both aging and diffuse brain damage (Hinton-Bayre et al., 2004; Tromp & Mulder, 1991). The sensitivity of Reaction Time to the effects of MTBI in respect of contact sports has been demonstrated in studies of boxing (e.g. Ramirez et al., 1998; Moriarity et al., 2004; Warden et al., 2001), ice hockey (e.g. Gaetz et al., 2000), soccer (e.g. Downs & Abwender, 2002), American football (Collins, Field, et al., 2003; Iverson et al., 2004b; Lovell, 2004), and Australian Football League (e.g. Collie et al., 2006; Cremona-Meteyard & Geffen, 1994; Maddocks & Saling, 1996; Makdissi et al., 2001). Of these studies only three have

investigated the chronic effects with respect to neuropsychological functioning following MTBI (Cremona-Meteyard & Geffen, 1994; Downs & Abwender, 2002; Gaetz et al., 2000), although none of these studies employed high school athletes in their samples. The implication from this aspect of the present study is that the ImPACT Reaction Time composite may be the most sensitive indicator of residual deleterious cognitive performance amongst high school contact sport players compared with any of the other cognitive measure employed. On the other hand, in that it was an isolated significant effect, its occurrence may be attributed to chance. Given the strong general tendency for nearly all the results to be in the same direction, however, it seems more likely that the Reaction Time composite presented more challenge to vulnerable participants than other tasks, hence causing deficit to be manifested to an extent that caused it to reach a level of statistical significance,

The general trends reported in the present study that failed to reach significance, but were predominantly in the hypothesized direction (i.e. that the Contact and 2+ Concussion groups would perform worse than the Non-Contact and 0 Concussion group) can be further explicated in terms of BRC theory. It was suggested in Chapter three that pre-morbid variables such as low education and low levels of IQ and education, smaller head size, female gender, the presence of a psychiatric or neurological disorder, prior head injury, learning disorders or a combination of these factors present as vulnerability factors predisposing individuals to the early onset of symptoms in association with neurological disease (Satz, 1993). In light of such vulnerability factors, the present sample can be seen to be a relatively protected one in that it consisted of male athletes, with no psychiatric or neurological disorders and with a relatively high level of education and estimated IQ level (all participants were completing Grade 12 in a relatively advantaged educational setting, and the sample's average Vocabulary scaled score was 12). Thus, it is likely that these protective factors served to buffer the exposure to multiple concussive trauma in this particular high school contact sport group. Over and above this, further protection from substantial neurocognitive decline for this sample was that exposure to multiple concussion was substantially less than that of older adults with many more years of exposure.

In light of all of these factors, therefore, the implication is that deleterious effects of concussion are likely to be occurring in this sample, but due to a large number of protective factors are still at a subclinical or very subtle level. Accordingly, the current pattern of results can be seen to be

highly commensurate with such a proposition, where there is only one result that reached significance despite an overwhelming set of consistent trends pointing in the direction of poorer neurocognitive performance for contact sports players. This argument is supported by prior research investigating residual effects amongst Rugby Union players at various level of play (Shuttleworth-Edwards et al., 2004). In this study both adult teams (Open Rugby and under 21 players) reported deficits on cognitive measures (Digit Symbol Substitution Test, Trails B, Digit Symbol Substitution Paired Associate Incidental Recall-Immediate and Delayed, WMS-Visual Reproduction and Associate Learning, Verbal Fluency and Hand motor function) in contrast to the school level players who reported deficits on only one test, which was on one trial of the hand motor function test.

### 9.2.2 Dependent t-test analyses

The dependent t-test comparison of all measures between the Contact group pre-season versus post-season and the Non-Contact group pre-season versus post-season, revealed that the results for the Contact group replicated the results for the Non-Contact group, in respect of a strong trend in the direction of both the Contact and Non-Contact groups performing better at post-season than at pre-season (five out of seven measures and six out of seven measures respectively, improving at post-season). There were no significant results indicating deterioration of function between pre- and post-season. However, indicating *improvement* at post-season compared with pre-season, two tests reached significance for the Contact group, viz. Digit Symbol Substitution and Digit Symbol Incidental Recall-Immediate ( $p = 0.000$  and  $p = 0.025$ , respectively), and three tests for the Non-Contact group, viz. Visual Motor Speed, Digit Symbol Substitution and Digit Symbol Substitution Incidental Recall-Delayed ( $p=0.011$ ,  $p = 0.000$  and  $p = 0.024$ , respectively). Substantial improvement at post-season versus pre-season for the Non-Contact group occurred as a strong trend on a fourth test, viz. Digit Symbol Substitution Incidental Recall-Immediate ( $p = 0.117$ ).

It is apparent from these results, therefore (see earlier Table 8.3.1, p.290), that there is significant improvement at post-season versus pre-season for both the Contact and Non-Contact groups on the Digit Symbol Substitution test. Further, there is a significant improvement at post-season versus pre-season for the Contact group on the Digit Symbol Substitution Incidental Recall-

Immediate that is replicated as a strong trend in the same direction for the Non-Contact group., However, the significantly improved scores for Visual Motor Speed and Digit Symbol Substitution Paired Associate Incidental Recall- Delayed that are in evidence for the Non-Contact group, are entirely absent for the Contact group, where the opposite trend of worsening scores at post-season versus pre-season is revealed.

The significant results and overall trends on the neurocognitive measures in the direction of the Contact group performing better at post-season than at pre-season do not support the hypotheses in respect of the Contact group performing worse at post-season in comparison to pre-season due to the effects of unreported concussive and sub-concussive events sustained over a sixth month sports season. The current findings, however, in respect of the significant practice effects and general trends are replicated elsewhere in the literature. For example Levin, Lippold et al. (1987) investigated the effects of boxing on cognitive performance in a group of amateur boxers and athlete controls. Their findings showed a significant improvement in the performances of most of the boxers and athlete controls six months after their initial assessment on measures including the Digit Symbol Substitution Test (DSST), the PASAT, Continuous performance task, microcomputer reaction time task, Digit Span test, Selective Reminding Test, Benton Visual Retention Test, Controlled Word Association test, Design Fluency, Trail Making Test and Purdue Pegboard. Putukian et al. (2000) investigated the concussive and sub-concussive effects of heading in a group of college soccer players. Their results demonstrated that regardless of whether the athletes were headers or not, both groups performed better on the post-season testing than on the pre-season testing on the Alphabet Backwards, the Trail Making Test, the Stroop Colour Word test and a modified version of the VIGIL/W that assesses reaction time and sustained attention and concentration. Similarly, Barr (2003) demonstrated significant practice effects in their group of high school athletes at a re-test interval of 60 days on the WAIS III Processing Speed Index, the Trail Making Test and the COWAT. Further, Pellman et al. (2004) in their investigation amongst NFL athletes, injured athletes performed significantly better on tests of attention and processing speed (Trails A, Digits forwards and the Symbol Digit Modalities Test) at post-injury than at pre-season. Researchers Collie et al. (2006), Hinton-Bayre et al. (1997) and Makdissi et al. (2001) in their studies investigating the effects of MTBI on AFL and Rugby League athletes respectively, all players demonstrated improvements on their cognitive measure performances and particularly on the DSST.

Whilst some studies have demonstrated improvements on tests with respect to contact sports players, others have shown no improvements in comparison with non-contact sports control groups that have shown improvements. For example, Maddocks and Saling (1996) demonstrated that amongst AFL athletes whilst concussed players performances were commensurate with their pre-season performances, the AFL referee controls demonstrated significant improvements from their pre-season performances on the DSST, Four-Choice Reaction Time Test and the PASAT. Similarly, amongst Rugby Union and non-contact sports athletes a significant practice effect was demonstrated for the control group on the Trail Making Test, Digit Supraspan, Finger Tapping Task and Purdue Pegboard that was not as strongly in evidence in respect of the rugby group (Shuttleworth-Jordan et al., 1993).

It would appear that generally some tests are more prone to the effects of practice than others, with measures of memory and visuomotor processing speed being particularly sensitive to practice effects (Lezak et al., 2004). In the present study two measures of visual motor processing speed were used, one a paper-and-pencil measure (DSST) and the other a component of the computerized neurocognitive test battery, ImPACT (Visual Motor Speed composite). In the research reported on above the DSST has consistently demonstrated its sensitivity to practice effects (e.g. Barr, 2003; Hinton-Bayre et al., 1997; Levin, Lippold et al., 1987; Macciocchi et al., 1996; Maddocks & Saling, 1996; Makdissi et al., 2001), which is consistent with the findings in the present study with respect to Contact and Non-Contact athletes showing improvement on the post-season testing relative to the pre-season testing. Whilst the DSST is a paper-and-pencil measure which consists of only one test form, the Visual Motor Speed composite of ImPACT test is a computerized measure comprising several tasks [i.e. X's and O's (average correct distracters), Symbol Match (average correct responses), Three letters (number of correct numbers correctly counted)]. The improvement on the DSST for both the Contact and Non-Contact groups is suggestive of a learning effect, and although the Visual Motor Speed component of the ImPACT neurocognitive battery demonstrated a significantly improved performance in the Non-Contact group ( $p=0.011$ ), this was not replicated for the Contact group ( $p=0.664$ ), where there was a tendency for their performance to worsen at post-season.

This finding is consistent with two studies investigating the effects of concussion in AFL athletes where a combination of paper-and-pencil measures (including the DSST and the Trail Making Test) and a neurocognitive test battery, CogSport, were employed. Whilst the paper-and-pencil measures demonstrated practice effects in both the concussion and control groups for the one study (Makdissi et al., 2001) and significant practice effects amongst the asymptomatic concussed athletes and controls in the other (Collie et al., 2006), the concussed and symptomatic concussed athletes in both studies demonstrated a significant deterioration in performance on the computerized neurocognitive battery, CogSport. This suggests the relative sensitivity of a computerized test battery over paper-and-pencil measures in the investigation of deficit amongst concussed athletes. Similarly, the lack of improvement for the Visual Motor Speed composite in the present study from pre- to post-season suggests that this measure provides a complexity and challenge for performance that is not present on the Digit Symbol Substitution Test, such that the more vulnerable Contact group with postulated exposure to multiple brain insults were not able to benefit from practice on this task, thereby differentiating this group from the Non-Contact group in a manner that did not occur for the paper and pencil task.

As indicated above, as with the Digit Symbol Substitution Test itself, substantial improvements from pre- to post-season were also noted for the Digit Symbol Paired Associate Incidental Recall task in the immediate form for both the Contact and Non-Contact groups, whereas in the delayed form the improvement was only in evidence for the Non-Contact group (see earlier Table 8.3.1, p.290). Although the Digit Symbol Incidental Paired Associate Recall task, particularly the Delayed Recall task, is very sensitive to subtle cognitive deficits associated with concussion (e.g. Heilbronner et al., 1991; Shuttleworth-Edwards et al., 2004) it is also prone to practice effects (e.g. DeMonte et al., 2005; Hinton-Bayre et al., 1997; Makdissi et al., 2001; Lezak et al., 2004). One could argue, however, that a learning effect for the Digit Substitution Paired Associate Incidental Recall – Immediate recall task is more likely in comparison to a delayed recall version of the task which is known to be more difficult for cerebrally compromised populations (see review in Shuttleworth-Edwards, 2002), and would therefore also be more cognitively challenging to the more cerebrally vulnerable Contact group. This serves to explain the absence of a practice effect for the Contact group on the delayed form of this task albeit it was in evidence for the Non-Contact sport group. These findings therefore support the alternative hypothesis in respect of the lack of significant improvement for the Contact group on the Digit

Substitution Paired Associate Incidental Recall – Delayed recall task and the Visual Motor Speed composite from the ImPACT test at the post-season interval. The overall implication of this failure to benefit from practice in the Contact group is that they are a more cerebrally vulnerable group due to their postulated increased risk of exposure to concussive and sub-concussive effects as a result of participation in a contact as opposed to a non-contact sport.

Satz (1993) postulates that cognitive deficit would remain undetected (sub-threshold) due to the protective factor of a greater BRC until such time that a sufficiently *challenging* task presents itself. Thus, cognitive deficit would not have otherwise been detected had the neuropsychological measures not been sufficiently robust to allow the individual to fall below the cognitive threshold level and present with symptoms. Taken together, the two tasks of Visual Motor Speed ImPACT composite, and the Digit Symbol Incidental Recall Delayed task appear to have provided optimal challenge on the neural networks involved in task completion in a cohort of Contact players exposed to cumulative MTBI, such that no improvement with practice was in evidence on these tasks for this cohort, albeit this occurred in the Non-Contact cohort without similar exposure to cumulative MTBI. Accordingly, in this way the contact sports players can be seen to have fallen below the threshold of symptom presentation in respect of learning ability with practice on these two challenging tasks, which allowed for the identification of incipient cognitive deficits that were apparently present in this group.

In sum, these results for the dependent statistical analysis of neurocognitive test performance between pre- and post-season do not provide support for worsening of neurocognitive performance in the Contact group compared with the Non-Contact group due to cumulative insults incurred during the season. However, they do provide support for the underlying theoretical hypothesis that exposure to cumulative MTBI in a contact sports group will result in compromised neurocognitive function. This is because on two tests with known sensitivity to diffuse brain damage that typically accompanies the concussive brain injury (ImPACT Visual Motor Speed composite and Digit Symbol Substitution Incidental Recall- delayed form), the Contact group failed to benefit significantly from practice on repeat testing following a six month interval, whereas the Non-Contact group did. The implication is that the Contact group have succumbed to deleterious effects of cumulative concussive and sub-concussive insults over years of exposure to the sport as well as possible more immediate deleterious effects sustained

during a season of participation in such sport, or a combination of both of these factors. Empirically this supposition is supported with prior research that indicates that individuals with traumatic brain injury tend not to benefit from practice effects on speeded cognitive tests (McCaffrey, Ortega, Orsillo, Nelles & Haase, 1992; Mitrushina & Satz, 1991; Rapport, Brookes-Brines, Axelrod & Theisen, 1997). Further, and of particular note is that more pronounced practice effects for the control group relative to the rugby group, at the post-season compared with the pre-season interval, were also noted by Shuttleworth-Jordan et al. (1993) on a test of visuomotor speed (Trails A) in their study on University level rugby players, with the implication of cerebral vulnerability in the rugby group who have a high risk of concussive injury.

### **9.3 EVALUATION OF FINDINGS ON THE POSTCONCUSSIVE MEASURES**

This section reports on the postconcussive findings for the independent analyses, between and within groups analyses for the Contact versus Non-Contact groups and 2+ Concussion versus 0 Concussion groups at both pre- and post-season occasions on both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire. This is followed by the findings in respect of the dependent analyses for the Contact group pre-season versus post-season and the Non-Contact group pre-season versus post-season on both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire. The Postconcussion Symptom Questionnaire comprised 26 symptoms at pre-season and was reduced to nine symptoms at the post-season interval.

#### **9.3.1 Independent t-test analyses**

The overall findings on the pre- and post-season comparisons between the Contact versus Non-Contact and between the 2+ Concussion versus 0 Concussion groups on both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire support the hypotheses in that both inventories consistently reveal significantly higher instances of symptoms for the Contact and 2+ Concussion groups at both the pre- and post-season intervals, on both inventories. None of the significant results were in the opposite direction (i.e. the Non-Contact or 0 Concussion groups reportedly being worse than the Contact or 2+ Concussion

groups). In all instances the total symptom score is either significantly lower or approaching significance for the Contact versus Non-Contact group ( $p = 0.024$ ;  $p = 0.056$ ;  $p = 0.050$  and  $p = 0.000$ ) and the 2+ Concussion versus 0 Concussion group ( $p = 0.020$ ;  $p = 0.116$ ;  $p = 0.000$  and  $p = 0.006$ ). Finally, the trends analyses also reflect a consistently predominant trend for the Contact and 2+ Concussion groups to be worse in respect of more symptoms than the Non-Contact and 0 Concussion group, across all analyses.

The findings on the pre- and post-season comparisons *within* the Contact group, between the 2+ and 0 Concussion groups on the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire support the hypotheses in that significantly higher instances of symptoms for the 2+ Concussion group were reported at pre-season and at post-season, exclusively on the Postconcussion Symptom Questionnaire (i.e. there were no significant effects revealed on the ImPACT Postconcussion Symptom Scale). There were no significant results in the opposite direction (i.e. the 0 Concussion group reportedly being worse than the 2+ Concussion group). Finally, in respect of both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire the trends analyses also support the hypotheses in that they reflect a consistent trend in the direction of the 2+ Concussion group reportedly being worse in respect of more symptoms than the 0 Concussion group, across all analyses.

On the between groups analyses for the Contact versus Non-Contact groups and the 2+ Concussion versus 0 Concussion groups, it is apparent that the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire provide relatively strong validation for each other in that in all analyses at both pre- and post-season there is significantly worse symptom outcome revealed for the Contact and 2+ Concussion groups and no significantly worse outcome revealed for the Non-Contact and 0 Concussion groups. In terms of the Contact versus Non-Contact analyses, there were four overlapping significant results at pre-season in respect of both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire for the total symptom score and the symptoms of fatigue, sleeping less, and nervousness, with no overlapping significant results occurring at post-season. In terms of the 2+ Concussion versus the 0 Concussion analyses there were six overlapping significant results at pre-season in respect of both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire for the total symptom score and the symptoms of

dizziness, fatigue, drowsiness, irritability, and concentration, with no overlapping significant results occurring at post-season. None of these overlapping symptoms reported above at pre-season, however, were included in the reduced Postconcussion Symptom Questionnaire at post-season, except for the symptom concentration, which may explain their lack of replication at the post-season interval.

On the ImPACT Postconcussion Symptom Scale the significant symptoms reported in respect of the Contact group at both pre- and post-season occasions included fatigue, sleeping less and nervousness. For the 2+ Concussion group the only symptom reported at both pre- and post-season intervals was dizziness on the ImPACT Postconcussion Symptom Scale. On the Postconcussion Symptom Questionnaire, significant symptoms reported in respect of the Contact group at both pre- and post-season included the total symptom score, and the symptoms of headache, fatigue, sleeping less, nervousness, feeling more emotional and aggression. For the 2+ Concussion group significant symptoms of dizziness, concentration and aggression were reported at both pre- and post-season intervals on the Postconcussion Symptom Questionnaire.

Compared with the between groups analyses, where there appears to be a relatively clear overall replication of findings for both inventories (i.e. higher symptom reporting for the Contact and 2+ Concussion groups and nothing for the Non-Contact and 0 Concussion groups), on the *within* Contact analyses, this is not apparent. In this case only the Postconcussion Symptom Questionnaire revealed any significant effects mainly at the pre-season interval (i.e. total symptom score, fatigue, difficulty falling asleep, drowsiness, sensitivity to noise, irritability and nervousness) and only one significant symptom (aggression) at post-season. Thus, whilst far fewer symptoms are reported on the *within* Contact group analyses the overall trends are still in the direction of the 2+ Concussion reporting more symptoms than the 0 Concussion group at both pre- and post-season.

It would appear that the two inventories provide relatively strong validation for each other in respect of the overlapping symptoms on the between groups analyses, which tends to suggest that these findings are not purely due to chance. Furthermore, the total symptom scores on both inventories, in respect of the Contact versus Non-Contact group ( $p = 0.024$ ;  $p = 0.056$ ;  $p = 0.050$  and  $p = 0.000$ ) and the 2+ Concussion versus 0 Concussion group ( $p = 0.020$ ;  $p = 0.116$ ;  $p =$

0.000 and  $p = 0.006$ ), between groups analyses were either significant or approaching significance. For the *within* Contact group analyses there is less cross-validation in that the total symptom score was significant for the PCS Questionnaire at pre-season only ( $p = 0.002$ ) and approaching significance at the post-season interval ( $p = 0.08$ ). However, taken together both the between and within group analyses, the results were predominantly in the direction of the Contact and 2+ Concussion group reporting more symptoms than the Non-Contact or 0 Concussion groups which suggests that the findings are attributable to more than chance occurrence, rather implying valid differences in symptomatic profiles between these groups in the hypothesized direction.

As previously mentioned in Chapter five, researchers draw a distinction between early onset PCS (i.e. headaches, dizziness, vomiting, nausea, drowsiness, and blurred vision), those appearing immediately or the morning after the injury and late onset PCS (i.e. irritability, anxiety, depression, poor memory, poor concentration, sleep disturbance, fatigue and hearing or visual disturbance), those which develop a few weeks or months later (King, 1997). Consistent with this classification, in the present study with the exception of the symptom "dizziness", more late onset symptoms were reported (i.e. fatigue, difficulty falling asleep, sleeping less, drowsiness, irritability, difficulty concentrating, difficulty remembering, visual problems and difficult hearing) than early onset PCS (i.e. headache, balance problems and dizziness). Across the two inventories and in respect of the between and *within* groups analyses for the Contact and 2+ Concussion groups, the most commonly reported symptom was fatigue, followed by the symptoms of aggression and nervousness, dizziness, sleeping less, feeling more emotional, and difficulty concentrating. These symptoms can also be classified according to the symptom clusters identified earlier in studies cited in Chapter five, i.e. somatic (fatigue, dizziness, sleeping less), cognitive (difficulty concentrating), and emotional (aggression, nervousness and feeling more emotional), which report that following MTBI individuals demonstrate a combination of cognitive, somatic and emotional effects (e.g. S.D. Anderson, 1996; Cicerone & Kalmar, 1995; King, 1997).

In comparison to investigations which have reported on the neurocognitive functioning of athletes following MTBI, relatively fewer MTBI studies have investigated PCS reporting amongst athletes. Of the sports studies reviewed in Chapter six which have included PCS

inventories in their studies, most have reported on symptoms in the acute condition, with fewer studies investigating persistent effects, and with particular reference to high school athletes (see Chapter six). In line with the most frequently reported symptoms (i.e. fatigue, aggression, nervousness, feeling more emotional, difficulty concentrating, dizziness and sleeping less) in respect of the Contact and 2+ Concussion groups in the present study, the following studies have also reported some of these PCS in their investigations of MTBI in sport in both younger and older athletes.

Jordan et al. (1997) reporting on professional boxers, used a Chronic Brain Injury scale which included symptoms such as agitation or aggression, delusions, hallucinations, dysphoria, anxiety, euphoria, apathy, disinhibition, irritability or lability. Their results showed that high exposure boxers had significantly higher CBI scores compared to those boxers with minimal exposure. Barnes et al. (1998) in their study on soccer players found that headache was the symptom that was most commonly reported in both gender groups (54 male, 55 female). Men reported more symptoms of blurred vision and numbness and tingling whereas females reported more symptoms of feeling dazed and dizzy. Abreau et al. (1990) in their study investigating the effects of neuropsychological functioning amongst college soccer players, demonstrated that the soccer players consistently reported more symptoms of headache, blurred vision and dizziness than controls. In a study investigating the effects of MTBI on neuropsychological functioning amongst professional and high school Rugby Union players, although more symptoms were reported amongst the professional players, both groups reported significantly more symptoms relative to the controls, with high school athletes reporting symptoms of clumsy speech, sleep difficulties, clumsiness, easily angered and hearing problems (Shuttleworth-Edwards et al., 2004). Wilberger et al. (1991) reported that at three months post-injury high school athletes were still reporting more symptoms of headaches, dizziness and poor concentration relative to controls, illustrating prolonged effects in high school athletes.

The MTBI literature reports that a prior history of MTBI increases the likelihood of persistent PCS (Carlsson, Svardstudd, & Welin, 1987; Lishman, 1988; Ryan & Warden, 2003; Sortir, 2001). The following studies support the present study in respect of concussion history, where athletes with a history of two or more and three or more concussions reported more PCS relative to groups reporting no concussion history. For example, Macciocchi et al. (2001), investigating

the acute and chronic effects of concussion amongst American college football athletes demonstrated that players who sustained two or more concussions reported more symptoms than those athletes who sustained a single concussion, with headache being the most commonly reported symptom. Similarly, Moser et al. (2005) investigated the effects of concussion amongst high school athletes and found that those athletes with a history of two or more concussions experienced prolonged neuropsychological deficits (no symptoms were specified) in comparison to those athletes with no concussion history. Researchers Gaetz et al. (2000) investigated the effects of MTBI amongst junior ice hockey athletes and showed that athletes who sustained three or more concussions reported more symptoms (memory and taking longer to think) relative to the controls. Further researchers Iverson et al. (2004a), demonstrated that at baseline, high school and college athletes with a history of three or more concussions reported more symptoms than athletes with no prior history of concussion (no symptoms, other than 'fogginess' specified). Similarly, in a study by Guskiewicz et al. (2005), retired athletes with a history of three or more concussions has a threefold prevalence of reported memory problems compared with retired athletes with no history of concussion. Thus, the above research findings support the PCS findings in the present study in that increased symptom reporting in these studies is suggestive of persistent PCS sequelae as a result of cumulative MTBI sustained as a consequence of exposure to a contact sport.

In the present study the most commonly reported symptom amongst the Contact and 2+ Concussion groups was 'fatigue' followed by the symptom 'aggression'. Whilst the symptom 'fatigue' appears to be included in many of the PCS inventories reviewed above (Collins, Field, et al., 2003; Collins, Grindel, et al., 1999; Ferguson et al., 1999; Field et al., 2003; Guskiewicz et al., 2000; Jordan, Green et al., 1996; Iverson et al., 2002; Iverson et al., 2004a, 2004b, 2006; Killam et al., 2005, Lovell et al., 2003; Lovell, Collins, Iverson, et al., 2004; McClincy et al., 2006; Mihalik et al., 2005; Pellman et al., 2006; Shuttleworth-Edwards et al., 2004; Shuttleworth-Jordan et al., 1993; Wilberger et al., 1991) to the author's knowledge the symptom 'aggression' in comparison has rarely been included in inventories, being identified in the inventories of only two sets of researchers (Jordan et al., 1997; Shuttleworth-Edwards et al., 2004; Shuttleworth-Jordan et al., 1993). This becomes an important issue given that it was the second most frequently reported symptom by athletes in this study.

In the present study the symptom 'aggression' was one of the symptoms incorporated into the Postconcussion Symptom Questionnaire and excluded from the ImPACT Postconcussion Symptom Scale (i.e. speech problems, poor appetite, hearing difficulties and aggression) and appeared to be significant in respect of the between and within groups analyses for the Contact and 2+ Concussion groups at both the pre- and post-season intervals. The only exception was for the *within* 2+ Concussion groups analyses at pre-season, where the symptom 'aggression' was not significant but the trends were in the right direction with the 2+ Concussion group reporting more symptoms of aggression than the 0 Concussion group. These significant findings are consistent with past research investigating the neuropsychological effects of MTBI in respect of Rugby Union players across all level of play (Shuttleworth-Edwards et al., 2004). In this study Rugby Union players reported greater percentages of aggressive type behaviour relative to the controls.

One could postulate that aggression is a pre-selected condition specific to athletes involved in contact sport in that they may generally constitute more aggressive personality types per se and are therefore more inclined to choose contact sports over non-contact sports. In the present study the symptom 'aggression' was consistently reported on the Postconcussion Symptom Questionnaire for the between groups analyses and in the *within* Contact group analyses. Whilst the symptom 'aggression' was significant at both the pre- and post-season intervals on the between groups analyses it was not significant in respect of the *within* Contact group analysis at the pre-season interval, although the trends were in the direction of the 2+ Concussion group reporting more symptoms of aggression than the 0 Concussion group. This would suggest that this finding has nothing to do with participation in a contact sport as opposed to a non-contact sport since *within* the Contact group one is seeing the same effects attributed to those athletes who sustained 2+ Concussions versus 0 Concussions. One still cannot entirely rule out a pre-selected effect, however, since those who sustain concussions may tend to be more aggressive because they are more inclined to put themselves into those high risk situations. Importantly, although the effects seen in contact sports players as well as those who have sustained two or more concussions may be due to a pre-selected sampling effect, one also needs to consider the organic basis of aggressive type behaviour and to what extent the symptom may be characteristic of frontal-type pathology associated with closed head injury (Lezak et al., 2004).

The association between frontal lobe dysfunction and aggressive type behaviour has been supported in research studies investigating the development of personality change following brain injury (e.g. Brower & Price, 2001; Max et al., 2000; Tateno, Jorge, & Robinson, 2003; Wood & Liossi, 2006). According to a recent study, aggressive behaviour was found in 33.7% of patients following TBI (Tateno et al., 2003). Personality change due to TBI is characterized by persistent personality disorder and is listed in the DSM-IV as consisting of five major subtypes which have been described as labile, aggression, disinhibited, apathetic, and paranoid (DSM-IV, 1994). In a sample of 57 mild to moderate TBI and 37 severe TBI children and adolescents, personality change occurred in 5% of the sample of mild to moderate TBI patients although it was always transient, whilst severe TBI patients seemed to suffer from persistent personality change (Max et al., 2000). However, Ganesalingam, Sanson, Anderson, and Yeates (2006) report that two to five years post injury significant deficits in the self-regulation of social and behavioural functioning were frequently reported in a study following moderate to severe childhood TBI, including increased aggression and poor temper control, and it is suggested from their research that such difficulties tend to increase over time and are not associated with injury severity. In a review by Silver, Yudofsky and Anderson (2005), it is reported that studies specifically in respect of MTBI and that have evaluated individuals at one year post injury, produce estimates of irritability, temper or agitation ranging from 5% to 70%. These authors also suggest from the research of Greve et al. (2001), that risk factors for such effects may include irritability, impulsivity, and a pre-injury history of aggression.

Given that lingering post-concussive symptoms generally are acknowledged to occur in association with cumulative sports concussion (Carlsson, Svardsudd, & Welin, 1987; Lishman, 1988; Ryan & Warden, 2003; Sortir, 2001), albeit not specifically researched one could postulate that aggressive behavioural effects might also persist, and in terms of the literature of Ganesalingam et al. (2006) cited above, might even become worse over time without any added brain trauma. However, a concerning factor is that one could further postulate that contact sports players who have aggressive personality types per se, and who in addition are exposed to multiple concussive events, would exhibit further aggressive type behaviour due to an additive organic effect. Whilst some authors argue that PCS may begin on an organic basis but that psychological factors contribute to their persistence (Levin et al., 1982), one could argue the converse, i.e. that symptoms begin on a psychological basis but that organic factors may

contribute to their persistence. Thus in this case one could postulate that individuals who are generally more aggressive types and who are drawn to more aggressive sports are at risk of prolonging, if not increasing their aggression levels through organic consequences of exposure to multiple concussive events. This supposition is particularly compelling given the suggestion of Silver et al. (2005) cited above, that risk factors for aggressive behaviours following TBI include a pre-injury history of aggression.

In summary, the PCS findings in respect of the ImpACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire provide relatively strong validation for each other and support the research hypotheses in that on both the between and within groups analyses all significant symptoms and trends were predominantly in the direction of the Contact and 2+ Concussion groups reporting more symptoms than the Non-Contact and 0 Concussion groups. The results are further supported by prior research (Abreau et al., 1990; Barnes et al., 1998; Ferguson et al., 1999; Gaetz et al., 2000; Guskiewicz et al., 2005; Hatfield et al., 2004; Iverson et al., 2006; Jordan et al., 1997; Killam et al., 2005; Moser et al., 2005 and Shuttleworth-Edwards et al., 2004) reporting on the long-term effects of MTBI in athletes and which have demonstrated an increase in symptom reporting suggestive of persistent PCS sequelae as a result of cumulative MTBI sustained as a consequence of exposure to a contact sport.

Given the fact that all arguments up to now have suggested that the deleterious neuropsychological consequences of concussion are relatively subtle at the high school level due to minimal exposure to the sport and due to the fact that the present sample can be seen as being a relatively protected one in respect of BRC theory (i.e. male athletes, with no psychiatric or neurological disorders and with a relatively high level of education and estimated IQ level), in comparison to the neurocognitive findings reported above, however, the PCS findings of the present study appear to be substantial. These findings are consistent with Shuttleworth-Edwards et al's (2004) study across the different levels of play where although more cognitive deficits and symptoms were reported for the older, professional athletes, amongst the high schoolboy rugby sample significantly higher percentages of symptoms were reported relative to the controls and relative to deficits reported on only one trial of a cognitive measure (hand motor function task).

In light of the findings for the present study, one could postulate that contact sports players and those athletes with a history of two or more concussions as a group are going to be reporting more symptoms as a result of being more symptom prone or as a consequence of increased exposure to a contact sport. Because of the considerable trends in the cognitive arena in the direction of the Contact and 2+ Concussion group performing worse than the Non-Contact and 0 Concussion group in addition to increased symptom reporting (many of which are replicated in the MTBI literature) this adds to the overall likelihood that what is being seen is rather as a consequence of increased exposure to concussive and sub-concussive events than as a result of being more symptom prone. Furthermore, it seems unlikely that the Contact and 2+ Concussion history groups as a whole would demonstrate deficits on a cluster of cognitive, somatic, emotional and behavioural effects sensitive to brain injury when factors such as age, education and IQ level are all similar.

### **9.3.2 Dependent t-test analyses**

The findings on the individual symptoms between the Contact group pre- versus post-season and the Non-Contact group pre- versus post-season on both the ImPACT Postconcussion Symptom Scale and the Postconcussion Symptom Questionnaire reveal instances of significantly higher reporting of symptoms for both Contact and Non-Contact groups at the post-season interval than at the pre-season interval with no significant results in the opposite direction (i.e. the Contact and Non-Contact groups reportedly being worse at pre-season than at post-season). The trends analyses also reflect a consistent trend in the direction of both the Contact and Non-Contact groups reportedly being worse in respect of more symptoms at post-season than at pre-season, across all analyses.

The 22-item ImPACT Symptom Scale elicits an equal number of significant symptoms being worse at post-season for the Contact and Non-Contact groups (four and four symptoms, respectively). In respect of the trend analyses, there was a predominant trend for the Non-Contact group reportedly being worse in respect of reporting more symptoms at post-season than at pre-season (17 out of the 22 symptoms), whilst there was a marginally predominant trend with the Contact group reportedly being worse in respect of reporting more symptoms at post-season than at pre-season (14 out of the 22 symptoms). Thus, there is no difference here between the Contact

and Non-Contact groups which does not support the hypothesis that the Contact group would be performing worse in comparison to the Non-Contact group.

On the other hand the analysis using the brief nine item Postconcussion Symptom Questionnaire elicited a powerful indication of worsening for the Contact group at post-season compared with minimal worsening for the Non-Contact group at post-season (total symptom score and five out of the nine symptoms for the Contact group, versus only one symptom for the Non-Contact group). The overall trends analyses also, revealed a predominant trend for both the Contact and Non-Contact groups reportedly being worse in respect of reporting more symptoms at post-season than at pre-season (seven out of the nine symptoms). Thus, although both Contact and Non-Contact groups performed worse at post-season than compared to pre-season, the significantly worsening of the Contact group relative to the Non-Contact group implies an increased vulnerability for the Contact group as a result of postulated exposure to concussive and sub-concussive effects sustained over a six month winter sports season.

The Postconcussion Symptom Questionnaire appears to have been substantially more sensitive to raised symptoms amongst the Contact group than the ImPACT Symptom Scale. Furthermore, the Postconcussion Symptom Questionnaire which was reduced from 26 symptoms at the pre-season interval to only nine symptoms at the post-season interval appears to have been particularly sensitive at the post-season interval in the two independent between groups analyses reported above, where eight out of nine and six out of nine symptoms respectively, reached significance. This is also the case for the dependent analyses where on the Postconcussion Symptom Questionnaire the results for the Contact group are similar to the results for the Non-Contact group, in respect of a predominant trend in the direction of both Contact and Non-Contact groups presenting with more symptoms at post-season than at pre-season. However, the total symptom score for the Contact group reveals a highly significant incidence of symptoms at the post-season interval that is not present for the Non-Contact group, including significantly worsening for five symptoms for the Contact group at post-season, compared with worsening for only one symptom at post-season for the Non-Contact group.

The significant worsening of symptom reporting for both the Contact and Non-Contact groups on both PCS inventories could be due to a repeat test artefact which results in higher reporting of

symptoms purely by virtue of a second exposure to the questionnaire, or it could be due to the current emotional and psychological status of the athletes evaluated at that time of the study, or a combination of both. In respect of the latter emotional explanation, the sample employed in the present study consisted of all final year high school athletes. The final year of high school, commonly referred to as Matric in South Africa, is an extremely stressful and demanding year for all the learners concerned. During this final year the learners are exposed to three sets of intensive examinations occurring between the months of May and June, August and September and the final examinations in November. In the present study the post-season testing coincided with the preliminary examinations in September. Many of the learners enlisted into the research were relying on the results of their preliminary examinations for placement in tertiary educational programmes. As a result of the added pressure incurred by the examinations this could have resulted in an increase in symptom reporting for both the Contact and Non-Contact groups.

For the Contact group the significant symptoms reported on the ImPACT Postconcussion Symptom Scale at post-season relative to pre-season were for the symptoms of nausea, balance problems, feeling slowed down and feeling mentally foggy and on the Postconcussion Symptom Questionnaire these were for the total symptom score, poor appetite, feeling slowed down, mentally foggy, difficulty remembering and speech problems. In respect of the Non-Contact group significant symptoms reported on the ImPACT Postconcussion Symptom Scale at post-season relative to pre-season were for the symptoms nausea, sleeping more than usual, sensitivity to noise and mentally foggy and on the Postconcussion Symptom Questionnaire this was for the symptom difficulty hearing. Whilst it can be argued that the symptoms reported by the athletes in both groups could largely be due to stress, it is interesting to see that a higher proportion of the symptoms more commonly reported by athletes following MTBI (e.g. balance problems, feeling slowed down, feeling mentally foggy, difficulty concentrating, nausea) were reported by the Contact group at post-season, with the symptoms 'feeling slowed down' and 'feeling mentally foggy' being reported on both inventories. Furthermore, all the significant results and trends are predominantly in the direction of a worsening of symptom reporting amongst the Contact group at post-season. Since arguably all participants would have been exposed to the same amount of work stress during the final year of high school, one could therefore postulate that in addition to this stress the cumulative exposure of unreported concussive and sub-concussive effects over a

six month season could have resulted in an additive effect causing persistent effects in respect of an increase in significant symptom reporting for the Contact group relative to the Non-Contact group.

In summary, the findings across both inventories do not support the hypothesis of poorer symptomatic outcome at post-season relative to pre-season, in that both Contact and Non-Contact groups demonstrate a worsening in respect of increased symptom reporting at pre-season relative to post-season. Whilst it has been postulated that the findings in both groups could be in response to a test-retest artefact or to the stress incurred as a result of being in the final year of high school, overall across both inventories the Contact group demonstrated significantly more symptoms than the Non-Contact group. Moreover, in respect of the inventories employed, more significant pre- versus post-season worsening of symptoms were reported by the Contact group on the Postconcussion Symptom Questionnaire in comparison to the Non-Contact group. This increase in significant symptom reporting in respect of the Contact group could be the result of an additive effect of experiencing stress due to the pressures of work plus the cumulative effects of concussive and sub-concussive events experienced over a six month winter sports season, as well as the effect of being cerebrally more vulnerable due to past participation in a contact sport.

#### **9.4 SYNTHESIS OF FINDINGS FOR THE COGNITIVE AND PCS MEASURES**

The overall findings for the independent analyses in respect of the neurocognitive and PCS measures for the between and within Contact and 2+ Concussion groups versus the Non-Contact and 0 Concussion groups do support the hypotheses in that all significant results were in the direction of the Contact and 2+ Concussion groups performing worse than the Non-Contact and 0 Concussion groups at both the pre- and post-season occasions. The overall trends also support the hypotheses in that all were in the direction of the Contact and 2+ Concussion groups performing worse than the Non-Contact and 0 concussion groups at both the pre- and post-season occasions, with the only exception being for the *within* Contact group analyses in respect of the cognitive measures at pre-season, where an overall opposite trend occurred (0 Concussion group performing worse than the 2+ Concussion group).

In respect of the neurocognitive findings for the between and within groups analyses reported on above there was very little significance and only in respect of the between groups analysis for the Contact group performing worse than the Non-Contact group at the pre-season interval on the cognitive measure Reaction Time ( $p = 0.014$ ) which presented as being more cognitively challenging to vulnerable participants than the other tasks. Caution needs to be adopted in over-emphasizing this significant finding in that it could be a random finding due to chance. However, in that all the trends (with the exception of the within group analysis at pre-season) are in the direction of the Contact and 2+ Concussion groups performing worse than the Non-Contact and 0 Concussion groups it would appear that this finding is not a chance effect but rather supports the hypothesis in respect of deleterious effects for both the Contact and 2+ Concussion groups. In addition to the neurocognitive findings, the PCS findings in respect of both between and within groups analyses provide substantial support for the hypothesis with significantly increased symptom reporting on both Contact and 2+ Concussion groups relative to the Non-Contact and 0 Concussion groups. In summary, it is therefore unlikely that these results are due to chance due to the cross-validation of findings across two vastly different forms of measurement (objective neurocognitive tests and self-reporting symptoms) all occurring in the same direction.

Thus, in support of the hypotheses, the significant results and overall trends for the independent analyses on the neurocognitive and PCS measures at both pre- and post-season, provide cross-validation for each other, and together demonstrate that athletes exposed to a contact sport as opposed to a non-contact sport generally tended to reveal deleterious neuropsychological sequelae as a result of postulated exposure to the effects of concussive and sub-concussive events over a high school career. Further, those athletes with a history of two or more concussions as opposed to those with no concussion history performed worse on neurocognitive testing and PCS inventories as a result of postulated additive, cumulative effects of being exposed to multiple concussions over a high school career.

Consistent with past research reporting on the neurocognitive effects of concussion amongst high school athletes (Lovell, Collins, Iverson, et al., 2004; Moser et al., 2005; Shuttleworth-Edwards et al., 2004; Webbe & Ochs, 2003; Wilberger et al., 1991; Witol & Webbe, 2003), the neurocognitive findings in respect of the Contact and 2+ Concussion groups appear to be subtle and less in evidence in comparison to older athletes due to the fact that high school athletes have

had minimal exposure to the concussive and sub-concussive cumulative effects of concussion compared with older athletes. In comparison to the neurocognitive findings, the PCS findings in respect of both PCS inventories appear to be substantial for both Contact and 2+ Concussion groups illustrating that these groups are reporting more symptoms in comparison to the Non-Contact and 0 Concussion groups. This suggests that the deleterious effects due to postulated exposure to the concussive and sub-concussive effects appear to be more pronounced in terms of symptomatic change rather than cognitive change. Thus, it would appear that these effects identified in the present study, although subtle especially on the neurocognitive dimension, are already initiated at this late high school stage in an athlete's career suggesting that as the number of years of exposure to contact sport increases so too does the risk of cumulative, deleterious effects of concussion.

With respect to the dependent analyses, the findings for the neurocognitive test performance between pre- and post-season do not provide support of worsening of neurocognitive performance in the Contact group compared with the Non-Contact group due to cumulative insults incurred during the season. However, they do provide support for the underlying theoretical hypothesis that exposure to cumulative MTBI in a contact sports group will result in compromised neurocognitive function. This is because on two tests with known sensitivity to diffuse brain damage that typically accompanies the concussive brain injury (ImPACT Visual Motor Speed composite and Digit Symbol Substitution Incidental Recall delayed form), the Contact group failed to benefit significantly from practice on repeat testing following a six month interval, whereas the Non-Contact group did. The implication is that the Contact group have succumbed to deleterious effects of cumulative concussive and sub-concussive insults over years of exposure to the sport as well as possible more immediate deleterious effects sustained during a season of participation in such sport, or a combination of both of these factors.

In respect of the results on the PCS measures for the dependent analyses, these do not support the hypotheses in that both the Contact and Non-Contact groups reported more symptoms at the post-season interval relative to the pre-season interval. The abbreviated Postconcussion Symptom Questionnaire in comparison to the ImPACT Symptom Scale however, appears to be more sensitive to the effects of concussion in that it revealed a greater number of significant

results indicating increased symptoms for the Contact group compared with the Non-Contact group at the post-season relative to the pre-season intervals.

Taking all the analyses together, the ImPACT neurocognitive measures, Reaction Time and Visual Motor Speed and the paper-and-pencil measure, DSSIR-delayed task, appear to be the most challenging neurocognitive tasks, and this is consistent with research indications that suggest their particular sensitivity to diffuse brain injury (Collie et al., 2006; Collins, Field, et al., 2003; Cremona-Meteyard & Geffen, 1994; Downs & Abwender, 2002; Gaetz et al., 2000; Iverson et al., 2004b; Lovell, 2004; Maddocks & Saling, 1996; Makdissi et al., 200; Moriarity et al., 2004; Ramirez et al., 1998; Warden et al., 2001). In respect of the PCS findings on both inventories, the symptoms tended to be those most commonly reported following concussion. The most predominant finding of the symptom 'aggression' appears to be most explicable as a combination of a pre-selected as well as an organic effect. In the final analysis on this cross-sectional study one cannot exclude the possibility that all the results are due to pre-selected sampling effects. This seems unlikely however, due to the conceptual coherence of the results in terms of expected neurocognitive and PCS outcome following repeated MTBI.

Finally, the above findings in respect of both neurocognitive and PCS measures on the dependent analyses, not only provide cross-validation for each other but also for the general direction of results for the neurocognitive and PCS on the independent test analyses. These results cumulatively provide compelling evidence for the presence of deleterious sequelae as a result of exposure to cumulative concussive and sub-concussive insults over years of participation in a contact sport as well as the more likely immediate deleterious effects sustained during a season of participation in such sport. In respect of BRC theory these effects appear relatively more subtle amongst this younger group of athletes owing to minimal exposure to concussive and sub-concussive effects as well as the fact that the sample as a whole is a relatively protected one (i.e. male athletes, no history of psychiatric or neurological disorders, relatively high level of education and estimated IQ level). Thus, it is likely that these protective factors served to buffer the exposure to multiple concussive trauma in this particular high school contact sport group. Given the expected subtlety of effects, this study can be seen to be particularly prone to Type II error such as may be problematic in MTBI research and neuropsychological research involving clinical populations in general (Demakis, 2006; Frencham et al., 2005; Reitan & Wolfson,

1999; Ruff, 2005; Woods, et al. 2006), and therefore increasing statistical stringency via Bonferroni's adjustment for multiple measures was considered inappropriate for this research. Rather, highly consistent findings albeit subtle need to be carefully extrapolated as has been done in the interpretation of the results of this research, and used heuristically as the basis for further research.

### **9.5 COMPARISON OF SOUTH AFRICAN AND UNITED STATES HIGH SCHOOL MALE NORMS ON THE ImPACT NEUROCOGNITIVE TEST BATTERY**

Normative data for South African high school male athletes were obtained from the performances of the Non-Contact athlete group on the composite neurocognitive test scores and the Postconcussion Symptom Scale total score included in the ImPACT test battery at the pre-season interval. For the purpose of this examination the pre-season cognitive composite scores of the Non-Contact group are delineated below in Table 9.5.1 together with the average ranges reported for the US sample. Perusal of Table 9.5.1 illustrates that the normative data obtained on a sample of male high school athletes in the United States is highly commensurate with the normative data derived for the South African high school male Non-Contact sports sample of the present study, in that all South African composite scores fell within the United States control group's average range. The similarity in cognitive findings between the South African high school population and the US high school population, demonstrate that both sample groups appear to be of similar education and IQ, as well as come from relatively similar socio-economic backgrounds. These findings are useful when applied to the South African context in that the normative data may be important for use on Grade 12 high school male athletes in South Africa.

**Table 9.5.1**  
**Pre-season Non-Contact group cognitive mean scores for high school male athletes in comparison with age equivalent US average normative ranges on the ImPACT test**

	<b>SA controls N=74</b>	<b>US control ranges N=158</b>
<b>Verbal Memory</b>	85.41	80 - 92
<b>Visual Memory</b>	77.77	71 - 88
<b>Visual Motor Speed</b>	37.79	33.7 - 42.5
<b>Reaction Time</b>	0.60	0.58 - 0.50

The findings also support the fact that the South African sample is not considered to be disadvantaged in comparison to first world high school standards in that one would expect the scores to be lower as a result of lower education, IQ or socio-economic levels and that cognitively their level of functioning is on the same level as an average American high school athlete.

In respect of the PCS findings obtained for the Non-Contact athletes on the ImPACT Symptom Scale (n=74), however, perusal of Table 9.5.2 illustrates that the average total symptom score reported by South African male athletes ranges from 10 to 15. In comparison the average total symptom score reported by high school male athletes in the United States (n=588) ranges from 1 to 6. Thus, if one were to compare the average total symptom score reported by American high school male athletes to the average total symptom score reported by South African male athletes it would appear as if South African high school male athletes have a general tendency to report more PCS than the average American high school male athlete. This is an interesting finding which may reflect the cultural differences between the two high school groups. As such differences in symptom reporting could reflect the different set of stresses imposed on high school children in South Africa or the different ways in which South African high school children respond to or cope with different stressors in comparison to the high school children in the United States.

**Table 9.5.2**  
Pre-season Non-Contact group total symptom raw scores for high school male athletes in comparison with age equivalent US average ranges on the ImPACT test

Classification	SA controls N=74	US Raw scores N=588
Low-Normal		0
Normal		1-6
Unusual	12.50	7-13
High		14-21
Very High		22+

(Lovell et al., in press)

In South Africa, as already mentioned earlier, the final year of high school is extremely stressful and is characterized by a series of intense examinations throughout the year, which will determine what tertiary programme the learner will be permitted to take. Considering that this is the final year of high school for the sample in the present study, the stress imposed as a result of examination pressure could have also resulted in more symptom reporting in this group as a whole. Conversely, the low average total symptom score reported by high school athletes in the United States may also reflect the underreporting of symptoms by athletes in that country, or a more stoic and less open attitude towards symptom reporting generally than amongst South African adolescents. Further investigation specifically in respect of cross-cultural differences in symptom reporting on normal populations is needed.

## **9.6 CRITICAL EVALUATION OF THE STUDY**

This research study sought to investigate the long-term neuropsychological effects of repetitive mild traumatic brain injury sustained as a consequence of participation in a contact sport amongst final year high school learners. Few studies up to now have focussed on the long-term neuropsychological effects of concussion amongst high school athletes, comprising only seven (Barr, 2003; Lovell et al., 2003, Lovell, Collins, Iverson et al., 2004; Moser et al., 2005; Shuttleworth-Edwards et al., 2004; Stephens et al., 2005; Wilberger et al., 1991) and only one of these studies demonstrated strict control for age, education and IQ (Shuttleworth-Edwards et al., 2004). None of these studies used a combination of both paper-and-pencil and computerized neurocognitive and postconcussion symptom inventories in their investigations of concussive and sub-concussive effects in this age group. Thus, in light of the above, it was decided to conduct an extensive investigation into the long-term consequences of male high school participation in a contact sport using a cross-sectional quasi-experimental design which was well controlled for age, gender, level of education and estimated IQ and employing both paper-and-pencil and computerized neurocognitive and postconcussion symptom inventories.

With respect to the above, the present study will be evaluated in the following way: (i) research design, (ii) research sample, (iii) comparative groups, (iv) research measures and (v) the potential for both Type I and Type II errors.

### (i) Research Design

A cross-sectional quasi-experimental design was employed for the purpose of this study. The study was quasi-experimental in that the participants were not randomly selected and already belonged to 'pre-selected' groups owing to their sports affiliation (Contact versus Non-Contact) and concussion history (2+ Concussion versus 0 Concussion). In order to evaluate the long-term concussive effects of being exposed to a contact sport as opposed to a non-contact sport, a cross-sectional research design was employed. In addition, because the sample was evaluated at post-season in order to investigate whether any deleterious effects that may have occurred over a six month sports season, it can be argued that the study is also slightly prospective. One major disadvantage of cross-sectional research is that the findings, in respect of the sample group, evident on objective testing could be pre-existing. Whilst one can never definitively rule this out, considering that the comparative sample groups were evaluated at both pre- and post-season and that the significant results and trends were all predominantly in the direction of a worsening for the Contact and 2+ Concussion groups relative to the Non-Contact and 0 Concussion groups on both neurocognitive and PCS measures, this seems unlikely to be the case. Furthermore, all groups were well matched for age, level of education and estimated IQ which supports the hypotheses in that the effects evident on testing are likely to be as a result of the concussive and sub-concussive effects due to exposure to a contact sport over a high school career and not to pre-existing conditions.

For the comparison of the different sports groups (Contact versus Non-Contact) a non-equivalent control group design was used in respect of the different sports groups for both pre- and post-season test measurement. Further, when comparing the different concussion history groups (2+ Concussion versus 0 Concussion) a non-equivalent comparison design was used in respect of the distribution of participants across sports types within the two comparison groups, for both pre- and post-season test measurement. In order to improve on the non-equivalence of this comparison in respect of sports types, a further analysis was performed on equivalent groups in respect of sport (equivalent control group design) comparing individuals who sustained 2+ Concussion versus 0 Concussion *within* the Contact group. The non-equivalent control group design is a pre-test, post-test design which uses a matched control for comparative purposes and is a powerful design in respect of providing the strongest inference about the relationship between concussions and neurocognitive and postconcussive sequelae (Macciocchi & Barth,

2004). Limitations however in respect of this design are practice effects that may occur between the testing intervals. This was evident in the present study in respect of the dependent group analyses for the Non-Contact group at the post-season interval. However, this potential limitation is alleviated by virtue of having a control group, and in this instance served to provide further compelling support for the underlying hypothesis in the present study. Here, the lack of practice for the Contact group at the post-season interval suggested deleterious effects as a result of concussive and sub-concussive effects sustained during a season of participation in a contact sport.

#### (ii) Sample

The sample employed for the purposes of this research was well-controlled for age, gender, level of education and estimated IQ. The sample size for all comparative groups was adequate ( $n > 30$ ) and all athletes were restricted to one level of play consisting of all high school athletes. Thus, the athletes were subjected to a similar length of exposure to a contact sport. The sample did not consist of only top team level athletes and arguably one may have been able to have seen more effects had only the top teams been included in the sample in that the level of play is generally faster and harder. Whilst factors such as age, level of education, IQ and gender are controlled for in the group analyses, it is questionable whether one can truly say that the only differences between the groups is due to sports participation and concussion history. Researchers are beginning to examine the possibility that differences in cognitive style or personality traits may also explain the variability between the test scores in contact sports players or visa versa, that differences in cognitive styles occur as a consequence of MTBI (Rutherford et al., 2003). For example, one could postulate that contact sports players are generally more impulsive than other athletes and would thus be more prone to making errors on performance tasks. However, when selected measures known to be sensitive to neuropsychological consequences of repeated MTBI show a tendency to be impaired (such as occurred in the present study), and these same impairments all revealed more findings in older cohorts after more years of exposure to a contact sport, the argument that all these effects can entirely be accounted for by pre-selection factors becomes less compelling.

The sample employed for the purposes of this study consisted of relatively high functioning, predominantly white (at least 90%), middleclass, English speaking participants and as a result

the sample is not truly representative of final year high school athletes in South Africa as a whole. South Africans have been referred to as the 'rainbow nation', which reflects the country's cultural diversity. The South African population totals about 45 million of which nearly 31 million are Blacks, five million are Whites, three million are Coloured and one million Indian (South African Government on line, n.d.). As a result of the diverse populations of varied cultural and socioeconomic backgrounds housed within South Africa, the present study would only represent a small proportion of high school athletes in the country and therefore the findings could not be generalized to other socio-economic and cultural groups within the country. With a more varied population of athletes where the control for IQ, educational attainment and socioeconomic status is less restrictive at a relatively high level, the neuropsychological effects may have been more apparent, in that in terms of the BRC theory these factors would have presented as vulnerability factors serving to lower pre-existing functional thresholds in athletes. In terms of BRC theory the implication is that these effects derived in respect of the current sample were relatively subtle and may have been substantially more pronounced on a less culturally advantaged sample. However, this would be subject to confirmation on further research. Furthermore, the sample also consisted exclusively of male athletes and owing to the known sex differences on testing reported amongst researchers in sport (e.g. Barr, 2003; Barnes et al., 1998; Downs & Abwender et al., 2002; Putukian et al., 2000), the findings cannot be generalized to the female population.

Whilst it can be assumed that by virtue of the nature of a contact sport, the risk of sustaining a concussion and even multiple concussions over the course of an athlete's career is very high, factors such as the player's position (Jakoet & Noakes, 1998), and the characteristics of the sport itself, for example heading amongst soccer players (e.g. Rutherford et al., 2005; Witol & Webbe, 2003) may contribute to this risk. In the present study, in respect of the contact sports of Rugby Union and soccer, the differentiation between players position in Rugby Union, between the forwards and backs (generally it is assumed that the forwards sustain a higher degree of injury than the backs), and between those soccer athletes who were headers and those who were not, was not made owing to the fact that generally at the school level positions are less entrenched than compared to the professional level. It was thus assumed that any effects would be balanced across the groups due to players alternating their playing positions. The above factors, should

however be considered in future investigations of MTBI in contact sport as they may also contribute to the variability of results demonstrated within and between sports studies.

Both the Contact and Non-Contact groups consisted of athletes participating in various different sports. In particular, the Contact group consisted of athletes participating in the sport of Rugby Union, soccer and the martial arts. Ideally, it would have been better to have had all athletes in pure groups because the incidences of concussion may be different in respect of the different sports, i.e. the incidence of concussion amongst Rugby Union players is known to be very high and reportedly less than the incidence in soccer. In the present sample however, at pre-season the Contact group ( $n = 115$ ) was made up of 105 athletes participating in rugby, nine athletes participating in soccer and one athlete participating in the martial arts and at post-season the Contact group ( $n = 90$ ) was made up of 81 individuals participating in rugby and nine individuals participating in soccer. Thus, since most of the Contact group consisted of athletes participating in Rugby Union it is possible to attribute the findings largely to concussive and sub-concussive effects due to participation in Rugby Union. It is therefore, not possible to generalize from the findings in the present study to athletes participating in soccer and the martial arts or to any other contact sport not represented due to their under-representation in the present study. The number of participants also decreased from 189 to 154 at the post-season interval due to sample attrition.

### (iii) Comparative sample groups

In the current study athletes were divided into groups on the basis of their sport affiliation and concussion history. In respect of the concussion history groups, these were based on the self-reports of the athletes themselves. Owing to the confusion around the allocation of grades of sports concussion, especially in a retrospective self-report history of sports concussive episodes, no differentiation was made in terms of severity of the sports-related concussions reported by those included in the study, and nor was any differentiation made within the concussion groups in terms of the presence or absence of loss of consciousness, confusion, PTA, or retrograde amnesia reported in association with the concussions. Duration of symptoms was not elicited on the questionnaires, but it is assumed that the concussions would mostly if not entirely have been in the milder spectrum of TBI as is generally the case in sports TBI, with most concussions not involving a loss of consciousness (Lovell et al., 1999).

The concussion history groups were divided into those athletes who reported to have sustained no concussions and those athletes who reported to have sustained two or more concussions. For the purposes of the prospective dependent t-test analyses only pre- versus post-season comparisons between the sports groups were made (Contact and Non-Contact). Due to the reduction in numbers at the post-season interval as a result of sampling attrition, the numbers became too reduced to warrant dependent t-test analyses with this 2+ Concussion group. Further it was considered that concussion history may be a less reliable way of evaluating effects than based on sports affiliation. This is largely due to the fact that concussion history was based on self-reports by the athletes themselves and may have been subjected to considerable bias, with athletes not reporting incidences of concussion history either on purpose or due to their inability to recognise or remember the signs and symptoms associated with concussion or as a result of varying definitions of what constitutes a concussion. Often researchers have suggested this to be a less reliable division (i.e. no concussion versus two or more concussions) than sports groups (Iverson et al., 2006; Macciocchi et al., 2001; Moser et al., 2005), however in terms of the present research the groups appear to be relatively equivalent in that the sports and concussion history groups reported similar findings on the independent t-test analyses, although further research is needed in order to support this on the basis of dependent t-test analyses.

As indicated, the current sample was divided into concussion history groups on the basis of those athletes who reported a history of zero concussions and those who reported a history of two or more concussions, in line with previous and current literature which have supported this division (Collins, Grindel, et al., 1999; Iverson et al., 2006; Macciocchi et al., 2001; Moser et al., 2005). It is possible that dividing groups up on the basis of no concussions versus three or more concussions may have yielded effects, however, due to insufficient numbers the sample could not be divided further in respect of three or more concussions which would have also increased the risk of Type II error (not finding significance when it does exist). Studies which have investigated athletes both in respect of those who have reported two or more and three or more concussions have produced varied results, with some reporting deficits in respect of two or more concussions (e.g. Collins, Grindel et al., 1999; Moser et al., 2005) and three or more concussions (e.g. Gaetz et al., 2000; Guskiewicz et al., 2003, 2005; Iverson et al., 2002, 2004a; McCrory et al., 2000) whilst others not in respect of two or more (e.g. Hinton-Bayre & Geffen, 2004; Iverson et al., 2006; Macciocchi et al., 2001; Maddocks, Saling, et al., 1995) and three or more

concussions (e.g. Pellman et al., 2004). Thus, it would appear that more research is needed to support the delineation of these groups in respect of number of concussions and the deleterious effects produced as a result.

#### (iv) Measures

To the author's knowledge, the current study is the first to include both paper-and-pencil and computerized neuropsychological measures to investigate the chronic and cumulative effects of MTBI amongst high school contact sports players. Despite this, in comparison to the major prior studies investigating chronic MTBI in sports relatively fewer measures were employed (e.g. Collins, Grindel, et al., 1999; Hatfield et al., 2004; Levin, Lippold, et al., 1987; Matser et al., 1998, 1999; Rutherford et al., 2005). In these prior chronic studies cited above, the model appeared to be the use of traditional wide-ranging paper-and-pencil neuropsychological tests, including up to as many as 17 tests. The present study in contrast employed only two paper-and-pencil tests known to be highly sensitive to the effects of diffuse brain damage in association with MTBI (Digit Symbol Substitution Test and Digit Symbol Paired Associate Incidental Recall), in conjunction with the economical ImPACT neurocognitive battery also specifically developed to target functions specific to diffuse brain damage. The reason for this was to provide a more focussed battery that is less prone to Type I error (finding significance when it does not exist). The inclusion of more test measures by researchers implies that their studies are more exploratory in that they are more prone to Type I error, whereas a more focussed battery makes the study less exploratory and thus less prone to Type I error of acquiring significance due to a chance effect.

Whilst more studies reporting on the neuropsychological outcome following MTBI do not include a PCS inventory, in the present study two postconcussion inventories were employed, one as part of the computerized neurocognitive test ImPACT and the other a paper-and-pencil measure. Further additional symptoms were added to the paper-and-pencil inventory which were not included in the ImPACT Symptom Scale and which demonstrated sensitivity to the effects of MTBI in prior research. The inclusion of extra symptoms into the paper-and-pencil inventory made the study more exploratory in that it was hoped that their sensitivity could be demonstrated. The findings of this study showed that the two inventories provided a measure of cross-validation each other, especially in terms of equivalent effects for the total symptom

scores, and to some extent with respect to similar symptoms being reported by the comparative groups on both inventories. Furthermore, the shorter Postconcussion Symptom Questionnaire used at the post-season interval appeared to be more sensitive in respect of eliciting a greater number of significant results than the longer ImPACT Postconcussion Questionnaire, suggesting that the athletes were able to respond with more certainty, and were less likely to provide moderate responses at random on this reduced inventory.

(v) Potential for Type I and Type II errors

Arguably the use of multiple measures on the same data set in this study might warrant the use of Bonferroni's adjustments so as not to incur Type I error (finding significance when it does not exist). However, as indicated in point (iv) above, the potential for Type I error was less in this study in comparison to other studies owing to the relatively fewer numbers of cognitive measures employed in comparison to a wide ranging battery of tests such as used by researchers Matser et al. (1998, 1999, & 2001) and Rutherford et al. (2003, 2005), and therefore Bonferroni's adjustments were not made on account of measurement parameters. Further justification for this lack of adjustment is the argument raised on a number of occasions above, that this study was more at risk of committing a Type II error (not finding significance when it does exist) for the following reasons: In addition to the relatively few measures employed in this research, the participants were expected to demonstrate subtle effects as a result of MTBI in sport at the high school level which are likely to be much less pronounced than at the professional level due to significantly less number of years of participation in the contact sport and therefore less exposure to the acute and sub-concussive effects. Furthermore, in light of BRC theory, the current sample was relatively high functioning and this could have further contributed to the subtlety of the results owing to their increased cognitive reserve. Thus, in the present study owing to the fact that relatively less measures were used in comparison to other MTBI studies, which have demonstrated their sensitivity to the effects of MTBI, the researcher had to find a balance between a lack of Type I error control due to multiple measures and not risking Type II error due to expected subtlety of effects (Johnson & Wichern, 2002).

Amongst the Contact group at least 70% of the athletes reported to have sustained a concussion which means that approximately 30% of these athletes reported to have never sustained a concussion. Because the Contact group did not represent a 'pure' concussion group, this further

contributed to the subtlety of the effects as significant individual effects are likely to be diluted in the analyses of group effects. Thus, in light of these findings it would appear that the risk of making a Type II error in the present study is greater than the risk of making a Type I error, and that making the level of significance too high through Bonferroni's adjustments would have been counterproductive in terms of heuristic possibilities for future research on the basis of any subtle indications of problems due to cumulative MTBI that might emerge from the present research. According to researchers studies such as the present study, which have not strictly implemented Type I control, must be seen as exploratory rather than confirmatory research in that the effects reported as significant do not meet standards for statistical significance although they do illustrate associations which could be investigated in a confirmatory way (Rutherford et al., 2003). However, there a growing tendency in the review literature that alerts to the potential for Type II error in MTBI research and neuropsychological research on clinical populations in general (Demakis, 2006; Frencham et al., 2005; Reitan & Woolfson, 1999; Ruff, 2005; Woods et al., 2006), and provides caution about being prematurely dismissive about the results of such research where there is intrinsically the potential for lack of statistical power in the identification of subtle effects.

## **9.7 IMPLICATIONS OF THE RESULTS**

### **9.7.1 General Implications**

Participation in sport is strongly advocated in all societies where both boys and girls are encouraged at an early age to participate in sport. In most countries it is the contact team sports that are the most popular amongst the athletes and those sports that attract the most attention and interest from supporters. Events such as the FIFA Soccer World Cup, the Rugby World Cup and the Super Bowl are just some of the major sporting events which are expected to draw capacity crowds. Whilst the enjoyment, excitement and challenge to win may represent prime motivations for sport participation, the reality in respect of injury and the risks associated with injury, particularly head injury are largely ignored amongst the younger athlete. The risks and deleterious effects associated with high impact contact sports such as boxing, the martial arts, ice hockey, soccer, American football, Australian Football League, Rugby League and Rugby Union amongst older athletes have been well supported and documented in the literature, whilst

amongst the younger athletes the same risks have not, until recently, received the same amount of interest. For children between the ages of 5 to 15 years rapid cognitive maturation is taking place and any interruptions in the neuronal maturation caused by the trauma can interfere with the normal maturation process (Anderson et al., 2001). Children and teenagers are also more vulnerable to rare but well recognised post-injury sequelae involving diffuse cerebral swelling that usually results in brainstem herniation and death, which is evident in second impact syndrome victims (Theye & Mueller, 2004). Research also supports the fact that younger athletes take longer to recover from concussion than older athletes, which warrants further research in this age group (Field, 2003; Pellman et al., 2006).

In the present study, the overall findings in respect of the significant results and trends for the Contact and 2+ Concussion between and within groups analyses provide subtle but compelling evidence that even at the school level deleterious, residual effects may be in evidence amongst high school athletes. The significant findings and overall trends analyses on both the cognitive measures and postconcussive symptoms provide support for the BRC theory hypothesis which suggests that the additive, cumulative effect of exposure to concussion over a high school career, serves to reduce the existing cognitive reserve in athletes. Owing to the strict control for age, education and IQ estimate, as well as the fact that the sample group has had minimal exposure to concussive and sub-concussive events (compared with adult athletes), the significant findings and trends in respect of the cognitive measures and PCS are subtle and less in evidence in comparison to the findings amongst older, more exposed athletes. Although these effects are more subtle than compared with older athletes, it would appear from the present research that they are already starting to emerge at the high school level. One could further postulate that had the sample consisted of athletes with a relatively low level of education or IQ, or with learning disabilities and neurological or psychiatric problems the findings would have probably been more in evidence due to the additive effect of an already compromised cognitive reserve with further lowering of the functional threshold as a result of exposure to concussive and sub-concussive effects. In addition, had the sample comprised of only top athletes involved in top level sports participation throughout their school careers, which is associated with more intense exposure, the deleterious effects would more likely be pronounced.

For the high school athlete who is preparing for the future, the above findings have important implications for successful achievement at school and may impact negatively upon learning as high school athletes who are exposed to the effects of concussions may have difficulty acquiring new knowledge and attending to schoolwork. Because the effects that have been demonstrated on testing are the combined effects of the sample group, it is difficult to know what the individual effects are. Thus, even though these effects are subtle at the group level it is difficult to know to what extent they impinge on the individual learner's performance in the classroom in particular cases. For example, in a highly competitive academic milieu these effects may take the edge off a very high functioning boy applying for scholarships with long-lasting effects further on in his career or these effects in a learner who may have a borderline pass, may make the difference between him or her passing or failing. Research on relatively subtle neurocognitive impairment in association with HIV-infection indicates that it is associated with impairment on everyday functioning, including unemployment, increased dependence in activities of daily living, and driving ability (Heaton et al., 2004; Marcotte et al., 1999). It is also uncertain to what extent such subtle changes in neuropsychological functioning evident on the findings will impinge on the high school athletes overall emotional, social and cognitive functioning.

The sample employed in this study constituted individuals between the ages of 16 to 18 years which according to theorists represents the latter part of adolescence which is approximately 18 to 22 years (Thomas, 1992). Some researchers believe that the main identity shifts occur in this period. According to Erikson's life-cycle theory, this period corresponds to his fifth stage in the life cycle, 'Identity versus Identity Diffusion' (or role confusion), where world views become important and the adolescent enters a psychological moratorium between childhood security and adult autonomy (Santrock, 1992). For most adolescents this is a time of internal conflict where the individual can temporarily lose their individuality and at the same time are faced with having to make important life altering choices and decisions concerning their future (Thomas, 1992). The implications for those adolescents, from lower socio-economic backgrounds, who struggle through this identity crisis and who over identify with poor social role-models (e.g. delinquents), are problematic in that these individuals may potentially be more inclined to choose more violent and aggressive contact sports over non-contact sports. The additive effects of an already vulnerable youth population (i.e. low education associated with low socio-economic and high delinquent environment) in addition to the exposure to cumulative MTBI as a result of

participation in a contact sport, could potentially be devastating in terms of aggregated effects of further cognitive decline and behavioural changes in the direction of enhanced aggressiveness. Clearly all of these suppositions of everyday consequences of cognitive and emotional/behavioural changes in association with sports MTBI warrant ratification by further research.

Nevertheless, it is important not to be too quick to fall into the trap of Type II error, by failing to take sufficient note of relatively subtle indications of effects such as identified in the present study, since this might cause an underestimate of the risk-benefit ratio of sports involvement and impact negatively on making appropriate clinical interventions (see arguments presented in Woods et al., 2006, in relation to investigations into other clinical entities that may result in subtle yet potentially harmful outcome were it overlooked). It would appear, rather, that the present findings together with the growing body of indications of persistent deleterious effects of sports concussion, call for careful management of the injury in terms of monitoring cognitive decline amongst younger athletes. It is of great importance to baseline individuals in respect of pre-season neuropsychological testing on both neurocognitive and PCS measures, and then to monitor and follow-up players who sustain concussions until such time that cognitively they are performing at or above their baseline scores and are not symptomatic before continuing with sport. When cognitive or emotional effects linger, this would alert to the advisability to withdraw from contact sport. In this way sports with high risk of repetitive concussion can be provided with a greater degree of comfort, in the knowledge that deleterious cognitive and emotional consequences of the type outlined above, are being prevented. Baseline evaluation is also important because it can identify at risk players (i.e. players who are initially compromised in terms of reduced cerebral capacity) who are potentially vulnerable to lowering their already compromised brain reserves due to being exposed to concussive and sub-concussive events.

### **9.7.2 Implications for future research**

Future studies investigating the effects of concussion amongst younger athletes should involve more case-based research. Case-based research will allow for a more in depth analysis in respect of the younger athlete by focussing on the individual effects as opposed to the group effects which obscure possible deficits on the individual basis. Athletes involved in a contact sport but

who have never reported any concussion may serve to further neutralize the individual effects in a group study. Since it is postulated that the inception of the deleterious effects of concussion begins during the early years of exposure to the sport, these groups should become prime targets for ongoing research. Prospective designs where data are collected at various points over a period of time are needed to supplement cross-sectional designs by providing confirmation of insidious changes over time that can be attributed with more certainty to repetitive MTBI rather than pre-selection sampling effects. Thus, in the future it is recommended that more case-based, prospective research designs be employed in order to elicit important information about the athletes thereby documenting changes in cognitive and PCS functioning over time further confirming the indications of this cross-sectional research.

Currently, studies investigating the neuropsychological effects of concussion amongst younger athletes are scarce. It would appear that the only study investigating the effects of concussion amongst junior athletes is that of Hatfield et al. (2004), investigating the chronic effects amongst junior ice hockey athletes. Generally, there is a need for more child and adolescent studies investigating the effects of MTBI in sport. Studies should include more varied and culturally diverse socio-economic populations of both sexes and across all ages, including younger athletes involved in top teams since these tend to be more exposed to the effects of concussion than the lower levels of play (see review in Shuttleworth-Edwards et al., 2004).

Whilst dividing the experimental groups into Contact and Non-Contact sports groups may be a more reliable way of capturing effects than dividing groups on the basis of concussion history, it may have been useful for athletes to have documented the length or duration of LOC and PTA when reporting on their concussion history which would have enabled the researcher to document their level of severity more exactly. It is uncertain, however, whether this type of reporting would have assisted the present study owing to the unreliability of retrospective reporting amongst athletes, in addition to the known underreporting of symptoms which commonly occurs. With this in mind and considering that the development of a universal concussion grading scale is a “work in progress” (McCrory, Johnston, et al., 2005), schools should be encouraged to retain medical records on all contact sports players who have sustained concussions by documenting the details of the athlete’s concussion in respect of length of LOC, PTA, symptom reporting and time to return to play.

In respect of the neuropsychological measures and in light of the current findings certain refinements to existing measures may need to be considered. The findings, consistent with prior research, suggest that cognitive measures of reaction time, visual motor speed and the delayed trial of the Digit Symbol Paired Associate Incidental Recall task are very sensitive to the effects of concussion. In this study, the following measures presented as being more challenging to the more vulnerable Contact group in comparison to the other tasks. Whilst the Reaction Time composite score was sensitive on the independent analysis in respect of the Contact group at the pre-season interval, on the dependent analysis the Visual Motor Speed composite and the Digit Symbol Paired Associate Incidental Delayed Recall task proved to be very sensitive by eliciting a practice effect on the Non-Contact group that was not evident for the Contact group at the post-season interval. In light of this, it is recommended that future cross-sectional research studies follow the model of the present study with more focussed batteries incorporating only tests known to be particularly sensitive to MTBI. A more limited and focussed test battery will reduce the incidence of chance effects in the study, and render the research less exploratory.

In respect of the PCS measures, the greater effects evident on the smaller inventory suggest that a more focussed inventory is less likely to dilute results using a Likert scale, such that there is less chance for participants to give random responses. The greater sensitivity of a smaller inventory consisting of nine items relative to a much larger inventory of 22 and 26 items, therefore may be a more valid way of eliciting effects from athletes who are known for their underreporting of symptoms. In view of the four symptoms added to the Postconcussion Symptom Questionnaire, it would appear that the symptom of 'aggression' is very sensitive to the effects of concussion and should be considered for inclusion in future batteries investigating the effects of PCS in athletes. Future research should also be performed to identify the less sensitive, more redundant symptoms that warrant exclusion from a PCS inventory, in order to develop a briefer, more focussed, and it would appear more sensitive PCS inventory.

## 9.8 FINAL WORD

The present study provides evidence for the possible presence of chronic deleterious effects on cognition and neurobehavioural symptomatic presentation as a consequence of being exposed to a contact sport amongst high school athletes, albeit the effects appear subtle relative to what is generally identified amongst older athletes. Nevertheless, these indications however subtle were derived on the basis of a group analysis of relatively high functioning individuals. This alerts to the fact that even at such an early age MTBI consequences may be present for a significant number of individuals in the general high school sports world, and it can be postulated that in many such athletes the 'seeds of destruction' have already been put in motion. Thus, the crucial implication arising out of the present research is that at the high school level there is an urgent need for consciousness raising around the broad theme underlying the motivation for this study which is that "glory is only temporary and brain injury may indeed be seen to last forever". Whereas fears about the potential for permanent brain injury might lead to diminished support of contact sports and create the potential for legal repercussions, this should not be a reason to underplay evidence for such possible effects.

## REFERENCES

- Abreau, F., Templer, D.I., Schuyler, B.A., & Hutchinson, H.T. (1990). Neuropsychological assessment of soccer players. *Neuropsychology*, *4*, 175-181.
- Adams, J.H., Graham, D.I., & Gennarelli, T.A. (1985). Contemporary neuropathological considerations regarding brain damage in head injury. In D.P. Becker & J.T. Povlishock (Eds.), *Central nervous system trauma. Status report-1985*. Washington, D.C.: National Institutes of Health.
- Adams, R.L., Parsons, O.A., Culbertson, J.L. & Nixon, S.J. (1996). *Neuropsychology for clinical practice*. Washington, DC: American Psychological Association.
- Ad Hoc Committee to Study Head Injury Nomenclature: Proceedings of the Congress of Neurological Surgeons in 1964. *Clinical Neurosurgery*, *46*, 586-588.
- Alexander, M.P. (1992). Neuropsychiatric correlates of postconcussive syndrome. *Journal of Head trauma Rehabilitation*, *7*, 60-69.
- Alexander, M.P. (1995). Mild traumatic brain injury: Pathophysiology, natural history and clinical management. *Neurology*, *45*, 1253-1260.
- Allam, M.F., del Castillo, A.S. & Navajas, R.F. (2003). Parkinson's disease risk factors. *Revista de Neurologia*, *36*(8), 749-755.
- Allsop, D., Haga, S., Bruton, C., Ishii, T., & Roberts, G.W. (1990). Neurofibrillary tangles in some cases of dementia pugilistica share antigens with amyloid beta-protein of Alzheimer's disease. *American Journal of Pathology*, *136*(2), 255-260.
- Alves, W.M., Colohan, A.R.T., O'Leary, T.J., Rimel, R.W., & John, J.A. (1986). Understanding post-traumatic symptoms after minor head injury. *Journal of Head Trauma Rehabilitation*, *1*, 1-12.
- Alves, W.A., Macciocchi, S.N., & Barth, J.T. (1993). Postconcussive symptoms after mild head injury. *Journal of Head Trauma Rehabilitation*, *3*, 48-59.
- American Academy of Neurology. (1997). Practice parameter: The management of concussion in sport (summary statement)- Report of the Quality Standards Subcommittee. *Neurology*, *48*, 581-585.
- American Academy of Orthopedic Surgeons.(1995) *Play it safe! A guide to Safety for Young Athletes*. Des Plaines, Illinois:American Academy of Orthopedic Surgeons
- American Academy of Paediatrics (US). (1997). Participation in boxing by children, adolescents, and young adults. Policy Statement of the Committee on Sports Medicine and Fitness. *Pediatrics*, *99*, 134-135.

- American Congress of Rehabilitation Medicine. (1993). Addressing the post-rehabilitation health care needs of persons with disabilities. The ACRM Committee on Social, Ethical, and Environmental Aspects of Rehabilitation. *Archives of physical medicine and rehabilitation*, 74, S8-S14.
- American Medical Association. (1966). *Subcommittee on classification of sports injuries: standard nomenclature of athletic injuries*. Chicago: American Medical Association.
- American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders*. 4<sup>th</sup> ed. Washington, DC: APA
- Anderson, S.D. (1996). Postconcussional Disorder and Loss of Consciousness. *Bulletin of American Academy of Psychiatry Law*, 24(4), 493-504.
- Anderson, S.J. (1996). Sports-related head injuries: a neuropsychological perspective. *Sports Medicine*, September, 23-27.
- Anderson, S.I., Housley, A.M., Jones, P.A., Slattery, J., & Miller, J.D. (1993). Glasgow Outcome Scale: An inter-rater reliability study. *Brain Injury*, 7, 309-317.
- Anderson, V. (2001). Outcome from mild head injury in young children: a prospective study. *Journal of Clinical Experimental Neuropsychology*, 23(6), 705-717.
- Anderson, V., Northam, E., Hendy, J., & Wrennall, J. (2001). *Developmental Neuropsychology*. Sussex, England: Psychology Press.
- Asarnow, R.F., Satz, P., Light, R., Zaucha, K., Lewis, R., & McCleary, C. (1995). The UCLA Study of Mild Closed Head Injury in Children and Adolescents. In M.E. Michel and S. Broman (Eds.), *Traumatic brain injury in children* (pp. 117-146). New York: Oxford University Press.
- 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 1<sup>st</sup> International Symposium on Concussion in Sport, Vienna 2001. *Clinical Journal of Sports Medicine*, 12, 6-11.
- Baddeley, A., 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, R.J. & Patel, D.R. (2000). Sports Related Mild Head Injury in Adolescents. *Indian Journal of Pediatrics*, 67(5), 317-321.

- Bangsboe, J., Norregaard, L., & Thorsoe, F. (1991). Activity profile of competition football. *Canadian Journal of Sports Science, 16*, 110-116.
- 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.
- 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 injury in sports: Neuropsychological sequelae and recovery of function in mild head injury. In H.S. Levin, H.M. Elsenberg & A.L. Benton (Eds.), *Mild Head Injuries* (pp.257-275). Oxford: Oxford University Press.
- Barth, J.T., Diamond, R., & Errico, A. (1996). Mild head injury and postconcussion syndrome: does anyone really suffer? *Clinical Electroencephalopathy, 24*(7), 183-186.
- 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.
- Bazarian, J.J. & Atabaki, S. (2000). Predicting postconcussion syndrome after minor TBI. *Academic Emergency Medicine, 8*(8), 788-795.
- Bazarian, J.J., McCling, 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*, 85-91.
- Belanger, H.G., Curtiss, G., Demery, J.A., Lebowitz, & 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.

- Bell, M.A. & Fox, N.A. (1992). The relations between frontal brain electrical activity and cognitive development during infancy. *Child Development*, 63, 1142-1163.
- Bender, S.D., Barth, J.T., & Irvy, J. (2004). Historical Perspectives. In M. Lovell, R.J. Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic Brain Injury in Sports* (pp. 3-21). The Netherlands: Swets & Zeitlinger Publishers.
- Bennett, T.L. & Raymond, M.J. (1997). Mild brain injury: An overview. *Applied Neuropsychology*, 4, 1-5.
- Benton, A.L. (1969). Constructional apraxia: Some unanswered questions. In A.L. Benton (Ed.), *Contributions to clinical neuropsychology*. Chicago: Aldine.
- Benton, A.L. & Hamsher, K. (1989). *Multilingual Aphasia Examination*. Iowa City: AJA
- Bernstein, D.M. (2002). Information processing difficulty long after self-reported concussion. *Journal of the International Neuropsychological Society*, 8, 323-346.
- Biasca, N., Simmen, H.P., Bartolozzi, A.R., & Trentz, O. (1995). Review of typical ice hockey injuries. *Unfallchirurg*, 98, 283-288
- Bigler, E.D. (1990). Neuropathology of traumatic brain injury. In E.D. Bigler (Eds.), *Traumatic brain injury*. Austin Texas: Pro-ed.
- Bigler, E.D. (2001). The lesion(s) in traumatic brain injury: Implications for clinical neuropsychology. *Archives of Clinical Neuropsychology*, 16, 95-131.
- Bigler, E.D. (2003). Neurobiology and neuropathology underlie the neuropsychological deficits associated with traumatic brain injury. *Archives of Clinical Neuropsychology*, 18, 595-621.
- Bigler, E.D. & Orrison, Jr, W.W. (2004). Neuroimaging in Sport-related brain injury. In M. Lovell, R.J. Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic Brain Injury in Sports* (pp71-94). The Netherlands: Swets & Zeitlinger Publishers.
- Binder, L.M. (1986). Persisting symptoms after mild head injury: A Review of the Postconcussive Syndrome. *Journal of Clinical and Experimental Neuropsychology*, 8(4), 323-346.
- Binder, L.M. (1997). A review of mild head trauma: Part II. Clinical 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.
- 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.

- Birrer, R.B. (1996). Trauma epidemiology in the martial arts: The results of an eighteen-year international survey. *Journal of Sports Medicine*, *24*, 72-78.
- 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., Cderequist, J., Reeves, D., & Lux, W. (1993). Effects of D-amphetamine on performance consistency following brain injury: A double-blind placebo crossover case study. *Neuropsychiatry, Neuropsychology, and Behavioural Neurology*, *6*, 245-248.
- Bleiberg, J., Garmoe, W.E., Halpern, E. Reeves, D.L., & Nadler, J. (1997). Consistency of within-day and across-day performance after mild brain injury. *Neuropsychiatry, Neuropsychology, Behavioural Neurology*, *10*, 247-253.
- Bleiberg, J., Kane, R.I, Reeves, D.L., Garmoe, W.E., & Halpern, E. (2000). Factor analysis of computerized and traditional tests used in mild brain injury research. *Clinical Neuropsychology*, *14*, 287-294.
- 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., Twijnstra, A., & Jolles, J. (1992a). Post-traumatic and emotional symptoms in differing subgroups of patients with mild head injury. *Brain injury*, *6*, 481-486.
- Bohnen, N., Jolles, J. & Twijnstra, A. (1992). Neuropsychological deficits in patients with persistent symptoms six months after mild head injury. *Neurosurgery*, *30*(5), 692-696.
- Borg, J., Holm, L., Cassidy, J.D., Peloso, P.M., Carroll, L.J., von Holst, H., et al. (2004). Diagnostic procedures in mild traumatic brain injury: results of the WHO collaborating centre task force on mild traumatic brain injury. *Journal of Rehabilitation Medicine, Suppl.* *43*, 61-75.
- Borgaro, S.R., Prigatano, G.P., Kwasnica, C., & Rexer, J.L. (2003), Cognitive and affective sequelae in complicated and uncomplicated mild traumatic brain injury. *Brain Injury*, *17*(3), 189-198.
- British Medical Association. (1984). *Board of Science and Education Working Party on Boxing. Report*. London: BMA.
- Broe, G.A., Henderson, A.S., Creasey, H., McCusker, E., Korten, A.E., Jorm, A.F., et al. (1990). A case control study of Alzheimer's disease in Australia. *Neurology*, *40*, 1698-1707.
- 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, J. (2004). Gender issues in brain injury. In M. Lovell, R.J. Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic Brain Injury in Sports* (pp. 443-466). The Netherlands: Swets & Zeitlinger Publishers.

- 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.
- 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
- Brooks, N. & McKinley, W. (1983). Personality and behavioural change after severe blunt head injury – a relative's view. *Journal of Neurology, Neurosurgery and psychiatry*, *46*, 336-344.
- Brooks, N, McKinley, W., Symington, C., Beattie, A. & Campsie, L. (1987). Return to work within the first seven years of severe head injury. *Brain Injury*, *1*, 5-19.
- 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., Kaushik, T., Erlanger, D., Webbe, F., & Barth, J.T. (2005). Sex differences in outcome following sports related concussion. *Journal of Neurosurgery*, *102*(5), 856-863.
- Brower, M.C. & Price, B.H. (2001). Neuropsychiatry of frontal lobe dysfunction in violent and criminal behaviour: a critical review. *Journal of Neurology, Neurosurgery and Psychiatry*, *71*, 720-726.
- Brown, A.W., Leibson, C.L, Malec, J.F., Perkins, P.K., Diehl, N.N., & Larson, D.R. (2004). Long-term survival after traumatic brain injury: A population-based analysis. *NeuroRehabilitation*, *19*, 37-43.
- Bruce, D.A., Schut, L., & Sutton, L.N. (1982). Brian and cervical spine injuries accruing during organized sports activities in children and adolescents. *Clinical Sports Medicine*, *1*(3), 495-514.
- Bruno, L.A., Gennarelli, T.A., & Torg, J.S. (1987). Management guidelines for head injuries in athletics. *Clinical Sports Medicine*, *6*, 17-29.
- 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.
- Buschke & Fuld (1974). Evaluation of storage, retention and retrieval in disordered memory and learning. *Neurology*, *11*, 1019-1025.
- 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 & Psychiatry*, *56*, 1055-1061.
- Campbell, D.T. & Stanley, J.C. (1966). Experimental and quasi-experimental designs for research. Skokie, IL: Rand McNally.

- Cantu, R.C. (1986). Guidelines for return to contact sports after cerebral concussion. *Physician Sports Medicine*, 14, 75-83.
- Cantu, R.C. (1992). Cerebral concussion in sport: management and prevention. *Sports Medicine*, 14, 64-74.
- Cantu, R.C. (1996). Head injuries in sport. *British Journal of Sports Medicine*, 30(4), 289-296.
- Cantu, R.C. (1997a). Reflections on head injuries in sport and the concussion controversy. (Editorial). *Clinical Journal of Sports Medicine*, 7, 83-84.
- Cantu, R.C. (1997b). Athletic head injuries. *Clinics in Sport Medicine*, 16(3), 531-542.
- 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. & Voy, R. (1995). Second impact syndrome: a risk in any contact sport. *Physician Sports Medicine*, 23(6), 27-28, 31-34.
- Carlsson, G.S., Svardsudd, K., & Welin, L. (1987). Long-term effects of head injuries sustained during life in three male populations. *Journal of Neurosurgery*, 67, 197-205.
- 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.
- 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., 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.
- 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.
- Centers for Disease Control and Prevention: Sports-related recurrent brain injuries, United States. (1997). *Morbidity Mortality Weekly Report*, 46, 224-227.
- Chandra, V., Kokmen, E., Schoenberg, B.S., & Beard, C.M. (1989). Head trauma with loss of consciousness as risk factors for Alzheimer's disease. *Neurology*, 39, 1576-1578.

- Cicerone, K.D. (1996a). Attention deficits and dual task demands after mild traumatic brain injury. *Brain Injury*, 10, 79-89.
- Cicerone, K.D. (1996b). Clinical sensitivity of four measures of attention to mild traumatic brain injury. *The Clinical Neuropsychologist*, 11, 266, 272.
- Cicerone, K.D. & Kalmar, K. (1995). Persistent postconcussion syndrome: The structure of subjective complaints after mild traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 10, 1-17.
- 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.
- Collie, A., Darby, D., & Maruff, P. (2001). Computerized cognitive assessment of athletes with sports related head injury. *British Journal of Sports Medicine*, 35, 297-302.
- Collie, A., Maruff, P., Makadissi, 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., 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.
- Collins, M.W., Echemendia, R.J., & Lovell, M.R. (2004). Collegiate and high school sports. In M. 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., et al. (2003). Relationship between post-concussion headache and neuropsychological test performance in high school athletes. *American Journal of Sports Medicine*, 31, 168-173.
- Collins, M.W., Grindel, S.H., Lovell, M.R., Dede, D.E., Moser, D.J., Phalin, B.R., et al. (1999b). 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 symptoms deficit following sport-related concussion. *Clinical Journal of Sports Medicine*, 13(4), 222-229.
- Collins, M.W., Lovell, M.R., Iverson, G.L., Cantu, R.C., Maroon, J.C., & Field, M. (2002). Cumulative effects of concussion in high school athletes. *Neurosurgery*, 51, 1175-1181.
- Collins, M.W., Lovell, M.R., & McKeag, D.B. (1999). Current issues in managing sports-related concussion. *Journal of the American Medical Association*, 282, 2283-2285.
- Congress of Neurological Surgeons. Committee on Head Injury Nomenclature: glossary of head injury. (1991). *Clinical Neurosurgery*, 12, 386-394.

- Cook, T.D. & Campbell, D.T. (1979). *Quasi Experimental; Design and analysis for field settings*. Boston: Houghton Mifflin.
- Courville, C.B. (1942). Coup-contrecoup mechanism of craniocerebral injuries: Some observations. *Archives of Surgery*, *45*, 19-43.
- 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.
- 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, & Behavioural Neurology*, *12*(3), 167-169.
- Davis, A. (2002). A predictor of symptoms after mild brain traumatic brain injury. Dissertation Abstracts International: Section B. *The Sciences and Engineering*, *62*, 3395.
- 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.
- Delaney, J.S. (2004). Head injuries presenting to emergency departments in the United States from 1990 to 1999 for ice-hockey, soccer and football. *Clinical Journal of Sports Medicine*, *14*(2), 80-87.
- Delis, D.C., Kaplan, E., Kramer, J.H., & Ober, B.A. (2000). California Verbal Learning Test-Second Edition (CVLT-II) Manual. San Antonio: Psychological Corporation
- 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 Renzi, E., Faglioni, P. & Sorgato, P. (1982). Modality-specific and supramodal mechanisms of apraxia. *Brain*, *105*(2), 301-312.
- 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.
- Diagnostic and Statistical Manual of Mental Disorders - Fourth Edition*. (1994). Washington D.C: American Psychiatric Association.
- Dick, R.W. (1994). A summary of head and neck injuries in collegiate athletics using the NCAA injury surveillance system. In E. Hoerner (Ed.), *Head and Neck Injuries in Sports* (pp. 13-19). Philadelphia: American Society for Testing and Materials.
- Dick, R.W. (2002). Football injury report. In: *2001-2002 NCAA Injury Surveillance System*. Indianapolis, Indiana: National Collegiate Athletic Association.

- Dick, R.W. (2003). *National Collegiate Athletic Association (NCAA) Injury Surveillance System 2002-2003*. Indianapolis, Indiana: National Collegiate Athletic Association.
- 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.H., 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., & 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.S., McLean, A., & Temkin, N.R. (1986). Neuropsychological and psychological consequences of minor head injury. *Neurosurgery, 48*, 1227-1232.
- Downs, D.S. & Abwender, D. (2002). Neuropsychological impairment in soccer athletes. *The Journal of Sports Medicine and Physical Fitness, 42*, 103-107.
- Drew, R., Templer, D., Schuyler, B., Newell, T.G., & Cannon, G. (1986). Neuropsychologic deficits in active licensed professional boxers. *Journal of Clinical Psychology, 42*, 520-525.
- Durkin, M.S., Olsen, S., Barlow, B., Virella, A & Connolly, E.S. Jnr. (1998). The epidemiology of urban pediatric trauma: evaluation of, and implications for, injury prevention programs. *Neurosurgery, 42*, 300-310.
- Duthie, G., Pyne, D., & Hooper, S. (2003). Applied Physiology and game analysis of Rugby Union. *Sports Medicine, 33(13)*, 973-991.
- Echemendia, R.J. (2004). Cultural aspects of neuropsychological evaluations in sport. In M. Lovell, R.J. Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic Brain Injury in Sports* (pp. 435-442). The Netherlands: Swets & Zeitlinger Publishers.
- Echemendia, R.J. & Cantu, R.C. (2003). Return to play following sports-related mild traumatic brain injury: The role for Neuropsychology. *Applied Neuropsychology, 10(1)*, 45-55.
- Echemendia, R.J. & Cantu, R.C. (2004). Return to play following cerebral brain injury. In M. 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. & Julian, L.J. (2001). Mild traumatic brain injury in sports: neuropsychology's contribution to a developing field. *Neuropsychological Review, 11*, 69-88.
- Echemendia, R.J., Petukian, M., Mackin, R.S., Julian, L., & Shoss, N (2001). Neuropsychological test performance before and following sports-related mild traumatic brain injury. *Clinical Journal of Sports Medicine, 11*, 23-31.

- Elson, L.M. & Ward, C.C. (1994). Mechanisms and pathophysiology of mild head injury. *Seminars in Neurology*, 14, 8-18.
- Engberg, A. & Teasdale, T.W. (1998). Traumatic brain injury in children in Denmark: a national 15 year study. *European Journal of Epidemiology*, 14, 165-173.
- Englander, J., Hall, K., Simpson, T., & Chaffin, S. (1992). Mild traumatic brain injury in an insured population: Subjective complaints and return to employment. *Brain Injury*, 6, 161-166.
- Erlanger, D.M., Feldman, D., Kutner, K., Kaushik, T., Kroger, H., Festa, J., et al. (2003). Development and validation of a web-based neuropsychological test protocol for sports-related return-to-play decision-making. *Archives of Clinical Neuropsychology*, 18, 293-316.
- Erlanger, D.M., Kutner, K.C., Barth, J.T., & Barnes, R. (1999). Neuropsychology of Sports-related head injury: Dementia Pugilistica to Post Concussive Syndrome. *The Clinical Neuropsychologist*, 13(2), 193-209.
- Erlanger, D.M., Saliba, E., Barth, J.T., Almquist, J., Webright, W., & Freeman, J. (2001). Monitoring resolution of post-concussion symptoms in athletes: Preliminary results of a web-based neuropsychological test protocol. *Journal of Athletic Training*, 36(3), 280-287.
- Evans, C.D. (1975). Discussion of the clinical problem. In Ciba Foundation Symposium, No.34 (new series), *Symposium on the outcome of severe damage to the CNS*. Amsterdam: Elsevier.
- Evans, R.W. (1992). Some observations on whiplash injuries. *Neurologic Clinics*, 10, 975-997.
- Evans, R.W. (2004). Post-traumatic headaches. *Neurologic Clinics*, 22, 237-249.
- Ewing-Cobbs, L., Miner, M.E., Fletcher, J.M., & Levin, H.S. (1989). Intellectual, motor and language sequelae following closed head injury in infants and preschoolers. *Journal of Pediatric Psychology*, 14, 531-547.
- Farace, E., Ferree, R.M., Hollier, J.A., Barth, J.T., & Shaffrey, M.E. (2003). Trails A: Neurocognitive effect of previous concussions in woman's rugby sample. *Journal of the International Neuropsychological Society*, 9(2), 207.
- Ferguson, R.J., Mittenberg, W., Barone, D.F., & Brown, L.M. (1999). Post-concussive syndrome following sports-related head injury; expectation as etiology. *Neuropsychology*, 13(4), 582-589.
- FIFA. (2002). FIFA survey. *Fédération Internationale de Football Association (FIFA)*, 11 Hitzigweg, 8030 Zurich, Switzerland 12.
- 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.

- Franzen, M.D., Frerichs, R.J., Iverson, G.L. (2004). Reliability, validity and the measurement of change in serial assessment so athletes. In M. 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.R., Fox, A.M., & Maybery, M.T. (2005). Neuropsychological studies of mild traumatic brain injury: A meta-analytic review of research since 1995. *Journal of Clinical and Experimental Neuropsychology*, 27, 334-351.
- 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.
- Fuster, J. (1993). Frontal lobes. *Current Opinion in Neurobiology*, 3, 160-165.
- Gaetz, M. (2004). The neurophysiology of brain injury. *Clinical Neurophysiology*, 115, 4-18.
- Gaetz, M., Goodman, D., & Weinberg, H. (2000). Electrophysiological evidence for the cumulative effects of concussion. *Brain Injury*, 14, 1077-1088.
- Ganesalingam, K., Sanson, A., Anderson, V., & Yeates, K.O. (2006). Self-regulation and social and behavioral functioning following childhood traumatic brain injury. *Journal for the International Neuropsychological Society*, 12, 609-621.
- Ganor-Stern, D., Seamon, J.G., & Carrasco, M. (1998). The role of attention and study time in explicit and implicit memory for unfamiliar visual stimuli. *Memory and Cognition*, 26, 1187-1195.
- Garraway, M. & MacLeod, D. (1995). Epidemiology of rugby football injuries. *Lancet*, 345, 229-233.
- Garraway, W.M., Lee, A.J., Hutton, S.J., Russel, E.B.A.W., & Macleod, D.A.D. (2000). Impact of professionalism on injuries in rugby union. *British Journal of Sports Medicine*, 34, 348-351.
- Geberich, S.G., Priest, J.D., Boen, J.R., Struab, C.P., & Maxwell, R.E. (1983). Concussion incidences and severity in secondary school varsity football players. *American Journal of Public Health*, 73, 1370-1375.
- Gennarelli, T.A., Adams, J.H., & Graham, D.I. (1981). Acceleration induced head injury in the monkey: The model, its mechanism and physiological correlate. *Acta Neuropathologia*, 7(Suppl.), 23-25.
- Gennarelli, T.A., Thibault, L.E., Adams, J.H., Graham, D.I., Thompson, C.J., & Marcincin, R.P. (1982). Diffuse axonal injury and traumatic coma in the primate. *Annals of Neurology*, 12(6), 564-574.
- 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.

- 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.
- 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.
- Gerberich, S.G., Finke, R., Madden, M., Priest, J.D., Aamoth, G., & Murray, K. (1987). An epidemiological study of high school ice hockey injuries. *Child's Nervous System*, 3(2), 59-64.
- 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.
- Giza, C.C. & Hovda, D.A. (2000). Ionic and metabolic consequences of concussion. In R.C. Cantu and R.I. Cantu, *Neurologic Athletic and Spine Injuries* (pp 80-100). Philadelphia: WB Saunders Co.
- Giza, C.C. & Hovda, D.A. (2004). The pathophysiology of traumatic brain injury. In M. Lovell, R.J. Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic Brain Injury in Sports* (pp. 45-69). The Netherlands: Swets & Zeitlinger Publishers.
- Gloning, K. & Hoff, H. (1969). Cerebral localization of disorders of higher nervous activity. In P.J. Vinken & G.W. Bruyn (Eds.), *Handbook of clinical neurology. Disorders of higher nervous activity. (Vol. 3)*. New York: Wiley.
- Goh, K.Y. & Poon, W.S. (1995). Children's head injuries in the Vietnamese refugee population in Hong Kong. *Injury*, 26, 533-536.
- Golden, C.A. (1978). *Stroop color and word test manual*. Chicago: Stoelting Company
- Goldstein, F.C., Levin, H.S., Goldman, W.P., Clark, A.N., & Altonen, T.K. (2001). Cognitive and neurobehavioural functioning after mild versus moderate traumatic brain injury in older adults. *Journal of the International Neuropsychological Society*, 7(3), 373-383.
- Graves, A.B., White, E., Koepsell, T.D., Reifler, B.V., van Belle, G., Larson, E.B., et al. (1990). The association between head trauma and Alzheimer's disease. *American Journal of Epidemiology*, 131, 491-501.
- Grindel, S.H., Lovell, M.R., & Collins, M.W. (2001). The assessment of sport-related concussion: the evidence behind neuropsychological testing and management. *Clinical Journal of Sports Medicine*, 11, 134-143.
- Gronwall, D. (1987). Advances in the assessment of attention and information processing after head injury. In H.S. Levin, J. Grafman, & H.M. Eisenberg (Eds.), *Neurobehavioural recovery from head injury*. (pp.355-371). New York: Oxford University Press.
- Gronwall, D. (1989). Cumulative and persisting effects of concussion on attention and cognition. In H.M. Eisenberg, & A.L. Benton (Eds.), *Mild head injury* (pp.153-162). New York: Oxford University Press.

- Gronwall, D. & Wrightson, P. (1974). Delayed recovery of intellectual functioning following mild traumatic brain injury. *Lancet*, *ii*, 1452.
- 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.
- Gualtieri, C.T. (1995). The problem of mild brain injury. *Neuropsychiatry, Neuropsychology, and Behavioural Neurology*, *8*, 127-136.
- 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.
- Guo, Z., Cupples, L.A., Kurz, A., Auerbach, S.H., Volicer, L., Chui, H., et al. (2000). Head injury and the risk of AD in the Mirage study. *Neurology*, *54*, 1216-1323.
- Guskiewicz, K.M., Bruce, S.L., Cantu, R.C., Ferrara, M.S., Kelly, J.P., McCrea, M., et al. (2004). National Athletic Trainers' Association Position Statement: Management of Sport-Related Concussion. *Journal of Athletic Training*, *39*(3), 280-297.
- Guskiewicz, K.M., Marshall, S.W., Bailes, J., McCrea, M., Cantu, R.C., Randolph, C., et al. (2005). Association between recurrent concussion and late cognitive impairment in retired professional football players. *Neurosurgery*, *57*(4), 719-26.
- Guskiewicz, K.M., Marshall, S.W., Broglio, S.P., Cantu, R.C., & Kirkendall, D.T. (2002). No evidence of impaired neurocognitive performance in collegiate soccer players. *American Journal of Sports Medicine*, *30*(2), 157-62.
- Guskiewicz, K.M., McCrea, M., Marshall, S.W., Cantu, R.C., Randolph, C., Barr, W., et al. (2003). Cumulative Effects Associated With Recurrent Concussion in Collegiate Football Players. *The Journal of the American Medical Association*, *290*(19), 2549-2555.
- 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*, Suppl.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*, 643-650.
- Haas, J.F., Cope, D.N., & Hall, K. (1987). Premorbid prevalence of poor academic Performance in severe head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, *50*, 52-56.

- Haglund, Y. & Bergstrand, G. (1990). Does Swedish amateur boxing lead to chronic brain damage? A retrospective study with cerebral computed tomography and magnetic resonance imaging. *Acta Neurologica Scandinavica*, 82, 297-302.
- Haglund, Y. & Persson, H.E. (1990). Does Swedish amateur boxing lead to chronic brain damage? A retrospective clinical neuropsychological study. *Acta Neurologica Scandinavica*, 82, 353-360.
- Haier, R.J., Siegel, A.V., Nuechterlein, K.H., Hazlett, E., Wu, J.C., Pack, J., et al. (1988). Cortical glucose metabolic rate correlates of abstract reasoning and attention studies with positron emission tomography. *Intelligence*, 12, 199-217.
- Halpern, D.F. (1997). Sex difference in intelligence: Implications for education. *American Psychologist*, 52, 1091-1102.
- Hammoud, D.A., & Wasserman, B.A. (2002). Diffuse axonal injuries: pathophysiology and imaging. *Neuroimaging clinics of North America*, 12, 205-216.
- Hart, R.P., Kwentus, J.A., Wade, J.B., & Hamer, R.M. (1987). Digit Symbol performance in mild dementia and depression. *Journal of Consulting and Clinical Psychology*, 55, 236-238.
- Hatfield, R., Bieliauskas, L., Bergloff, P., Steinberg, B., & Kauszler, M. (2004). Youth Hockey. In M. Lovell, R.J. Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic Brain Injury in Sports* (pp.129-147). The Netherlands: Swets & Zeitlinger Publishers.
- Heaton, R.K., Marcotte, T.D., Mindt, M.R., Sadek, J., Moore, D.J., Bentley, H., Mccutchan, J.A., Reicks, C., Grant, I., & the HNRC group. (2004). The impact of HIV-associated neuropsychological impairment on everyday functioning. *Journal of the International Neuropsychological Society*, 10, 317-331.
- 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*. The Netherlands: Swets & Zeitlinger Publishers.
- 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.
- High, W.M., Levin, H.S., & Gary, H.E. (1990) Recovery of orientation following closed-head injury. *Journal of Clinical and Experimental Neuropsychology*, 12, 703-714.
- Hill, L.R., Klauber, M.R., Salmon, D.P., Yu, E.S., Liu, W.T., Zhang, M., et al. (1993). Functional status, education, and the diagnosis of dementia in the Shanghai survey. *Neurology*, 43(1), 138-145.
- 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 ruled football and rugby league. In M.R. Lovell, R.L. Echemendia, J.T. Barth, & M.W. Collins (Eds.). *Traumatic Brain Injury in Sports*. The Netherlands: Swets & Zeitlinger Publishers.
- Hinton-Bayre, A.D., Geffen, G., & Friis, P. (2004). Presentation and mechanisms of concussion in professional Rugby League football. *Journal of Science Medicine and Sport*, 7(3), 400-404.
- Hinton-Bayre, A.D., Geffen, G., Geffen, L.B., McFarland, K.A., & Friis, P. (1999). Concussion in contact sports: reliable change indices of impairment and recovery. *Journal of Clinical Experimental Neuropsychology*, 21(1), 70-86.
- Hinton-Bayre, A.D., Geffen, G., & McFarland, K.A. (1997). Mild head injury and speed of information processing: a prospective study of professional rugby league players. *Journal of Clinical and Experimental Neuropsychology*, 19, 275-289.
- 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.
- Honey, C.R. (1998). Brain injury in ice hockey. *Clinical Journal of Sports Medicine*, 8, 43-46.
- 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.
- Howell, D.C. (1989). *Fundamental statistics for the behavioural sciences* (2<sup>nd</sup> Ed.). Boston: PWS-Kent Publishing Company.
- 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.
- Hughenoltz, H. & Richard, M.T. (1982). Return to athletic competition following concussion. *Canadian Medical Associate Journal*, 127, 827-829.
- Hughenoltz, 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.
- Inglis, J. (1959). A paired-associate learning test for use with elderly psychiatric patients. *Journal of Mental Science*, 105, 440-443.
- International Classification of Diseases (ICD)*. (2006). Retrieved March 10, 2006, from <http://icd9.chrisendres.com/index.php?action=child&recordid=7942>.
- International Classification: International Headache Society Classification and diagnostic criteria for headache disorders, cranial neuralgias and facial pain. (1988). *Cephalalgia*, 8(Suppl.7), 1-96.

- 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. (2004). Construct Validity of Computerized Neuropsychological Screening in Athletes with Concussions. Paper presented at the annual conference of the National Academy of Neuropsychology, November 17-20, 2004, Seattle, Washington.
- 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 foginess and neuropsychological testing following concussion. *Journal of the International Neuropsychological Society*, 10, 904-906.
- Iverson, G.L., Gaetz, M., Lovell, M.R., & Collins, M.W. (2005). Validity of ImPACT for measuring processing speed following sports related concussion. *Journal of Clinical and Experimental Neuropsychology*, 27, 683-689.
- Iverson, G.L., 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., & 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.
- 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.
- 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 Neuroradiology*, 25, 738-745.
- 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.
- Jellinger, K.A. (2004). Head injury and dementia. *Current Opinion in Neurology*, 17, 719-723.

- Jennett, B. (1976). Assessment of the severity of head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, 39, 647-655.
- Jennett, B. & MacMillan, R. (1981). Epidemiology of head injury. *British Medical Journal*, 282, 101-104.
- Jennett, B. & Galbraith, S. (1983). *An Introduction to Neurosurgery*, 4<sup>th</sup> ed. London: Heinemann.
- Jernigan, T.L. & Tallal, P. (1990). Late childhood changes in brain morphology observable with MRI. *Developmental Medicine and Child Neurology*, 32, 379-385.
- Johnsom, R.A. & Wichern, D.W. (2002). *Applied Multivariate Statistical Analysis*, 5<sup>th</sup> ed. (pp. 305-312). Upper Saddle River, New York: Prentice-Hall
- Johnston, K.M., Lassonde, M., & Ptito, A. (2001). A contemporary neurosurgical approach to sport-related head injury: the McGill concussion protocol. *Journal of American College of Surgery*, 192, 525-524.
- 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, 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.
- 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., Jahre, C., Hauser, W.A., Zimmerman, R.D., Zarelli, M., Lipsitz, E.C., et al. (1992). CT of 338 active professional boxers. *Radiology*, 185(2), 509-512.
- Jordan, B.D., Matser, E., Zimmerman, R. & Zazula (1996). Sparring and cognitive function of professional boxers. *Physician Sports Medicine*, 24(5), 87-98.
- Jordan, B.D., Ravdin, L.D., Jacobs, A.R., Bennett, A., & Gandy, S. (1997). Apolipoprotein E e4 association with chronic traumatic brain injury in boxing. *Journal of the American Medical Association*, 276, 136-140.
- 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.
- 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.

- Junger, E.C., Newell, D.W., Grant, G.A., Avellino, A.M., Ghatan, S., Douville, C.M., et al. (1997). Cerebral autoregulation following minor head injury. *Journal of Neurosurgery*, 86, 425-432.
- Kaplan, E., Fein, D., Morris, R., & Delis, D. (1991). *WAIS-R-NI manual*. San Antonio, TX: The Psychological Corporation.
- 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.
- Katz, D.I. & Alexander, M.P. (1994). Traumatic brain injury: Predicting course of recovery and outcome for patients admitted to rehabilitation. *Archives of Neurology*, 51, 661-670.
- Katz, L. Goldstein, G., Rudisin, S. & Bailey, D. (1993). A neuropsychological approach to the Bannatyne recategorization of the Wechsler Intelligence Scales in adults with learning disabilities. *Journal of Learning Disabilities*, 26, 65-72.
- Kelly, J.P., Nichols, J.S., Filley, C.M., Lillehei, K.O, Rubinstein, D., & Kleinschmidt-DeMasters, B.K. (1991). Concussion in sports: guidelines for the prevention of catastrophic outcome. *Journal of the American Medical Association*, 226, 2867-2869.
- Kelly, J.P. & Rosenberg, J.H. (1997). Diagnosis and management of concussion in sports. *Neurology*, 48, 575-580.
- Kerlinger, F.N. (1986). *Foundations of behavioural research*. (3<sup>rd</sup> ed.). USA: Harcourt Brace Jovanovich College Publishers.
- Kibby, M.Y. & Long, C.J. (1996). Minor head injury: attempts at clarifying the confusion. *Brain Injury*, 10(3), 159-186.
- King, N. (1997). Mild Head Injury: Neuropathology, sequelae, measurement and recovery. *British Journal of Clinical Psychology*, 36, 161-184.
- 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.
- Kirkendall, D.T., Jordan, S.E., & Garrett, W.E. (2001). Heading and head injuries in soccer. *Sports Medicine*, 31, 369-386.
- Klinberg, T., Viadya, C., Gabrieli, J., Moseley, M., & Hedehus, M. (1999). Myelination and organisation of the frontal white matter in children: A diffusion tensor MRI study. *NeuroReport*, 10, 2817-2821.
- Koh, J.O. & Cassidy, J.D. (2004). Incidence study of head blows and concussions in competition taekwondo. *Clinical Journal of Sports Medicine*, 4(2), 72-9.
- 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.
- Kozora, E. & Gerber, D. (2004). Special considerations and implications of

neuropsychological testing in professional athletes. In M.Lovell, R.J.Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic Brain Injury in Sports* (pp. 358-374). The Netherlands: Swets & Zeitlinger Publishers.

- Kraus, J.F. (1995). Epidemiological features of brain injury in children: occurrence, children at risk, causes and manner of injury, severity and outcomes. In S. Broman and M. Michel (Eds.), *Traumatic head injury in children*. New York: Oxford University Press.
- Kraus, J.F., Fife, D., & Conroy, C. (1987). Pediatric brain injuries: the nature clinical course and early outcomes in a defined United States population. *Pediatrics*, *79*, 501-507.
- Kraus, J.F., Fife, D., Cox, P., Ramstein, K., & Conroy, C. (1986). Incidence, severity, and external causes of pediatric brain injury. *American Journal of Diseases in Children*, *140*, 687-693.
- Kraus, J.F. & Nourjah, P. (1988). The epidemiology of mild uncomplicated brain injury. *Trauma*, *28*, 1637-1643.
- Kreiter, K.T., Copeland, D., Bernardini, G.L., Bates, J.E., Peery, S., Claasen, J., et al. (2002). Predictors of cognitive dysfunction after subarachnoid hemorrhage. *Stroke*, *33*, 200-208.
- Kutner, K.C., Erlanger, D.M., Tsai, J., Jordan, B., Relkin, N.R. (2000). Lower cognitive performance of older football players possessing apolipoprotein E  $\epsilon$ 4. *Neurosurgery*, *47*(3), 651-657.
- Lampert, P.W., & Hardman, J.M. (1984). Morphological changes in the brains of boxers. *Journal of the American Medical Association*, *251*(20), 2676-2679.
- 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.
- 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.
- Lee, J.H. (2003). Genetic evidence for cognitive reserve: Variations in memory and related cognitive functions. *Journal of Clinical and Experimental Neuropsychology*, *25*, 594-613.
- 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.
- Leininger, B.E., Gramling, S.E., Farrel, 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.
- Lendon, C.L., Harris, J.M., Pritchard, A.L., Nicoll, J.A., Teasdale, G.M., & Murray, G. (2003). Genetic variation of the APOE promoter and outcome after head injury, *Neurology*, *61*(5), 683-685.

- Levin, H.S., Amparo, E.G., Eisenberg, H.M., Williams, D.H., High, W.M., McArdle, C.B., et al. (1987). Magnetic resonance imaging and computerized tomography in relation to neurobehavioural sequelae at mild and moderate head injury. *Journal of Neurosurgery*, 66, 706-713.
- Levin, H.S., Benton, A.L., & Grossman, R.G. (1982). *Neurobehavioural consequences of closed head injury* (2<sup>nd</sup> 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., 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., Mendelsohn, D., Lilly, M., Fletcher, J., Culhane, K., Chapman, S., et al. (1994). Tower of London performance in relation to magnetic resonance imaging following closed head injury in children. *Neuropsychology*, 8, 171-179.
- 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., Song, J., Scheibel, R., Fletcher, J., Harward, H., Lilly, M., et al. (1997). Concept formation and problem solving following closed head injury in children. *Journal of the International Neuropsychological Society*, 3, 598-607.
- 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.
- Lezak, M.D. (1976). *Neuropsychological Assessment*. New York: Oxford University Press.
- Lezak, M.D. (1983). *Neuropsychological Assessment*. (2<sup>nd</sup> ed.). New York: Oxford University Press.
- Lezak, M.D. (1995). *Neuropsychological Assessment*. (3<sup>rd</sup> ed.). New York: Oxford University Press.
- Lezak, M.D., Howieson, D.B., & Loring, D.W. (2004). *Neuropsychological Assessment*. (4<sup>th</sup> ed.). New York: Oxford University Press.
- Lichtman, S.W., Seliger, G., Tyckl, B., & Marder, K. (2000). Apolipoprotein E and

functional recovery from brain injury following postacute rehabilitation, *Neurology*, 55, 1536-1539.

- Lindsey, K., Pasaoglu, A. & Hirst, D (1990). Somatosensory and auditory brain stem conduction after head injury: A comparison with clinical features in predicting outcome. *Neurosurgery*, 26, 278-285.
- Lishman, W.A. (1987). *Organic Psychiatry* (2<sup>nd</sup> Ed.). Oxford: Blackwell.
- Lishman, W.A. (1988). Physiogenesis and psychogenesis in the 'post-concussional syndrome'. *British Journal of Psychiatry*, 153, 460-469.
- Lord-Maes, J. & Obrzut, J.E. (1996). Neuropsychological consequences of traumatic brain injury in children and adolescents. *Journal of Learning Disabilities*, 29(6), 609-617.
- Lovell, M.R. (1996). *Evaluation of the professional athlete*. Paper presented at the New Developments in Sports-Related Concussion Conference, Pittsburgh, PA.
- Lovell, M. R. (1999). Evaluation of the professional athlete. In J.E. Bailes, M.R. Lovell & J.C. Maroon (Eds.), *Sports Related Concussion* (pp. 200-214). St Louis: Quality Medical Publishing.
- Lovell, M.R. (2002). The relevance of neuropsychological testing for sports-related head injuries. *Current Sports Medicine Reports*, 1, 7-11.
- Lovell, M.R. (2004). *ImPACT Version 2.0 Clinical User's Manual*. Accessed 3.12.2004 at <http://www.impacttest.com/clients.htm>
- Lovell, M.R. & Burke, C.J. (2000). Concussion management in professional hockey. In R. E. Cantu (Eds.), *Neurologic Athletic Head and Spine Injury*. Philadelphia: W.B. Saunders.
- Lovell, M.R. & Collins, M.W. (1998). Neuropsychological assessment of the college football player. *Journal of Head Trauma and Rehabilitation*, 13, 9-26.
- Lovell, M.R. & Collins, M.W. (2002). New developments in the evaluation of sports-related concussion. *Current Sports Medicine Reports*, 1, 287-292.
- Lovell, M.R., Collins, M.W., & Bradley, J. (2004). Return to play following sports-related concussion. *Clinical Sports Medicine*, 23, 421-441.
- Lovell, M.R., Collins, M.W., Iverson, G.L., Field, M., Maroon, J.C., Cantu, R., et al. (2003). Recovery form mild concussion in high school athletes. *Journal of Neurosurgery*, 98, 296-301.
- Lovell, M.R., Collins, M.W., Iverson, G.L., Johnston, K.M., & Bradley, J.P. (2004). Grade 1 or "ding" concussions in high school athletes. *Journal of Neurosurgery*, 98(2), 296-301.
- 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. 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, 193-198.
- Lovell M.R., Iverson, G.L., Collins, M.W., Podell, K., Johnston, K.M., Pardini, D., et al. (in press). Measurement of symptoms. *Applied Neuropsychology*.
- Lye, T.C. & Shores, A. (2002). Traumatic brain injury as a risk factor for Alzheimer's disease: A Review. *Neuropsychology Review*, 10(2), 115-129.
- Macciocchi, S.N. & Barth, J.T. (2004). Methodological concerns in traumatic brain injury. In M.Lovell, R.J.Echemendia, J.T. Barth, & M.W. Collins (Eds.), *Traumatic Brain Injury in Sports* (pp. 281-298). The Netherlands: Swets & Zeitlinger Publishers.
- Macciocchi, S.N., Barth, J.T., Alves, W., Littlefield, L., Jane, A., & Cantu, R.C. (2001). Multiple concussions and neuropsychological functioning in Collegiate Football Players. *Journal of Athletic Training*, 36(3), 303-306.
- Macciocchi, S.N., Barth, J.T., Alves, W., Rimel, R.W., & Jane, J.A. (1996). Neuropsychological functioning and recovery after mild head injury in collegiate athletes. *Neurosurgery*, 39, 510-514.
- Macciocchi, S.N., Barth, J.T. & Littlefield, L. (1998). Outcome after mild head injury. *Clinics in Sports Medicine*, 17(1), 27-36.
- MacFarlane, D.P., Nicoll, J.A.R., Smith, C., & Graham, D.I. (1999). APOE-ε4 allele and amyloid β-protein deposition in long-term survivors of head injury. *NeuroReport*, 10, 3945-3948.
- MacFlynn, G., Montgomery, E.A., Fenton, G.W., & Rutherford, W. (1984). Measurement of reaction time following minor head injury. *Journal of Neurology, Neurosurgery and Psychiatry*, 47, 1326-1331.
- MacLeod, D.A.D. (1993). Risks and injuries in rugby football. In G.R. McLatchie and C.M.E. Lennox (Eds.), *The soft tissues. Trauma and sport injuries* (pp. 371-381). London: Butterworth-Heinemann Ltd.
- Maddocks, D.L. Dicker, G.D., & Saling, M.M. (1995). The assessment of orientation following concussion in athletes. *Clinical Journal of Sports Medicine*, 5, 32-35.
- Maddocks, D.L. & Saling, M.M. (1996). Neuropsychological deficits following concussion. *Brain Injury*, 10, 99-103.
- 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.
- Makdissi, M., Collie, A., Maruff, P., Darby, D.G., Bush, A., McCrory, P., et al. (2001).

Computerized cognitive assessment of concussed Australian Rules Footballers. *British Journal of Sports Medicine*, 35, 354-360.

- Mandel, S. (1989). Minor head injury may not be 'minor'. *Postgraduate Medicine*, 85, 213-225.
- Marcotte, T.D., Heaton, R.K., Wolfson, T., Taylor, M.J., Alhassoon, O., Arfaa, K., Grant, I., & the HNRC group. (1999). *Journal of the International Neuropsychological Society*, 5, 579-592.
- Marshall, S.W., Loomis, D.P., Waller, A.E., Chalmers, D.J., Bird, Y.N., Quarrie, K.L., et al. (2004). Evaluation of protective equipment for prevention of injuries in rugby union. *International Journal of Epidemiology*, 34, 113-118.
- Martland, H.S. (1928). Punch-Drunk. *Journal of the American Medical Association*, 19, 1103-1107.
- Maroon, J.C., Field, M., Lovell, M., Collins, M., & Post, J. (2002). The evaluation of athletes with cerebral concussion. *Clinical Neurosurgery*, 49, 319-332.
- Maroon, J.C., Lovell, M.R., Norwig, J., Podell, K., Powell, J.W., & Hartl, R. (2000). Cerebral concussion in athletes: Evaluation and neuropsychological testing. *Neurosurgery*, 47(3), 659-669.
- Maroon, J.C., Steele, P.B., Berlin, R. (1980). Football head and neck injuries: an update. *Clinical Neurosurgery*, 27, 414.
- 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.
- 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., & 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.
- Max, J.E., Koele, S.L., Castillo, C.C., Lindgren, S.D., Arndt, S., Bokura, H., et al. (2000). Personality change disorder in children and adolescents following traumatic brain injury. *Journal of the International Neuropsychological Society*, 6, 279-289.
- 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.
- Mayeux, R., Ottman, R., Tang, M.X., Noboa-Bauza, L., Marder, K., Gurland, B, et al. (1993).

Genetic susceptibility and head injury as risk factors for Alzheimer's disease among community-dwelling elderly persons and their first-degree relatives. *Annals of Neurology*, 33, 494-501.

- McAllister, T.W. & Arciniegas, D. (2002). Evaluation and treatment of postconcussive symptoms. *Neurorehabilitation*, 17, 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.
- McCaffrey, R. J., Ortega, A., Orsillo, S. M., Nelles, W. B., & Haase, R. F. (1992). Practice effects in repeated neuropsychological assessments. *The Clinical Neuropsychologist*, 6, 32-42.
- McClincy, M.P., Lovell, M.R., Pardinin, 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., Guskiewicz, K.M., Marshall, S.W., Barr, W., Randolph, C., Cantu, R.C., et al. (2003). Acute Effects and Recovery Times Following Concussion in Collegiate Football Players. *Journal of the American Medical Association*, 290(19), 2556-6563.
- McCrea, M., Kelly, J.P., Kluge, J., Ackley, B., & Randolph, C. (1997). Standardized assessment of concussion in football players. *Neurology*, 48, 586-588.
- McCrea, M., Kelly, J.P., Randolph, C., Cisler, R., & Berger, L. (2002). Immediate neurocognitive effects of concussion. *Neurosurgery*, 50, 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 & Rehabilitation*, 13, 27-35.
- 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.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. (1998a). Second impact syndrome. *Neurology*, 50, 677-683.
- McCrory, P.R. & Berkovic, S.F. (1998b). Concussion Convulsions. *Sports Medicine*, 25(2), 131-136.

- McCrorry, 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.
- McCrorry, P.R., Johnston, K., Meeuwisse, W., Aubry, M., Cantu, R., Dvorak, J., et al. (2005). Summary and agreement statement of the 2<sup>nd</sup> International Conference on Concussion in Sport, Prague 2004. *Clinical Journal of Sports Medicine*, 15(2), 48-55.
- McCrorry, P.R., Makkdissi, M., Davis, & Collie, A. (2005). Value of neuropsychological testing after head injuries in football. *British Journal of Sports Medicine*, 39(Suppl 1), i58-i63.
- McIntosh, A.S., McCrorry, 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.
- McLatchie G., Brooks, N., Galbraith, S., Hutchinson, J.S.F., 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.
- McLean, A., Temkin, N., Dikmen, S., Wyler, A., & Gale, J.L. (1984). Psychosocial functioning at one month after head injury. *Neurosurgery*, 14, 393-399.
- McLean, D.A. (1992). Analysis of the physical demands of international rugby union. *Journal of Sports Science*, 10, 285-296.
- McMahon, K.A., Nolan, T., Bennett, C.M., & Carlin, J.B. (1993). Australian Rules football injuries in children and adolescents. *The Medical Journal of Australia*, 159, 301-306.
- McMillan, T. M. (1997). Minor head injury. *Current opinion in Neurology*, 10, 479-483.
- Meerhoff, S.R., de Kruijk, J.R., Rutten, J., Letters, P., & Twijnstra, A. (2000). (Incidence of traumatic head or brain injuries in catchment area of Academic Hospital Maastricht in 1997). *Nederlands tijdschrift voor geneeskunde*, 144(40), 1915-1918.
- Mehta, K.M., Ott, A., Kalmijn, S., Slooter, A.J., van Duijn, C.M., Hofman, A., et al. (1999). Head trauma and risk of dementia and Alzheimer's disease: the Rotterdam study. *Neurology*, 53, 1957-1962.
- Metzl, J.D. (1999). Sport-specific concerns in the young athlete: Football. *Pediatric Emergency Care*, 15 (5), 363-367.
- Micheli, P.T. & Riseborough, E.M. (1974). The incidence of injuries in rugby football. *Journal of Sports Medicine*, 2, 93-97.
- 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.
- Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest

- Group of the American Congress of Rehabilitation Medicine. (1993). Definition of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation*, 8, 86-87.
- Miller, E.N. (1987-1999). California Computerized Assessment Package (CalCAP).
- Miller, H. (1961). Accident neurosis. *British Medical Journal*, 1, 919-925.
- Miller, J.D. (1986). Minor, moderate and severe head injury. *Neurosurgical Review*, 9(1-2), 135-139.
- Miller, L.S., & Rohling, M.L. (2001). A statistical interpretative method for neuropsychological test data. *Neuropsychology Review*, 11, 143-169.
- Millsbaugh, J.A. (1937). Dementia Pugilistica. *U.S. Naval Medical Bulletin*, 35, 297.
- Minderhoud, J.M., Boelens, M.E.M., Huizenga, J., & Saan, R.J. (1980). Treatment of minor head injuries. *Clinical Neurology and Neurosurgery*, 82, 127-140.
- 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., Grossman, R., Hiehle, J., Hurst, R., Kauder, D., Gennarelli, T., et al. (1994). Prevalence of MR evidence of diffuse axonal injury in patients with mild head injury and normal CT findings. *American Journal of Neuroradiology*, 15, 1583-1589.
- Mitrushina, M., & Satz, P. (1991). Effect of repeated administration of a neuropsychological battery in the elderly. *Journal of Clinical Psychology*, 47(6), 790-801.
- Morrison, R.G. (1986). Medical and public health aspects of boxing. *Journal of the American Medical Association*, 255(18), 2475-2480.
- Moriarty, 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. *American Academy of Neurology*, 62(9), 1497-1502.
- Mortimer, J.A. (1994). What are the risk factors for dementia? In F.A. Huppert, C. Brayne, & D.W. O'Connor (Eds.), *Dementia and Normal Aging*. Cambridge University Press: Cambridge.
- Mortimer, J.A. (1997). Brain reserve and the clinical expression of Alzheimer's disease. *Geriatrics*, 52 (Suppl. 2), 50-53.
- Mortimer, J.A., French, L.R., Hutton, J.T., & Schuman, L.M. (1985). Head injury as risk factor for Alzheimer's disease. *Neurology*, 35, 264-267.
- Moser, R.S., Schatz, P., & Jordan, B.D. (2005). Prolonged effects of concussion in high school athletes. *Neurosurgery*, 57, 300-306.
- Mrazik, M., Ferrara, M.S., Perterson, C.L., Elliott, R.E., Courson, R.W., Clanton, M.D., et al. (2000). Injury severity and neuropsychological and balance outcomes of four college athletes. *Brain Injury*, 14(10), 921-931.

- Murelius, O., & Haglund, Y. (1991). Does Swedish amateur boxing lead to chronic brain damage? A retrospective neuropsychological study. *Acta Neurologica Scandinavica*, 83, 9-13.
- 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.
- Naugle, R.I. (1990). Epidemiology of traumatic brain injury in adults. In E.D. Bigler (Ed.), *Traumatic brain injury*. Austin, TX: Pro-ed.
- Nell, V. & Brown, S.O.D. (1991). Epidemiology of traumatic brain injury in Johannesburg: II. Morbidity, mortality, and etiology. *Social Science and Medicine*, 33, 289-296.
- 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.
- Newcombe, F., Rabbitt, P., & Briggs, M. (1994). Minor head injury: pathophysiology or iatrogenic sequelae? *Journal of Neurology, Neurosurgery, and Psychiatry*, 57, 709-716.
- Noakes, T. & Jakoet, I. (1995). Spinal cord injuries in rugby union. *British Medical Journal*, 310, 1345-1346.
- Northern Transvaal Rugby Union. (1982). *Rugbybeserings: 'n Studie van 'n Statistiese Opname*. Pretoria: The Northern Transvaal Rugby Union.
- Nortje, J. & Menon, D.K. (2004). Traumatic brain injury: physiology, mechanisms, and outcome. *Current Opinion in Neurology*, 17, 711-718.
- O'Connor, T.A., & Burns, N.R. (2003). Inspection time and general speed of processing. *Personality and Individual Differences*, 35, 713-724.
- Office of Population Census and Surveys (1976-1987). *Hospital In-patient Enquiry 1974-1987* (Series MB4, Nos. 1-27). London: Her Majesty's Stationery Office.
- Ommaya, A.K. & Gennarelli, T.A. (1974). Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. *Brain*, 97, 633-654.
- Oppenheimer, R.D. (1968). Microscopic lesions in the brain following head injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, 31, 299-306.
- 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).
- Oxford Concise Colour Medical Dictionary. (1996). Oxford: Oxford University Press.
- Pang, D. (1985). Pathophysiologic correlates of neurobehavioural syndromes following closed head injury. In M. Ylvisaker (Ed.), *Head injury rehabilitation: Children and adolescents* (pp.3-70). London: Taylor & Francis.

- Pang, D. (1989). Physics and pathology of closed head injury. In M.D. Lezak (Ed.), *Assessment of the behavioural consequences of head trauma*. Vol. 7. *Frontiers of clinical neuroscience*. New York : Alan. R. Liss.
- Parasuraman, R., Mutter, S.A., & Molloy, R. (1991). Sustained attention following mild closed-head injury. *Journal of Clinical and Experimental Neuropsychology*, 13(5), 789-811.
- Parker, R.S. (2001). *Concussive brain trauma. Neurobehavioural impairment and maladaptation*. Boca Raton, FL: CRC Press.
- Parkinson, D., Stephensen, S., & Phillips, S. (1985). Head Injuries: A prospective, computerized study. *The Canadian Journal of Surgery*, 28, 79-82.
- Pearn, J. (1998). Boxing, youth and children. *Journal of Paediatrics & Child Health*, 34, 311-313.
- Peek-Asa, C., McArthur, D., Hovda, D. & Kraus, J. (2001). Early predictors of mortality in penetrating compared with closed brain injury. *Brain Injury*, 15, 801-810.
- Pellman, E.J., Lovell, M.R., Viano, D.C., Casson, I.R., & Tucker, A.M. (2004). Concussion in Professional Football: Neuropsychological Testing –Part 6. *Neurosurgery*, 55(6), 1290-1305.
- Pellman, E.J., Lovell, M.R., Viano, D.C., & Casson, I.R. (2006). Concussion in Professional Football: Recovery of NFL and high school athletes assessed by computerized neuropsychological testing–Part 12. *Neurosurgery*, 58(2), 1-10.
- 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.
- Pettersen, J.A. & Skelton, R.W. (2000). Glucose enhances long-term declarative memory in mild head-injured varsity rugby players. *Psychobiology*, 28, 81-89.
- Piaget, J. (1963). *The origins of intelligence in children*. New York: W.W. Norton.
- Pieter, W. & Zemper, E.D. (1998). Incidence of reported cerebral concussion in adult Taekwondo athletes. *Journal of the Royal Society of Health*, 118(5), 272-279
- Pieter, W. & Zemper, E.D. (1999). Head and neck injuries in young taekwondo athletes. *The Journal of Sports Medicine and Physical Fitness*, 39(2), 147-153.
- 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.
- 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., Ayton, G., et al. (1997). Cognitive and behavioural outcome following mild traumatic brain injury in children. *Journal of the International Neuropsychological Society*, 3, 225.
- Ponsford, J., Wilmott, C., Rothwell, A., Cameron, P., Ayton, G., Nelms, R., et al. (1999). Cognitive and behavioural outcome following mild traumatic brain injury in children. *Journal of Head Trauma and Rehabilitation*, 14(4), 360-370.
- 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.
- Porter, M. & O'Brien, M. (1996). Incidence and severity of injuries resulting from amateur boxing in Ireland. *Clinical Journal of Sports Medicine*, 6, 97-101.
- 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. & Barber-Foss, K.D. (1999). Traumatic brain injury in high school athletes. *Journal of the American Medical Association*, 282, 958-963.
- Putukian, M., Echemendia, R.J., and Machin, 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. Practice Parameter. The management of concussion in sports (summary statement). Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology*, 48, 581-585.
- Rabadi, M.H. & Jordan, B.D. (2001). The cumulative effect of repetitive concussion in sports. *Clinical Journal of Sport Medicine*, 11, 194-198.
- Ramirez, J.R. (1998). Attentional deficits in youth boxing: Effects of repeated mild closed head injuries. *The Sciences and Engineering*, 58, 5138.
- 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., 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.
- Rapport, L. J., Brook-Brines, D., Axelrod, B. N., & Theisen, M. E. (1997). Full scale IQ as mediator of practice effects: The rich get richer. *The Clinical Psychologist*, 13(3), 283-380.

- 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.
- Rasmusson, D.X., Brandt, J., Martin, D.B., & Folstein, M.F. (1995). Head injury as a risk factor in Alzheimer's disease. *Brain Injury*, *9(3)*, 213-219.
- 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.
- Reid, S.R. & Losek, J.D. (1999). Factors associated with significant injuries in youth ice hockey players. *Pediatric Emergency Care*, *15(5)*, 310-313.
- 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.
- Report of the Ad Hoc Committee to Study Head Injury Nomenclature: Proceedings of the Congress of Neurologic Surgeons in 1964. *Clinical Neurosurgery*, *12*, 386-394.
- Rey, A. (1941). L'examen psychologique dans les cas d'encephalopathie traumatique. *Archives de Psychologie*, *28*, 286-340 (see Corwin & Bylsma, 1993b for translation).
- Rey, A. (1964). *L'examen clinique em pschologie*. Paris: Presses Universitaires de France.
- Richards, M., & Sacker, A. (2003). Lifetime antecedents of cognitive reserve. *Journal of Clinical and Experimental Neuropsychology*, *25*, 614-624.
- Richardson, J.T.E. (1990). *Clinical and Neuropsychological aspects of closed head injury*. Great Britain: Burgess Science Press.
- Rimel, R.W., Giordani, B., Barth, J.T., Boll, T.J., & Jane, J.A. (1981). Disability caused by minor head injury. *Neurosurgery*, *9(3)*, 221-228.

- Roberts, G.W., Allsop, D., & Bruton, C. (1990). The occult aftermath of boxing. *The Journal of Neurology, Neurosurgery and Psychiatry*, 53, 373-378.
- Roberts, W.O., Brust, J.D., & Leonard, B. (1999). Youth ice hockey tournament injuries: rates and patterns compared to season play. *Medicine & Science in Sports and Exercise*, 31, 46-51.
- Ropper, R.H., & Miller, D.C. (1985). Acute traumatic midbrain hemorrhage. *Annals of Neurology*, 18, 80-86.
- 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.
- 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.
- 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., Camenzuli, I, Mueller, J. (1996). Miserable minority: emotional risk factors that influence the outcome of mild traumatic brain injury. *Brain Injury*, 10(8), 551-65.
- 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.
- Ruijs, M., Gabreels, F., & Thijssen, H. (1994). The utility of electroencephalography and cerebral computed tomography in children with mild and moderately severe closed head injuries. *Neuropediatrics*, 25, 73-77.
- Russell, W.R. (1932). Cerebral involvement in head injury: A study based on the examination of two hundred cases. *Brain*, 55, 549-603.
- Russell, W.R. & Nathan, P.W. (1946). Traumatic amnesia. *Brain*, 69, 280-300.
- Russell, W.R. & Smith, A. (1961). Post-traumatic amnesia in closed head injury. *Archives of Neurology*, 5, 16-29.
- Rust, J. & Golombok, S. (1989). *Modern Psychometrics*. London: Routledge.
- Rutherford, W.H. (1989). Post-concussion symptoms: Relationship to acute neurological indices, individual differences and circumstances of injury. In H. Levin, H.M. Eisenberg, & A.L. Benton (Eds.), *Mild Head Injury* (pp 217-228). New York: Oxford University Press.
- Rutherford, W.H., Merrett, J.D. & McDonald, J.R. (1979). Symptoms at one year following concussion from minor head injuries. *Injury*, 10, 225-230.

- 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.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, A.J. (1987). Intracranial injuries resulting from boxing: a review (1918-1985). *Clinics in Sports Medicine*, 6(1), 31-39.
- Ryan, L.M. & Warden, D.L. (2003). Post concussion syndrome. *International Review of Psychiatry*, 15, 310-316.
- Salcido, R. & Costich, J.F. (1992). Recurrent traumatic brain injury. *Brain Injury*, 6(3), 293-298.
- Santrock, J.W. (1992). *Life-span Development*. (4<sup>th</sup> ed.). United States: Wim. C. Brown Publishers.
- 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.S., Alfano, M.S., Light, R.F., Morgenstern, H.F., Zaucha, K.F., Asarnow, R.F., et al. (1999). Persistent Post-Concussive Syndrome: A proposed methodology and literature review to determine the effects, if any, of mild head and other bodily injury. *Journal of Clinical and Experimental Neuropsychology*, 21(5), 620-628.
- Satz, P., Zaucha, K., McCleary, C., Light, R., Asarnow, R., & Becker, D. (1997). Mild head injury in children and adolescents: A review of studies (1970-1995). *Psychological Bulletin*, 122(2), 107-131.
- Saunders, R.L. & Harbaugh, R.E. (1984). The second impact in catastrophic contact-sports head trauma. *Journal of the American Medical Association*, 252, 538-539.
- Schatz, P. & Chute, D.L. (1995). Predicting level on independence following moderate and severe traumatic brain injury. *Archives of Clinical Neuropsychology*, 11 (5), 444-445.
- Schatz, P., Pardini, J., Lovell, M., & Collins, M. (2006). Sensitivity and Specificity of the ImPACT Test Battery for concussion in athletes. *Archives of Clinical Neuropsychology*, 21, 91-99.
- Schofield, P.W., Logroscino, G., Albert, S., & Stern, Y. (1997). An association between head circumference and Alzheimer's disease in a population-based study of aging and dementia. *Neurology*, 49, 30-37.

- Schofield, P.W., Tang, M., Marder, K., Bell, K., Dooneief, G., Chun, M., et al. (1997). Alzheimer's disease after remote head injury: an incidence study. *Journal of Neurology, Neurosurgery and Psychiatry*, 62, 119-124
- 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.
- Segalowitz, S.J. & Brown, D. (1991). Mild head injury as a source of developmental disabilities. *Journal of Learning Disabilities*, 24, 551-559.
- Segalowitz, S.J. & Lawson, S. (1995). Subtle symptoms associated with self-reported mild head injury. *Journal of Learning Disability*, 28, 309-319.
- 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.
- Servadei, F., Ciucci, G., Piazza, G., Bianchedi, G., Rebutti, G., Gaist, G., et al. (1988). A prospective clinical and epidemiological study of head injuries in northern Italy: the Commune of Ravenna. *Italian Journal of Neurological Sciences*, 9, 449-457.
- Seward, H., Orchard, J., Hazard, H., & Collinson, D. (1993). Football injuries in Australia at the elite level. *The Medical Journal of Australia*, 159, 298-301.
- Shallice, T. & Burgess, P.W. (1991). Deficits in strategy application following frontal lobe damage in man. *Brain*, 114, 727-741.
- 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.
- Sherer, M., Krull, K.R., & Adams, R.L. (1995). Interpretive significance of variability of performance on neuropsychological tests. *Applied Neuropsychology*, 2, 39-41.
- Shewchenko, N., Withnall, C., Gittens, R., & Dvorak, J. (2005). Heading in football. Part 1: Development of biomechanical methods to investigate head response. *British Journal of Sports Medicine*, 39, (Suppl 1), i10-i25.
- 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.B., Border, M., Reid, I., & Radloff, S. (2004). South African Rugby Union. In M.R. Lovell, R.L. Echemendia, J.T. Barth, & M.W. Collins (Eds.). *Traumatic Brain Injury in Sports*. The Netherlands: Swets & Zeitlinger Publishers.
- Shuttleworth-Jordan, A.B. (1997). Age and education effects on brain damaged subjects: "Negative" findings revisited. *The Clinical Neuropsychologist*, 11, 205-209
- Shuttleworth-Jordan, A.B., & Bode, S.G. (1995a). Taking account of the age effect on Digit

Symbol and Incidental Recall for diagnostic purposes. *Journal of Clinical and Experimental Neuropsychology*, 17, 439-448.

- Shuttleworth-Jordan, A.B., & Bode, S.G. (1995b). Use of the SAWAIS Digit Symbol test of Incidental Recall. *South African Journal of Psychology*, 25, 57-60.
- Shuttleworth-Jordan, A.B., Puchert, J., & Balarin, E. (1993). Negative consequences of mild head injury in rugby: a matter worthy of concern. In R. Plunkett & S. Anderson (Eds.). *Proceedings of the 5<sup>th</sup> National Neuropsychology Conference*. Durban: South African Clinical Neuropsychological Association (SACNA).
- 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.
- Simon, B., Letourneau, P., Vitorino, E., & McCall, J. (2001). Pediatric minor head trauma: indications for computed tomography scanning revisited. *Journal of Trauma*, 51(2), 237-238.
- Snowdon, D.A., Ostwald, S.K., & Kane, R.L. (1989). Education, survival and independence of elderly Catholic sisters, 1936-1988. *American Journal of Epidemiology*, 130, 999-1012.
- Solomon, G.S., Johnston, K.M., & Lovell, M.R. (2006). The Heads-up on sport concussion. United States: Human Kinetics.
- Sorensen, S.B. & Kraus, J.F. (1991). Occurrence, severity and outcomes of brain injury. *Journal of Head Trauma Rehabilitation*, 6 (2), 1-10.
- Sortland, O. & Tysvaer, A.T. (1989). Brain damage in former association football players. *Neuroradiology*, 31(1), 44-48.
- Sortir, C. (2001). Complications of Mild Traumatic Brain Injury. *Advance News Magazines*, February, 42-49.
- South African Wechsler Adult Intelligence Scale manual*. (1969). Johannesburg: National Institute for Personnel Research.
- South African Government online*. (n.d.). Retrieved May 28, 2006, from <http://www.gov.za/>
- Sosin, D.M., Sniezek, J.E., & Thurman, D.J. (1996). Incidence of mild and moderate brain Injury in the United States, 1991. *Brain*, 10, 47-54.
- Sowell, E. & Jernigan, T. (1998). Further MRI evidence of late brain maturation: Limbic volume increases and changes asymmetries during childhood and adolescence. *Developmental Neuropsychology*, 14, 599-618.
- Sparks, J.P. (1981). Half a million hours of rugby football. *British Journal of Sports Medicine*, 15(1), 30-32.

- Spreeen, O. (1981). The relationship between learning disability, neurological impairment and delinquency. *Journal of Nervous and Mental Disease*, 169, 791-799.
- Spreeen, O., & Straus, E. (1998). *A compendium of neuropsychological tests* (2<sup>nd</sup> ed.). New York: Oxford University Press.
- Stablum, F. Mogentale, C., Umilta, C. (1996). Executive functioning following mild closed head injury. *Cortex*, 32, 261-278.
- Stambrook, M., Moore, A.D., Peters, L.C., Deviaene, C., & Hawryluk, G.A. (1990). Effects of mild, moderate and severe closed head injury on long-term vocational status. *Brain Injury*, 4, 183-190.
- Stapells, D.R. (2004, 2005). What are auditory evoked potentials? Retrieved from <http://www.audiospeech.ubc.ca/haplab/aep.htm>.
- 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
- 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. (2006). *Cognitive Reserve. Theory and Applications*. NewYork: Taylor & Francis.
- Stern, Y., Zarahn, E., Hilton, H.J., Flynn, J., DeLaPaz, R., & Rakitin, B. (2003). Exploring The neural basis of cognitive reserve. *Journal of Clinical and Experimental Neuropsychology*, 25, 691-701.
- Stewart, W., Gordon, B., Selnes, O., Bandeen-Roche, K., Zeger, S., Tusa, R.J., et al. (1994). Prospective study of central nervous system function in amateur boxers in the United States. *American Journal of Epidemiology*, 139, 573-588.
- Stillings, N.A., Feinstein, M.A., Garfield, J., Rissland, E., Rosenbaum, A. Weisler, S., et al. (1987). *Cognitive Science*. Cambridge, MA: MIT Press.
- Strauss, I. & Savitsky, N. (1934). Head injury: neurological and psychiatric aspects. *Archives of Neurological Psychiatry*, 31, 893.
- 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 (Suppl 1), i70-i77.
- Strich, S.J. (1961). Shearing of nerve fibers as a cause of brain damage due to head injury. *The Lancet*, ii, 446-448.

- 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.
- Stuss, D.T. (1987). Contribution of the frontal lobe injury to cognitive impairment after Closed head injury: Methods of assessment and recent findings. In H.S. Levin, J. Grafman, & H.M. Eisenberg (eds.), *Neurobehavioural recovery from head injury* (pp. 166-177). New York: Oxford University Press.
- Suhr, J.A. & Gunstad, J. (2002). Postconcussive symptom report: The relative influence of head injury and depression. *Journal of Clinical and Experimental Neuropsychology*, *24*(8), 981-993.
- Susman, M., DiRusso, S.M., Sullivan, T., Risucci, T., Nealon, P., Cuff, S., et al. (2002). Traumatic brain injury in the elderly: increased mortality and worse functional outcome at discharge despite lower injury severity. *Journal of Trauma*, *53*(2), 219-223.
- Sweet, J.J., Peck, E.A. III., Abromowitz, C., & Etzeiler, S. (2003). National Academy of Neuropsychology/Division 40 of the American Psychological Association practice survey of clinical neuropsychology in the United States Part II: reimbursement experience, practice economics, billing practices and incomes. *Archives of Clinical Neuropsychology*, *18*, 557-582.
- Tate, R.L., McDonald, S., & Lulham, J.M. (1991). Incidence of hospital-treated traumatic brain injury in an Australian community. *Australian New Zealand Journal of Public Health*, *22*, 419-423.
- Tateno, A., Jorge, R.E., & Robinson, R.G. (2003). Clinical correlates of aggressive behaviour after traumatic brain injury. *Journal of Neuropsychiatry and Clinical Neuroscience*, *15*, 155-160.
- Teasdale, G & Jeannett, B. (1974). Assessment of coma and impaired consciousness: A practical scale. *Lancet*, *2*, 81-84.
- Teasdale, G. & Mendelow, D. (1984). Pathophysiology of head injuries. In. Brooks (Ed.), *Closed head injury. Psychological, social and family consequences*. Oxford : Oxford University Press.
- Teasdale, G., Nicoll, J.A., Murray, G., & Fiddes, M. (1997). Association of apolipoprotein E polymorphism with outcome after head injury. *Lancet*, *350*, 1069-1071.
- Teasdale, G., Murray, G., & Nicoll, J.A. (2005). The association between APOE, age and outcome after head injury: a prospective cohort study. *Brain*, (Epub head of print).
- Tegeler, C., Kim, J.Y., Collins, G., Steelman, D., Westwood, K., Reynolds, P. et al. (n.d.). *Transcranial Doppler Ultrasound for concussion in amateur athletes*. Retrieved April 16<sup>th</sup>, 2006, from Wake Forrest University, WFU Sports Medicine Web site: <http://www.wfu.edu/athletic/medicine/concussion2.html>.
- Tegeler, C. (2004). *New developments in neuroscience*. Paper presented at the New Developments in Sports-Related Concussion Conference. Pittsburgh, July 2004.

- Tegner, Y. & Lorentzon, R. (1996). Concussion among Swedish elite ice hockey players. *British Journal of Sports Medicine*, 30, 251-255.
- Temkin, N.R., Machamer, J.E., & Dikmen, S.S. (2003). Correlates of functional status 3-5 years after traumatic brain injury with CT abnormalities. *Journal of Neurotrauma*, 20(3), 229-241.
- Theye, F. & Mueller, K.A. (2004). "Heads up": Concussions in High School Sports. *Clinical Medical Research*, 2(3), 165-171.
- Thomas, R.M. (1992). *Comparing Theories of Child Development (3<sup>rd</sup> Ed)*. California: Wadsworth, Inc.
- Thompson, J., Sebastianelli, W., & Slobounov, S. (2005). EEG and postural correlates of mild traumatic brain injury in athletes. *Neuroscience Letters*, 377, 158-163.
- Thornhill, S., Teasdale, G.M., Murray, G.D., McEwan, J., Roy, C.W., & Penny, K.I. (2000). Disability in young people and adults one year after head injury: prospective cohort study. *British Medical Journal*, 320, 1631-1635.
- Thurman, D., Branche, C., & Sniezek, J. (1998). The epidemiology of sports-related traumatic brain injuries in the United States: recent developments. *Journal of Head Trauma and Rehabilitation*, 13, 1-8.
- Tiret, L., Hausherr, E., Thicoipe, M., Garros, B., Maurette, P., Castel, J.P., et al. (1990). The epidemiology of head trauma in Aquitaine (France), 1986: a community-based study of hospital admissions and deaths. *International Journal of Epidemiology*, 19, 133-140.
- Tromp, E. & Mulder, T. (1991). Slowness of information processing after traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology*, 13(6), 821-830.
- Tysvaer, A. & Storli, O.V. (1989). Soccer injuries to the brain: a neurologic and electroencephalographic study of active football players. *American Journal of Sports Medicine*, 17, 573-578.
- Tysvaer, A., Storli, O.V., & Bachen, N.I. (1989). Soccer injuries to the brain: a neurologic and electroencephalographic study of former players. *Acta Neurologica Scandinavica*, 80, 151-156.
- Tysvaer, A. & Lochen, E. (1991). Soccer injuries to the brain: A neuropsychologic study of former soccer players. *The American Journal of Sports Medicine*, 19, 56-60.
- 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 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.
- 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.

- Vásquez-Barquero, A., Vásquez-Barquero, J.L., Austin, O., Pascual, J., Gaité, L., & Herrera, S. (1992). The epidemiology of head injury in Cantabria. *European Journal of Epidemiology*, 8, 832-837.
- Vieth, A.Z., Johnstone, B., & Dawson, B. (1996). Extent of intellectual, cognitive, and academic decline in adolescent traumatic brain injury. *Brain Injury*, 10(6), 465-470.
- Walker, R.D. (1985). Sports injuries: rugby league may be less dangerous than union. *The Practitioner*, 229, 205-206.
- 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.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). *Manual for the Wechsler Adult Intelligence Scale – Third Edition*. San Antonio: The Psychological Corporation.
- Weight, D.G. (1998). Minor Head Trauma. *The Psychiatric Clinics of North America*, 21(3), 609-624.
- Wennberg, R.A. & Tator, C.H. (2003). National Hockey League reported concussions, 1986-1987 to 2001-2002. *Canadian Journal of Neurological Sciences*, 30, 206-209.
- Wikipedia, the free encyclopedia*. (n.d.). Retrieved March 31, 2006, from [http://en.wikipedia.org/wiki/Contact\\_sport](http://en.wikipedia.org/wiki/Contact_sport)
- Wikipedia, the free encyclopedia*. (n.d.). Retrieved May 28, 2006, from [http://en.wikipedia.org/wiki/Ice\\_hockey](http://en.wikipedia.org/wiki/Ice_hockey)
- Wikipedia, the free encyclopedia*. (n.d.). Retrieved May 30, 2006 from [http://en.wikipedia.org/wiki/Construct\\_validity](http://en.wikipedia.org/wiki/Construct_validity)
- 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.
- Wilberger, J.E. (1993). Minor head injuries in American football. *Sports Medicine*, 15(5), 338-343.

- Willer, B., Dumas, J., Hutson, A., Leddy, J. (2004). A population based investigation of head injuries and symptoms of concussion of children and adolescents in schools. *Injury Prevention, 10*(3), 144-148.
- Williams, D.H., Levin, H.S., & Eisenberg, H.M. (1990). Mild head injury classification. *Neurosurgery, 27*, 422-428.
- Wills, S.M. & Leathem, M. (2001). Sports-related brain injury research: Methodological difficulties associated with ambiguous terminology. *Brain Injury, 15*(7), 645-648.
- 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.
- Woods, S.P., Rippeth, J.D., Conover, E., Carey, C.L., Parsns, T.D., & Troster, A.I. (2006). Statistical power of studies examining the cognitive effects of subthalamic nucleus deep brain stimulatoin 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.
- Wrightson, P. & Gronwall, D. (1989). Management of disability and rehabilitation services after mild head injury. In H. Levin, H.M. Eisenberg, & A.L. Benson (Eds.), *Mild head Injury*. New York: Oxford University Press
- 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.
- Yarnell, P.R. & Lynch, S. (1970). Retrograde memory immediately after concussion. *Lancet, 1*, 863-864.
- Young, H.A., Gleave, J.R.W., Schmidek, H.H., & Gregory, S. (1984). Delayed traumatic intracerebral hematoma: report of 15 cases operatively treated. *Neurosurgery, 14*, 22-25.
- Zhang, M., Katzman, R., Salmon, D., Jin, H., Cai, G., Wang, Z., et al. (1990). The prevalence of dementia and Alzheimer's disease in shanghai, China: The impact of age, gender and education. *Annals of Neurology, 27*, 428-437
- Zemper, E.D. & Pieter, W. (2004). Injury rates during the 1988 United States Olympic trials for taekwondo. *British Journal of Sports Medicine, 23*, 161-164.

## Appendix A

### Consent forms

Dear Parents

#### **CONCUSSION IN SCHOOL SPORT**

In keeping with our goals to maximize the safety of sports players generally, and particularly in the contact sports where there is a known risk of concussion, we plan to implement the latest internationally approved risk prevention strategies for concussion.

It is generally considered that computer-based screening of reaction times and memory function provides easily accessible, yet *crucial* information for concussion management especially with respect to return-to-play decisions. Our objective is to support an innovative study that aims to develop such facilities within the South African school context, which are already extensively in place in sports playing institutions in countries such as the USA, Australia and New Zealand. Consequently, your son will have the unique opportunity to participate in the groundbreaking research by clinicians at Rhodes University, who are working in collaboration with the University of Pittsburgh Medical School, USA and the MRC/UCT Research Unit for Exercise Science and Sports Medicine, Newlands, South Africa.

All Grade 12 schoolboys will be the first to participate in this monitoring and risk prevention project. These schoolboys will be monitored pre and post winter sport season using scientifically valid computerized screening systems developed in the USA specifically for concussion management. Screening will involve the evaluation for functions such as memory, reaction time and processing speed and will take the form of simple paper and pencil exercises and computer games. These are usually enjoyed by the boys and take between 40 minutes to complete. In addition, pupils will be requested to fill out a brief demographic questionnaire with medical background and a symptom checklist, with relevance to the research.

There will be ongoing sports concussion monitoring through the winter season and appropriate intervention. In the event of a concussion, follow-up evaluation of concussed players will take place within 6 days of injury and then again at weekly intervals, until acute symptoms resolve. Pre and post season testing will take place on the school property. Concussion follow-ups will take place at the Sports Science Institute of South Africa, Boundary Road Newlands. The data will be examined by researchers at Rhodes University and if the outcome proves favourable in terms of minimizing risk to players, the strategy will be introduced to other grades. The results of the research will be used for scientific publication purposes only by the collaborating universities.

It is important to be aware that this study does not interfere with or substitute for good medical practice. We therefore advise that all schoolboys with concussion should be seen as soon as possible by their general practitioner or other medical practitioners and should not return to contact sport for at least 3 weeks from time of injury and thereafter on the advice of the medical practitioner. The information collected on individual schoolboys will be strictly confidential and will only be made available to the parents and /or a medical practitioner on request. This information may form part of the management decision in individual cases. However, the researchers will not be held accountable for medical decisions made by medical practitioners or parents on the basis of that information.

We believe that it is in your son's best interest to participate in this concussion monitoring and risk prevention project although participation is voluntary and you have the right to withdraw from the entire project or part thereof if you so wish. Indication of your desire to do so must be done by signing a withdrawal from before the 14<sup>th</sup> February 2003, which will be made available by contacting the sports head. At the same time any further queries that you may have can be discussed.

Yours sincerely  
Deputy Principal

## BRIEF TO PARENTS REGARDING CONCERNS ABOUT CHILD'S PARTICIPATION IN RESEARCH

We believe that it is in your son's best interest to participate in this concussion monitoring and risk prevention project, although participation is voluntary.

1. The parent has the right to withdraw the child from the entire project or any part thereof. Withdrawal of the child from the project will not prejudice him in any way in terms of his position in team sport.
2. Withdrawal of the child from the project must occur in writing. The parent must complete the withdrawal form below.
3. We emphasize that the project is neither invasive nor harmful to the child's physical, mental and/or emotional well-being. For research purposes, the identity of the participants will be kept strictly confidential and individual data will be made available for clinical purposes only with parental permission.
4. By not participating in the project, in the event of a concussion, no base-line scores or concussion follow-up by the researcher will be available for that child.
5. Should your son not participate in any formal sports activity, his participation is still of crucial benefit to the research. This is in order to make comparisons between the boys who are exposed to sports concussion, and those who are not exposed. Moreover, should your son sustain a head injury for any other reason, any deterioration in cognitive functioning would be more accurately assessed in relation to baseline data derived from the study.

---

### WITHDRAWAL FORM

I \_\_\_\_\_ hereby wish to withdraw my son  
\_\_\_\_\_ from participating in the school's concussion project. I am  
aware of the possible negative consequences to my son's well-being in declining computer-based monitoring in respect  
of concussion.

SIGNED: \_\_\_\_\_

DATE: \_\_\_\_\_

**RHODES UNIVERSITY  
DEPARTMENT OF PSYCHOLOGY  
HEADMASTER CONSENT FORM**

I, \_\_\_\_\_ headmaster of \_\_\_\_\_ have been informed of the nature of the research which will be conducted by Rhodes University doctoral student, Victoria Whitefield on the effects of concussion in school rugby.

**I understand that:**

1. The above mentioned students are conducting the concussion management research as a requirement for a PhD degree at Rhodes University in collaboration with the University of Pittsburgh Medical School, USA and the MRC/ UCT Research Unit for Exercise Science and Sports Medicine, Newlands, South Africa.
2. The research will involve all Grade 12 schoolboys, who will be assessed using internationally validated computer-based neuropsychological screening batteries, pre and post winter sport season, during school hours. The initial pre-season assessments will take approximately one hour; all subsequent assessment will take 30-40 minutes. Follow-up assessments of concussed players will take place within 6 days, of injury and then again at weekly intervals, until acute symptoms resolve. Pre and post season testing will take place on the school property. Concussion follow-ups will take place at the Sports Institute of South Africa, Boundary Road, Newlands. In addition, pupils will be requested to fill out a brief demographic questionnaire with medical background and a symptom checklist, with relevance to the research.
3. The study does not interfere with or substitute for good medical practice. It is therefore advised that all schoolboys with concussion should be seen as soon as possible by their general practitioner or other medical practitioners and should not return to contact sport for at least 3 weeks from the time of injury and thereafter on the advice of the medical practitioner.
4. Participation in the research is strictly voluntary and parents have the right to withdraw their sons from the study at any stage. Parents must contact the school in order to sign a withdrawal from should they not wish their sons to participate in the study.
5. The information collected on individual schoolboys will be strictly confidential and will only be made available to the parents and /or a medical practitioner on request. This information may form part of the management decision in individual cases. However, the researchers will not be held accountable for medical decisions made by medical practitioners or parents on the basis of that information.
6. Data arising out of this project will be used for thesis and publication purposes only by the collaborating universities.

I, hereby give consent for those pupils who will be participating in this research project to be assessed by the mentioned researchers.

Signed on: \_\_\_\_\_

## Appendix B

### Pre-season Questionnaire and Pre- and Post-season paper-and-pencil Postconcussion Symptom Questionnaires

Name: \_\_\_\_\_

Age: \_\_\_\_\_

Date of Birth: \_\_\_\_\_

Grade: \_\_\_\_\_

Class: \_\_\_\_\_

Contact number during the school term: \_\_\_\_\_

#### *A: Background Information*

Name of school: \_\_\_\_\_

Height: \_\_\_\_\_

Weight: \_\_\_\_\_

Right handed:

Left handed:  (Please tick)

Country of birth, other than South Africa: \_\_\_\_\_ First language: \_\_\_\_\_

Second language: \_\_\_\_\_ Years speaking second language: \_\_\_\_\_

Please tick if you have received any of the following:

- Speech therapy
- Attended special classes or remedial classes
- Occupational therapy
- Repeated any grades at school
- Diagnosed with ADD or Hyperactivity
- Diagnosed with a learning disability

Name your current sport(s): \_\_\_\_\_

What is your position? \_\_\_\_\_

What team were you in last year? \_\_\_\_\_

How many years have you played at this level (not including this year)? \_\_\_\_\_

How many times have you be diagnosed by with a concussion (i.e. felt dazed, dizzy or confused, however briefly or unconscious)? \_\_\_\_\_

If you have sustained a concussion please complete the following:

Concussion	Approximate date	Reason for concussion
1.		
2.		
3.		
4.		
5.		
6.		

From the list above, name the total number of concussions that resulted in a loss of consciousness: \_\_\_\_\_

List the total number of concussions that resulted in confusion: \_\_\_\_\_

List the total number of concussions that resulted in difficulty with memory for events occurring immediately after the injury: \_\_\_\_\_

List the total number of concussions that resulted in difficulty with memory for events occurring immediately before the injury: \_\_\_\_\_

Please indicate whether you have experienced the following:

- |                              |                             |   |
|------------------------------|-----------------------------|---|
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Treatment for headaches by physician                                |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Treatment for migraine headaches by a physician                     |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Treatment for epilepsy/seizures                                     |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | History of meningitis   |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Treatment for substance/alcohol abuse                               |
| <input type="checkbox"/> Yes | <input type="checkbox"/> No | Treatment for psychiatric condition (e.g. depression, anxiety etc.) |

***B: Current Symptoms and Conditions***

Hours of sleep last night: \_\_\_\_\_

Current medications: \_\_\_\_\_

Please tick the appropriate number below that indicates the degree to which you are currently experiencing any of the following:

**a) Headache**

not experiencing this symptom

- |                                  |                         |                                     |                         |                                   |
|----------------------------------|-------------------------|-------------------------------------|-------------------------|-----------------------------------|
| <input type="radio"/> 1<br>Minor | <input type="radio"/> 2 | <input type="radio"/> 3<br>somewhat | <input type="radio"/> 4 | <input type="radio"/> 5<br>severe |
|----------------------------------|-------------------------|-------------------------------------|-------------------------|-----------------------------------|

**b) Nausea**

not experiencing this symptom

- |                                  |                         |                                     |                         |                                   |
|----------------------------------|-------------------------|-------------------------------------|-------------------------|-----------------------------------|
| <input type="radio"/> 1<br>Minor | <input type="radio"/> 2 | <input type="radio"/> 3<br>somewhat | <input type="radio"/> 4 | <input type="radio"/> 5<br>severe |
|----------------------------------|-------------------------|-------------------------------------|-------------------------|-----------------------------------|

**c) Vomiting**

not experiencing this symptom

- |                                  |                         |                                     |                         |                                   |
|----------------------------------|-------------------------|-------------------------------------|-------------------------|-----------------------------------|
| <input type="radio"/> 1<br>Minor | <input type="radio"/> 2 | <input type="radio"/> 3<br>somewhat | <input type="radio"/> 4 | <input type="radio"/> 5<br>severe |
|----------------------------------|-------------------------|-------------------------------------|-------------------------|-----------------------------------|

**d) Poor appetite**

not experiencing this symptom

- |                                  |                         |                                     |                         |                                   |
|----------------------------------|-------------------------|-------------------------------------|-------------------------|-----------------------------------|
| <input type="radio"/> 1<br>Minor | <input type="radio"/> 2 | <input type="radio"/> 3<br>somewhat | <input type="radio"/> 4 | <input type="radio"/> 5<br>severe |
|----------------------------------|-------------------------|-------------------------------------|-------------------------|-----------------------------------|

**e) Balance problems**

not experiencing this symptom

1  
Minor

2

3  
somewhat

4

5  
severe

**f) Dizziness**

not experiencing this symptom

1  
Minor

2

3  
somewhat

4

5  
severe

**g) Fatigue/Tiredness**

not experiencing this symptom

1  
Minor

2

3  
somewhat

4

5  
severe

**h) Trouble falling asleep**

not experiencing this symptom

1  
Minor

2

3  
somewhat

4

5  
severe

**i) Sleeping more than usual**

not experiencing this symptom

1  
Minor

2

3  
somewhat

4

5  
severe

**j) Sleeping less than usual**

not experiencing this symptom

1  
Minor

2

3  
somewhat

4

5  
severe

**k) Drowsiness**

not experiencing this symptom

1  
Minor

2

3  
somewhat

4

5  
severe

**l) Sensitivity to light**

not experiencing this symptom

1  
Minor

2

3  
somewhat

4

5  
severe

**m) Sensitivity to noise**

not experiencing this symptom

- 1 Minor      2      3 somewhat      4      5 severe

**n) Difficulty hearing**

not experiencing this symptom

- 1 Minor      2      3 somewhat      4      5 severe

**o) Irritability**

not experiencing this symptom

- 1 Minor      2      3 somewhat      4      5 severe

**p) Aggression**

not experiencing this symptom

- 1 Minor      2      3 somewhat      4      5 severe

**q) Sadness**

not experiencing this symptom

- 1 Minor      2      3 somewhat      4      5 severe

**r) Nervousness**

not experiencing this symptom

- 1 Minor      2      3 somewhat      4      5 severe

**s) Feeling more emotional**

not experiencing this symptom

- 1 Minor      2      3 somewhat      4      5 severe

**t) Numbness or tingling**

not experiencing this symptom

- 1 Minor      2      3 somewhat      4      5 severe

**u) Feeling slowed down**  not experiencing this symptom

1 Minor      2      3 somewhat      4      5 severe

**v) Feeling mentally foggy**  not experiencing this symptom

1 Minor      2      3 somewhat      4      5 severe

**w) Difficulty concentrating**  not experiencing this symptom

1 Minor      2      3 somewhat      4      5 severe

**x) Difficulty remembering**  not experiencing this symptom

1 Minor      2      3 somewhat      4      5 severe

**y) Visual problems (e.g. double vision or blurring etc.)**  not experiencing this symptom

1 Minor      2      3 somewhat      4      5 severe

**z) Speech problems**  not experiencing this symptom

1 Minor      2      3 somewhat      4      5 severe

## POST-SEASON QUESTIONNAIRE

NAME: \_\_\_\_\_

Please tick the number below that indicates the degree to which you are currently experiencing the following:

**a) Headache**  not experiencing this symptom

1                      2                      3                      4                      5  
Minor                      somewhat                      severe

**b) Poor appetite**  not experiencing this symptom

1                      2                      3                      4                      5  
Minor                      somewhat                      severe

**c) Feeling mentally foggy**  not experiencing this symptom

1                      2                      3                      4                      5  
Minor                      somewhat                      severe

**d) Difficulty hearing**  not experiencing this symptom

1                      2                      3                      4                      5  
Minor                      somewhat                      severe

**e) Difficulty concentrating**  not experiencing this symptom

1                      2                      3                      4                      5  
Minor                      somewhat                      severe

**f) Difficulty remembering**  not experiencing this symptom

1                      2                      3                      4                      5  
Minor                      somewhat                      severe

**g) Speech problems**  not experiencing this symptom

1                      2                      3                      4                      5  
Minor                      somewhat                      severe

**h) Aggression**  not experiencing this symptom

1                      2                      3                      4                      5  
Minor                      somewhat                      severe

**i) Feeling slowed down**  not experiencing this symptom

1                      2                      3                      4                      5  
Minor                      somewhat                      severe

