Cumulative Mild Head Injuries in football (soccer): a comparison of cognitive deficit and Post - concussive symptomology between University of Pretoria (AmaTuks) football players and University of Limpopo volleyball controls in South Africa.

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Cumulative Mild Head Injuries in football (soccer): a comparison of cognitive deficit and Post-concussive symptomology between University of Pretoria (AmaTuks) football players and University of Limpopo volleyball controls in South Africa

by

Patricia Maite

RESEARCH DISSERTATION

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ABSTRACT

The aim of the study was to investigate the effect of Cumulative Mild Head Injury (CMHI) or concussion and Post-Concussive symptomatology of football players. For this purpose, a non-equivalent quasi experimental design of fifteen (15) professional football players and non-contact control group of fifteen (15) university volleyball players were assessed on 4 reaction time tasks and Post-Concussion Symptom questionnaire. The main findings of the study showed no significant difference on cognitive changes among football players and the control group. However, the two sample t-test, ANOVA and ANCOVA analysis in respect of both football players and the volleyball control group, provided significant results of a probability of CMHI or concussion on the CALCAPôs Sequential 1 Reaction Time Test. The study makes important theoretical and practical contributions to the understanding of Post-Concussion syndrome. The study suggests that some of the Post-Concussion symptoms persist after an initial concussion.

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To my other daughter Lerato, your everlasting love, patience and unceasing support were invaluable. You helped me persevere when the journey was difficult. I thank you for giving me the strength throughout by helping me to remember my goal.

DECLARATION

I declare that Cumulative Mild Head Injuries in football (soccer): a comparison of cognitive deficit and Post-concussive symptomology between University of Pretoria (AmaTuks) football players and University of Limpopo volleyball controls in South Africa Thesis hereby submitted to the University of Limpopo, for the degree of Doctor Of Philosophy has not previously been submitted by me for a degree at this or any other university; that it is my work in-design and in execution, and that all material contained herein has been duly acknowledged.

_____ _____

Ms. P. Maite **Date**

TABLE OF CONTENT

Abstract ..... **i**

Acknowledgements ..... **ii**

Declaration ..... **iii**

Content List ..... **iv**

List of Tables, Figures, Diagrams and Checklist ..... **viii**

Chapter 1: Introduction to the study

1.1 Introduction 1

1.2 The research question 2

1.3 Background to the study 3

1.4 Aim of the study 7

1.5 Objectives of the study 7

1.6 Significance of the study 7

1.7 Summary 8

Chapter 2: Literature Review

2.1 Introduction 9

2.2 Head Injury 9

2.2.1 Frontal lobes of the brain 9

2.2.2 Open Head Injury 10

2.2.3 Closed Head Injury 12

2.3 Mild Head Injury (MHI) 13

2.3.1 Cumulative Mild Head Injury 15

2.3.2 Concussion 17

2.4 Severity of brain injury 19

2.4.1 Coma classification 20

2.4.2 Post-Traumatic Amnesia (PTA) 21

2.5 Incidence of Mild Head Injury 22

2.6 The mechanisms of Mild Head Injury.....26
2.6.1 Primary brain injury.....26
2.6.2 Secondary brain injury.....27
2.6.2.1 Haemorrhage and Haematoma 28
2.6.2.2 Diffuse Brain Swelling.....28
2.6.2.3 Abnormalities in Brain Structure 29
2.6.2.4 Neuropsychological abnormalities.....30
2.7 Pathophysiology of Mild Head Injury (MHI) 30
2.7.1 Diffuse Axonal Injury 30
2.7.2 Post-Concussive Syndrome (PCS).....32
2.7.3 Acute symptoms and symptoms recovery from post-concussive.....36
syndrome (PCS)
2.7.4 Post concussive syndrome (PCS) Diagnostic Criteria 37
2.7.5 Reliability and validity of post-concussion criteria 39
2.7.6 Treatment for headaches and dizziness related to post-concussion 39
syndrome
2.8 Neuropsychological sequelae of MHI..... 40
2.9 Neuropsychological recovery following MHI..... 42
2.10 Cognitive Deficits 43
2. 10.1 Attention 43
2.10.2 The three models of attention.....46
2.10.2.1 Mensulamôs (2000) selective and Spatial Model.....46
2.10.2.2 Posnerôs (2001) Anterior and Posterior Attention Model.....47
2.10.2.3 Mirskyôs (1996) Elements of Attention Model 48
2.10.3 Attention shift.....49
2.10.4 Arousal 50
2.10.5 Alertness.....50
2.10.6 Sustained attention.....51
2.10.7 Selective attention.....52
2.10.8 Focused attention.....53
2.10.9 Divided attention 54
2.10.10 Visuo-spatial orienting of attention 55
2.10.11 Attention and the concept of Information Processing 56

2.10.12 Reaction Time 56
2.11 Use of Neuroimaging to diagnose Mild Head Injury (MHI).....59
2.12 Neuropsychological assessment 61
2.12.1 Neuropsychological assessment for evaluating attention deficits 62
2.12.2 Arousal and Alertness 62
2.12.3 Selective Attention 63
2.12.4 Focused Attention 63
2.12.5 Divided Attention 63
2.12.6 Sustained Attention.....64
2.12.7 Computerised neuropsychological assessment.....64
2.13 Cross-cultural neurological assessment.....65
2.14 Summary.....66

Chapter 3: Theoretical framework of the study

3.1 Introduction 67
3.2 Theoretical context and hypothetical framework for the present 67
3.3 MHI in sport 69
3.4 Epidemiology of MHI and concussion 70
in sport
3.5 Pathophysiology of (MHI in sport 73
3.5.1 Neurochemical Changes after head injury.....74
3.5.1.1 Hormonal Imbalances after head injury 75
3.5.2 Second Impact Syndrome (SIS) 77
3.5.3 Epidemiology of Second Impact Syndrome (SIS) 77
3.5.4 Dementia Pugilistica 78
3.6 American Football 79
3.7 Boxing 80
3.8 Australian Rules Football 81
3.9 Rugby League.....84

3.10 Rugby Union 87
3.11 Football (Soccer) 89
3.12 Under-reporting or Non-recognition of MTBI.....92
3.13 Concussion management.....92
3.13.1 Modifying factor in concussion management.....94
3.13.2 The significance of loss of consciousness (LOC).....94
3.13.3 The significance of amnesia and other factors.....94
3.13.4 Depression.....95
3.14 Return to play.....96
3.14.1 Rehabilitation after brain injury.....96
3.14.2 Recovery and rehabilitation after brain injury.....97
3.14.3 The role of pre-participation concussion evaluation.....102
3.15 The child and adolescent athlete.....102
3.16 Summary.....104

Chapter 4: Research Methodology

4.1 Introduction.....105
4.2 Selection Criteria.....105
4.2.1 Inclusion Criteria.....106
4.2.2 Exclusion Criteria.....106
4.2.3 Final Sample.....108
4.2.4 Sample characteristics.....108
4.2.5 Age, language, education and race.....108
4.3 Data administration.....109
4.3.1 Pre-season (Baseline) Testing.....109
4.3.2 Post-season (end of season) Testing.....111
4.4 Instruments used in the study.....111
4.4.1 Validity and Reliability of The California Computerised Package (CALCAP).....112
4.4.2 Additional questionnaire.....114
4.4.2.1 Reliability and validity of the Rivermead PCS Questionnaire (RPQ).....115
4.4.2.2 Threats to internal validity of the study.....116

4.5 Data analysis.....117
4.5.1 Significance level.....118
4.6 Hypothesis.....118
4.7 Ethical considerations.....120
4.8 Summary.....121

Chapter 5: Analysis of Results

5.1 Introduction.....122
5.2 Demographic characteristics of the sample.....122
5.2.1 Age.....122
5.2.2 Education.....123
5.2.3 Pre-Season (Baseline) Testing: Two sample t-test comparison for.....124
the experimental and control groups on CALCAP
5.2.4 Post-Season assessment: Two sample t-test comparison for.....125
the experimental and control groups on CALCAP
5.2.5 Pre and post-season assessment: two sample t-test comparison on.....126
CALCAP
5.2.6 Repeated measures analysis of variance (ANOVA) for.....127
post-test mean scores for football and volleyball control groups
on CALCAP
5.2.7 Analysis of Covariance (ANCOVA) with a single covariance.....128
comparison between football and volleyball control groups
5.2.8 Summary of ANOVA and ANCOVA on CALCAP.....130
5.2.9 Figure representation of the mean RT of the football and.....130
volleyball control groups
5.3 Post-Concussive Symptom Checklist comparisons for the.....135
experimental football group and the volleyball controls
5.4 The combination of results for the football experimental group and.....157
the volleyball control group
5.4.1 Neurocognitive measures, CALCAP.....157
5.4.2 Post-Concussion Symptom Checklist.....158
5.5 Summary.....159

Chapter 6: Discussion of Results, Evaluation and Recommendation of the Research

6.1 Introduction.....160
6.2 Broad outline of this study.....160
6.3 Interpretation of findings for football and the control groups.....163
6.3.1 Interpretation of findings on neurocognitive measures.....163
6.3.2 Interpretation of findings on the Rivermead PCS measure.....165
6.4 Combination of findings for the neurocognitive symptoms.....167 measures
6.5 Critical evaluation of the study.....168
6.5.1 Methodological Strengths of the study.....168
6.5.2 Limitations of the study.....169
6.6 Comparative groups.....169
6.7 Research measures.....171
6.8 Other methodological and statistics concerns for research.....172 on MTBI in sport
6.8.1 Practice effects.....172
6.8.2 Sample size.....173
6.8.3 Lack of control for extraneous variables.....173
6.8.4 Under-reporting of MTBI.....174
6.9 Implications of the results.....174
6.9.1 General implications.....174
6.9.2 Implications of Satzôs Brain Reserve Theory (1993).....175
6.10 Implications of future research.....176
6.11 Summary..... 177

List of Tables

Table 1: The American Academy of Neurology Guidelines (adapted from Maroon et al., 2000)

Table 2: Mathesonôs (1994) Post Traumatic Amnesia (PTA) classification

Table 3: Posner and Rothbart (2006) Attentional Networks

Table 4: Mirskyôs (1996) Elements of Attention

Table 5: University of Pittsburgh side-line mental status testing card (on field cognitive testing procedure)

Table 6: Concussion modifiers

Table 7: Graduated return-to-play protocol (McCrory et al., 2009)

Table 8: Exclusions from the experimental Football and Volleyball control groups

Table 9: Age distribution for football and the control group

Table 10: Education distribution for football playing group

Table 11: Education distribution for the volleyball playing control group

Table12: Pre-Season two sample t-test mean scores comparison on CALCAP for the football group and the volleyball controls

Table 13: Post-season two sample t-test mean scores comparison on CALCAP for the football group and the volleyball control group

Table 14: Pre-season versus post-season comparison of means for the football and control groups on the CALCAP using a two sample t-test

Table 15: Post-season CALCAP repeated measure analysis of variance (ANOVA) for the football players and volleyball controls

Table 16: Analysis of covariance (ANCOVA) between football players and the volleyball control group

Table 17: Summary of pre and post-assessments of Post-Concussion Symptoms: experimental group

Table 18: Summary of pre and post-assessments of Post-Concussion Symptoms: Control group

Table 19: Summary of all pre and post-assessment of Post-Concussion Symptoms change: football playing experimental group

Table 20: Summary of all pre and post-assessment of Post-Concussion Symptoms change: volleyball control group

Table 21: Frequency of all pre and post-assessment of Post-Concussion Symptom change: football playing group

Table 22: Frequency of all pre and post-assessment of Post-Concussion Symptom change: Control group

Table 23: Football and volleyball control groupsô count on symptom intensity (worsened) at pre-season versus post-season

Table 24: Football and volleyball control groupsô count on symptom intensity (unchanged) at pre-season versus post-season

Table 25: Football and volleyball control groupsô count on symptoms intensity (improved) counts pre-season versus post-season

List of Diagrams

Diagram 1: Frontal lobes of the brain (Headway, 2012)

Diagram 2: Open Head Injury, penetration of skull with foreign object (British Medical Journal, 2011)

Diagram 3: Closed Head Injury (CT scan of left frontal lobe acute epidural)

Diagram 4: Concussion injuries (Lucas & Coertzen, 2010)

Diagram 5: Diffuse Axonal (DAI)

Diagram 6: The Pituitary gland (Headway, 2012)

List of Diagrams

Diagram 1: Frontal lobes of the brain (Headway, 2012)

Diagram 2: Open Head Injury, penetration of skull with foreign object (British Medical Journal, 2011)

Diagram 3: Closed Head Injury (CT scan of left frontal lobe acute epidural)

Diagram 4: Concussion injuries (Lucas & Coertzen, 2010)

Diagram 5: Diffuse Axonal (DAI)

Diagram 6: The Pituitary gland (Headway, 2012)

List of Checklists

Checklist 1: Management of concussion card (New Zealand Rugby Concussion Policy, 2010)

Checklist 2: Return to play protocol from the internationally approved guidelines (New Zealand Rugby Concussion Policy, 2010).

Checklist 3: The SCAT card (Patricios et al., 2012)

CHAPTER 1: INTRODUCTION

1.1 Introduction

The study will explore and describe how Cumulative Mild Head Injury (CMHI) can pose cognitive problems amongst football players who have experienced this type of repetitive head injury (CMHI). Studies on mature players have noted a decline in cognitive function resulting in repetitive head trauma for instance, heading the ball (Janda, Bir & Cheney, 2002). Players use the heading technique to pass the ball to each other and to move the ball up and down the field in order to score a goal. In South Africa during and after the football world cup the sport has been promoted as one that is a healthy pastime and a nation building game. The study is seen as relevant as it investigates possible cognitive injuries that occur as a result of the sport. Professional football players were compared to a control group of volleyball players. Volleyball players were deemed appropriate as a control group because they do not engage in any physical contact nor do they use their head to pass, or head the ball to each other. The cognitive and post-concussion symptoms were assessed pre (baseline) and post (end of) season for both football and the volleyball control group to acquire baseline information. At present, there is very limited research on the subject and none in South Africa.

The post-concussion symptoms were evaluated through Rivermead Post-Concussion Self-Report Questionnaire (King, Crawford, Wenden, Moss & Wade, 1995). The California Computerised Assessment Package (CALCAP) was used to identify cognitive decline (Miller, 1990). CALCAP identifies deficits in components of attention such as focused, divided attention and sustained attention. In this study, CALCAP was used as a measure of reaction time (RT), changes in RT and the speed of information processing. In the information processing approach, the researcher measures the speed with which the participants perform specific and elementary cognitive operations. The speed of performance on these operations was assessed through simple, choice and Sequential Reaction Time (RT). Results were analysed by direct comparison of the football players and volleyball playersô mean levels on the CALCAP and the post-concussive symptoms self-report questionnaire.

1.2 The research question

The research question arose out of the findings from a relatively new body of research in the field of mild head injury in contact sport. The pivotal question is, ñWhat is the effect of sports-related MHI on cognitive functioning amongst football players?ò There is a well-documented high incidence of this type of injury in other sports such as rugby union (Bennet & Raymond, 1997; Echemendia & Julian 2001; Gardner, Shores & Batchelor, 2010; Garraway, Lee, Hutton, Russell & Macleod, 2000). MHI is often accompanied by residual cognitive symptomatology that may interfere with for instance, the cognitive skills necessary for academic success at university (Peterson & Bernhardt, 2011). In addition, even though a lot of research has investigated the sequelae of single uncomplicated MHI, there is a lack of consensus with regard to the chronic effects of CMHI (Binder, Rohling, & Larrabee, 1997; Satz, Zaucha, McCleary, & Light, 1997).

Players of contact sport run the risk of head injury. Lezak, Howieson and Loring (2004), state that many sports injuries, especially contact sports, fall into the category of Mild Traumatic Brain Injury (MTBI). Research into the cumulative effects of mild head trauma in contact sports suggests that diffuse brain damage may be present. This type of injury has been found in players of American Football, Boxing, Australian Rules football, Rugby league, Rugby Union and Football players (Matser, Kessels, Lezak, Jordan, & Troost, 2009). Individuals who sustain MTBI often report a cluster of unfavourable events, commonly referred to as post-concussive symptoms. Post-concussive symptoms are usually divided into three categories namely, physical, (which may occur in the absence of cognitive deficits) and include headaches, blurred vision and inability to concentrate (Kibby & Long, 1996; Paré, Rabin, Fogel, & Pépin, 2009) emotional symptoms which include increased anxiety, emotional lability, depression and attention and memory deficits (Evans, 1992; Paré, et al., 2009; Szymanski & Linn, 1992).

According to Arciniegas, Anderson, Topkoff and McAllister (2005) the main dysfunction in MBTI which is often referred to as Mild Head Injury (MHI) is slowed information processing. They suggest that the dysfunction of the attentional control system reduces the rate of information processing after head injuries. Individuals frequently have difficulty with aspects of attention when required to analyse many items simultaneously. They appear to be slow, easily

distracted, forgetful and inattentive because of the extra effort required to process information. It may take weeks to months (and sometimes longer) for all symptoms to abate. In the case of contact sport players this can be problematic as they are often pressurised to return to the field of play when not properly rehabilitated (Nel, 2009).

1.3 Background to the study

The study will explore and describe how Cumulative Mild Head Injuries CMHI can pose cognitive problems amongst South African professional football players who have experienced mild repetitive head injuries during one season. In general there is lack of knowledge and scientific study about the topic in South Africa. The study data was collected pre and post the 2011 football season as the focus is on CMHI sustained during one season. Attention and memory deficits are the most evident neuropsychological sequelae after MHI however, attention was chosen as the one of the focuses of this study because it is the least well understood (Niemann, Ruff & Kramer, 1996; Nobre & Coull, 2010). The study is situated within the theoretical context of Satzôs (1993) Brain Reserve Capacity (BRC) theory.

The different types of head injury are described to contextualise the study. According to Burton and Volpe (1994) cited in Mureriwa (1997) Traumatic Brain Injury (TBI) is one of the main sources of brain injury both in South Africa and globally. One of the complications arising from TBI is cognitive deficit. TBI is categorised as either an open or penetrating injury or a closed injury. Open TBI injury occurs when there is penetration by a foreign object in the brain and is associated with a fractured or perforated skull (Levin, Benton & Grossman, 1982; Lezak et al., 2004 & Richardson, 1990). Closed TBI occurs when the brain undergoes either a noticeable acceleration or deceleration or both (Bohnen, Jolles & Twinstra, 1992; Zillmer, Spiers & Culbertson, 2008). Closed TBI occurs in two stages, namely primary and secondary injury. Primary injury occurs at the time of the impact and secondary injury is the physiological process that sets in after the primary injury (Lezak et al., 2004). The severity of closed TBI depends on how much diffuse axonal damage is present and can cover a range of severity from mild, moderate to severe (Dikmen, Temkin & Armsden, 1989; Elson & Ward, 1994; Lezak et al., 2004). Classification of TBI is accomplished through using the Glasgow Coma Scale (GCS) an

initial score of 13 to 15, without subsequent deterioration, places patients in the mild category for head trauma (Davidhizar & Bartlet, 1997; Evans, 1992; Menascu & Tshechmer, 2011).

MTBI which, in sporting circles, is commonly referred to as concussion falls under closed head injury. According to Menascu and Tshechmer (2011), MTBI is a brief or negligible loss of consciousness (LOC) and memory which doesnôt last for more than an hour. In the sports arena where concussion or concussive injury are the favoured terms for MTBI a series of definitions has evolved. However, there has been a lack of a universally accepted definition for concussive injuries and many of the common symptoms that occur. In addition, relatively minor impact injuries that result in either persistent physical or cognitive symptoms have not been included in these definitions. A World Health Organisation (WHO) study estimated that between 70% and 90% of head injuries that receive treatment are mild. However, due to under-reporting it is difficult to determine how common the condition is.

Elson and Ward (1994) state that MTBI is similar to moderate and severe head injury can lead to Diffuse Axonal Injury (DAI). DAI is caused by shearing forces generated by sudden brain acceleration or deceleration (Alexander, 1995; Lezak et al., 2004). DAI is the primary neuropathology of head injury. Alexander (1995) and Lezak et al., (2004) support Holbourn (1943) in stating that diffuse axonal damage is the main causal mechanism for injury and ensuring behavioural dysfunction in MTBI. In the acute phase of MTBI, a period of one to three months post injury, a range of complications from mild to severe can occur. The areas in which MTBI is associated with problems in the acute phase post-injury are firstly, cognitive deficits such as difficulty in concentrating, memory problems and impaired problem solving (Arciniegas, Anderson, Topkoff & McAllister, 2005; Binder, 1986; Dischinger, Ryb, Kufera & Auman, 2009). Secondly, Post Traumatic Amnesia (PTA) and thirdly, physical symptoms including dizziness, fatigue, sleep difficulty, nausea, headaches, blurred vision and insensitivity to intense light and sound (Arciniegas et al., 2005; Bernstein, 1999; Sundström, 2006). The same symptoms that are present under physical symptoms are also present in PCS or Post - Concussive Syndrome (Anderson, 1996; Dischinger, Ryb, Kufera & Auman, 2009). Emotional sequelae have also been identified as an MTBI problem area with the symptomology which includes irritability,

anger outbursts, depression, anxiety and poor social functioning (Arciniegas et al., 2005; Bernstein, 1999).

There is also the possibility of long term harmful effects incurred because of mild repetitive head injuries, particularly those sustained in contact sport (Macciocchi, Barth & Littlefield, 1998). De Villiers (1987) states that one of the most disturbing features of mild concussive or sub-concussive brain injury is that the effects are cumulative. Gronwall and Wrightson (1975) as cited in Janda, Bir and Cheney (2002) introduced the concept of cumulative effects of concussion after they had conducted a study of twenty people with CMHI. They found that there was a decline in the ability of the personôs ability to process information when compared to a control group of individuals who had incurred only one concussion**.** Dacey, Vollmer and Dikmen (1993) and Larrabee (1999) state that evidence suggests that the risk of permanent cognitive deficits may increase as a result of CMHI.

The effect of CMHI can be theoretically understood in terms of BRC and Threshold Concept Theory. Satzôs (1993) BRC theory postulates a threshold factor that exists before the manifestation of symptoms caused by disease in the Central Nervous System (CNS). This threshold represents the critical amount of brain tissue at which normal functioning can be sustained. The model holds that BRC capacity thresholds differ between individuals. Educational levels represent an indirect measure of an individualôs BRC threshold; it suggests that the higher the education levels the more protection against negative CNS symptomology. In terms of the theory, a higher BRC usually acts as a protective factor, decreasing the risk of functional impairment and the likelihood of an individual exhibiting symptomology associated with neurological impairment. Individuals with lower BRC have a higher vulnerability factor creating greater risk of functional impairment. In terms of this theory a reduction in BRC due to neurological pathology is likely to increase an individualôs functional impairment. An individual with less BRC is thus more likely to exhibit neuropsychological impairment.

Players of contact sport run the risk of repeated head injury. According to Lezak et al., (2004) most sports injuries, especially contact sports, fall into the category of MHI. American Football is the most notable protagonist, as cerebral concussions occur frequently, more than 250 000 concussions occur yearly in this sport. One in five high school American Football players suffers a concussion annually. Physicians have become more conservative in treating these patients, as

they are aware that the ability to process information is reduced after a concussion, and the duration and severity of functional impairment is greater with repeated concussions. Rugby Union is a sport where impact injuries to the neck, back and head are common and where MHI can occur as a result thereof (Gardner, Shores & Bachelor, 2010; Shuttleworth-Jordan, Balarin & Pucheret, 1993). Research indicates that MHI occurs frequently in Rugby Union players who

suffer many concussive injuries, particularly frontline players **(**Barnes, Cooper, Kirkendall, McDermott, Jordan & Garrett, 1998; Kaplan, Goodwillie, Strauss & Rosen, 2008; Reid, 1998).

Football is a sport where CMHI frequently occurs and can have serious outcomes. Although football was once designated a non-contact sport contemporary literature refers to football as a contact sport (Giannotti, Al-Sahab, McFaull & Tamim, 2010). It is estimated that between 4% and 22% of injuries in football are head injuries involving trauma to the brain (Tysvaer & Storli, 1989). Evidence indicates that concussion in football goes unrecognized and undiagnosed therefore players donǿt usually seek medical attention (Al-Kashmiri & Delaney, 2006). In normal play, head trauma frequently arises from on-field collisions. It may also arise as a consequence of blows to the head through frequent heading of the ball which results in numerous sub-concussive blows to the head from impact with the soccer ball (Matser, Kessels, Lezak, Jordan & Troost, 2009). An investigation using a sample of 31 football players, and a control group of 31non-contact sport tennis players, showed evidence of poorer information processing in the football players (Abreau, Templer, Schuyler, & Hutchison, 1990). Frequent heading of a hard and fast moving ball by soccer players is a concern with regard to the lasting effects of CMHI (Ruchinskas, Francis & Barth, 1997). The cognitive consequences of heading a ball were studied in adult football players in Norway who had played football from early childhood. Eighty one percent of the players who were tested showed mild to severe deficits in attention span, concentration and memory. The research concluded that players who head the ball more frequently during football competitions have the highest rates of these cognitive deficits.

1.4 Aim of the study

The aim of the study is to investigate the effects of Cumulative Mild Head Injury (CMHI) and Post-Concussive symptomology of football players and compare them to a non-contact sport control group (volleyball players).

1.5 Objectives of the study

The study objectives are:

- to determine if there is a correlation between symptoms of Mild Head Injury (MHI) and cognitive impairment between football players and volleyball players (control group);
- to examine the extent of cognitive impairments amongst football players and the volleyball players pre and post season;
- to compare the experimental (football players) group with the control (volleyball players) group;
- to inform and impart knowledge to the football fraternity, specifically in Gauteng, and generally in South Africa about CMHI.

1.6 Significance of the study

The significance of the study is as follows:

- to contribute to the limited scientific knowledge regarding the relationship between CMHI and cognitive impairments;
- to help increase safety for football players;
- to broaden the knowledge base about the effects of CMHI.

1.7 Summary

This chapter contextualised the present research. The background to the study gave an overview of head injuries and a theoretical understanding of Cumulative Mild Head Injury (CMHI) in terms of Brain Reserve Capacity (BRC) and Threshold Concept theory. The following chapter focuses on a further understanding of CMHI within the broader context of head injuries and contextualises the problem using relevant literature.

CHAPTER 2: LITERATURE REVIEW

2.1 Introduction

The different types of MHI are defined within the broader context of (TBI). The phrase TBI refers to neural damage resulting from open and/or closed head injuries following an accident involving the brain which is often catastrophic (Mureriwa, 1997) whilst MHI describes a type of head injury that is not catastrophic (Menascu & Tshecmer, 2011). A brief overview of the anatomy and neuro-anatomy of the brain will be given to contextualise the study, diagrams or photographic representations will be provided where appropriate to illustrate the mechanisms involved in head injuries. Neuropsychological assessment and deficits associated with mild cognitive injury (particularly those related to attention span) will also be discussed.

2.2 Head Injury

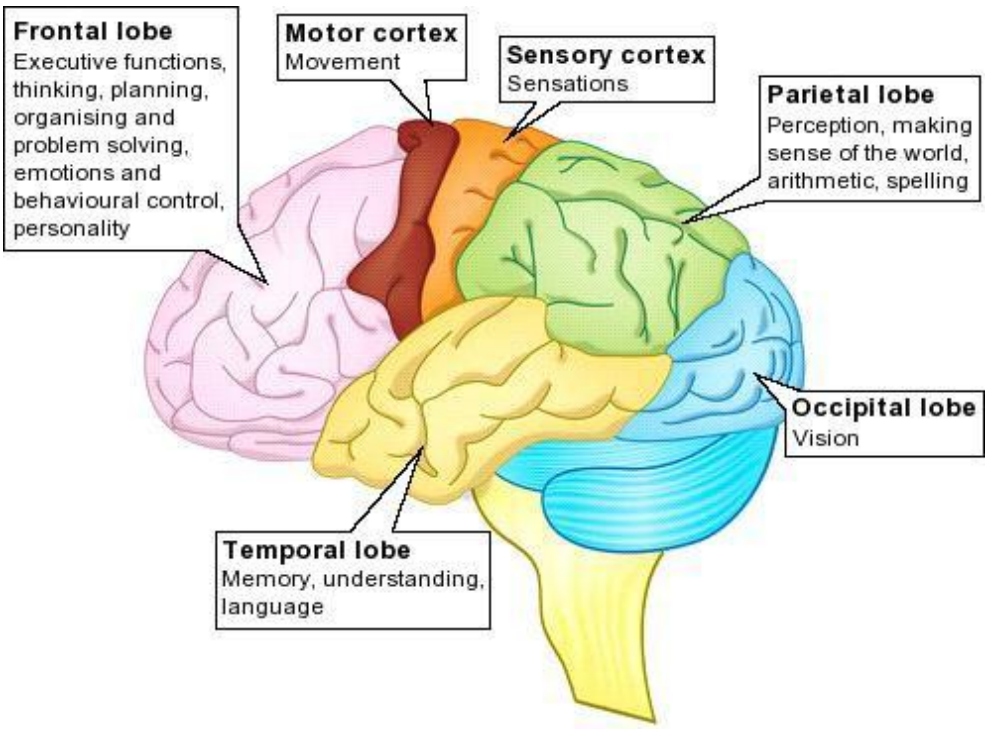
(TBI) refers to a catastrophic or serious injury involving the brain. Most TBIôs are closed which means the skull remains intact and the brain is not exposed. Closed head injuries (CHIôs) can also be referred to as blunt head trauma injuries. The skull can be fractured and the injury may still be closed. Penetrating head injuries (PHIôs) are sometimes called open head injuries. PHI may include all injuries from any source in which the skull and dura are penetrated by missiles or other objects. The term TBI can include other aetiologies like stroke and anoxia (lack of oxygen to the brain) which are also catastrophic in nature (Lezak et al., 2004).

2.2.1 Frontal lobes of the brain

The frontal lobes of the brain because of their position at the front of the skull (behind the forehead) are particularly susceptible to damage. This type of damage can occur because of illness (for instance, viral meningitis) or any TBI for instance, a blow to the head incurred because of falling hard, being hit with an object or repeated blows to the head. Blows that hit the back of the skull can cause damage to the frontal lobes of the brain. This is because the brain is

not attached to the skull and moves backwards and forwards in the skull if a blow to the head is received. This results in the brain hitting the bony protuberances of the skull behind the eyes, which causes bruising or bleeding to the brain (Headway, 2011; Lezak et al., 2004; Shuttleworth-Jordan, 1991). A diagram (See diagram 1) is provided to illustrate where the frontal lobes lie, how large they are (also making them more susceptible to damage), and what role they play in the functionality of the brain.

Diagram 1: Frontal lobes of the brain (Headway, 2012)

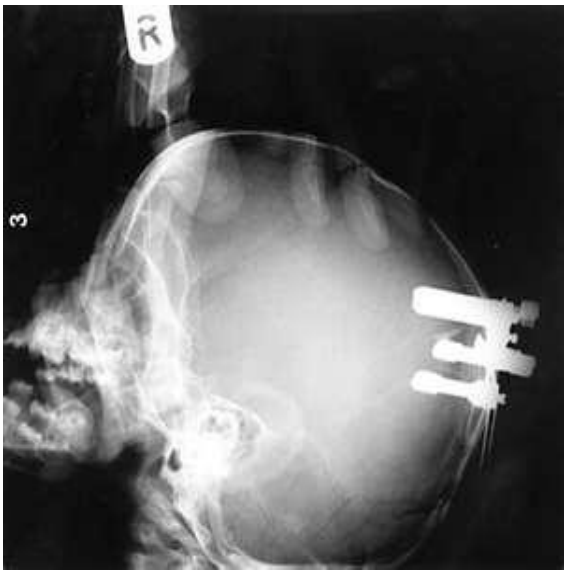


2.2.2 Open Head Injury

Open head injury occurs when there is forceful penetration of a foreign object to the brain (See Diagram 2) and is associated with a fractured or perforated skull (Levin, Benton & Grossman, 1982; Lezak et al., 2004; Richardson, 1990). The damage to brain tissue is normally concentrated in the path of the intruding object. These injuries often result in the exposure of the intra-dural contents of the brain to the atmosphere (Lezak et al., 2004; Mureriwa, 1997). Open head injuries can also result from a tangent injury in which an object glances off the skull and bone fragments are driven into the brain. Many of these injuries have been reported to be as a result of strange

sources for instance, ball-point pens, chopsticks, door keys and metal door display stands that have penetrated the skull (See Diagram 3). Some of these objects become rooted in the head and others, including bullets, cause through and through injury with both entry and exit wounds (Lezak et al., 2004). The neurological signs and effects of these injuries are highly specific (Kolb & Wishaw, 2003). For, example widespread scalp wounds may cause so much blood loss that hypotension (abnormally low blood pressure) and hypovolemia (abnormally low blood volume) occur. Some injuries, for instance, gunshot wounds result in brain contusions (bruises) especially at entry and countercoup sites. Intracranial haematomas (swelling filled with blood) usually develop three to eight hours post injury and can cause catastrophic injury to the brain. The leading cause of open head injury is gunshot wounds to the head. The mortality rate of open head injury is much higher than closed head injury, around 6.6 to 1. In a study of open head injury outcomes 36% of patients were already dead on arrival or died in the emergency trauma unit. Of the open head injuries admitted for inpatient care, 52% were severe, 7% moderate and 42% were mild. Forty one percent of these admissions died in the first 48 hours after being admitted to hospital (Lezak et al., 2004).

Diagram 2: Open head injury, penetration of skull with foreign object (British Medical Journal, 2011)

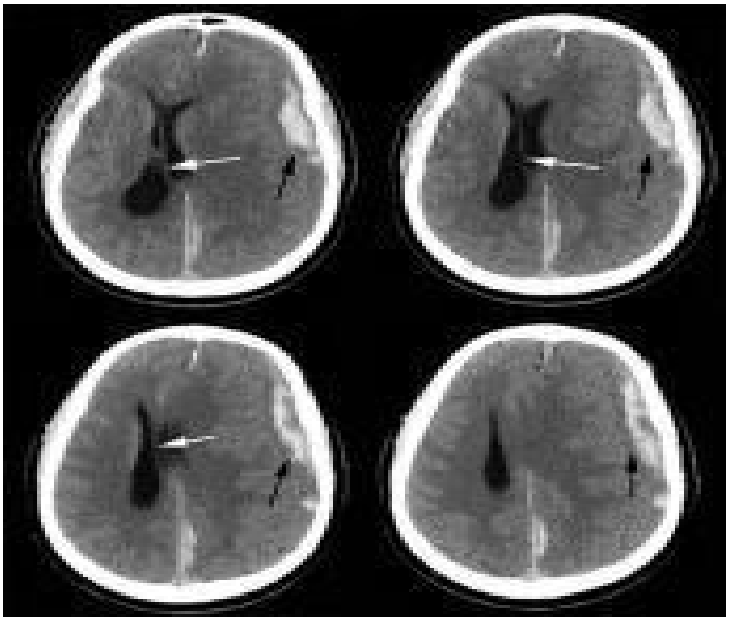


2.2.3 Closed Head Injury

Bohnen, Twijnstra and Jolles (1992) and Zillmer, Spiers and Culbertson, (2008) postulate that closed head traumas have many different causes, but the most common is when the brain undergoes either a noticeable acceleration or deceleration or both. Zillmer et al., (2008) state that in acceleration, the brain experiences a significant physical force that propels it quickly from being stationery to rapid movement. This can happen when the brain is hit by a moving object such as a hard ball or a bat or racquet of some kind. In deceleration, the brain is already in motion and then it suddenly stops, often immediately. Most accidents cause acceleration and deceleration of the brain but some are more traumatic than others for instance, a motor vehicle accident (MVA) where a vehicle is moving at fast speeds and then suddenly stops, can cause massive brain trauma. On the other hand repeated blows to the head in sport (concussive injury) cause milder injuries to the brain (which may be chronic rather than acute), the effects of which may not be immediately apparent (Nel, 2009; Shuttleworth-Jordan, et. al., 1993).

Closed head injury results from a blow to the head which can subject the brain to a number of mechanical forces (See Diagram 3). Firstly, there is damage at the site of the blow; a bruise (contusion) named a coup. Coups are incurred where the brain has been compacted by the skull pushing inwards, even when it is not fractured. Secondly, the pressure that produces the coup may push the brain against the opposite side of the skull, producing an additional bruise, known as a countercoup. Thirdly, the movement of the brain may cause a twisting or shearing of fibres producing microscopic lesions. These lesions may occur throughout the brain but they are most common in the frontal and temporal lobes. The twisting and shearing may damage the major fibre tracts of the brain, especially those crossing the midline (Kolb & Wishaw, 2003). This type of injury can occur when the brain undergoes an obvious injury or one that is not as apparent, and includes acceleration and deceleration forces (Bohnen et al., 1992; Zillmer et al., 2008).

Diagram 3: Close head injury (CT scan of left frontal lobe acute epidural haematomas (Rangel-Castilla, 2011)



2.3 Mild Head Injury (MHI)

The field of MHI has evoked much controversy with regard to its definition and classification. Lack of a single universally accepted system of severity classification has severely limited both research and clarification on this area. Satz et al., (1997) assert that the determination of head injury classification, particularly in the mild to moderate category, represents one of the most fundamental problems confronting researchers of head injury**.** Binder et al., (1997) support this statement and note that different definitions of MHI cause problems for researchers in both analysing and understanding data. MHI is commonly referred to as concussion falls under closed head injury. The term MHI is broadly understood to refer to head injuries in which loss of consciousness (LOC) and/or Post Traumatic Amnesia (PTA) is relatively brief and in which there is an absence of any structural pathology of the skull (Binder, 1986). Criteria used in defining MHI are usually based on definitions of consciousness, measured by Glasgow coma scale (GCS), changes in orientation and memory (duration of PTA) and length of unconsciousness (Arciniegas et al., 2005; Satz et al., 1997). However, whilst these symptoms are successfully used to define the more severe range of head injuries, they become unreliable or not

applicable in the mildest range of head injury severity. Whilst the criteria used in defining MHI has differed considerably in literature through the decades it has been stressed that an appropriate definition, globally accepted, is necessary to ensure the exclusion of confounding variables (Arcniegas et al., 2005; Evans, 1992). Evans (1992) specified a classification of MHI in terms of the duration LOC of 30 minutes or less without further neurological complications.

At an Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (1993) the spectrum of MHI was broadened to include different grades of injury severity. It was recommended that MHI be defined by the measure of at least one of the following: (a) any period of LOC for less than 30 minutes, with GCS of 13 to 15 following the LOC; (b) any loss of memory for events immediately before or after the accident with PTA of less than 24 hours; (c) any alteration in mental state at the time of the accident (for instance, double vision, loss of balance, taste or smell) that may or may not be transient. This definition encompassed a broader range of injury severity than was traditionally used (Satz et al., 1997).

2.3.1 Cumulative Mild Head Injury (CMHI)

There is evidence that indicates that cumulative and more permanent neuropsychological impairments arise from repeated MHI, as a consequence of neural attrition, which may be harmful to an athleteôs well-being (Gronwall, 1989; Rutherford, Stephens, Potter & Fernie, 2005). A study on neuro-trauma indicated that repeated head injuries result in cumulative damage to the brain and cause cumulative damage to hippocampal cells (Packard, 2008). Further, repeated CMHI occurring over an extended period of time (months or years) is likely to result in cumulative neurological and cognitive deficits (Silver, McAllister & Arciniegas, 2009). These studies support earlier findings by Gronwall and Wrightson (1975) on twenty young adults after they had experienced at least two concussions. Results found that the rate at which participants were able to process information was reduced more than that of the control group which had experienced only one concussion. The participants who had experienced a second concussion also took longer to recover than the controls. The effects of concussion are thus likely to be cumulative which has important implications for contact sports where concussion and CMHI injuries are common.

The previous sections attempted to highlight the complex and yet unresolved issues of the long term effects following MHI. Researchers suggest that it is safe to conclude that a single MHI to individuals with no prior compromising condition probably produces mild, clinically insignificant difficulties up to one month Post - injury, as yet no clearly demonstrable permanent side effects have been identified (Dikmen, McLean & Temkin, 1986; Levin, 1995; Shuttleworth - Edwards & Radloff, 2008). However, the reversibility of sub-acute cognitive deficits after MHI does not exclude the presence of microscopic lesions which may reduce an individualôs cerebral reserve in response to a later head injury (Opperheimer, 1968 Shuttleworth ï Edwards & Radloff, 2008). Gronwall and Wrightson (1975) report that individuals who incurred two or more concussions showed a decreased rate in information processing and slower reaction time than individuals with only one concussion. The same authors conclude that cognitive deficits are more persistent in patients with a history of multiple head injuries. A later study by Gronwall (1989) using participants with MHI corroborates his earlier findings in older individuals and individuals with previous head injuries. These individuals displayed impairment in speed of information processing and took longer to recover than the group with a single MHI. However, a recent study conducted in the United Kingdom (UK) compared three independent groups for CMHI on neuropsychological test scores. In this research school team football players, school team rugby players were the experimental groups and school team non-contact sport players were the controls. The hypotheses predicted poorer neuropsychological performance in the experimental groups after a number of head injuries. However, the results did not support this. CMHI amongst the football playing group did not appear to affect their neuropsychological performance (Stephens, Rutherford, Potter & Fernie, 2010). Another study by Straume-Naesheim, Andersen, Holme, McIntosh, Dvorak and Bahr (2009) identified a total of 228 impacts in players of contact sport (which were noted as causing mild trauma to the brain). They followed up 44 of these impacts with a computerised testing programme for cognitive functioning. The programme tested, amongst other things, simple reaction time, choice reaction time and congruent reaction time. The results indicated reduced neuropsychological performance after MHI in football players even in reportedly asymptomatic players. However, it was stated that the long term cognitive consequences are unclear. These findings provide support for the hypothesis that the effects of MHI may be cumulative and indicate that the course of recovery is prolonged after successive injuries (which are thought to inflict progressive diffuse axonal

injury). It appears from the above-mentioned studies that the sequelae following MHI may be cumulative even if an individual has recovered clinically and, in fact, that the course of recovery is prolonged after successive injuries. It is thus reasonable to conclude that there is a risk of increasingly negative consequences from subsequent head injuries (Levin et al., 1987; Shuttleworth-Edwards et al., 2008). The most dangerous of these consequences is referred to in the literature as second impact syndrome. This is when a minor second impact, sustained before full resolution of the symptoms of the first concussion, may result in fatal brain swelling (Echemendia & Julian, 2001; Mueller & Colgate, 2009; Saunders & Harbaugh, 1984). The concept of cumulative damage is integral to this research as players of contact sports such as football run the risk of sustaining CMHI.

One of the most disturbing features of mild concussive or sub-concussive brain injury is that the effects are cumulative (Ancer, 2000; Echemendia & Julian, 2001; McCrory et al., 2009). According to Gronwall and Wrightson (1975) as cited in Janda et al., (2002) the concept of the cumulative effects of concussion was postulated after they conducted a study of 20 participants who had several MHI or concussive injuries. They found that these individuals experienced a decline in the ability to process information when compared to individuals who had only suffered one concussion. This evidence suggests that the risk of permanent deficit may increase as a result of CMHI (Dikmen et al., 1989; Larrabee, 1999; Lezak et al., 2004; McAllister, 2005). However, a study of football players in Australia by Maddocks, Saling and Dicker (1995) does not support the idea of the cumulative effects of repeated MHI. The authors argue that the Gronwall and Wrightsonôs (1975) study included participants who had been injured in MVAôs involving acceleration or deceleration forces of greater consequence than the acceleration or deceleration forces involved in head injuries incurred in playing contact sport. However, the Maddocks et al., (1995) study had methodological limitations which included that a retrospective concussive head injury history was obtained from both the football players and the control group who supposedly had no previous head injuries, this may have confounded results. Another limitation was noted as the difficulty in getting football players to accurately report their history of head injuries because of pressure to remain in their teams. Another factor is that many knocks to the head, which occur on the field of play, are not reported because players donôt notice them (McCrory et al., 2009; Roux, Goedeke & Visser, 1987; Ruchinskas, Francis, & Barth, 1997).

2.3.2 Concussion

Concussion is a brain injury that may result in a bad headache and/or changed levels of alertness or unconsciousness. Lezak et al., (2004) define concussion as immediate disturbances in neurological functioning created by the mechanical forces of rapid acceleration or deceleration of the brain inside the skull as a result of a shock, jarring, or blow to the head. It is more properly classified under diffuse axonal injury. In defining the parameters of concussion, the Committee of Head Injury of the Congress of Neurological Surgeons (1966) proposed that even transient impairment of neural function following head injury, such as the alteration of consciousness and disturbances of vision and equilibrium, constitutes concussion. Cantu (1986) raised concerns about the wide range of differing categories of concussion which make the comparison of research data difficult. He then proposed guidelines cerebral concussion. The American Academy of Neurology Concussion Grading Scale, adapted from Maroon et al. (2000) follows below (see Table 1). In this study, terms like concussion, Mild Head Injury and Mild Traumatic Brain Injury (MBTI) and Cumulative Mild Head Injury (CMHI) are often used interchangeably and to all intent and purpose mean the same thing. However, the study will focus on the use of the latter term, as it is appropriate for the nature of the study, which emphasises repeated head injuries incurred while playing a contact sport.

Table 1: The American Academy of Neurology Guidelines (adapted from Maroon et al., 2000)

Grade 1 - Mild	Transient confusion No loss of consciousness Symptoms resolve in less than 15 minutes
Grade 2 - Moderate	Transient confusion No loss of consciousness Symptoms last longer than 15 minutes
Grade 3 ï Severe	Any loss of consciousness (brief or prolonged)

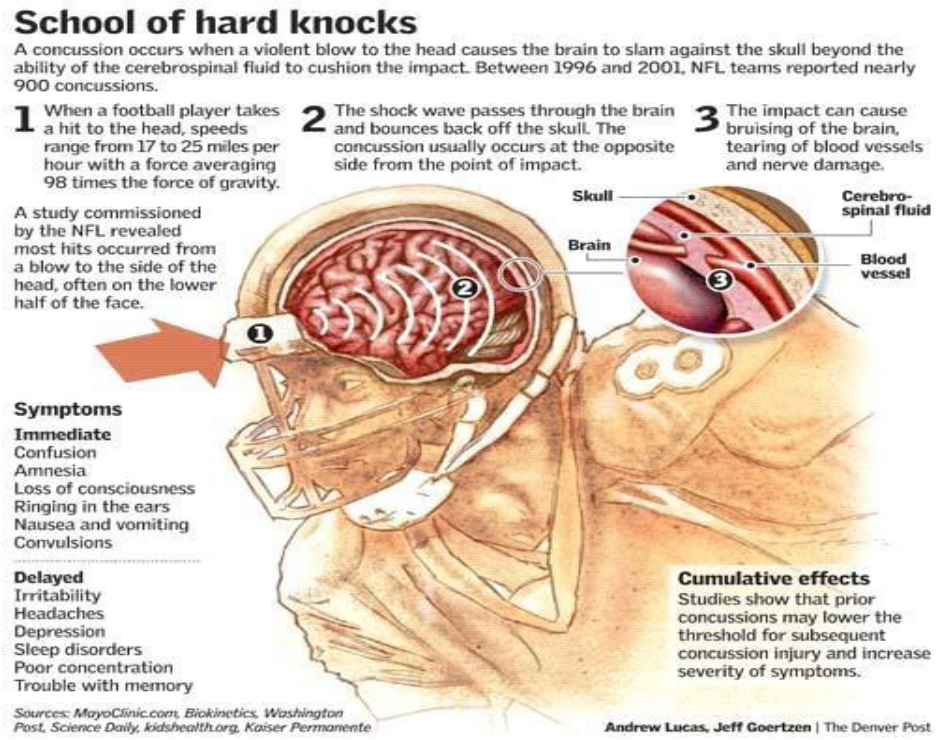
Schatz, Pardini, Lovell, Collins and Podell (2006) postulate that although these grading scales are beneficial they are not empirically based and are mostly based on subjective clinical experience rather than on objective research. The Concussion in Sport Group proposed a further definition of concussion at the first International Conference of Concussion which was held in

Vienna in 2001 (Aubry et al., 2002). Concussion was stated as a complex pathophysiological process that affects the brain which is induced by biochemical forces. The definition included several common features that incorporate clinical, pathological and biochemical injury concepts that are used to explain the nature of a concussive head injury, including : (1) concussion may be caused by a direct blow to the head face, neck, or elsewhere on the body with an impulsive force transmitted to the head; (2) concussion typically results in the rapid onset of short-lived impairment of neurological functions that resolves spontaneously; (3) concussion may result in neuro-pathological changes but acute clinical symptoms largely reflect functional disturbances rather than structural injury; (4) concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course; (5) concussion is typically associated with grossly normal structural neuroimaging studies. At the Second International Conference of Concussion held in Prague in 2004 it was decided to endorse the earlier definition of the Vienna Conference with one important addition, namely that in some cases post-concussive symptoms may be prolonged and persistent (McCrory et al., 2009). At the third International Conference of Concussion held in Zurich it was unanimously agreed that concussion be defined as a complex pathophysiological process affecting the brain induced by traumatic bio-mechanical and biochemical forces.

The term concussion is used descriptively to refer to a type of mild closed head injury resulting from a blunt impact injury, such as those frequently sustained in a contact sport like football. The term concussion enables the use of additional description within the spectrum of MHI, namely sub-concussive head injuries. These are explained in the literature as involving subtle changes in consciousness and are difficult to detect as they usually lasts from seconds to minutes (Ancer, 2000; Lezak et al**.,** 2004). In this study, sub-concussive head injuries refers to the blows to the head which go unnoticed and which frequently occur in a contact sport like football**.** Bio-kineticists and sports medicine physicians, developed a comprehensive definition of concussive injury in sport, as a complex pathophysiological process affecting the brain induced by traumatic biochemical forces. The definition includes the following parts: (i) concussion may be caused by a direct blow to the head, face, neck or elsewhere on the body with an impulsive force transmitted to the head; (ii) concussion typically results in the rapid onset of short lived impairment of neurological function that resolves spontaneously; (iii) concussion may result in

neurological changes but acute clinical symptoms largely reflect a functional disturbance rather than a structural injury; (iv) concussion results in a graded set of clinical and cognitive symptoms which typically follows a sequential course and (v) concussion is typically associated with grossly normal structural neuroimaging studies (Herring et al., 2006). This definition is inclusive and is used by sports scientists thus it is the one adopted for use by the study.

Diagram 4: Concussion injury (Lucas & Coertzen, 2010)



2.4 Severity of brain injury

According to Zillmer et al., (2008) the three main measures of severity of brain injury are the duration of coma, the depth of coma and the duration of Post-Traumatic Amnesia (PTA). The following discussion will concentrate on the disadvantages and advantages of the various ways of assessing the severity of head injury. The duration of loss of consciousness (LOC) or coma is related to the severity of any brain damage. Symonds, (1924) as cited in Mureriwa (1997), was one of the first to suggest that the duration of unconsciousness depends on how many injuries there are rather than the location of the brain lesions (injuries). MHI however, has a brief LOC and/or memory loss lasting for no more than an hour (Davidhizar & Bartlet, 1997; Echemendia

& Julian, 2001; Parker, 2009). An individual with MHI may suffer from cognitive deficits such as memory and reduced attention span and may experience post-concussive symptomology. Repeated concussion and MHI can also cause cumulative brain damage such as second impact syndrome in contact sports (See Chapter 3, paragraph 3.5.2).

2.4.1 Coma classification

The Glasgow Coma Scale (GCS) is a widely used measure of severity of brain injury based on impaired consciousness (Jennett & Teasdale, 1977; Kolb & Wishaw, 2003). To determine a patientôs GCS score the doctor, nurse or paramedic assesses the patientôs eye opening response, verbal output and motor responses. A person, who is fully conscious, shows spontaneous eye-opening, is well oriented and obeys commands. On the other hand, a patient who is in a deep coma will show no eye opening, no verbal response and no motor response. Researchers have proposed different cut-off points on the GCS to classify patients into mild, moderate and severe head injuries. It has proved to be of considerable predictive value in pointing to long-term outcomes in terms of both survival and ultimate levels of cognitive disability (Jennett, 1991). Numerical scores are summated for the best responses obtained under each category at a defined time. In this way useful predictions can often be made within 24 hours of injury and prognosis within the first week (Dacey, Vollmer & Dikmen, 1993; Kolb & Wishaw, 2003). Patients whose GCS is less than 8 on admission, or 6 after injury are considered to have sustained severe head injuries and those with a GCS of 9 to 12 are considered to have sustained a moderate head injury (Lishman, 1988). An initial score of 13 to 15 on the GCS, without subsequent deterioration, places patients in the mild category for head trauma (Arciniegas et al., 2005; Evans, 1992). The benefit of the GCS is that it has low inter-observer variability and high reliability when applied by medical, nursing or paramedic personnel (Lishman, 1988). The validity of the GCS with respect to differentiating levels of severity of head injury is supported by the finding that the patientôs length of hospital stay is inversely proportional to GCS admission (Dacey et al., 1993; Lezak et al., 2004).

In spite of its demonstrated usefulness the GCS has specific intrinsic problems. Some trauma patients are coherent in the first hours after MHI but may subsequently deteriorate. An early GCS score may not be obtained in patients who have been anaesthetised and intubated at the scene of an accident, or if they have surgery before regaining consciousness (Lezak et al., 2004). GCS scores are also frequently influenced by alcohol, drug ingestion and other neurological organ system trauma (Stambrook, Moore, Lubusko, Peters & Blumenschein, 1993). The GCS was also not designed to make a subtle distinctions in patients categorised as having a mild head trauma.

2.4.2 Post-Traumatic Amnesia (PTA)

(Russell, 1932) was the first to conduct a systematic study of significance of PTA in closed head injuries. He described PTA as an early stage of recovery from head injury during which the patient is not sufficiently aware of the environment to be able to commit events to memory (Russell, 1932; Russell & Smith, 1961). Clinical features experienced during PTA may include defective-attention, disorientation, lethargy and agitation as well as inappropriate and disinhibited behaviour and speech (Levin, 1990; McMillan, Jongen, & Greenwood, 1996). For the purpose of classifying the severity of head injury using PTA, Russell (1971) as cited in Gronwall and Wrightson (1980), suggested that concussion with a PTA of less than one hour is mild. He proposed that concussion is moderate if the PTA is 1 to 24 hours in duration and is severe if the PTA exceeds 24 hours. In most studies, the cut-off point for severe head injury is a PTA of 24 hours or more (McClelland, 1988). This in all probability over classifies severe cases, to overcome this, Matheson (1994) suggested the following (See Table 1).

Table 2: Mathesonôs (1994) Post Traumatic Amnesia (PTA) classification

Less than five minutes	Very mild
Five to sixty minutes	Mild
One to twenty four hours	Moderate
One to seven days	Severe
One to four weeks	Very severe

The more recent assessment procedures for PTA are prospective and exclude periods of coma. The first such prospective measure of PTA was the Galveston Orientation and Amnesia Test (Levin, Oôdonnell & Grossman, 1979). In the prospective assessment of PTA, the measurements begin while the patients is in, and emerging from, PTA (Forrester, Encel & Geffen, 1994). This is in contrast to traditional assessments that were carried out after the state of PTA had resolved. Retrospective assessments of PTA depend on the patientôs subjective recollections, which make them unreliable (Mureriwa, 1997). The Westmead Post Traumatic Amnesia test (Ponsford et al., 2000) was designed and found useful for people with a history of psychiatric illness, developmental or intellectual disability, substance abuse and people who experienced more than one head trauma or trauma to the CNS. It can also be used with people who have complex communication needs. Despite any criticism PTA is still considered to be the best yardstick for predicting the severity of catastrophic brain injury or TBI (Wilson, Teasdale, Hadley, Wiedmann, & Lang, 1994). PTA is caused by diencephalic damage which is a focal injury. Diencephalic damage occurs only with moderate to severe head injury. It is for this reason that PTA measures are not suitable measures for MHI (Mureriwa, 1997).

2. 5 Incidence of MHI

Accurate statistics regarding the prevalence of closed head injuries are relatively difficult to obtain. Terms such as mild, minor, moderate, and minimal are applied to head injuries without accurate or universal definitions in hospitals, trauma rooms and medical practices. The causes of MHI are similar to those that are more severe. In the early 1990ôs in the United States of America (USA), it was reported that MVAôs caused 46% of MHI, whereas sports, accidents, falls and assaults caused 28%, 10% and 23% respectively. This was supported by Cassidy et al., (2004) who reported that the majority of causes of MHI globally are MVAôs accidents, falls and assaults while sports injuries comprise the bulk of the remainder. It is also notable that death rates are elevated amongst young adults who suffer severe or moderate to MHI with an incidence of 15% per 1000 per year contrasted with 2% per 1000 per year in other age groups (McMillan, Teasdale, Weir & Stewart, 2011).

There are many reasons why the exact incidence of MHI is difficult to determine, for instance:

- most health surveys concentrate on patients who have been hospitalised. However, many patients who sustain MHI are not hospitalised but are evaluated in hospital emergency rooms or doctorsô offices;
- the International Classification of Diseases (ICD, 2010) and other descriptive tools are often applied ambiguously to injuries (like maxillofacial fractures and scalp lacerations) by including them in the overall statistics for injuries to the head;
- patients who have sustained multiple injuries that include a MHI may be classified according to their most severe or complex injury and the occurrence of MHI injury may be ignored (Dacey et al., 1993)

Wrightson and Gronwall (1998), carried out a study on the incidence of MHI in New Zealand, estimating the incidence to be 1 769 per 100 000 population per year for people aged 15 and beyond. For those under 15 years of age, the incidence was put at 2 929 per 100 000 population per year. These figures were supported by another study completed by Van der Naalt (2001). It has been estimated that 80% to 95% of all head injuries experienced in Europe can be considered mild (Vos et al., 2002). According to Cassidy et al., (2004) in their overview of the incidence of MHI in Europe the incidence amongst hospital treated adults for MHI was between a 100 and 300 per 100 000. However, since the majority of MHI are not documented in emergency or medical departments, these figures probably underestimate the true incidence. They postulate that a more plausible estimate exceeds 600 out of 100 000. In a study conducted in Sweden, MHI ranged from 191 out of 100 000 to 718 out of 100 000. Accident data in Sweden indicates that 50% - 60% of MHI patients admitted to a hospital were injured in a fall and 25% received their injury while operating a motor vehicle. It must be noted that MVAôs in Sweden are relatively low as compared to the rest of Europe (Cassidy et al., 2004).

A study conducted by Gökyar, Yildiz, Bostanci and Ozdemir (2011) in Eastern Europe analysed 851 patients younger than 14 years old with head injuries incurred between January 2003 and June 2008. Five hundred and fifty participants were male and 301 were female. According to the GCS, 74% of patients had an MHI with a score of 13-15 on the scale.

As stated earlier an additional factor confounding estimates of the incidence of MHI injury is lack of a universal definition for this type of head injury. This is illustrated by Ryu, Feinstein, Colantonio, Streiner and Dawson (2009) who conducted a study on the variability in diagnosis of MTBI. They wanted to determine the incidence of MTBI in Canada. Potential MTBI cases were identified through reviewing three months of Emergency Department (ED) and Family Physician (FP) health records. Potential cases were selected from ED records using the International Classification of Disease, 10th revision (2010). Documented diagnoses of MTBI were compared to expert reviewer diagnosis. Incidence of MTBI was determined using the documented diagnosis and data from hospital catchment areas and the population census. The results reported that 876 potential MTBI cases were identified with 25 cases from FP records. Key indicators of MTBI were missing on many records and only 308 of 876 patients reviewed had GCS scores. The expert reviewer disagreed with the documented diagnosis in 380 of the 876 cases. The expert calculated incidence rates of hospital treated MTBI as 426 or 545 out of a 100 000. Family physician cases increased the rate to 653 cases of MTBI out of 100 000. The research concluded that health record documentation of key indicators for MTBI is often lacking and some patients with MTBI appear to be missed or misdiagnosed by primary care physicians. The study yielded a more comprehensive case definition that resulted in estimated incidence rates higher for MTBIôs than previous reports or research.

The Congress Report on MTBI in the USA (Maroon et al, 2000) reported that in 2003 more than 1.5 people experience (TBI) each year. Of the 1.5 who survive a TBI 392 000 are hospitalised, 543 000 are treated in emergency departments and released inside several hours and 221 000 are treated in clinics and physiciansô offices. The remaining 381 000 do not receive medical care. Of those who were hospitalised, 146 000 stayed in hospital for only one night. These data suggests that as many as 75% of people who sustain a brain injury incur a MHI. In 2001, the TBI Surveillance Programme in the state of South Carolina Department of Health, USA (SCDOH, 2001) identified 56 780 cases of TBI from 1996 to 2000. From the data that was collated 86% or

49 099 were MHIôs. Of these 85% were identified through ED surveillance. However, it was postulated that overall national surveillance systems underestimate the occurrence of MTBI because they do not include injured people who received medical care in other facilities for instance, outpatient clinics, family physicians or those who receive no medical care for their injuries (SCDOH, 2001).

Menascu and Tshecmer (2011) in a recent study in the USA state that about 85% of all head injuries requiring medical treatment are mild in nature, about 8% are moderate and the remaining 6% are severe. They also state that most MHI do not come to the attention of health care personnel. The incidence is thus likely to be underestimated rather than overestimated. Additional difficulty in determining an accurate incidence of MHI is confounded by the inclusion of contusions to the face and scalp in these statistics which do not necessarily constitute an MHI.

According to Menascu and Tshemer (2011) the United States National Coma Bank Document reports that about 85% of all head injuries requiring medical treatment are mild in nature, about 8% are moderate and the remaining 6% are severe. The incidence of closed head injuries varies significantly according to different demographic factors including gender, age and socio-economic status. The ratio of boys to girls rises from approximately 1.5 to 1 for preschool children to approximately 2 to 1 for school-age children and adolescents. These changes appear to reflect the sharp increase in head injuries among males and a gradual decrease among females. The incidence of closed head trauma also varies with age. Data demonstrates that the incidence is relatively stable from birth to age 5 years, with injuries occurring in about 160 per 100 000 children in this age group. After age 5 the overall incidence gradually increases until early adolescence and then shows rapid growth, reaching a maximal peak of 290 per 100 000 by the age of 18 years. It is also reported that incidence of MHI varies with familial socio-economic status. It seems that more middle class families are likely to report MHI than the working classes and/or that they have more access to medical facilities.

Research carried out by Nell and Brown (1991) in South Africa reported an average of 316 per 100 000 incidents of brain injuries per year. In this study Black and Coloured men were more at risk of brain injury than White males. Black males in the 25 to 44 year age groups were most at risk of brain injury. More recent research reports that 89 000 cases of brain injuries are reported

annually in South Africa (Durban Hospital, Department of Neurosurgery, 2010**)**. The report also states that in South Africa the three most common causes of head injury are MVAôs, motorbike, or vehicle-pedestrian accidents (more than 50%). Approximately 25% of head injuries are due to falls and other types of violence. These contribute to nearly 20% of the total head injuries reported per year.

2.6 The mechanisms of MHI

The mechanism of MHI is divided into two categories, namely primary and secondary brain injury. Primary injury occurs immediately on impact whilst secondary injury occurs after the impact. Secondary brain injury can result from complications of primary brain injury. The duration during which the secondary damage can occur differs from seconds to days (Edlow, 2006).

2.6.1 Primary brain injury

A number of damaging mechanical forces have been identified as contact forces and inertial forces in primary brain injury. The inertial forces typically involve translational acceleration in which the head moves in a straight line with the brainôs centre of gravity, or rotational acceleration in which the brain rotates around the centre of gravity. The contact force (force of impact) is the main cause of brain damage in still injuries, in which a motionless victim receives a blow to the head. Movement of the head and neck on impact results in angular acceleration, a combination of translational and rotational acceleration (Lezak et al., 2004).

According to Lezak et al., (2004) and Werner and Engelhard (2007) cerebral contusions consist of focal damage to the brain tissue, which may result in laceration (tissue covering the brain is torn) as a result of head trauma. They also state that the coup is an injury that results from a direct blow at impact and is expected to appear below the site of impact. Countercoup is an injury in which the brain sustains contusion(s) in an area opposite the blow. It most frequently occurs in the frontal and temporal lobes regions of the brain and in most cases the injuries occur because of occipital injuries (Lezak et al., 2004).

According to Mureriwa (1997) ancient Greek physicians were aware of the coup-countercoup phenomenon. Holbourn (1943) was the first to apply rotational forces to gelatine models of the brain and observed that contusions were produced and were most severe at the front of the frontal and temporal lobes. He explained these early results by stating that when the head receives a blow the behaviour of the skull during, and immediately after the blow, is determined by the physical properties of the skull and the brain and Newtonôs laws of motion. Thus, following the blow, the brain rotates and forcibly comes into contact with the bony prominences inside the skull. He suggested that rotation and skull deformation were responsible for some intracranial haemorrhages and probably for some concussions. This was supported by later studies (Lezak et al., 2004). Coup and countercoup lesions account for specific and behavioural localised changes that accompany closed head injuries.

Six main theories are postulated to explain countercoup injuries to the brain. These are the Vibration or Echo theory, Transmitted Force Theory, Brain Displacement Theory, Pressure Gradient Theory and Rotational Theory. Vibration or Echo theory states that the traumatic impact sets up vibrations that are reflected in damage to the opposite pole of the brain. The theory posits that vibrations in the skull occur like an echo across space. The proponents of Transmitted Force Theory suggest that traumatic impact results in a transmission of applied force through the tissue of the brain. This force causes the contralateral structures of the brain to be thrust against the wall of the skull. Brain Displacement Theory states that countercoup injuries result from the avulsion of the cerebral cortex from the overlying meninges. Pressure Gradient Theory is based on the observation that there is a sudden fall in intracranial pressure opposite to the point of impact. This fall in pressure causes blood vessels at that point to rupture. Proponents of Rotation Theory suggest that, after a blow to the head, the brain is set in centrifugal motion in the direction of the line of the original force. The brain is then pushed against the irregularities (bony protuberances) on the interior of the skull (Edlow, 2006; Lezak et al., 2004).

2.6.2 Secondary brain injury

Secondary brain injury occurs at different lengths of time after head trauma. It is important to recognise MBTI including CMHI is a dynamic process because the pathologic picture continues to evolve for hours and days after impact (Salazaar, 1992). Much of the eventual damage from

head trauma appears to occur in a delayed or secondary fashion (Smith, Casey & McIntosh, 1995). The central mechanisms of brain cell injury are hypoxia or low oxygen supply to cells and an insufficient supply of blood to the brain or ischemia (Bennett, OÕBrien, Phillips & Farrell, 1995). Other secondary effects are haematoma, oedema (swelling) in the white matter of the brain next to focal mass lesions, intracranial haemorrhage, diffuse brain swelling, ischaemic brain damage, raised intracranial pressure, brain shift and herniation (Kolb & Whishaw, 2003; Werner & Engelhard, 2007).

2.6.2.1 Haemorrhage and Haematoma

Haemorrhage means intracranial bleeding and this occurs as a result of the rupture of blood vessels in different sites in the cranium. The main three areas affected during trauma to the brain are the space between the dura and the skull, the space between the dura and the arachnoid matter and the substance of the brain itself (Banich, 2004; Lezak et al., 2004). Haematoma are blood clots that result from the haemorrhage. Like haemorrhage, haematoma is also divided into extradural, subdural and intracerebral. In extradural haemorrhage, the meningeal arteries and veins, more especially the anterior division of the meningeal are injured. Subdural haemorrhage results from the tearing of the superior cerebral veins in which they enter the superior sagittal sinus (Lezak et al., 2004). This happens when a blow to the head causes excessive anteroposterior displacement of the brain within the skull. Bleeding takes place under high pressure but the pressure is less than in the case of extradural haemorrhage. Subdural haemorrhage, unlike extradural haemorrhage spreads diffusely over the affected hemisphere. Intracerebral haemorrhage occurs as a result of a rupture of the thin wall of the lenticulostriate artery, a branch of the middle cerebral artery. This type of haemorrhage occurs into the substance of the brain and is found in more severe injuries (Banich, 2004).

2.6.2.2 Diffuse brain swelling

Brain swelling refers to the increase in the total volume of the brain, such swelling can occur in some cases as a result of bleeding and/or oedema. Diffuse Brain Swelling can also occur in areas adjacent to haemorrhages, haematomas and contusions (Werner & Engelhard, 2007). They

suggest that cerebral swelling involves a disturbance of the vasomotor tone and that there is evidence of reduced cerebral flow in the brain areas surrounding the swelling. Brain swelling also leads to raised intracranial pressure, which results in brain shift. A suggestion was also made that axonal injury causes localised transport failures in the axons, which leads to swelling. Swelling in the brain is serious as there is little room for expansion or swelling within the parameters of the skull.

2.6.2.3 Abnormalities in brain structure

Radiological techniques are relied upon for investigations of abnormal structures after brain injury. The most commonly used are the computerised tomography (CT) scans and magnetic resonance imaging (MRI). These techniques produce three dimensional images of the brain. The CT and MRI (See Appendix E) are both equally sensitive in detecting superficial soft tissue injury (Lezak et al., 2004). The differences between the CT and MRI as noted by Christo (2008) are the following: The CT scan has advantages over the MRI in that it requires less time to acquire images and is superior to the MRI for visualising bone structures, it is less sensitive to motion effects (created by agitated patients), is less expensive and is generally appropriate in making early diagnostic and treatment decisions. Focal and multifocal contusions as revealed by CT scan are associated with an increase in the frequency of memory problems, speech problems, weakness of the limbs and consciousness. The CT scan is thus more useful than an MRI in identifying acute brain damage. Although the CT scan has the aforementioned advantages it also has disadvantages for instance, CT scans fail to identify many cases of brain injury. Christo (2008) and Lezak et al., (2004) postulate that a CT scan fail to identify lesions because it is only sensitive to focal injuries. CT scans fail to identify a noteworthy number of closed head injuries because most of these injuries lead to diffuse brain damage. CT scans do not identify diffuse injuries of the brain as this type of injury occurs mostly at the cellular level, leaving gross anatomy unchanged, thus on a CT scan the brain looks normal. The MRI has been found to be more sensitive than CT scans in identifying subtle neurological damage like that found in MHIôs. It has also been shown to be more superior to the CT scan in identifying cerebral contusions, shearing injuries and hematomas (Lezak et al., 2004; Paterakis, Karantanas & Komnos, 2000).

Electroencephalography (EEG) is another radiological procedure which was designed to assess brain function rather than brain structure. EEG (See Appendix E) involves measurements of voltage changes from the brain which often indicates that there is a slowing of brain function after an MHI.

2.6.2.4 Neuropsychological abnormalities

Neuropsychological assessments aim to identify abnormalities in a wide variety of psychological processes associated with brain injury. All psychological processes are potential targets for these assessments. The psychological processes include consciousness, sensation, perception, attention and memory, intellectual function, emotional and motivational behaviour and personality change. A number of tests are currently in use to assess neuropsychological deficits after different types of brain injury (Lezak et al., 2004). According to Shuttleworth-Jordan (1999) there is an association between the presence of post-concussion symptoms and deficits on pen and paper neuropsychological tests. The tests included finger tapping, the continuous performance test, pattern comparison test and symbol digit substitution (See Appendix F). Memory problems were most consistently associated with neuropsychological deficits identified by such tests. The assessment of neuropsychological deficits can also be carried out using the information processing paradigm, computerised testing and various neuropsychological tests.

2.7 Pathophysiology of MHI

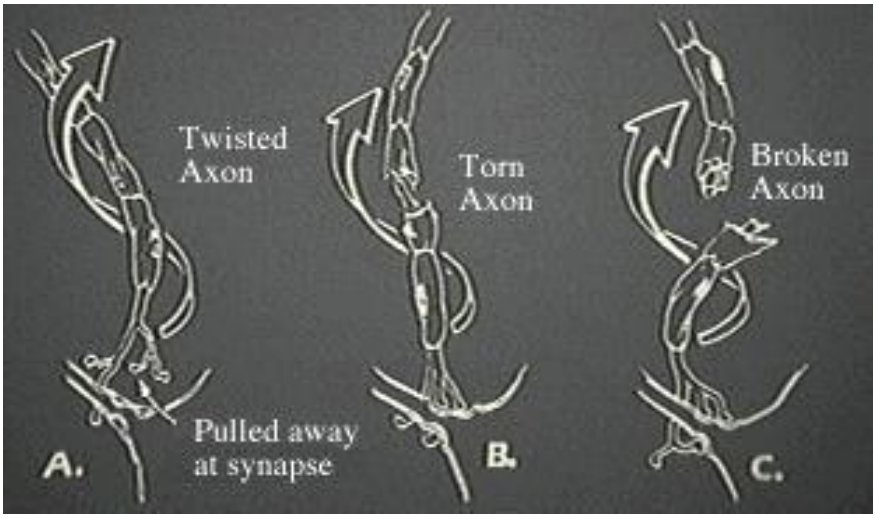
2.7.1 Diffuse Axonal Injury (DAI)

According to Lezak et al., (2004) acceleration-deceleration trauma, particularly when the brain twists or rotates within the skull (rotational acceleration), may cause axonal strain and tensile stress which may be focally diffuse (See Diagram 5). This process was referred to in the 1940ôs by Holbourn as shear-strain or more recently Diffuse Axonal Injury (DAI). This is one of the most common and devastating types of injury in brain trauma. It is reported as being one of the major causes of unconsciousness and persistent vegetative states after a serious head trauma (Edlow, 2006).

Strich (1956; 1970) was the first to comprehensively describe DAI after the post-mortems of people who had died after severe head injury. DAI is the most consistent pathology reported after moderate to severe head injury (Mureriwa, 1997). However, MTBI also involves distortion, stresses and strains of the vascular brain system and neural brain systems. These stresses and strains are typically caused by acceleration injuries resulting from movement of the head and skull which result in stretching and shearing of nerve fibres causing axonal damage (Lezak et al., 2004). It has been postulated that axonal fibres are stretched in mild brain injury occurrences, resulting in fibres becoming swollen, beaded and varicose. This effect renders the neuron dysfunctional, even though the neuron is still alive (Echemendia & Julian, 2001; Edlow, 2006; Lezak et al., 2004). These strains are often higher at the surface, than at the depth of the brain, and decrease drastically toward the centre of the brain. Autopsy investigations have indicated that the mass of the cerebral hemisphere above the cerebella structures and the stalk of the midbrain are especially vulnerable to these rotational shear strains. This type of diffuse, non-localised injury is typical of MTBI. Alexander (1995) and Holbourn (1943) noted that diffuse axonal damage is the main causal mechanism for injury and ensuring behavioural dysfunction in MTBI. Gennarelli, Thibault and Graham, (1998) describes three types of DAI grades. Grade one is widespread axonal damage in the corpus callosum, the white matter of the cerebral hemisphere and the brain stem. In Grade two DAI, the focal abnormalities in the corpus callosum are often associated with small tissue tear haemorrhages and exist in addition to the injuries in Grade 1. In Grade 3 DAI, the injuries noted in Grade 2 are present with additional axonal abnormalities, commonly in the rostral brain stem, resulting from tissue tear haemorrhages. Grade 1 DAI occurs most often with MBTI and does not result in loss of consciousness but, as noted, there is axonal damage.

DAI alters numerous executive functions including the speed of information processing, working memory, attention span and interference control (McAllister, Sparling, Flashman & Saykin, 2001; Niogi et al., 2008; Turner & Levine, 2008; Wallesch et al., 2001). It is also likely that DAI contributes to persistent post-concussive symptomology and attentional deficits following MTBI (Niogi et al., 2008). They also note that frontal-temporal areas and anterior and inferior regions of the brain are highly susceptible to focal damage which contributes to attentional, executive and emotional control difficulties.

Diagram 5: Diffuse Axonal Injury ï DAI (Headway, 2012)



2.7.2 Post-Concussive Syndrome (PCS)

After a relatively minor impact to the head a common pattern of complaints termed post-concussive syndrome is found (Parker, 2009). A cluster of self-reported or subjective symptoms may persist long after the injury, even after neuropsychological testing has indicated that there has been resolution. These clusters are referred to in the literature as post-concussion syndrome (Sundström, 2006). This cluster of symptoms may be both acute and long term and they fall into three broad categories namely cognitive, physical and psychological symptoms.

MHI is described as a multi-dimensional and multi-factorial disorder and the sequelae following damage caused by such an injury will vary from person to person depending on educational level, age, premorbid neuropsychological integrity, injury characteristics and psychological reaction to the injury (Arciniegas et al., 2005). Generally, individuals recover from these symptoms without any residue within one to three months (Alexander, 1995; Ponsford, et al., 2000). Several studies however, report persistent symptomology months (sometimes years) after the initial injury (Alexander, 1995; Lezak et al., 2004; Ryan & Warden, 2003). The estimated

prevalence of symptoms lasting months or longer is unclear. Binder et al. (1997) report prevalence of 7% to 8%, Ponsford et al., (2000) at 10% to 25% and Bohen and Jolles (1992) at 80%. When symptoms persist a Post-Concussive Syndrome exists (PCS). According to King (1997) such symptomatic reactions to MHI are mediated by a number of issues from concerns about compensation claims to emotional reactions. Broadly, many persistent post-concussive symptoms are an interaction between organic and psychological factors. They usually start on an organic basis and persist and are experienced by the individual on a psychological level.

During the 1970ôs it was argued within medical and neurological circles whether or not the different post-concussive symptoms could constitute a syndrome. These symptoms did not appear to form clear-cut clusters and therefore, according to some pundits, could not really constitute a syndrome (Rutherford, Merrett & Mcdonali, 1977). However, research particularly factor analytic studies have demonstrated symptom clusters within post-concussive syndrome. Two clusters of complaints were identified. The first group of complaints was categorised as impairments and consisted of forgetfulness, slowness, poor concentration and inability to divide attention between two concurrent activities. These complaints were found to be related to the severity of brain injury. The second cluster of complaints consisted of all other symptoms for instance, headaches and anxiety which were labelled intolerances as they were not related to the severity of brain injury (Van Zomeren, Brouwer & Deelman, 1984).

Jacobson (1995) classified post-concussive indicators into somatic and psychological symptoms. The somatic symptoms included headaches, dizziness and fatigue. He further suggested that psychological symptoms are sub-classified into cognitive (poor memory and concentration) and psychological or affective symptoms (irritability, emotional lability, depression and anxiety). Mureriwa (1997) noted that there is a strong correlation between headaches and dizziness and between poor concentration and memory. He suggests that this demonstrates that there are strong correlations between such post-concussive symptomology. Bohnen and Jolles, (1992a) identified two sub-groups of patients with MHI. They categorised these into two sub-groups after administering a 26 item questionnaire which included post-concussive, cognitive and emotional symptoms. Principal component analysis with various rotations was carried out on the responses

of 71 patients 10 days after they received a head trauma. The results of the analysis indicated that the symptoms of headache, dizziness and intolerance to environmental stimuli resulted in decreased cognitive and work performance. This group of symptoms is referred to as post-concussive or cognitive complaints. The second subgroup was categorised as emotional. It consists of emotional lability and depression as well as specific symptoms like heart palpitations and clammy hands. Boulind (2005) conducted a meta-analysis of research that focused exclusively on the acute phase of PCS in the sports context. He noted a number of physical and neurological symptoms experienced by athletes. These included headaches and dizziness, difficulty concentrating and memory problems plus poor problem solving ability and feelings of mental fogginess.

The American Psychological Association (APA, 1994) encouraged more research and communication among researchers with regards to PCS which were included in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (APA, 1994). The criteria which are subject to change as research is on-going (See criteria list 2, p. 40) include a history of head trauma, cognitive problems plus symptomology that lasts 3 months or longer and which doesn't improve. These symptoms must impair day-to-functioning (American Psychological Associasion, 2000). Arciniegas et al., (2005) describe PCS or Post-Concussion Disorder in the nosology of the DSM-IV as generally signifying the development of a constellation of physical, cognitive, and emotional or behavioural post-concussive symptoms. Since there are differences in the symptomology across patients it has been suggested that PCS is a group of illnesses rather than one illness (Arciniegas et al., 2005). Diagnosing the syndrome is quite difficult because virtually all symptoms listed in PCS criteria have a high basal rate in the general population (Dikmen & Levin, 1993; Satz et al., 1999). These symptoms are also exhibited in other conditions, not involving injury to the head, including: a) Post-Traumatic Stress Disorder (Hickling, Gillen, Blanchard, Buckley & Taylor, 1998); b) anxiety and/or depressive disorders (King, 1997; McCauley, Boake, Levin, Contant & Song, 2001) and c) chronic pain (Gasquoine, 1997; Smith-Seemiller, Fow, Kant & Franzen, 2003). It must be noted that the problems experienced by individuals with MHI are more precisely understood as post-concussive symptoms rather than post-concussive syndrome per se (Niogi et al., 2008).

Females may be more at risk for developing PCS, even though men are more likely to sustain head injury (Gasquoine, 1997; McCauley et al., 2001). A small but significant effect size was noted in a meta-analysis of eight studies on mild to severe head injuries, finding an overall outcome for women are worse than men (Farace & Alves, 2000). One explanation of this might be related to differences in the mechanisms of injury. In particular, a greater percentage of females sustain head injuries in MVAôs 57% versus 22%, whereas a greater percentage of males sustain head injuries whilst participating in sports, 33% versus 9%. Research also indicates that sports injuries exhibit less persistent sequelae than MVAôs (Bazarian et al., 1999; Bring, Björnstig, & Westman, 1996). This difference might also be due to under-reporting of sports related MHI because participants do not want to be removed from play (Carroll et al., 2004). The older the individual is the more susceptible they are to persistent sequelae. The risk of persistent sequelae post-head injury is twice as high at age 40 than at age 30 (Arciniegas et al., 2005; Fenton, McClelland, Montgomery, MacFlynn & Rutherford, 1993; Kashluba et al., 2004). Injuries tend to differ with age for instance, younger adults are more likely to be injured in MVAôs whereas older adults are more likely to be injured in falls (Thurman, Alverson, Dunn, Guerrero & Sniezek, 1999). Pre-injury factors may also play a role in any age-related differences because the elderly injured in falls are more likely to have other ailments impacting on PCS outcomes (Lange, Iverson & Rose, 2010). However, some studies did not identify a significant relationship between age and outcomes after head injuries (Breed, Flanagan & Watson, 2004). There are no clear explanations for the inconsistent relationships between age and PCS. Some studies suggest that differences are related to the level of injury severity which impacts on positive PCS outcomes. The relationship between pre-injury personality characteristics and MHI has been suggested as an explanation for persistent symptomology. Parker (2009) suggests that personality change can be a consequence of MHI. Other studies believe poor coping skills may explain prolonged PCS (Bohnen & Jolles, 1992; Ponsford, et al., 2000). Persistent symptoms after MHI can be associated with malingering especially if litigation is involved (Mickeviļiene et al., 2002; Kashluba et al., 2004). This is underpinned by a study by Binder et al., (1997) who performed a meta-analysis to assess the relationship between potential financial gain and clinical outcomes after head injury. They concluded that financial incentives had a positive effect on the development of persistent PCS, especially in patients with MHI.

2.7.3 Acute symptoms and symptom recovery from post-concussive syndrome (PCS)

McCrea (2007), reports that the diagnosis of concussion or MTBI is to a large extend based on the subjective symptoms reported by the patient. It is the same for the recovery after MTBI, as most often this is determined by the patientôs self-reported resolution of concussion symptoms. The diagnosis of post-concussion syndrome (PCS) is reserved for those patients with persistent complaints after MTBI. The importance of symptoms diagnosis and prognosis highlights the need to establish empirically defined parameters for the expected course of recovery. Some studies have generated evidence that is based on the natural history of symptom recovery after MTBI which guides the clinician in interpreting a patientôs persistent complaint at the individual level.

The World Health Organization (WHO), in 2004 collaborated on a study of MTBI which published a detailed review of literature on the prognosis after a MTBI (Carroll et al., 2004). One hundred and twenty studies of best-evidence on prognosis after MTBI met their criteria for inclusion in the review. There was consistent and methodologically sound evidence that childrenôs prognosis after MTBI is good, with quick the resolution of symptoms and with little evidence of residual cognitive, behavioural or academic deficits. They found that adultsô cognitive deficits and symptoms were common in the acute stage and most of the studies reported that recovery for most adults resolves within 3 to 12 months. Where compensation and litigation were reported as factors in sustained symptomology however, there was very little consistent evidence for other predictors of long term effects. The report concluded that symptoms after MTBI are typically temporary in nature for both children and adults, with quick or gradual resolution within days to weeks post injury in the majority of patients. MTBI symptoms are highly non-specific and often are the same to those reported after other types of injury for example, orthopaedic injury. The WHO (2004) task force summarised the results of several studies on self-reported symptoms on MTBI and noted that headaches, blurred vision, dizziness, subjective memory problems and sleep problems were the most commonly experienced symptoms (Carroll et al., 2004).

2.7.4 Post-Concussive Syndrome (PCS) Diagnostic Criteria

The two most commonly cited systems for defining and diagnosing PCS come from the 10th edition of the International Classification of Disease (ICD-10, 2010) and the Diagnostic and Statistical Manual of Mental Disorders - DSM-IV (APA, 1994**)** as both sets of criteria cite the occurrence of head injury as the principal prerequisite for the eventual diagnosis of PCS. ICD-10 (2010) diagnostic criteria for PCS represents a revised criteria that the syndrome occurs after head trauma and is characterised by symptoms in three or more categories that are present not later than four weeks post injury (McCrea, 2007).

The DSM-IV (APA, 1994) criteria for post-concussional disorder require a history of head trauma that has caused a significant cerebral concussion with LOC, Post- Traumatic amnesia or seizures (See Criteria list 1). Based on the latest findings on the acute injury characteristics of MTBI, the reference to the loss of consciousness (LOC) requirement would exclude about 90% of patients from the eventual diagnosis of PCS because there was no LOC associated with the injury. The DSM-IV (See criteria list 2) criterion in contrast to the ICD-10 system requires neuropsychological evidence of difficulties with attention and memory (McCrea, 2007).

Criteria list 1: ICD ï 10 Post-Concussive criteria (ICD-10, 2010)

A. History of head trauma with loss of consciousness precedes symptoms onset by maximum of four weeks.

B. Symptoms in three or more of the following symptom categories.

- Headache, dizziness, malaise, fatigue, noise tolerance.
- Irritability, depression, anxiety, emotional lability.
- Subjective concentration, memory, or intellectual difficulties without neuropsychological evidence of marked improvement impairment.
- Insomnia.
- Reduced alcohol tolerance.
- Preoccupation with above symptoms and fear of brain damage with hypochondriacal concerns and adoption of sick role.

The DSM-IV (APA, 1994) criteria for PCS requires three or more symptoms that have at least three month duration and an onset shortly after head trauma or should represent a substantial worsening of previous symptoms. The DSM-IV criteria also need that this disturbance should cause a significant impairment in social or occupational functioning and represent a significant decline from the patientôs previous level of functioning. The criteria of ICD-10 and DSM-IV are subjective in nature, even to the extent that there are self-reported cognitive problems but no evidence of impairment on objective neuropsychological testing when it comes to the cognitive symptom category. The general features of these criteria suggest that there is the potential for a psychological or emotional basis to the symptoms of PCS as they are subjective in nature (McCrea, 2007).

Criteria list 2: DSM-1V - Post-Concussive criteria (APA, 1994)

A. History of head trauma that has caused significant cerebral concussion. 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 recall of information)

C. Three (or more) of the following occur shortly after trauma and last at least three months:

    1. Becoming fatigued easily.
    2. Disordered sleep
    3. Headache
    4. Vertigo or dizziness
    5. Irritability or aggression on little or no provocation
    6. Anxiety, depression, or affective instability
    7. Changes in Personality (e.g. social or sexual inappropriateness)
    8. Apathy or lack of spontaneity

C. The symptoms in criteria B and C have their onset following head trauma or else

represent a substantial worsening of pre-existing symptoms.

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

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

2.7.5 Reliability and validity of post-concussion criteria

A number of studies have evaluated the reliability of clinical utility of the DSM-IV (APA, 1994) and ICD-10 (2010) diagnostic criteria for PCS Boake et al. (2004) compared diagnoses of PCS between the classification systems. They studied 178 adults with mild to moderate brain injury based on a structured interview at three months post-injury. Their results showed that, despite concordance of the two sets of symptom criteria, agreement between overall DSM-IV (APA, 1994) and ICD-10 (2010) diagnoses was slight because few patients met the DSM-IV (APA, 1994) cognitive deficit and clinically significant criteria. This lack of concordance has been supported by other studies. This type of incongruity between the two most widely used classification criteria adds to the probable under-reporting of PCS and/or confusion in reporting symptoms which lead to difficulties for researchers in reporting its incidence and prevalence.

2.7.6 Treatment for headaches and dizziness related to Post-Concussion Syndrome (PCS)

According to Doble, Feinberg, Rosner and Rosner, (2010) after a MHI many people experience on-going symptoms of PCS. Some of the most common and unbearable effects of this condition are headaches, dizziness, anxiety and neck pain, which could have a significant effect on the quality of an individualôs life. Headache is the most common MHI symptom, occurring in 70 to 92% of individuals sustaining MHI (Lovell et. al., 2007; McCrory, Ariens & Berkovic, 2000; Packard, 2008). Headache is also the most persistently reported MHI symptom (Rimel, Giordani, Barth, Boll & Jane, 1981). Arciniegas et al. (2005), assert that dizziness is an extremely common

somatic complaint post a MHI and may confound neuropsychological assessment. Doble et al., (2010) report that people with these symptoms can be treated with different types of medication and therapy however, on the whole these treatments are not helpful and can take months, sometimes years to complete. A study that they conducted in the USA investigated a new treatment for headaches and dizziness resulting from PCS. The 43 participants in the study, who had on-going symptoms from a concussion injury, wore glasses with specialised lenses containing prisms. It was found that in most of the participantsô post-concussive symptomology is reduced. These results suggest that one of the causes of post-concussion symptoms, such as headaches, is strain to the eye muscle and not actually the direct brain injury. The injury disturbs or damages the nerves of the brain, causing the eyes to align differently, therefore stopping the eyes from working together in unison and thus causing specific PCS (headaches, double vision and dizziness). The special lenses in the glasses force the eyes back into proper alignment preventing muscle strain. This is a cutting-edge discovery which has positive implications for the treatment of post-concussive symptoms in some individuals.

2.8 Neuropsychological sequelae of MHI

Individuals who sustain MTBI often report a group of adverse events, usually referred to as post-concussive symptoms. As previously stated post-concussive symptoms are commonly divided into three categories physical, emotional and cognitive. The latter include problems with memory, concentration, initiation, planning, and problem solving as well as difficulties with attention and slowed information processing speed (Ruff, 2005). A total of 10-20% of MTBI patients experience post-concussive problems or symptoms that persist beyond the recognised recovery period of approximately 6-12 months (Alexander, 1995; Ruff, 2005). Physical symptoms include headache, fatigue, dizziness, blurred vision, light and sound sensitivity as well as sleep disturbance (Pare et al., 2009; Ziino & Ponsford, 2006). Fatigue is one of the most reported symptoms after a MHI. Severe fatigue may affect cognition interfering with return to work, exercise and sports programmes and it may also limit social interactions (Borgaro, Baker, Wethe, Prigatano & Kwasnica, 2005; Sundström, 2006). Immediately after an MHI nearly 50% of individuals report that they become fatigued easily, this impacts on their quality of life (Carroll et al., 2004). Fatigue may be the primary effect of CNS dysfunction, or a secondary

effect of sleep disturbance or depression post MHI, and may also confound neuropsychological assessments (Arciniegas et. al., 2005). Psychological and emotional symptoms such as depression are also commonly reported after a MHI (Busch & Alpern, 1998; Holsinger et al., 2002). Other symptoms frequently reported after all types of head injury are anxiety and irritability (Alexander, 1995; Bernstein, 1999; Pare, et al., 2009). As these symptoms often co-exist they can be difficult to assess and separate (Van der Linden et al., 1999).

Interestingly, these symptoms are similar to patients who experience TBI. For instance, an investigation in the USA by Englander, Bushnik, Oggins and Katznelson (2010), attempted to define any association between fatigue and abnormalities in sleep, mood, cognitive, physical and hormonal functioning after TBI. The study was conducted in a rehabilitation centre hospital and there were 119 participants who had experienced a head injury at least one year before the investigation. The findings of the study noted that 53% of the sample reported fatigue on the multidimensional Assessment of Fatigue (MAF), while one third reported it on the Fatigue Severity Scale (FSS). Sixty five percent (65%) were found to have moderate to severe growth hormone deficiency and 15% of the men had a testosterone deficiency. The study concluded that fatigue after brain injury is linked to gender, depression, pain, memory problems, hormonal deficiencies and motor dysfunction. It was suggested that investigation of post TBI fatigue should include the screening for depression, pain and sleep disturbances.

Kerr (2010) conducted a pilot study into the cognitive and neuropsychological sequelae of MHI in children, aged between 6 and 12 years old, with a GCS of 15. They were assessed using the CANTAB or Cambridge Neuropsychological Test Automated Battery (See appendix F) to examine their neuropsychological performance after sustaining MHI. The outcome of the study was that children with MHI had a significantly poorer performance with regards to problem-solving on the CANTAB. It was postulated that this played a part in their impulsive tendencies and poor planning ability. The results suggested that children with MHI may be at risk of developing problems with high-level cognitive functioning post injury.

2.9 Neuropsychological recovery following MHI

Whilst the more acute sequelae of MHI are widely accepted, researchers report conflicting results with regard to the course of recovery post-injury. Some studies suggest that the resolution of neuropsychological deficits occurs four or five weeks post-injury although a disruption of psychosocial functioning may still remain (Bohnen, Jolles & Twijnstra, 1992; Lezak et al., 2004; Pare et al., 2009).

According to Lezak et al., (2004) mild cognitive deficits are primarily associated with slowed processing of information and they persist in many patients who experience MHI. For example, in a study they conducted 1 of 57 mildly injured patients in hospital trauma rooms had post-concussional complaints immediately after the injury. One month after injury, most patients showed evidence of attentional deficits and reduced visuo-motor speed. These problems were associated with complains of headache, fatigue, and dizziness which diminished significantly in two months. However, at three months, almost all of these patients still complained of headaches, fatigue and dizziness. These were reported by 22% of the patients in the study. These continuing symptoms are often subtle and may become evident only with appropriate testing and may not become evident at all if an appropriate examination, sensitive to these problems, is not given. However, an early study by (Gentilini et al., 1985) reported that no conclusive evidence was found that MHI causes cognitive impairments one month after the trauma.

De Boussard et al., (2005) also reported that MTBI is associated with signs of cognitive impairment such as problems of recall, speed of information processing and attention problems that, in most cases, resolve within 1 to 3 months after the injury. A later study by Mfiller et al., (2009) investigated the relationship between predictors and outcomes. They identified predictors influencing the time trend in recovery after this type of brain injury. Fifty nine patients with MHI underwent a comprehensive assessment with neurological and neuro-radiological examinations. The assessments were performed before and 6 months after discharge from hospital on patients with brain injury with a GCS score of less than 15. The neuro-radiological procedures predicted impaired cognitive performance both at baseline and 6 months post-injury. However, Riggio & Wong (2009) refute the above period of recovery and state that cognitive deficits in MTBI generally resolve within days of the acute injury and rarely last longer than 3 months post injury.

According to Lezak et al., (2004) there is a complex inter-relationship between head injury, individual differences and interpersonal adjustment. These variables complicate the debate on neuropsychological recovery following MHI. There are also concerns about different research methodology which contribute to conflicting research findings for instance using poorly controlled studies and a failure to account for pre-morbid factors (Partington-Nel, 2001).

2.10 Cognitive Deficits

As the objective of this study is to determine if there is a correlation between symptoms of CMHI and cognitive impairment amongst football players and a non-contact sport control group, cognitive functions related to those being tested in the study are discussed briefly. Evidence of highly specific areas of deficit following MHI emerging from research includes impairments of attention, memory and information processing. The cognitive tasks that are most likely to show deficits are those that require fast processing, working memory and attention and executive functioning (Alexander, 1995; Frencham, Fox, & Maybery, 2005). Attention and components and models of attention are briefly discussed below as they are relevant to the present study in terms of reaction time.

2.10.1 Attention

Lezak et al., (2004) and Zillmer et al., (2008) assert that people are confronted by an overflow of information and that the nervous system cannot treat all information equally. The brain must target specific material to process and tune out irrelevant information. For example, when talking to someone an individual hears competing sounds or can be preoccupied by his or her inner thoughts. When the individual focuses attention he or she orients a small sample of the incoming information and ignores most of the other input. In this way, attention operates as a gateway for information processing. Attention allows orientation in selecting and maintaining focus on specific information and makes that information available for cortical processing.

According to Zillmer et al., (2008) the history of the neuropsychology of attention has been a confusing subject because of the many definitions of attention. The term attention can refer to a general level of alertness or vigilance, a general state of arousal, orientation versus habituation to stimuli, the ability to focus, divide or sustain mental effort, the ability to target the processing of specific information within a precise sensory arena (such as visual attention or auditory attention) or it can be defined as a measure of capacity.

Definitions of attention, that are in some cases are still used today, were noted formally in medical literature in the twentieth century. For instance, (James, Burkhardt & Skrupskelis, 1981) stated that attention necessitates an individual to have the ability to be selective in their focus. This explanation is supported by later generations of authors (Mureriwa, 1997; Niemann, Ruff & Kramer, 1996; Whyte, Hart, Ellis & Chervoneva, 2008). Focus of attention requires that the individual should be able to sustain several trains of thought simultaneously. They should then have the ability to focus and concentrate on the most important or pressing train of thought so that they can deal with a situation or problem effectively (Whyte et al., 2008). At present, most researchers view attentional processing as a multifaceted multiple behavioural state and cortical process that a range of subsets of cerebral structures control, and not as a unified system as was previously implied (Zillmer et al., 2008). Attentional issues generally concern the higher levels of attentional processing which are co-ordinated by the cerebrum, including focused attention, the ability to alternate and divide attentional processes and the ability to sustain attention (Lezak et al, 2004; Zillmer et al., 2008). Attention can further be characterised by task or information processing demands. Tasks that are routinely processed or over learned can be performed automatically, with negligible conscious thought these place minimal demands on attentional resources. This means that as the demands of this type of information processing is low, other tasks can be performed concurrently (Zillmer et al. 2008). It must be noted that the majority of studies target traumatic or catastrophic brain injury and results in more severe attentional deficits. However, there is more literature that suggests attentional deficits in patients suffering from MHI which can cause difficulties in day-to-day living (Niemann, Ruff & Kramer, 1996).

Kolb and Wishaw (2003) state that the concept of attention has a difficult history in psychology in that there was an era when attentional processes were simply assumed to exist. This contrasted with periods when it was posited that specific attentional systems existed and could be compromised. For example, the behaviourists in psychology held the view that personality differences were fully responsible for behaviour and did not recognise the concept of attention as a cognitive area which could, with injury, suffer deficits leading to uncontrolled or changed behaviour patterns.

Attention refers to a number of different processes that are related to aspects of how an individual becomes receptive to stimuli and the processing of interpreting incoming or attending to information, whether internal or external (Lezak et al., 2004). Mirsky (1989) as cited in Lezak et al., (2004) places attention within the broader category of information processing while Wells and Matthews (1994, p.19) define attention more simply as ñthe selection or prioritisation for the processing of certain categories of information, signals for focal (conscious) attention, and maintaining a vigilant and alert stateò Mirsky, Anthony, Duncan, Ahearn and Kellam (1991) cited in Niemann et al., (1996) take a more clinical approach and identified four elements of attention as the ability to focus, sustain attention, encode details and shift attention. Niemann et al., (1996) proposed a framework of separating attention into the following subdivisions (a) arousal/alertness and sustained attention, (b) selective attention and (c) energetic aspects of attention which encompass concepts such as effort, resource allocation and information speed processing.

According to Drew et al. (2007) orienting attention requires disengagement from one point of fixation to another attentional focus or to a new location of interest. It then requires a process of re-engagement (or re-focus) at the new location of interest. Deficits in any, or all of these processes, could lead to difficulties with orienting attention or moving attention from one location to another. This pattern has been observed in individuals who have suffered a MTBI. (Oken, Salinsky & Elsas, 2006). According to Whyte et al., (2008) phasic arousal, focussed attention and sustained and divided attention are responsible for most information processing impairments.

2.10.2 The three models of attention

As attention span is linked to reaction time (an inability to pay attention to a stimulus leads to an inability to react) the three models of attention posited by Mesulam, Posner and Mirsky as outlined by (Zillmer et al., 2008) are briefly described below to contextualise the concept in terms of the research. Each model represents a different theoretical orientation, type of attention, method of study and degree of empirical verification. The following models provide information pertaining to the neuropsychological conceptualisation of attentional functioning.

2.10.2.1 Mensulam's (2000) Selective and Spatial Model

According to Zillmer et al., (2008) Mesulamôs model, which was developed in 2000, is a model of selective and spatial attention that has improved the understanding of the neuropsychological signs of patients who show symptoms of attentional neglect. Based on clinical and empirical research it was found that a neural network which involves the frontal, parietal, and cingulated cortices supports spatial attention to the extra-personal world (the outside world). Each of these regions makes a different contribution to spatial attention. The parietal regions generate an internal spatial representation (sensory map) of the extra-personal environment, whereas the cingulated cortex assigns and regulates motivational and emotional significance.

The frontal cortex, particularly the frontal eye fields and surrounding areas, adjusts and co-ordinates motor programming for exploring, scanning, fixating and manipulating extra-personal stimuli (Mirsky et al., 1991). Mesulamôs (2000) extended his model from spatial attention to information held within working memory. Research participants underwent neuroimaging while performing a spatial working memory task and a spatial orientation task. The spatial working memory task required the orientation of attention, while the spatial orientation task required attentional orientation to extra-personal stimuli. Neuroimaging demonstrated that both tasks required overlapping networks involving the occipital, parietal and frontal cortices within the brain. With regard to the frontal lobes, orienting to extra-personal stimuli activated the premotor and dorsal prefrontal cortex of the brain, while the anterior prefrontal regions were selectively engaged in orienting attention to internally represented stimuli. It was concluded that lesions in any neural components that support spatial attention may lead to hemi-spatial neglect, essentially

a failure to attend to the contralateral visual field. Hemi-spatial neglect (inability to see both sides of the visual field) usually relates to right brain hemisphere injury and not the left.

2.10.2.2 Posner and Rothbart (2006) Anterior and Posterior Attention Model

According to Posner and Rothbart (2006) models of attention are derived from a cognitive psychology and neuroscience perspective. They assert that attention can be defined by three major functions, namely (1) orienting of events to locations in visual space; (2) achieving and maintaining a vigilant or alert state; and (3) the ability to coordinate voluntary actions. Each attentional function is supported by separate neural networks, namely the orientation, vigilance and executive networks. These attention-neural networks operate interactively with each other and other cortical and subcortical regions. The three basic cognitive operations are active when visually orienting to a new event in the environment. First attention will be disengaged from the present event of focus and will then be moved to the new point of focus. Parietal, midbrain and the thalamic regions of the brain are linked to the operation of disengagement, movement or engagement. The visual orienting system (posterior attention system) plays a role in conscious attention and also directs the attention of the eyes to a point in space. The posterior parietal lobe mediates conscious attention to spatial targets and the midbrain superior collicus plays a role in eye movement, from one position to another, while the pulvinar (of the thalamus) helps to select and filter important sensory information for processing.

Table 3: Posner and Rothbart (2006) Attentional Networks

Attentional Networks	**Functions**	**Neural Correlates**
Posterior orienting system	Orienting stimuli	
	Disengage attention from a stimuli	Temporoparietal, superior, temporal, superior parietal
	Move to stimuli Engage new stimuli	Superior colliculus Thalamus
Vigilance attention system	Achieving and maintaining an alert state	Right frontoparietal
Anterior or executive attention system	Orchestrating voluntary actions	Anterior cingulated lateral and orbit frontal prefrontal cortex, basalganglia, and thalamus.

2.10.2.3 Mirsky's (1996) Elements of Attention Model

Mirsky (1996) developed a neuropsychological model that identified the elements of attention which he related to neuropsychological measures and the underlying neural system. His model has three elements of attention, namely focus and execute and sustain and shift. A study was conducted on adult neuropsychiatric patients who were compared with a non-neuropsychiatric control group. A battery of neuropsychological measures was administered and the outcome of the tests helped him conceptualise and operationalize his model. Later, the battery was extended to healthy children, with measures appropriate for their age groups. Four factors were identified as elements of attention in both studies.

Later he identified another element that represented the consistency of attentional effort (stable or stability). The five elements of attention are supported by distinct neuroimaging regions (See table 3). He postulated that undamaged neural regions can provide some degree of compensation to the specific function of attention when they are compromised by injury. Zillmer et al., (2008) state that there is an agreement that at a cortical level, the right hemisphere, in particular the parietal and the frontal regions, play an important role in attentional control. Subcortically, the anterior cingulated thalamus, colliculi and the basal ganglia contribute to attentional functioning. These cortical and subcortical regions do not operate independently but rather perform their functions via interconnecting neural system. The following table (4) presents the five elements of attention and their assumed supportive neural substrates.

1. Focus-execute attention entails the selective attention quick perceptual-motor output.
2. Shifting attention depicts the ability to move or change attentional focus in a flexible and adaptive manner.
3. Sustained attention pertains to the attention function of vigilance.
4. Encode attention details the capacity to momentarily maintain the information in memory whilst performing other related actions.
5. Stable ï was added later and represents the consistency of attentional effort.

Table 4: Mirskyôs (1996) Elements of Attention

Subject Group	**Factor1: Focus-executive**	**Factor 2: Shift**	**Factor3:Sustain Added later Factor 5: Stable**	**Factor 4: Encode**
Adult	WAIS-R Digit Symbol, Stroop test, Letter Cancellation and TMT A & B	WCST	CPT	WAIS-R Digit Span and Arithmetic
Child	WISC-R Coding and digit Cancellation	WCST	CPT	WAIS-R Digit Span and Arithmetic
Supporting substrate	Focus: Inferior parietal and superior temporal cortexes. Executive: Inferior parietal and corpus striatum	Prefrontal cortex	Rostral midbrain structures and brainstem	Hippocampus and amygdala

2.10.3 Attention shift

The shift element is the ability to change attentional focus in a flexible and adaptive manner, with the capacity to shift from one salient aspect of the environment to another. The shift mechanism of attention is situated within the prefrontal cortex of the brain. Rafal and Robertson (1995) found that the right parietal lobe is critical for shifting attention between locations, whilst the left parietal lobe is critical for shifting attention between objects, damage to either lobe results in significant attentional deficits. Mirsky et al., (1991) suggest that the encoding of stimuli depends upon the hippocampus and amygdala. There is very little consensus that exists with regard to a model that would best explain attentional phenomena, regardless of substantial efforts within several disciplines (Lezak et al., 2004).

2.10.4 Arousal

Luria (1966) proposed that there are three hierarchically integrated and interdependent anatomical systems which regulate attentional processes. These are the brainstem, the diffuse thalamic system and the thalamofrontal gating system. The brainstem is responsible for the tonic arousal of the telencephalon. The thalamic projection system is responsible for the phasic activation of the cerebral cortex, especially the associative cortex. The thalamofrontal gating system is responsible for controlled or selective attentional processes. Duffy (1962) however, simply referred to arousal as a range of physiological states between coma and excitation. Whereas Banich (2004) states that arousal represents the most basic levels of attention without which an individual would be unable to extract information from the environment or to select a particular response. According to Niemann et al., (1996) at first arousal was considered to be the general drive state of the organism that enhances all behaviour. The concept of arousal has since shifted from behaviourism to cognitive psychology, with the growth of interest in the information processing model in psychology. Nobre and Coull (2010) note that there are many different neurotransmitters associated with arousal. They define arousal as the tonic and phasic levels of mediated noradrenergic activation. This action enhances signal to noise ratios of neural signals underpinning perceptual and cognitive representations.

2.10.5 Alertness

Posner, Nissen and Klein (1976), introduced the concept of alertness which was defined as the state of general receptivity of the organism to external and internal information. He clarified alertness as a cognitive state and arousal as a physiological state reflecting the attentiveness of the organism. Alertness, like attention, can also be divided into tonic and phasic levels (Mureriwa, 1997). Van Zomeren et al. (1984) differentiated between tonic and phasic arousal. Tonic alertness is the continuing responsiveness of the organism to stimulation for minutes or hours and changes occur slowly and involuntarily as a result of physiological changes in the organism for instance, diurnal rhythms. Phasic alertness occurs in anticipation of an event. The changes in phasic alertness occur rapidly and depend on the individualôs interests and intentions (Papanicolaou, 1987).

A study using 108 women athletes suggest that levels of alertness are likely to be lower in athletes that are involved in heavy in-season training Brown, Guskiewicz, and Bleiberg (2007). They noted that subjects from a wide-range of athletic activities including lacrosse and womanôs soccer (football) showed poorer performance on neuropsychological testing particularly on visual memory and response on vigilance tasks compared to out-of-season athletes. Males and females however, may show differences in this regard due to different physiological make-up. A later study, using a computerised battery that had simple reaction time tasks found that a number of factors affected scores particularly alertness at the time of testing, the type of sport the athlete was involved in and the athletesô gender. Females had poorer scores generally however, it was noted that clinicians should be wary of misinterpreting scores because of differences in athletes base-line performances (Brown et al., 2007). This was supported in a study by Colvin et al. (2009) who found that football players with a history of concussion performed worst on a computerised neuropsychological test and female soccer players generally performed worst on neurocognitive testing than males.

2.10.6 Sustained Attention

Mirsky et al., (1991) describe sustained attention as meaning vigilance in the sense of the ability to maintain focus and alertness over time. Lezak et al., (2004) describes it as a capacity to maintain an intentional activity over a period of time. Whereas Zillmer et al., (2008) describes sustained attention as the ability to maintain an effortful response over time, which is related to the ability to persist and sustain an appropriate level of vigilance. A recent description of sustained attention is the capacity to maintain accurate responses across tasks which can be effortful and demanding or monotonous (Nobre & Coull, 2010). According to Zillmer et al., (2008) it is the major responsibility of rostral brain structures to sustain focus on a particular element in an environment and sustained attention is attributed to the right fronto-parietal-thalamic neural network. Van Zomeren et al., (1984) propose that sustained attention deficits present as time-on-task effects, lapses of attention and intra-individual variability. Time-on-time task effects refer to a decrease in performance over time. For instance, on reaction time tasks an individual takes longer to respond to a stimulus. Intra-individual variability means that in a continuous task the individual shows fluctuations in performance. Such lapses are defined by

Van Zomeren et al., (1984, p. 38) as ñsudden dips in level of performance lasting a few seconds at the most.ò A study conducted on individual differences in general cognitive ability might be related to observable differences in the activity of brain system. Electroencephalograms of 80 healthy young adults were recorded during a working memory task. Measures of task-related neurophysiological and behavioural variables were derived from the data and compared to scores on test battery commonly used to assess general cognitive ability namely, on the WAIS-R or the Wechsler Adult Intelligence Scale (See Appendix F). The results suggested that subjects who scored high on the WAIS-R tasks were better able to focus and sustain attention than those with lower scores (Gevins & Smith, 2000).

2.10.7 Selective attention

Selective attention is the ability to set priorities in information processing by means of evaluation which enables the individual to make optimal use of limited sensory capacities (Niemann et al., 1996). In many types of brain dysfunction the brain competence or ability to process information is reduced, because of this an individual cannot sustain attention on one particular stimulus for long periods, or cannot select information from competing sources. This impairment may be minimally present and only be detected through formal neuropsychological testing.

Initially, the theories that were proposed to explain selective attention problems assumed bottlenecks or impediments in the processing of information. These bottlenecks occurred when too much information (sensory overload) occurred at any one time (Mureriwa, 1997). However, the different theories assumed that these bottlenecks occurred at different stages of information processing. These models were found to be inappropriate as they could not explain many research results. According to Zillmer, et al., (2008) contemporary neuropsychological theories of attentional processing consider the role of the reticular activating system (RAS) in cortical arousal, subcortical and limbic system structures in regulating the information to be attended to, the posterior parietal lobe system in focusing conscious attention and the frontal lobes in directing attentional resources. The right hemisphere is given prominence as guiding attentional

processes. It must be noted that theorists can only describe the general subsets of the brain system related to attentional functioning.

Broadbent (1971; 1982) as cited in Niemann et al., (1996) proposed a model with two modes of selection (stimulus-set and response-set). The stimulus-set controls the source of stimuli (right ear versus left ear) and response-set controls the range or properties of stimuli (digits versus letters). Three strategies (filtering, categorising and pigeon-holing) are associated with the above model. Filtering is a type of selection based on physical or sensory features of the stimulus. It is a process that is fast and takes place in a hierarchical fashion and is susceptible to sensitivity changes (spatial proximity of stimuli). Categorising implies selection that connects a set of stimuli with a set of responses. It is a process that requires focus and an increase in the number of irrelevant stimuli slows down processing time, suggesting serial processing. Pigeon-holing is a strategy which operates by applying bias to specific categories. It is also fast, but is not a hierarchical, process (for instance, digits versus letters are processed without analysis of the first feature). It is assumed that since pigeon-holing involves simultaneous processing an increase in the number of relevant stimuli does not affect processing time. Nobre and Coull (2010) also report that dopamine signals and top-down processing mediate selective attention in the cortex and are both believed to direct information processing appropriately.

2.10.8 Focused attention

According to Zillmer et al., (2008) focused attention is the ability to respond and pick out the important elements of attention from the ground or background of external and internal stimuli. Focused attention also implies a measure of effort processing. Lezak et al., (2004) describes focus attention as the capacity to respond directly to specific stimuli. Mirsky et al., (1991) describe focus attention as symbolising the ability to select target information from a broader stimulus field for additional processing. They also found that the ability to focus depends largely on the activities of the superior temporal and inferior parietal cortices, as well as the structures of the corpus callosum. These investigators adopted an earlier suggestion by Mureriwa (1997) that the execution of responses relies on the integrity of the inferior parietal and the corpus striatal

regions. Focused attention requires the ability to ignore distraction. Lezak et al., (2004) postulate that the encoding of stimuli depends on the hippocampus and amygdala, whilst the capacity to shift from one salient aspect of the environment to another is supported by the pre-frontal cortex. The Stroop Word Colour Test (Stroop, 1992) is one of the oldest measures of focused attention and usually assesses the auditory and visual area of an individual (Van Zomeren et al., 1984). More recently computerised testing for instance, The California Assessment Computerised Assessment such as the CALCAP measures attention, specifically, divided and focused attention (Miller, 1993a).

2.10.9 Divided attention

Divided attention refers to doing two or more things simultaneously and vigilance, also known as sustained attention, is the capacity to maintain accurate responding over time (Nobre & Coull, 2010). Divided attention requires using attentional resources at the same time rather than switching backwards and forwards (Zillmer et al., 2008). Divided attention deficits are the most salient and commonly reported cognitive disruptions following MTBI and they may also comprise the most sensitive indicator of cognitive dysfunction (Binder et al., 1997; La Berge, 1995; Pare et al., 2009). According to Lezak et al, (2004) divided attention involves the ability to respond to more than one task at a time or two multiple elements or operations within a task, as in a complex problem solving. It is thus very sensitive to any condition that reduces attentional capacity. Attention improves the speed and accuracy of many tasks and according to La Berge (1995) the major benefit of attention is that it allows individuals to engage in the sustained processing of any mental activity. Deficits in divided attention are frequently evidenced by reduced speed in the performance of tasks (Miller, 1993a; Ponsford & Kinsella, 1992). First indications of divided attention deficits after MTBI were reported over 30 years ago by Gronwall and Wrightson (1975), who measured performance on the PASAT (See Appendix E), a task which requires individuals to add consecutive pairs of numbers as the listen to a string of numbers read out to them. MacFlynn, Montgomery, Fenton and Rutherford (1984) and Wang, Chan and Deng (2006), subsequently obtained the same results using a more complex task which required divided attention between two tasks. Patients with mild concussive symptoms produced performances which were three times slower than patients who were not concussed. A recent

study notes that divided attention deficits are still in existence several months after MTBI which supports earlier research (Paré et al., 2009). The research recruited MTBI patients from hospital emergency departments. The researchers utilised a computerised programme which measured divided attention as a neurological function. The three administration conditions for divided attention were: 1) a complex reaction time (RT) task in which participants provided different motor responses; (2) participants continued to carry out the RT task while simultaneously repeating aloud a different series of four numbers (digit span task) presented verbally by the researcher and (3) participants had to carry out complex computerised RT task while simultaneously repeating aloud a different series of five numbers (digit span task) presented verbally by the experimenter. These tasks are similar to those used in the present study minus the verbal component.

2.10.10 Visuo-spatial orientation of attention

Halterman et al., (2006) report that attention deficits are regularly observed in individuals who have recently suffered an MTBI. Research has illustrated that participants with MTBI often struggle to maintain or allocate appropriate attentional resources when performing one or more concurrent tasks. The visuo-spatial orienting of attention is comprised of disengagement, movement and re-engagement components that are associated through unique but interconnected neural networks. The parietal, frontal, temporal and cingulated cortices, in addition to the midbrain, have specific roles that are played in these attentional networks. The lesions of the parietal lobe lead to deficits in the ability to disengage attention from the sign location, whereas lesions in the frontal or temporal lobes and midbrain create no such deficits. Based on previous studies, they believe that it is possible to identify regions of the brain that are susceptible to injury induced by a traumatic blow to the head. Those regions of the brain which might be at greater risk for injury may be associated with specific deficits when performance of concussed individuals is compared with non-concussed matched controls in the same age group. The authors conducted a study with 20 participants who had incurred MTBI who were recruited for testing within two days following and MTBI. The Attentional Network Test (ANT) (See Appendix F) was used to assess specific aspects of the alertness, orientation and executive components of attention. The course of injury ranged from impacts to the head occurring during

sporting activities to accidental falls and collision with stationery objects. The outcome of the study demonstrated that the orienting and executive components of visuo-spatial attention are vulnerable to injury that is caused by MTBI and that the executive component of attention will exhibit dysfunction a month post injury. In contrast, the alertness component of attention is relatively immune to the negative effects of MTBI. It is widely held that these components of attention do involve networks within the brain that function differently, even though they are interconnected. Halterman et al., (2006) state that, based on this new evidence, they believe that these regions of the brain are more at risk than others after an individual incurs an MTBI.

2.10.11 Attention and the concept of Information Processing

The main assumption of the information processing approach is that there are a few symbolic, computational operations like encoding, comparing, locating, storing, retrieving and making decisions which ultimately account for intelligence. According to Mulder, (1983, p.38) cognition is ñthe basic ability of the brain to analyse, store, retrieve and manipulate information in order to solve problems.ò Theoretical constructs of attention place it within the framework of information processing. There are three suggested stages of information processing serial comparison, binary decision and choice that all require controlled attentional processes. Whyte (1992) postulates that attention can be expected to affect the speed and accuracy of information processing because of its controlling function in reaction time tasks. This was also postulated by Van Zomeren et al., (1984) who stated that nearly all neurological impairment can be expressed in reaction time tasks, such as slow information processing, Since reaction time is important in the present study, it will be discussed briefly.

2.10.12 Reaction Time

Reaction time (RT) is a favourite subject of experimental psychologists (Kosinski, 2008). Helmholtz was the first to introduce reaction time (RT) in 1980 and carried out RT experiments in order to measure the speed of nerve conduction. His method entailed stimulating the nerve of a frogôs leg at different points, near and far from target muscles and measure the time it took for the animal to respond to the stimulation, which was labelled as the reaction time. Later RT was

used in the assessment of patients with TBI, in all probability, because it reflects either information processing speed or speed of attentional capacity (Segalowitz, Dywan & Unsal, 1997).

Current RT experiments are designed in different ways and use diverse equipment, both pen and paper and computerised testing. Miller (1995) carried out reaction time experiments in order to study the subtle cognitive changes that occur in the early stages of many kinds of brain disease. A study was conducted on a homogenous sample of well-educated, gay and bisexual men. They were tested at semi-annual intervals using both computerised and traditional neuropsychological tests. These tests were carried out at the beginning of the HIV and Aids pandemic to see if the retrovirus had any impact on brain systems (which was subsequently found to be the case). Brouwer and Van Wolffelaar (1985), as cited in Niemann et al., (1996) administered a 40 minutes low event rate task with memory load to 8 patients with moderate to severe TBI. They were tested twice at intervals of 2 months and 5 months after the initial injury. No difference in performance over time was noted between the patients and a control group of individuals who hand not incurred TBI, either in terms of criterion and sensitivity shifts or mental effort as measured by heart rate variability. However, the mean heart rate was elevated and RTôs were prolonged in the experimental group as compared to the control group.

Computerised RT measures are often viewed as being more sensitive than traditional neuropsychological tests for studying the subtle kinds of cognitive changes that occur in the early stages of brain disease (Miller, 1995). For the purpose of this research, simple and choice reaction time are briefly explained in order to clarify the concepts. Reaction Time abbreviated as RT is the duration, in milliseconds, from the time the test stimulus is presented, to the time the subject reacts. In simple reaction time experiments, using computerised testing, there is only one stimulus and one response and it measures psychomotor skills (Kosinki, 2008; Miller, 1995). In choice RT experiments, the user must give a response that corresponds to the stimulus on the computer screen by pressing the key corresponding to a letter as it appears on the screen. Choice RT occurs when the task is more complex (Kosinski, 2008). A study conducted by Miller (1995) found that simple and choice RT tasks measure at least two domains of cognitive functioning

that are relatively independent of the psychomotor skills assessed by traditional neuropsychological tests. Computerised RT programmes expect individuals to use the space bar when responding to a stimulus (Kosinski, 2008; Miller, 1993a; Wintink, Segalowitz & Cudmore, 2001). Today, computerised assessment is generally used to measure RT as its advantages are that test-packages are fast, accurate and easy to use. The main disadvantage found previously was that not all individuals were computer literate. However, computer literacy is not a necessity as a stimulus appears on the screen and the individual presses the space bar. This should not disadvantage rural or illiterate groups. The cost of such computerised packages is similar to pen-paper tests. There are many such computerised tests that in post-20th century assessment have assumed a dominant place in the neuropsychological assessment repertoire (Kosinski, 2008).

Two main measurements are taken during RT experiments. These are reaction time (RT) and movement time (MT) (Jensen & Munro, 1979; Miller, 1995). RT is the duration, in milliseconds, from the time the subject lifts his finger to react to the time he presses the computer key. According to Wintink et al. (2001), some researchers suggest that RT reflects decision time, the length of time required for stimulus evaluation and response programming. Movement time (MT), on the other hand is a measure of the time it takes to complete a response. Whilst RT reflects cognitive processes MT reflects the motor component of the reaction time. Other researchers have defined reaction time as the sum of RT and MT which is then referred to as Total Reaction Time (Dunlop, Björklund, Abdelnoor & Myrvang, 1993; Miller, 2001). According to Miller (2001) the California Computerised Assessment Package (CALCAP) is designed to assess specific facets of cognition, including processing speed, language skills, rapid visual scanning, form discrimination, recognition memory and divided attention and as it emphasises processing efficiency, RT is critical to its tasks.

The central issue in the information processing approach after head injury is that patients are significantly slower than uninjured individuals. The main cause of cognitive slowness is a delay in access to stored knowledge (Brouwer & Van Wolffelaar, 1985). According to Tromp and Mulder (1991), mental slowness after head injury is due to reduced redundancy of memory representations, causing a delay in the retrieval of information stored in memory. Redundancy

implies that, in the normal brain, knowledge is stored in multiple ways and that multiple access routes can be taken to reach an item of knowledge. Other factors influencing RT are arousal, fatigue, alcohol and brain damage (Kosiniski, 2008). RT is fast with an intermediate level of arousal and it deteriorates when the subject is either too relaxed or too tense (Broadbent, 1971; Kosiniski, 2008). RT gets slower when the subject is fatigued, mental fatigue especially sleepiness, has the greatest effect. The slowing of RT by alcohol is due to the slowing of muscle activation and not muscle action. Kosiniski (2008) reviewed Bashore and Ridderinkhof's (2002) study and reported that, as might be expected, brain injury slows RT but different types of responses are slowed to different degrees. A study by Collins et al., (2003) found that high school athletes with concussion and headache, a week after injury, had worse performance on RT and memory tests than athletes with concussion but did not have a headache a week after injury. Kaminski, Cousino and Glutting (2008) found that hitting the ball with the head in soccer had no significant effect on the RT of female football players. However, Dvorak, McCrory and Kirkendall (2007) reported, in a study of 2340 male and female American high school and collegiate athletes, that 155 individuals sustained sports-related concussions. This was determined by using standardised cognitive tests. Female athletes had a markedly greater decline in simple and complex reaction times relative to pre-season baseline levels and also reported more post-concussive symptoms compared with age-matched male athletes.

2.11 Use of neuroimaging to diagnose MHI

Echemendia and Julian (2001) postulate that traditional neuroimaging techniques**,** Magnetic Resonance Imaging (MRI) and Computerised Tomography (CT) scans (See Appendix E), do not allow clinicians and researchers to view many of the pathophysiological processes described earlier. It is common that athletes report somatic and cognitive symptomatology even when MRI and other imaging techniques reveal no gross abnormality in the appearance of brain tissue. Players who have sustained MTBI present with many different symptoms or deficits ranging from temporary confusion to death, as seen in second impact syndrome (See 3.5.2). Since MTBIôs causes axonal and biochemical changes that are difficult to visualise with traditional neuroimaging techniques, individuals involved in the care of athletes (at all levels from school athletes to elite athletes) have been unable to obtain objective, reliable information on the

severity or course of such injuries. According to McCrory et al., (2009) newer structural MRI modalities, which include gradient echo perfusion and diffusion imaging have a greater sensitivity for structural abnormalities. However, the absence of published studies as well as absent pre-injury neuroimaging data limits the usefulness of this approach in clinical management at present. In contrast to the limited published studies of newer radiologic techniques, neuropsychological measures (pen and paper tests and computerised tests) have demonstrated utility in the assessment of MTBI and are sensitive to diffuse axonal damage. Researchers in United States used an MRI on 32 amateur football players, with an average age of 30.8 years, who had played soccer (football) since childhood. The results of the MRI were that heading a ball repeatedly can lead to brain damage (Pretoria News, 2011).

According to Lovell (2008) concussion occurs on a physiologic rather than a structural level. Traditional neuro-diagnostic technique (CT scan, MRI and neurologic examination) display almost consistently normal results after concussive injuries. It should however, be emphasised that these techniques are valuable in ruling out more serious pathology (for instance, cerebral haematoma or skull fractures) that may arise with head trauma. Recent research has examined the potential usefulness of the Functional MRI (FMRI) as a feasible tool for the assessment of neural processes after concussion. The technology is based on measurement of specific correlates of brain activation, such as cerebral blood flow and oxygenation. FMRI has also promoted the assessment of specific neuropsychological test paradigms through which cerebral blood flow changes can be linked to specific tests which measure memory and other cognitive processes. Since FMRI does not involve exposure to radiation, it can be safely used in children and repeat evaluations can be undertaken with minimal risk. This promotes the assessment of changes in neural substrata that may occur with mild concussion, permitting the tracking of injured athletes throughout the recovery process. One of the most important potential uses of FMRI scanning is the ability to provide validity data, with regard to the sensitivity and specificity of neuropsychological testing, for the detection of subtle changes in brain function. Although it is a it is a promising tool, the FMRI is still has to be implemented in most clinical settings.

2.12 Neuropsychological assessment

Lovell (2008) states that the use of neuropsychological testing in sports medicine was developed in the mid-1980ôs when professional sport became the international money making entity it is today. Lovell (2008) states that Barth et al. (1989), and his colleagues at the University of Virginia demonstrated the usefulness of neuropsychological test procedures to document cognitive recovery within the first week of concussion. A series of events transpired in the early 1990ôs and that shifted the use of neuropsychological testing into clinical sports medicine. A number of high profile professional athletes in American Football were injured and did not recover timeously, costing their franchises much money. This resulted in the implementation of baseline neuropsychological testing by several American Football clubs in the mid 1990ôs. The National Hockey League (NHL), also in the USA, mandated baseline neuropsychological assessments for all athletes for similar reasons. A number of large scale studies of collegiate athletes in America were completed, in addition to the increased use of neuropsychological testing in sports. These studies provided further evidence that neuropsychological tests yielded useful clinical information. Neuropsychological testing has specifically allowed a baseline and post injury analysis of the subtle cognitive functions likely to be affected by concussive injury. This has provided objective data to help compile return-to-play and treatment protocols for all kinds of professional sports. It can be stated that the use of traditional neuropsychological testing (pencil and paper testing) and later computerised testing has resulted in the rapid expansion of knowledge regarding concussion.

According to McCrory et al., (2009) the application of neuropsychological (NP) testing in concussion is of clinical value and contributes important information in contemporary evaluations of concussion. It has been demonstrated that cognitive recovery may occasionally precede, or more commonly follow, clinical symptom resolution. This indicates that the assessment of cognitive function is an important part in any return to play protocol in professional sport. It should be emphasised that NP testing should not be the only basis for the management of return-to-play decisions. It should be seen as a part of the clinical decision making process in conjunction with a range of various clinical domains and investigations. In most cases, NP testing is used to assist return-to-play decisions, and is not carried out until an

individual is symptom free. Neuropsychologists are in the best position to interpret NP test results by virtue of their background and training. In instances where a neuropsychologist is not available other medical professionals, particularly neurologists, perform or interpret NP screening tests. All-inclusive concussion management protocols have been advocated in a number of consensus statements and should include baseline cognitive and postural stability (utilising a bio-kineticist) testing, especially for athletes in high-risk sports (Tommasone & McLeod, 2006).

2.12.1 Neuropsychological assessment for evaluating attention deficits

Whyte (1992) and Lezak et al., (2004) suggested that in order to assess attention the clinician has to rely on measures that assess motor, perceptual, and cognitive activity. However, little attention has been given to the development of psychological tests of attention. The most currently used methods for assessment of attentional deficits are grouped under the different components of attention, namely arousal and alertness, selective, focused, divided and sustained attention as briefly presented in the following paragraphs.

2.12.2 Arousal and Alertness

Arousal can be ascertained from EEG (See appendix E) spectral analysis. The hippocampal theta rhythm is associated with heightened attention (Lezak et al., 2004). Reaction time has been used to assess phasic arousal by comparing an individualôs performance with and without a warning signal. De Brabander, De Clerk and Boone (2002) showed that in normal controls, the presence of a warning led to a reduction in reaction time, but this was not the case with brain damaged individuals. The difference between brain damaged individuals and a normal control group on warned versus not warned task is more evident in choice reaction time (RT) tasks.

2.12.3 Selective Attention

Whyte (1992) and Lezak et al. (2004), describe the different assessment measures of selective attention. They included pencil and paper tests of hemi-spatial inattention such as Line Bisection, Letter cancellation, and the drawing of symmetrical figures like clocks (See Appendix F). Illiterate individuals (those who cannot read or write) are assessed by using tests such as the Motor Free Visual Perception test (See Appendix F).

2.12.4 Focused Attention

According to Lezak et al., (2004) the Stroop Word Colour test (See Appendix F) is one of the oldest measures of focused attention. Focused attention is normally assessed in the visual and auditory areas. It can be assessed using a dichotic listening task. Fundamentally, a dichotic listening task is used to measure the selective and focused attention of the auditory system. It is used to test for hemispheric lateralisation of speech and sound perception. In the usual type of test a subject is presented with different types of auditory stimuli over a headphone and has to make specific choices (Ingram, 2007). Other measures of focused attention are visual tests that include the Letter Cancellation Task, the Trail Making Test and Reaction Time with Distraction **(**See Appendix F).

2.12.5 Divided Attention

Reduced speed in the performance of tasks is normally evidenced as deficits in divided attention. The reduced speed in performance can be confirmed through the use of reaction time tests. The Paced Auditory Serial Addition Test (PASAT - See Appendix F) is a commonly cited measure of divided attention. The degree of impairment on the PASAT correlates positively with the severity of injury (Lezak et al., 2004). Patients with mild concussion produced performances which are three times slower than a control group with no concussion and the severely injured were found to be five times slower than normal controls. The WISC-R (See Appendix F) can be also used to diagnose attentional problems.

2.12.6 Sustained Attention

Bonnelle et al., (2011) state that sustained attention deficits present as time-on-task effects, lapses of attention and intra-individual variability. Time-on-task effects refer to a poorer (decreased) performance over time. For example, in a reaction time task, the patient takes longer to respond and to complete the task. Intra-individual variability means that in a continuous task the individual shows fluctuations in performance. According to Lezak et al. (2004), other tests for sustained attention are cancellation tasks like the Letter cancellation test, Vigilance tests and Perceptual Speed tests (See Appendix F).

2.12.7 Computerised neuropsychological assessment

According to Zollman (2011) a number of computerised cognitive tests (CCTôS) have been developed to assess changes in cognition. Lovell (2008) also notes that CCTôs are widely used to assess sports related injuries for example, the Post-Concussion Assessment and Cognitive Test (ImPACT). Foxcroft and Roodt (2001) assert that computerised assessment programmes have been multiplying and may be on the verge of assuming a dominant place in psychological and neuropsychological testing. The advantages are that good levels of standardisation of assessment are achieved and the potential bias effect of the assessment practitioner is eliminated, as the computer administers and scores the measure in an objective way. There are disadvantages in computerised assessment, for instance lack of computer literacy on the part of some test takers which could impact negatively on their performances. It is also true that important qualitative information about test taking behaviour cannot be readily accessed during computerised assessment. It is also true that some computerised scoring routines can have errors or may be poorly validated. Such problems are often difficult to detect within the software. Lezak et al., (2004) report that guidelines for appropriate and ethical computerisation of neuropsychological assessments were first published in 1987 and are still valid today (they have been updated periodically). These ethical guidelines should be reviewed by anyone who is considering the introduction of computerised programmes into their neuropsychological examination procedures. However, a perusal of published articles, books, and test publisherô catalogues suggests that by and large, most clinicians and researchers, continue to rely primarily on traditional clinical

assessment techniques with limited use of specialised computerised programmes, for example ImPACT and Cognate (See Appendix F).

2.13 Cross-cultural neurological assessment

Lezak et al., (2004) state that the cultural background of patients should always be considered when planning and interpreting assessment data. Clinicians should be aware of cross-cultural influences and bias that are vital for the assessment of individuals who come from different cultural backgrounds. They state that a leading assessment problem is the lack of well-standardised, culturally relevant tests for minority groups, or in the case of South Africa majority groups. Nell (1999) states that it should be standard that the construct underlying the test and interview questions should have a common shared existence in the minds of the test maker and the test taker. The absence of construct equivalence is often clear for clinicians assessing clients from cultures other than those a test has been standardised and validated on. Lezak et al., (2004) report that the evaluation of a patientôs responses in a neuropsychological examination must take into account the contributions of their social and cultural experiences and attitudes to test performance, plus their feelings about and understanding of, their physical or psychological conditions. When characteristics of cultural backgrounds or socio-economic status are overlooked, test score interpretations are subject to confusion. This can lead to inappropriate culturally determined analysis of results by the tester. This gives rise to false positive errors and to the missing evidence of deficit on over learned or over practiced behaviours, resulting in false negative errors. The test performance of a patient whose cognitive development is irregular and who has sustained brain injury, that involved his or her strongest abilities, may show variability on test results that suggest impairment but who may be cognitively intact. Nell (1999) also recommends that for the achievement of a threshold of functional literacy an individual must have completed at least 12 years of formal education. This minimum is required for tests that require well-entrenched numbering, reading or reasoning skills.

2.14 Summary

This section of the study, focused on literature which place MHI, MBTI and CMHI in the broader scope of head injury through discussing the dominant types of head injuries and assessment of their severity. The definitions of MHI, CMHI, MTBI and concussion were also discussed. Prevalence studies were reviewed which indicated that MHI was likely to go unreported and often, even if reported, not assessed with proper concern. Injuries were divided into primary and secondary occurrences. The incidence, demographic characteristics, pathophysiology and neuropsychological sequelae of MHI were also discussed. The chapter discussed cognitive deficits which can occur in MHI and CMHI and lastly, a brief discussion was given as to how neurological impairment can be expressed through RT. The complexity of the neurological substrates of attention was also highlighted. From the review in chapter two (2.7.1) it was pointed out that closed head injury tends to be associated with diffuse brain injuries. This implies that there is a wider range of deficits possible because of the involvement of widespread areas of the brain, particularly the frontal lobes, because of their size and position. Neuropsychological assessment, pen-paper and computerised testing were discussed as was neuroimaging in the context of MHI. Attention deficits were discussed as they relate to the type of neuropsychological deficit incurred in MHI and CMHI.

CHAPTER 3: THEORETICAL FRAMEWORK FOR THE STUDY

3.1 Introduction

The following chapter gives a theoretical framework for the study as well as an in-depth overview of literature that focuses on MHI and CMHI in contact sports. The epidemiology of MHI, diagnosis, management and rehabilitation thereof is also discussed. Whilst the focus of the present study is on the Cumulative effects of MHI (CMHI) in adult football players, research pertaining to brain injury in contact sports is also discussed as it provides a framework within which to contextualise this type of injury. Reference is also made to relevant research on brain injury in children, females and/or adolescents as this adds depth to the review. Lastly, methodological issues that are encountered during research for MTBI in sport are highlighted

3.2 Theoretical context and hypothetical framework for the present study

The main contention of Shuttleworth-Edwards, Ackerman, Beilinsohn, Border and Radloff (2001) is that almost all literature pertaining to MHI is empirically based and not theoretically articulated. In order to comprehend research results in this area she postulates that a theoretical context is required. Shuttleworth-Jordan used Satzôs (1993) Brain Reserve Capacity Threshold Theory (BRC) and Jordanôs (1997) model of inter-individual variability to explain how the use of theoretical concepts allow for the identification of cognitive patterns that might not otherwise be apparent. BRC refers to a threshold factor within each human being which represents a crucial point at which normal functioning is sustained, preceding the manifestation of symptoms caused by injury or disease to the brain. Built into this model is the idea that there are individual differences that exist with regard to BRC that account for variable instances of vulnerability and symptom onset. The model holds that BRC thresholds differ between individuals. Educational levels represent an indirect measure of an individualôs BRC threshold. A higher BRC is therefore likely to act as a protective factor by decreasing the risk of functional impairment and the likelihood of exhibiting symptoms related to neurological impairment. Lower BRC acts as a vulnerability factor which increases the risk of functional impairment. In terms of this theory any

reduction in BRC, due to neurological pathology, is likely to increase an individualôs functional impairment and will more likely show neuropsychological impairment or symptom onset. Certain risk factors such as previous head injuries, age and a lower education-level may lower an individualôs BRC threshold and increase his or her vulnerability to functional impairment. Shuttleworth-Jordan (1999) proposes that even in the absence of observable or testable functional outcomes, mild brain injury may result in a reduction in BRC.

According to Shuttleworth-Edwards et al., (2001), BRC theory holds that there is a functional impairment or cut-off point that differs between individuals depending on the presence of differing vulnerability and protective factors. Pre-existing differential vulnerability factors will be a leading variability factor in symptom presentation following MHI. Jordan (1997) developed a *Shuttle* model of variability within the context of BRC. She postulates that owing to different levels of pre-existing cerebral reserve in association with the onset of neural attrition, due to normal ageing, the presentation of symptomatology in brain damaged groups will occur differently between individuals. This will be reflected in an increased variability of cognitive scores associated with the ageing process and declining raw scores. Specifically, due to protective factors which raise the threshold of symptoms onset, a notable percentage of individuals will not present with much cognitive fall-off. Alternatively, due to vulnerability factors which lower the threshold of symptom onset, a notable percentage of individuals will show a marked fall-off. This will result in a substantially expanded distribution of scores. As the brain ages, protective factors which cause high BRC thresholds in some individuals become less effective and previously good scores will perform closer to the norm. This results in marked variable symptom presentation between individuals. The distribution of scores will narrow again and be reflected in the reduced variability of cognitive test scores in association with the continued lowering of mean scores.

Shuttleworth-Jordan (1999) asserts that Jordanôs (1997) model of Inter-Individual Variability, which developed within the context of BRC theory to delineate cognitive ageing, can be extrapolated to describe outcomes following MHI sustained in rugby and other contact sports. She conceptualises normal ageing as a form of progressive mild brain injury and identifies a

pattern of variability, much the same as can be expected from mild brain injury. The model proposes that inter-individual variability in cognitive reserves (BRC) in association with the onset of neural attrition, results in the differences of symptom presentation and the variability of cognitive test scores between individuals. In other words, due to protective factors, certain individuals may not present with cognitive dysfunction whilst others, due to threshold lowering factors, may show a noticeable fall-off in functioning. This may lead to a wide distribution of scores within a group, a fact which is not represented by average group effects.

3.3 MHI in sport

Echemendia, Putukian, Mackin, Julian and Shoss (2001) estimated that between 70% and 90% of brain injuries that receive treatment are mild, many of which occur in sports related injuries. However, due to under-reporting it is difficult to determine how common the condition is.

MHI and MTBI will be used interchangeably to denote how the brain is injured in sports because the literature in sports often refers to the more clinical MTBI and not MHI. Sports related brain injuries in the form of cerebral concussion or MTBI or MHI have recently become the focus of attention for the media and medical community. Historically, sports related injuries have been dismissed as, part of the game, with little cause for concern (Echemendia & Julian, 2001). However, in the last three decades, with emphasis on athletes being in peak condition at all times and the advent of the sport bio-kineticist and sports scientist, this is no longer the case.

In the sports arena where concussion tends to be the favoured term for MTBI or MHI, a series of definitions have evolved. However, there has been a lack of an accepted definition due to the numerous limitations in accounting for the common symptoms present. In addition, relatively minor impact injuries that result in either persistent physical or cognitive symptoms have not been included in these definitions. Herring et al. (2006) developed the following definition seeking to surpass any limitations to previous descriptions. They define concussion as a complex pathophysiological process affecting the brain, induced by traumatic biochemical forces which include (a) concussion which may be caused by a direct blow to the head, face, neck or elsewhere on the body with an impulsive force transmitted to the head; (b) concussion which

typically results in the rapid onset of short lived impairment of neurological function that resolves spontaneously; (c) concussion may result in neurological changes but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury; (d) concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness where the resolution of the clinical and cognitive symptoms typically follows a sequential course and (e) concussion is typically associated with grossly normal structural neuroimaging studies.

3.4 Epidemiology of MHI and concussion in sport

In the latter part of the twentieth century and the early part of the twenty first century there was an exponential increase in the reporting of sports-related injuries, particularly concussive injury. The recent estimates of concussion by the Center for Disease Control in 2006, are approximately 300 000 injuries per year during the 1990ôs to a recent range of 1.6 to 2.3 million per year (Lovell, 2008). There are two critical factors that explain this tenfold increase in injury prevalence and awareness: 1) increased awareness at medical and public levels of the risk of sports injures and 2) the increased identification and better reporting of these injuries. Despite the above mentioned trend there is however, a high probability that concussion is still under-reported (Aubry, Cantu & Dvorak 2002, as cited in Lovell, 2008; Gerberich, Priest, Boen, Straub & Maxwell, 1983). It is postulated that in the next few decades increases in concussion rates will continue to rise (Lovell, 2008). Although not all sports related, a study conducted by Cunningham (2007) examined the prevalence and nature of concussive symptoms up to one month post presentation. They conducted the study amongst ED patients diagnosed with MHI (injuries included falling and banging the head and sports related concussions amongst other aetiologies). Ninety four who presented with minor head injury were recruited. The Rivermead Post-Concussion Symptoms Questionnaire (See Appendix B) was administered within one or two days of presentation and again at one month post injury. The proportion of patients who reported concussive symptoms were 68 out of 94, that is 72% at first assessment and 59 out of 94 that is 63% at the second assessment. A worrying finding indicated that the majority of patients with MHI from the sample suffered from cognitive symptoms that did not resolve quickly. The common symptoms reported after one month post injuries were headaches (41.5%), dizziness (28.7%), impatience, (26.6%) and taking longer to think (28.7%). It was suggested that all

patients with concussive symptoms should be informed of possible post-injury symptomology as part of routine care.

According to Echmendia and Julian (2001) The National Head Injury Association of the USA reported that brain injuries sustained in athletic competition account for approximately 18% of head injuries. The data gathered at high school, college and professional levels indicate comparatively high rates of MTBI in many sports. American Football alone generates over 250,000 head injuries per year and an estimated 20% of all high school football players sustain cerebral concussions annually (Gerberich, Priest, Boen, Straub & Maxwell, 1983; Pellman, Viano, Casson, Arfken & Feuer, 2005). The incidence of concussion in young American Football players has been estimated to be from 4 to 5%. Echmendia & Julian (2001) collected data on head injuries on high school American Football teams from the National Athletics Trainers Association (NATA) in the USA involving 351 football teams which comprised of over 21, 000 players over a 3 year period (1986-88). It was found that a total of 12, 796 injuries were reported. Five hundred and ninety eight percent of these injuries were concussions or MHI. Projections made from the data, of the USA population of high school football players estimated that an average of 25 520 concussions will occur per season, with an injury rate of 2 460 concussions per 100 000 population (Powell & Barber-Foss, 1999).

Injury data was collected from 1984 to 1991 by the National Collegiate Athletic Association Injury Surveillance System (Dick, Agel & Marshall, 1992) in the USA. It was found that cerebral concussions accounted for 1.8% to 4.5% of all injuries, with an injury rate ranging from 0.11% to 0.27% of injuries per 1 000 athletes. From 1995 to 1996, the NCAA ISS data disclosed slightly higher rates of injury from cerebral concussions when compared to previous years. Concussions accounted for 1.6% to 6.4% of all injuries with an injury rate of 0.06% to 0.55% injuries per 1 000 athletes. The NCAA ISS data for male sports from 1997 - 1998 indicated that ice hockey had the highest injury rate as compared to other sports with 0.56% per 1 000, followed by wrestling with 0.0494% per 1 000. This was followed by American Football with 0.428% per 1 000 population (1997). The increase in injury between 1984 and 1998 may be the function of the improved reporting of MTBI. It may also be that there has been an increase in the

frequency of MTBI because of the increased weight and speed of athletes which increase the force of collisions between players.

A study of American high school and college football players demonstrated 94 catastrophic head injuries (significant intra-cranial bleeding or oedema) over a period of 13 years (Boden, Tacchetti, Cantu, Knowles & Mueller, 2007). Seventy-one percent (71%) of high school players, who had suffered such injuries, had experienced concussion in the same season with 39% of them playing with residual symptoms. On the other hand, results from a study of concussion by the National Football League demonstrated no cases of catastrophic head injury in players returning to play after resolution of symptoms relating to post-concussive injuries (Pellman et al., 2005).

Tommasone and McLeod (2006) reviewed 23 incidence articles on contact sport concussion which revealed that high school male ice hockey athletes showed the highest incidence of concussion at 3.6% per 1 000 athlete exposures. They also found that soccer (football) players had the lowest incidence of concussion namely, 0.18% per 1000 athletesô exposure. At the professional level however, concussion incidence rates were found to be higher for instance, in professional ice hockey players suffered injury at a prevalence of 6.5% per 1000 player-games.

Interesting data was presented by Delaney, Lacroix, Leclerc and Johnston (2000), which is obtained from The Canadian American Football League professionals during the 1997 season. They used a retrospective survey and found that 45% of the sample of American Football players experienced symptoms of concussion but only 19% of the players realised that the symptoms they had were commensurate with concussive or sub-concussive injury. Sixty nine point six percent (69.6%) of the sample reported more than one concussion in the season but were unaware of the dangers linked to this type of injury. Echemendia and Julian, (2001) warn that players should be informed about the symptoms and effects of concussion. They state that this data underscores the comparatively high rate of brain injury in athletic competition and the fact that it is difficult to estimate the true incidence of concussions. Most studies rely on retrospective data, which have problems related to a number of factors, including (a) the athleteôs accurate

memories with regard to previous brain injuries; (b) inconsistent definitions of concussion; (c) inconsistent application of diagnostic criteria; (d) lack of knowledge among treatment personnel regarding concussion; (e) lack of information on whether the concussion was diagnosed by a physician or an athletic trainer and (f) a strong tendency on the part of athletes to under-report symptoms and concussive events. McCrea, Hammeke, Olsen, Leo and Guskiewicz (2004) as cited in Tommasone and McLeod (2006) found that more than 50% of high school American Football players did not report their concussions. The reason for not reporting their concussions included 66% believing that the injury was not serious enough and 36% not knowing they had experienced a concussive injury. Similarly, 56% of college athletes reported no knowledge of concussion consequences, 28.2% reported playing while dizzy and 30.4% reported continuing to play, despite having a headache after a blow to the head. They also stated that they did not report concussions for fear of being dropped from the team.

3.5 Pathophysiology of MHI in sport

Dischinger, Ryb, Kufera and Auman, (2009) as cited in (Echmendia & Julian, 2001) state that the three basic mechanisms through which MHI injuries occur in sports include the following: a stationary hit with a forceful blow (impact or comprehensive force); a moving head hitting a non-moving object (acceleration or tensile force) and the head being struck parallel to its surface (shearing and rotational force). Factors that predict the quantity of axonal damage involves an interaction between the magnitude and force as determined by the mass, surface area, velocity, and hardness of the impacting object. Common dynamic loading forces cause injury that last less than 200 milliseconds and in many cases that last less than 20 milliseconds, but will still cause neural tissue to break at these strain levels as they are under rapidly applied loads**.** It appears that the brain will suffer less axonal strain if the impact is applied at a slower rate. The brain appears to endure sagittal movements and motions in the horizontal plane best, which contrast to it being most vulnerable when moved laterally (Bailes & Hudson, 2001).

Axonal injuries have been labelled DAI or Diffuse Axonal Injuries (See 2.7.1 Diffuse Axonal Injury). These injuries change and/or disrupt the capability of the brain to maintain ionic gradients resulting in neurochemical changes, which is why depression and other psychiatric illnesses may occur (Lezak et al., 2004: Lovell, 2008).

According to Lovell (2008) recent research concerning the subtle neuro-metabolic effects of concussion has led to new insights into the pathophysiology of concussion. Lovell (2008) using a rodent model described metabolic dysfunction that occurred at the intracellular and extracellular levels. They hypothesized that these changes are the result of excitatory amino acid-induced ionic shifts with increased Na/K-ATPase (a protein membrane that is expressed in each cell) activation and resultant hyperglycolysis (increased glucose metabolism). This results in a high energy demand within the brain immediately after a concussive injury. Hovda, Prins and Becker (1999) as cited in Lovell, (2008) had previously demonstrated that hyperglycolysis is accompanied by a decreased blood flow to the brain which results in widespread cerebral neurovascular constriction. The resulting metabolic mismatch between energy supply and demand within the brain is hypothesized as leading to cellular vulnerability after concussive injury (from a few days to several weeksô post-injury).

3.5.1 Neurochemical changes after head injury

According to Hovda et al., (1999) as cited in Echemendia and Julian (2001), in addition to mechanical axonal changes that occur in the brain subsequent to MHI, a sequence of neurochemical changes occur resulting in paralysed and dysfunctional brain cells which create increased susceptibility to further injury. This neurochemical and metabolic cascade begins within the first hour of injury and continues for up to 10 days post injury. These metabolic changes create cells that are not necessarily irreversibly destroyed but are alive and exist in a vulnerable state. This enhanced vulnerability is characterised by an increase in the demand for glucose (fuel) and a reduction in cerebral blood flow (CBF) or fuel delivery. As a result, the neurovascular system is unable to respond to demands for the energy required to return to normal neurochemical and ionic environments. Given the cellular changes described, it has been concluded that the brainôs vulnerable state is identified as a metabolic dysfunction created by an imbalance between energy demand and the ability of the brain to work at its normal capacity. The inadequacy of knowledge regarding this time period of vulnerability can lead to an underestimation of the time required for the metabolic crisis to resolve, and may be partially responsible for the phenomena called Second Impact Syndrome or SIS (Boden et al., 2007). It was also found that higher serum levels of protein S-100 were found in patients who suffered

MHI which impairs neuropsychological function a year post injury. It was also found that computerised neuropsychological assessment are more sensitive for finding small differences or signs of neurocognitive abnormalities after MHI than conventional test batteries (Waterloo, Ingebrigstein & Romner, 1997).

3.5.1.1 Hormonal imbalances after head injury

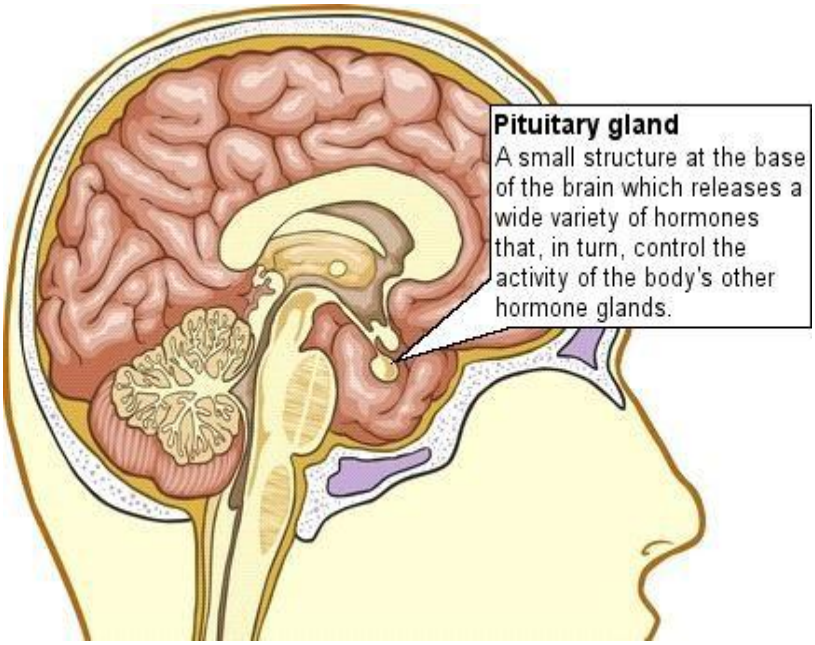
Head injuries may occasionally damage the hypothalamus and/or pituitary gland, which are small structures at the base of the brain responsible for the regulation of body hormones (Johnson & Criddle, 2004). The damage to these areas can lead to increased or insufficient release of one or more hormones, which in turn causes disruption of the bodyôs ability to maintain a stable internal environment (homeostasis). Hypopituitarism which is a condition in which the pituitary gland does not produce normal amounts of some, or all of its hormones, can be caused by damage to the pituitary gland after a TBI, leading to a reduction in hormone production (Carlson et al., 2009).

According to Carlson et al., (2009) most peopleôs hormone levels are severely affected in the early stages of head injury, thus making the diagnosis of hypopituitarism or any other hormonal problem difficult. These are only clarified during the recovery period when it becomes clear that some of the symptoms are probably due to hormonal imbalances caused by the brain injury. Since some of the symptoms are the same as the more common effects of head injury the problem may be under diagnosed. Examples of symptoms that have common characteristics with the effects of head injury are depression, sexual difficulties (like impotence and altered sex drive), mood swings, fatigue, headaches and visual disturbance. These symptoms can be caused by a change in the level of a particular hormone that is produced by the pituitary gland. Some of these symptoms also present in MHI and CMHI.

A study conducted by Carlson et al., (2009) investigated the association between hormone levels and recovery after TBI. The sample consisted of 43 adult males with moderate to severe TBI who were admitted to an acute rehabilitation unit within six months after sustaining the injury. Each participant had a blood test which measured the levels of the different hormones on

admission to the rehabilitation facility. Their cognitive and physical impairments were measured using the (FIM) Functional Independence Measure (which measures levels of independence on a range of cognitive and physical items). The results confirmed previous findings that indicated that 60% of participants had at least one abnormal hormonal value on admission and that testosterone (in males) was lower than the normal levels of testosterone. It was recommended that a thorough assessment is needed before any diagnoses can be made as there are many possible causes for the symptoms given above. The authors note that another hormonal problem, in the early stages of recovery after head injury, is neurogenic diabetes insipidus (essentially, it mimics diabetes) which is characterised by increased thirst and excessive production of dilute urine. This occurs as a result of the reduction in secretion of a vasopressin hormone and can be treated by administering desmopressin (a manufactured anti-diuretic hormone) and replacing lost fluids.

Diagram 6: The Pituitary gland (Headway, 2012)



3.5.2 Second Impact Syndrome (SIS)

Echemedia and Julian (2001) and Boden et al., (2007) postulate that SIS is uncommon but can arise in any sport that produces consecutive head blows. Heilbronner et al., (2009) report that despite the dramatic effects of the knockout (KO) punch, the cumulative effects of multiple sub-concussive head blows appear to be the primary cause of neurologic injury in boxers (especially professional boxers with extensive fight histories). It is characterised by a massive cerebral oedema that occurs when the injured brain sustains a secondary injury prior to the non-resolution of the first injury, and this condition is often fatal. Cantu and Voy (1995) indicate that this is seen in American Football approximately one to two times per year. This syndrome is considered to result from an abnormal cerebral vascular sensitivity resulting from the first injury. The metabolic dysfunction, vascular congestion and intracranial hypertension can leave the individual symptomatic from the first injury. The catastrophic second impact can be extremely mild, even a blow to the chest, side or back that may cause a whiplash injury to the athleteôs head. When an individual is subsequently re-injured, a rapid and fulminant cerebral oedema with brain herniation occurs. Usually within several minutes, the individual will collapse and the onset of a life-threatening neurological crisis will ensue. The patient will immediately be intubated and given medication to reduce cerebral swelling. Athletic populations are often identified as the primary group affected with SIS due to their often premature return to competition after MTBI. A case study documented that young athletes, most often those of high school age, are susceptible to this type of injury. The disastrous events of SIS have been attributed to cerebrovascular dysregulation, vascular engorgement and herniation of brain tissue (Prins & Hovda, 2003, as cited in Heilbronner et al., 2009). In contrast to the acute effects of SIS, Dementia Pugilistica results from a more chronic exposure to repeated head blows.

3.5.3 Epidemiology of Second Impact Syndrome (SIS)

Second Impact Syndrome seems to be a fairly rare occurrence and most of the work on SIS has been on sports-related head injuries. From 1983-1993 the National Center for Catastrophic Sports Injury Research in the USA, identified 35 possible cases amongst American football players (Cantu, 1986). According to Boden et al., (2007) 71% of high school players suffering head injuries had a previous concussion in the same season, with over a third known to have

been playing with some post-concussive symptomology. SIS has been found mostly in young males engaged in contact sports such as boxing and American Football. The number of published cases that confirmed SIS is quite small. However, the situation in which a young, previously healthy young adult succumbs suddenly (and often dies) indicates that this syndrome must be taken seriously to avoid potentially catastrophic consequences (Zollman, 2011).

3.5.4 Dementia Pugilistica

Dementia Pugilistica is a type of neurodegenerative dementia which affects amateur or professional boxers. Persistent and pervasive deficits resulting from the cumulative effects of multiple head injuries have been described within the context of the syndrome known as Dementia Pugilistica, Chronic Traumatic Encephalopathy and/or Punch Drunk Syndrome. The characteristic symptomatology pertaining to this syndrome includes headaches and problems with dizziness, fatigue and dysarthia (motor speech disorder). Psychosocial and neurocognitive symptom presentations include deficits in memory, attention, concentration and speed of information processing, deficits in judgements, irritability, emotional distress and inability to maintain employment Jordan et al. (1997) and Lezak et al. (2004). In their research Erlanger, Kutner, Barth and Barnes (1999) into post-mortem autopsies of boxers whose clinical histories were_consistent with the syndrome of traumatic encephalopathy, five categories of damage were revealed. These were recorded as: 1) abnormalities of the septum pellucidum; 2) cerebellar abnormalities; 3) cerebral scarring and atrophy; 4) degeneration of specific nuclear groups and 5) the presence of neurofibrillary tangles. Further, a study of randomly selected ex-boxers suggested a 17% prevalence of this syndrome in boxers (Erlanger et al., 2003). Thirty professional boxers aged between 35 ï 76 years underwent a neurologic and behavioural assessment. Amongst the 30 boxers, 11 were found to be normal (according to age related criteria), 12 showed mild cognitive deficits, 4 had moderate cognitive impairment and 3 showed signs of severe cognitive impairments (Jordan et al., 1997). Heilbronner et al., (2009) reported a trend between poorer neuropsychological test scores and past number of fights and rounds fought. They suggested that there might be a latency period for symptoms to show. This is consistent with the theory that a preceding brain injury increases vulnerability to future neuropathology.

3.6 American Football

American Football has been the subject of much scrutiny regarding the incidence of MTBI, as has been noted. Even though a number of researchers have examined the incidence and prevalence rates in American Football, there is still little information that exists on the medium to long term effects of MTBI in players of the game. Jordan (1987) and Barth et al., (1989) were the first to methodically research and evaluate the effects of MTBI in American Football. Their project introduced the innovative technique of obtaining baseline data by testing each athlete before the season commenced and after the season ended. Neuropsychological test instruments used in this investigation included The Trail Making Test, Symbol Digit Modalities Test (SDMT), and the Paced Auditory Serial Addition Test (PASAT). The results suggested that MTBI results in cognitive and information processing deficits which were detectable within 24 hours. They also suggested that swift, yet incomplete recovery, proceeds 5-10 days post-injury, specifically on performance of the Paced Auditory Serial Addition Test (PASAT) and the Symbol Digit Modalities Test (See Appendix E**)**

Collins et al., (1999) examined the effects of concussion on the neuropsychological test performance in a sample of 393 American Football players (college) who were administered a battery of neuropsychological tests. Significant baseline differences were found between those players who reported a previous history of concussion (54%) and those who did not report any such injury (46%). This finding is important because it suggests that players who sustained two or more concussions may have poorer long-term neuro-cognitive consequences than those who do have not suffered more than one concussion. The New York Times (2011) reported a study that was conducted in 2000 from a sample of former and retired National Football League (NFL) players. The study surveyed 1 090 former NFL players and found that around 60% had suffered at least one concussion in their career and 26% suffered three or more concussions. Those who had suffered more concussions reported more problems with memory, concentration, speech impediments, headaches and other neurological problems than those who did not suffer any (or only one concussion). The New York Times (2011) report that in a study conducted by The North Carolina Center for the Study of Retired Athletes in 2007, of the 595 retired NFL players who recalled sustaining 3 or more concussions on the American Football field, 20.2% said that

they also suffered depression (this is three times the rate of players who had not sustained concussion). These investigations support the assumption that athletes who have sustained two or more concussions are likely to have poorer long-term neurocognitive consequences than those who do not. These neurocognitive changes appear not to resolve for most players up to 10 days post-injury but instead they become chronic.

3.7 Boxing

Boxing is a sport where the aim is to knock-out an opponent rendering him or her unconscious. Boxers (See 3.5.2) who suffered several knock-outs and display symptoms like headache, dizziness, poor co-ordination, speech difficulties, resting tremors and even memory difficulties were regularly diagnosed as suffering from Dementia Pugilistica (Erlanger et al., 1999; Jordan, 1987, Lezak, Howieson & Loring, 1995; Ruchinskas et al., 1997). The mechanisms involved in brain injury sustained from boxing are that a punch can cause rotational acceleration of the head, the veins and long axon fibres may be stretched and torn resulting in subdural haematomas or axonal damage. In addition, falling against the ropes may cause impact deceleration and blows to the neck may injure an artery (Haglund & Eriksson, 1993).

All boxers are at risk of progressive consequences of tissue damage resulting from repeated head trauma. Researchers have become aware that the most important factor contributing to the severity and long term consequences of head trauma in boxing is not necessarily the number of knock outs, but rather the subtle and chronic cumulative effect of multiple blows sustained over a period of time. In the 1980ôs, Ross, Casson, Siegel and Cole (1987) found neuropsychological test impairment correlated with the number of professional fights a boxer had. Studies using other neurological examinations also indicated the adverse cognitive effects following punishment to the head in boxing. In this regard, Casson et al. (1984) found that 87% of active and former boxers demonstrated abnormal results on two of four indices utilised in their study using computerised tomography (CT) scans, electroencephalography (EEG) and neurological tests (See Appendix E). A study investigating 23 amateur boxers before and after an amateur

boxing event found verbal and incidental memory was diminished (Butler, 1994). However no matched control group was used in this study.

In contrast to the studies supporting the presence of cognitive difficulties, other researchers have reported null outcomes when examining the neurocognitive effects of amateur boxing. A study of amateur boxers which used tests of information processing, reaction time and learning and memory, found the boxers exhibited no significant differences when compared with non-contact sport controls (Brooks, Kupshik, Wilson, Galbraith & Ward, 1987). The authors further concluded that amateur boxing appears to be well-controlled and thus neurologically safe. Porter (2003) also conducted a study on 20 amateur boxers over a period of 9 years and found no evidence of decreased neurocognitive test performance.

Heilbronner et al. (2009) report that studies investigating the neuropsychological effects of amateur boxing revealed few, possibly negligible, neurocognitive deficits. Moriarity et al., (2004) examined amateur boxers who were participating in a number of bouts during a 7 day tournament and found that, with the exception of those whose bouts were stopped by the referee, there was no evidence of cognitive dysfunction in the immediate post-bout period. It was noted that the majority of studies on amateur boxing indicate that it does not lead to the extent and degree of deficits found in professional boxers. It must be noted that these studies researched amateur boxers who often wear protective head gear and frequently do not have the pressure to have as many fights as professional boxers. This may account for the aforementioned research findings. The small samples used also limit any specific conclusions

3.8 Australian Rules Football

Australian Rules Football is a variant of rugby league and is played with eighteen rather than thirteen players on a bigger field. Although the game is thought to involve less body contact than rugby league; kicking, running and jumping is also involved (Gibbs, 1993). However, the players of Australian football run the risk of sustaining MHI, as they are often involved in interceptions and tackles while the oval ball is either kicked or punched around the field,

frequently causing collisions between players (Cremona-Meteyard & Geffen, 1994; Verrall, Slavotinek & Barnes, 2005).

According to Cremona-Meteyard and Geffen (1994) in research investigating the acute and long-term effects of concussion on professional Australian Rules Footballer players who had sustained MHI (using cued reaction time tasks, at two weeks and one year Post - injury) the results were significant. Although there were no test differences between players and controls during the acute phase, at one year post-injury the MHI group showed a reduced benefit of valid cueing relative to controls. The control group was made up of elite sportsmen with no history of concussion. The research also controlled for, and managed, potentially confounding variables. The authors concluded that their research provides convincing evidence of persistent impairment in the ability to direct visuo-spatial attention following MHI in Australian Rules football. The implication is that players have a reduced ability to act quickly in response to expected spatial events (such as a ball thrown towards a player), which puts them at risk of further head injury or CMHI.

A neuropsychological study investigating whether the effects of MHI were measureable utilised the PASAT, the Digit Symbol Substitution Test and Four-Choice Reaction Time (See Appendix F). The sample consisted of 130 professional players, 10 of whom sustained MHI and were subsequently re-assessed five days post-injury (along with members of the control group). Results suggested that persisting neuropsychological changes in information processing and decision time are detectable at five days post-injury for players who sustained MHI. Although the study focused solely on the effects of single MHI, which precludes any comments on the neuropsychological sequelae of CMHI, the results are still valuable in demonstrating the deleterious effects of MHI sustained as a result of concussion (Maddocks, Saling & Dicker, 1995).

Orchard & Seward (2011) assert that the 19th annual Australian Injury Report for 2010 revealed that the rates of concussion have been low and remained stable over the past decade. At the beginning of 2011, The Medical Officers Association for Australian Rules Football introduced new guidelines for the management of concussion. The guidelines promote a conservative approach to managing concussion whereby a player who is diagnosed with concussion cannot return to the field. The introduction of rules to penalise a player who makes forceful contact with another player, and reduced tolerance for any head-high contact, has contributed to the low rates of concussion reported in the sport. More tightening of these rules occurred before the 2011 season. It is difficult to achieve a full record of concussion as retired players report that on some of the occasions, when they received concussions, they did not report the full extent of symptoms to the team medical staff to prevent being dropped from the team. The consistent low incidence and prevalence for concussion (consistently less than one injury per team per season) is noted over a ten year period, in 2001 concussion incidence was 0.7 and in 2010 was 0.5, concussion prevalence in 2001 was 1.3% and in 2010 it was 0.8%. This may be due to the stringent rules in the sport or under-reporting of concussion because of fear of being dropped from the team for up to 6 weeks.

Greenhow (2011) asserts that concussion will always remain concussion regardless of whether it occurs in Australian Rules Football or Rugby League, or any other contact sport. However, a unified approach to concussion management could send a strong message to all sportsmen and sports administrators that a playerôs health and safety must come first and for that reason commercial considerations should take second place. He further noted that if players are not protected then an increase of concussion-related litigation could occur. The Australian Rules Football governing body has mandated the use of the CogState Sports System by all clubs for baseline measures of player cognition and has adopted the SCAT2 (See Appendix F) for determining if a player has suffered a concussion.

3.9 Rugby League

The game of Rugby League is extremely physical and players need to have speed, stamina, strength and agility. It is faster and the players are lighter than Rugby Union players but there is still a lot of contact. Since the ball can only be carried or kicked down the field, players often collide with each other or tackle each other (the latter being an intrinsic part of the game), which results in players being repeatedly exposed to head injuries. The ball must be kicked down the field or carried forward over the goal line to score a try. Players run the risk of being knocked over backwards and sustaining whiplash when they bang their heads. The forward players are involved in sustained bodily contact throughout the game as they are involved in rucks and reportedly incur more concussive injuries than backline players (Gibbs, 1993; Gissane, Jennings, Cumine, Stephenson & White, 1997; Kaplan, Goodwillie, Strauss & Rosen, 2008; Seward, Orchard, Hazard &, Collinson, 1993).

The most frequently injured body parts in this game according to Gissane et al. (1997) are the head and neck which account for 33% of all injuries. According Seward et al., (1993) incidence studies show that concussion accounts for 5% to 8.5% of all injuries sustained amongst Rugby League players. However, there have been few studies investigating the neurocognitive effects of MHI in the sport. Hinton-Bayre and Geffen and McFarland (1997) examined the hypothesis that an impairment of speed of information processing underlies the poor neuropsychological performance subsequent to MHI in the game. The first phase of the study measured the sensitivity of players on specific neuropsychological tests, the Symbol Digits Modalities Test, the Digit Symbol Substitution Test and the Speed of Comprehension Test (See Appendix F). The second phase of the study used measures of speed of information processing which are sensitive in the post-acute phase subsequent to MHI. Speed of Comprehension was more sensitive to cognitive impairment than the other two tests.

According to the summary review of the New Zealand Rugby Concussion Policy (2010), the New Zealand Rugby League (NZRL) has recognised the potential for concussion or head injuries occurring, which could have potentially devastating effects in Rugby League. As a result of this

it recommended that appropriate consideration be undertaken by everyone who participates, manages or administers the sport at all levels, in both the training and the match environment. The summary review states that there has long been a perception that concussion occurs only when there is loss of consciousness. This perception is incorrect as concussion can arise without loss of consciousness and differs in severity from brief periods of confusion through to a significant loss of consciousness. A policy to ensure that concussion is properly managed and all stakeholders are aware of the potential for on-going deficits was adopted by the NZRL in 2010. The purpose of this policy is to ensure player safety and to reduce the risk of repeated concussion and the development of SIS. They also introduced mandatory assessment of any player who seemed concussed as it is important that a quick and accurate assessment is made when assessing an injured player on the sports field. Appropriate questions and the signs and symptoms of concussion can be found in the side-line concussion checklist provided by the NZRL. All referees, coaches, managers and trainers were advised that they should carry out the following.

Checklist 1: Management of Concussion Card (New Zealand Rugby Concussion Policy, 2010)

Management of Concussion

Acute injury

When a player shows any symptoms or signs of concussion, the following should be applied.

1. The player should not be allowed to return to play in the current game or practice.
2. The player should be left alone, and regular monitoring for deterioration is essential over the initial few hours after injury.
3. The player should be medically evaluated after the injury.
4. Return to play must follow a medically supervised stepwise process.

A player should never return to play while symptomatic.

"When in doubt, sit them out"

The policy of NZRL (New Zealand Rugby Concussion Policy, 2010) makes it mandatory that when concussion is suspected for players over 16 years of age, a 21 day stand down period must be observed according to Checklist 2.

Checklist 2: Return to play protocol from the internationally approved guidelines (New Zealand Rugby Concussion Policy, 2010)

Level	**Activity undertaken**	**Time Post-Concussion (approximate) Guidelines**
1.	No activity, complete rest, once symptom free and cognitive recovery is demonstrated, proceed to level 2.	2-3 days
2.	Light aerobic exercise such as walking or stationary cycling	4-10 days
3.	Sports specific training (e.g. running, drills, ball handling skills)	11-15 days
4.	Non-contact training drills	16-20 days
5.	Full contact training after medical clearance	21 days
6.	game play	21+ days

Those players who are aged 16 or below must observe a 28 day stand down period the same as the adolescent and younger players. This is because the developing brain takes longer to recover from the damage and symptoms of brain injury. Players should be able to progress through each step towards the next level without any symptoms occurring. The player should drop back to the previous symptom free level and try to progress again in the next 42 hours if any post-concussive symptoms occur in accordance with international guidelines (New Zealand Rugby Concussion Policy, 2010).

3.10 Rugby Union

The majority of research on CMHI in contact sport has been carried out on Rugby Union players. Rugby Union is the traditional form of the sport and has thirteen players. It has high contact and is noted as a tough physical game. Even though countries including the United Kingdom (UK), New Zealand, Australia and South Africa play Rugby Union the majority of research on CMHI in the sport has been conducted in South Africa by Shuttleworth-Jordan. According to Shuttleworth-Jordan et al., (1993) her research began with analysis of pre and post-season differences between reportedly non-head injured rugby players and non-contact playing controls. An analysis of repeated test differences between rugby players with MHI and matched controls was also performed. The test battery administered was the Denckla Finger Tapping, Purdue Pegboard, Digit Span, Digit Supraspan and Trail Making Tests (See Appendix F). The pre-season comparison between the rugby players and controls suggested the presence of impairments in working memory, verbal skills, new learning ability and hand motor dexterity in the rugby players, a pattern of deficits typically associated with closed head injury due to the effects of diffuse brain damage. One inconsistent finding with the general trend of these results was a significantly faster Finger Tapping test score in the rugby group compared to the controls. It was postulated that the differences amounts to points of a second and it was considered that this test was not scored rigorously enough to ensure reliable differences with respect to points of a second (fundamentally, the researchers were not accurate enough as this is a pen-paper test reliant on researcher observations). They thus concluded that the direction of the Finger Tapping test (in isolation) had little interpretive validity. Shuttleworth-Edwards and Radoff (2008) investigated the residual effects of concussion among players of Rugby Union from school through to the national adult level, with pre-season testing on tests for visuomotor processing speed (Digit symbol and Trail Making A & B). The comparison group included 124 male rugby players versus 102 non-contact sports controls and, 71 rugby forwards versus 53 backline players. There was equivalence across the groups for age, education, and estimated IQ and hand motor dexterity. The results indicated vulnerability amongst players on visuo-motor processing which was linked to years of exposure to repetitive concussion and sub-concussion injuries. The post-season comparison revealed that the rugby players demonstrated significantly less capacity on many of the tests in contrast with the control group. These findings were attributed to the permanent effects of a MHI sustained in previous rugby seasons, or the recent effects of sub-

concussive or unreported concussive effects during the rugby season in which the testing took place, or combination of both. Also of significance, when the rugby playing group was compared with regard to position of play, the forwards showed greater cognitive impairments than the backline players.

A study by Ancer (2000) investigated the effects of cumulative concussive and sub-concussive MHI on the cognitive functioning of professional rugby players. A comprehensive battery of neuropsychological tests was administered to 26 professional Rugby Union players and a non-contact sport control group of 21 professional cricket players. Within the rugby group, forward and backline players were compared. There was a significant increased variability of scores for the rugby players compared with the cricket players on tests particularly sensitive to cognitive deficit, associated with MHI. This invalidates the null indications of average effects, indicating that a notable proportion of rugby playersô performances were falling off relative to the rest of the rugby players on tests vulnerable to the cognitive effects of diffuse brain damage. Mean score comparisons within the rugby group indicated that it was the sub-group of forward players in particular, whose test performances revealed deficits suggestive of cerebral damage. Deficits were specifically found in working memory, visuo-perceptual tracking, verbal memory and visual memory, a pattern of deficits commensurate with CMHI.

Gardner, Shores and Bachelor (2010) conducted a study that sought to examine possible detrimental cognitive effects in a sample of adult male Rugby Union players, (n=34) who reported a history of 3 or more concussions. These were compared with rugby players who reported no previous concussion, (n=39). A neuropsychological test battery and a traditional neuropsychology measure of processing speed were administered. The results revealed that there were differences between groups on two processing measures for both sets of tests. Players with a history of multiple concussions performed systematically lower on these measures than those with no history of concussion. These results provide further evidence to suggest that a history of three or more self-reported concussion in active Rugby Union players may have a detrimental effect on cognitive function.

Ackermann's (2000) study was part of on-going research at Rhodes University (South Africa) that investigated the cognitive effects of CMHI in Rugby Union players which focussed specifically on high school rugby players. Comprehensive neuropsychological tests were administered to a sample of 47 high school rugby players and 34 non-contact schoolboy hockey playing controls. The results revealed no significant relationship between the number of reported MHIôs and cognitive performance.

A smaller study conducted by Nel (2009), on high school rugby players, using a computerised test (CALCAP) did not support the results of prior research which have mostly used standard neurological test batteries (pen-paper tests). This body of research indicates that concussive injury and CMHI results in cognitive deficits in schoolboy Rugby Union players, particularly the forwards (Shuttleworth-Jordan et al., 1993). Even with the relatively small sample of 32 players, over the short schoolboy rugby season, it would be anticipated that the CALCAP would yield results that would support this assumption. Seventeen of the experimental participants showed no effects as compared to the hockey playing controls. There was also no difference between the performance of the forward and backline players. It was concluded that it may be that repeated concussive injuries in adolescent boys do not have a cumulative effect as previously postulated, factors such as education and age may mitigate against this. However, the sample was small and it was recommended that a larger study be undertaken.

3.11 Football

Whilst there have been a number of studies with regard to boxing and rugby, football related sports research into CMHI remains sparse. Football is a sport where MHI frequently occurs and can have serious outcomes. Although historically it was a designated non-contact sport contemporary literature refers to it as a contact sport (Giannotti et al., 2010; Kolodziej, Koblitz, Nimsky & Hellwig, 2011). Evidence indicates that concussion in football often goes unrecognized and undiagnosed and therefore the players donôt usually seek medical attention (Al-Kashmiri & Delaney, 2006). Football players may sustain CMHI through head - to - head contact, head to goal-post contact, head - to- ground contact and head-to-ball contact. The potential for cumulative neurological and cognitive consequences of heading the ball have been the focus of increased interest in the field of cognitive deficits incurred in sport. Tysvaer and

Storli (1989) report that a ball kicked with half an individualôs power can travel between 22 kilometres and 83 kilometres per hour. A strike to the head (headed ball) could occur with a force of 116 kilometres per hour. This impact is increased to 200 kilometres per hour when a ball is kicked at full power. Naunheim, Standeven, Richter and Lewis (2000) demonstrated that among high school soccer (football is referred to as soccer in the USA) players, peak accelerations (measured on the surface of the head) were 160-180% greater from heading the ball in soccer than from routine, no injurious contacts in American Football or Ice Hockey.

Reilly (1997) estimated that approximately 5 heading opportunities exist per team member in any given football match. This frequency of head blows, plus additional exposure to blows outside of the game play (practice), lead to concerns regarding the acute and chronic effects of heading related to CMHI. This is underpinned by studies in the eighties by Tysvaer and Storli (1989) who utilised an electroencephalography (EEG - See Appendix E), a test that measures and records the electrical activity of the brain by using sensors (electrodes) attached to the head and hooked by wires to a computer. The authors did this to evaluate neurologic functioning in former soccer players. They found that 12 of the 37 players evaluated had either slightly abnormal or abnormal EEG results as compared to 4 of the 37 controls (who had never played soccer).

A sample of Norwegian football players was evaluated for concussive symptomology and reported symptoms of headaches, neck pain, dizziness, irritability, insomnia, and weakened memory after repeated heading of balls (Tysvaer & Storli, 1989). Another study by Barnes, Cooper, Kirkendall, McDermott, Jordan and Garrett (1998) surveyed male and female football players who competed in the 1993 Olympics. These participants were asked to report on the frequency of heading the ball, previous head injury, and symptoms experienced either after incurring a concussion or after heading the ball. Participants reported increased neurocognitive symptoms after heading the ball as compared to normal play. Reported symptoms and frequencies in men and women included headaches 54% amongst males and 55% amongst females. Males who felt dazed after repeated heading of the ball were 31% of the samples while 49% of the female sample felt dazed after repeated heading of the ball. Eighteen percent (18%) of the male sample reported decreased concentration after repeated heading of the ball while

39% of the female sample experienced this. This supports the findings of Kross, Ohler and Barolin (1983) who reported that 2 out of 10 football players had abnormal EEGôs after 15 minutes of header training.

A study was conducted on 31 football players and a control group of 31 tennis players and the results indicated possible evidence of poorer information processing in football players (Abreau et al., 1990). Furthermore, Meecham and Bachur (2009) researched the cognitive consequences of heading the ball in adult football players in Norway who had started playing football in the junior leagues. Eighty one percent (81%) of the sample who were tested showed mild to severe deficits in attention, concentration and memory. They also reported that players who headed the ball more frequently during football matches had the highest rate of cognitive deficits.

Putukian, Echemendia and Mackin (2000) found no significant deficits in female and male college soccer players in America after a twenty minutes heading drill on the following measures (See Appendix F). The Continuous Performance test (Vigil), Stroop Colourï word Test, Alphabet Backwards and The Trail Making Test (TMT). They found an increase in reported headaches amongst those players who headed the ball more frequently but no neurological impairments were indicated in the test results.

A study conducted by Echemendia and Julian (2001) found that there were significant differences in impaired performance on planning and memory function in amateur soccer players in the USA. Significant differences were found when soccer players were compared to athlete controls (swimmers and long distance runners) on the Complex Figure Test, the Wisconsin Card Sorting Test and the Wechsler Memory Scales (See Appendix F). The number of concussions sustained was inversely related to neuropsychological performance. Their findings however, should be interpreted cautiously because the soccer players were found to have consumed alcohol at a rate that was significantly higher than that of the non-soccer playing controls.

Kirkendall and Garrett (2001) state that it is difficult to blame purposeful heading of the football for reported cognitive deficits when actual heading exposure and details of the nature of head-ball impact are unknown. They report that concussions are a common head injury in football (mostly from head to head or head to ground impact) and this is more likely a factor in any

cognitive deficits. These results were reported after a meta-analysis of literature regarding the effect of heading a ball amongst football players.

3.12 Under-reporting or non-recognition of MHI

According to Bailes and Hudson (2001), it is acknowledged that athletes under-report MHI and CMHI for a number of reasons. These include fear of being withheld from competition and motivation to participate in sport which is related to unwillingness to let the team down. Another factor is that under-reporting is frequently due to athletes not recognising that a concussion has occurred and not realising that concussion is an injury severe enough to deserve medical attention (Cunningham, 2007; Iverson, Gaetz, Lovell & Collins, 2004; Shuttleworth-Edwards et al., 2008).

3.13 Concussion management

Lovell (2008) asserts that a concussed athlete should firstly receive appropriate care undertaken during the initial on-field evaluation (See example, checklist 2). No sporting event should take place without trained medical or paramedical personnel. The first priority is to evaluate the athleteôs level of consciousness (LOC), ensure he or she has open airways and that breathing is not obstructed and circulation to any affected area is not obstructed. The medical staff attending to the concussed athlete should be properly prepared with an emergency action plan for the evacuation of a critically head or a neck-injured athlete. All the medical, management, coaching and support staff should be familiar with the plan, each member must be given a role to play that is well defined in advance of any game-play.

Lovell (2008) states that even though LOC is not common in contact sport players, confusion and amnesia are common sequelae to head and body contact injuries. Confusion (disorientation) represents impaired awareness and orientation to surroundings. It often manifests in athletes who are described as appearing stunned, confused, or glassy-eyed on the side-lines. Teammates are usually the first to notice a confused athlete who is in difficulties and often, are the first to inform

the coach. The presence of concussion can be assessed properly by asking simple orientation questions such as (name, current stadium, opposing team, current month and day). In the diagnosis and management of concussed athletes, a careful evaluation of amnesia is of particular importance as amnesia may be associated with loss of memory for events preceding (retrograde) or after injury (post-traumatic). To assess on-field retrograde amnesia properly athletes must be asked questions pertaining to details occurring just before the trauma that caused the injury.

Table 5: University of Pittsburgh side-line mental status testing card on field cognitive testing procedure (2004)

Orientation-ask the athlete the following questions:

- What stadium is this?
- What city is this?
- Who is the opposing team?
- What month is this?
- What day is it?

Posttraumatic amnesia-ask athlete the following words:

- Girl, dog, green.

Retrograde amnesia-ask athlete the following words:

- What happened in the prior quarter or half?
- What do you remember just before the hit?
- What was the score of the game before the hit?
- Do you remember the hit?

Concentration-ask athlete the following words:

- Repeat the days of the week backwards, starting with today
- Repeat these numbers backwards: 63, 419

Word list memory

- Ask athlete to repeat the three words from earlier (girl, dog, green)

3.13.1 Modifying factors in concussion management

McCrory et al., (2009) reported that a number of factors may influence the investigation and management of concussion, and in some of the cases they may predict the potential for long-term or persistent symptomology. Important modifiers to consider in a detailed concussion history are outlined in table 5. In the context of professional sport there may be additional management considerations beyond simple return to play advice. Additional investigations utilising neuropsychological testing, neuroimaging and balance assessment may be necessary. A multidisciplinary management team, co-ordinated by a medical doctor with specific expertise in sports medicine should always be available to appropriately modify any interventions. The female gender is a modifying factor which must be taken into consideration, but as yet it has not been added to concussion management regimes due to lack of research and ambiguous research findings. However, it is generally accepted that females may be more at risk for injury and/or the severity of MHI due to their inherent physiology.

3.13.2 The significance of loss of consciousness (LOC)

The duration of LOC is an acknowledged predictor outcome in the overall management of moderate to severe TBI. The description of LOC is associated with specific cognitive deficits in published findings of concussion but it has not been noted as a measurement of severity of injury. It has been determined that prolonged (over 1 minute duration) LOC would be considered a factor that may modify concussion management of any brain injury (McCrory et al., 2009***).***

3.13.3 The significance of amnesia and other factors

Evidence in published research suggests that the duration, nature and length of post-concussive symptoms could be more important than the presence or duration of amnesia alone. It should also be noted that that retrograde amnesia differs, with the time measurement post-injury, and thus is a poor reflection of severity of injury (McCrory et al., 2009).

3.13.4 Depression

McCrory et al., (2009) state that depression has been reported as a long-term consequence of TBI, including sports related MHI and concussive injuries generally. A depressed mood following concussion may reflect an underlying pathophysiological abnormality consistent with a limbic-frontal model of depression as suggested by neuroimaging studies. It could also be a result of hormonal imbalances as a result of the brain injury.

Table 6: Concussion modifiers (McCrory et al., 2009)

Factors	**Modifiers**
Symptoms	Number Duration (> 10 days) severity
Signs	Prolonged loss of consciousness (>1 Min), amnesia
Sequelae	Concussive convulsions
Temporal	Frequency- repeated concussions over time Timing-injuries close together in time ñRecencyò-recent concussion or traumatic brain injury
Threshold	Repeated concussions occurring with progressively less impact force or slower recovery after each successive concussion
Age	Child and adolescent (< 18 years old)
Co- and pre-morbidities	Migraine, depression or other mental health disorders, attention deficit hyperactivity disorder, learning disabilities, sleep disorders
Medication	Psychoactive drugs, anticoagulants
Behaviour	Dangerous style of play
Sport	High risk activity, contact and collision sport, high sporting level

Tommasone and McLeod (2006) also report that comprehensive concussion management protocols have been advocated in a number of consensus statements. It is important to emphasise to the athlete, while still symptomatic following an injury, that physical and cognitive rest is required during the period of recovery. This implies that activities that require concentration and attention (for instance, scholastic work, videogames and even text messaging) may exacerbate symptoms and possibly delay recovery. If the recovery programme is designed by professionals and is followed properly no further intervention is required and the athlete will typically resume sporting activities without further problems. If the athlete continues to train, and take part in other high risk activities, while still symptomatic, the risk of further or re-injury, and resultant depression, is significant (McCrory et al., 2009).

3.14 Return to play

There are different considerations concerning the return of athletes who have had a brain injury. The following must be taken into account.

3.14.1 Rehabilitation after brain injury

According to Headway (2012) it must be noted that brain cells do not regenerate like cells in other parts of the body when they are destroyed although recent evidence suggests some, but not full regeneration occurs in these cells. This does not imply that an individual who suffers injury to the brain and therefore to the brain cells cannot recover. The brain, is to some extent flexible, and is able to reorganise itself to an extent in order to regain lost function. This process is known as brain plasticity. During the recovery process the injured part of the brain cannot do the tasks it usually does so other areas of the brain take over those activities and new nerve pathways can be established using undamaged brain cells. The aims of rehabilitation are to help the brain learn different ways of working (wearing a skull cap when playing contact sport, for instance) in order to reduce the long-term impact of any (but particularly severe) brain injuries.

3.14.2 Recovery and rehabilitation after brain injury

Shuttleworth-Edwards et al., (2008) compared the seasonal concussion incidence of school, university, club and provincial level Rugby Union in South Africa, between 2002 and 2006. They used concussion management programmes which utilised computerised neuropsychological assessments. Of 1366 rugby players who received baseline testing, 175 concussive episodes were reported for 165 rugby players. There was a wide disparity in the manner in which the concussion follow ups were managed by the different organisations. With broadly comparable groups, tighter control was associated with a comparatively higher concussion management incidence for athletes per rugby playing season, with average incidence figures ranging from 4% to 14% at school level and 3% to 23% at adult level. Tighter control and management meant that Rugby Union players were better monitored and less at risk of returning to play too soon and running the risk of further or more serious injury (for instance, SIS).

McCrory et al., (2009) updated recommendations for the management of concussive injury in sports. They noted that the cornerstone of concussion management is physical and cognitive rest. This means that until symptoms have resolved athletes must be introduced to a graded programme of exertion (preceding medical clearance and return to play). A number of factors that require sophisticated management strategies may modify the recovery period and outcomes of any injury. However, the majority of injuries will recover spontaneously over several days. McCrory et al., (2009) state that in these situations, it is wise that athletes proceed progressively through a stepwise return to play.

Lovell (2008) reports that concussion may occur without direct trauma to the head and that concussed athletes are only occasionally rendered unconscious. Athletes may be unaware that they are injured and may not show any obvious immediate signs or symptoms of injury, such as motor incoordination, gross confusion or amnesia. In re-assessing the common signs and symptoms of concussion, it is necessary to understand that an athlete may only have a few signs or symptoms of injury or a group of symptoms. Regarding the frequency of post - concussion

signs and symptoms, the most commonly reported symptom is headaches which occur in approximately 70% of athletes who are concussed. Even though it is true that musculoskeletal headaches and other pre-existing headache syndromes may complicate the assessment of post-concussion headache, any headache presented after a blow to the head or body should be conservatively managed. A concussion is most frequently described as a sensation of pressure in the skull which is most often localised to the frontotemporal regions of the head. It might not develop immediately after injury but may develop over time.

It is not possible to estimate the length of time for recovery and outcome of any brain injury however, tables indicating the usual timescales for recovery present the normal or average time most individuals need for recovery. Brain injury, like any other injury, is also reliant on factors that affect the individual both psychologically, psychosocially and physically which must be taken into account by physicians and coaches involved in the management of the concussed athlete (McCrory et al., 2009). According to Headway (2012) The British Society of Rehabilitation Medicine (BSRM) has produced rehabilitation guidelines after acquired brain injury. These guidelines recognise the important role family members play in the rehabilitation process of brain injury. This suggests that athletes who suffer concussive injury need support both on and off the field as they may be prone to depression, stress and anxiety about their ability to compete, particularly in an elite (professional) sporting environment.

Table 7: Graduated return-to-play protocol (McCrory et al., 2009)

Number of stages	**Rehabilitation stage**	**Functional exercise at each stage of rehabilitation**	**Objective of each stage of protocol**
1.	No activity	Complete physical and cognitive rest	Recovery
2.	Light aerobic	Walking, swimming or stationary cycling keeping intensity <70% miles per hour. No resistance training	Increase Heart Rate
3.	Sport-specific exercise	Running drills in soccer. No head impact exercises	Add movement
4.	Non-contact training drills	Progression to more complex training e.g. passing drills in football. May start progressive resistance training	Exercise, co-ordination and cognitive load
5.	Full contact practice	Following medical clearance participate in normal training	Restore confidence and assessment of functional skills by coaching staff
6.	Return to play	Normal game play	

McCrory et al., (2009) state that with the above stepwise progression programme, an athlete will continue to proceed to the next level if asymptomatic at each of the levels. Each step usually takes about 24 hours. This means that an athlete takes approximately one week to proceed through the full rehabilitation protocol once they are asymptomatic. If any post-concussion symptoms occur whilst the athlete is in the stepwise programme he or she should go back to the previous asymptomatic level. The athlete should try to progress again after a further 24 hour period of rest has passed. Cantu (2010) reports that a major league soccer star in America, one of the youngest to reach a 100 goal plateau, had his dream of playing in the 2010 Soccer World Cup de-railed. This occurred, when after being diagnosed with PCS (after a ninth concussion), he returned to play prematurely. He was unable to play in the world cup and, due to his injuries, had to stop playing soccer altogether.

Patricios, Collins, Branfield, Roberts and Kohler (2012), of the Sports Concussion Institute of South Africa, report that over the last decade sports concussion research and clinical guidelines have evolved exponentially. The sports assessment tool (SCAT-1 & 2, see Appendix F), that originated from the Prague consensus meeting held in 2004, was enlarged on at the Zurich consensus meeting in 2008 to provide clinicians with the SCAT2 (See chart 3, as a clinical template for the assessment of acute concussion). The SCAT2 was designed as a practical assessment tool specifically for clinical evaluation and of the concussed athlete. The South African sports physicians institute have put its use into practice at international, provincial, club, school and recreational levels in both team and individual sports between 2009 and 2011. Important changes in concussion categorisation are an indication that concussion management guidelines are a work in progress. Other groups have suggested modifications to the clinical concussion evaluation protocol and more recent modifications to the tool have been developed. Patricios et al., (2012) assert that the SCAT2 represent the best attempt at converting the principles of the international concussion consensus meetings into a practical clinical tool which will be revaluated in December 2012. Another tool developed by the South African Sports Physicians Institute the SCOAT (See Appendix F) represents the relevant aspects of clinical care and is, regarded by some pundits, as more practical.

Checklist 3: The SCAT Card (Patricios et al., (2012)

The SCAT Card (Management of Sport Concussion Assessment) Athlete Information **What is concussion**? **How do you feel**: You should score yourself on the following symptoms based on how you feel now. Post-Concussion symptom Scale Headache Pressure in the head Neck pain Balance problems or dizzy Nausea or vomiting Hearing problems/ ringing Donôt feel right Feeling ñdingedò or ñdazedò Confusion Feeling like ñin a fogò Drowsiness

Fatigue or low energy
More emotional than usual
Irritability
Difficulty concentrating
Difficulty remembering
(**Follow up symptoms only**)
Sadness
Nervous or Anxious
Trouble falling asleep
Sleeping more than usual.
Sensitive to noise
Other:
What should I do?
Signs to watch out for:
What can I expect?

Medical Evaluation

Name: Date:
Sport/ Team

1.SIGNS

Loss of consciousness?
Seizure or convulsive activity?
Balance problems/ unsteadiness?
Pulse
BP

2. MEMORY

Modified Maddocks questionnaire

3. SYMPTOM SCORE

Total number of positive symptoms

4. COGNITIVE ASSESSMENT

5 word recall
Digit backwards

5.NEUROLOGICAL SCREENING

Speech
Eye motion and pupils
Balance test
Gait Assessment
Any neurologic screening abnormality necessitates formal Neurologic or Hospital assessment

6. RETURN TO PLAY

Athletes should not return to play on the same day of injury. When returning to play, athletes should follow the stepwise symptom limited program, with stages of progression.

A common cause of stress after a MHI is that athletes worry about the symptoms they have. Eight out of ten patients with a mild brain injury show some symptoms during the first week to a month after the accident. These symptoms are part of the normal recovery process and are not

signs of permanent damage or medical complications. For a trouble free recovery, athletes who have suffered any kind of head injury on the field of play should be advised of this. The majority of patients with MHI recover completely in a week to three months. However, if the athlete is older than 40, or is in his or her late thirties, and has suffered CMHI it may take longer to return to normal. It is important to remember to impress on the injured athlete that many of these symptoms particularly, tiredness and headaches, occur in a non-head injured groups (Hanks et al., 2008).

3.14.3 The role of pre-participation concussion evaluation

McCrory et al., (2009) note that a detailed concussion history is of value as it will pre-identify athletes that fit into the high risk category for concussive injury. This also provides an opportunity for the healthcare provider to educate athletes regarding the significance of concussive injury. A structured concussion history should include specific questions as to previous symptoms of a concussion and not just the perceived number of past concussions. It is worth noting that dependence on others to recall concussive injuries for instance, teammates or coaches, has been demonstrated to be unreliable. A clinical history should also include information about all previous head injuries, face or cervical spine injuries and any co-existent concussive injuries that may be missed unless specifically asked about. Questions pertaining to disproportionate impact versus symptom severity may alert the clinician to an athleteôs progressively increasing vulnerability to injury (CMHI). The benefit of a comprehensive pre-participation concussion evaluation allows for modification and optimization of protective behaviour (for instance, wearing headgear in rugby matches and practices) and an opportunity for educating the athlete on the issues relating to PCS and CMHI.

3.15 The child and adolescent athlete

A brief description of concussion in the child and adolescent athletes, as discussed at the International Concussion Conference in Geneva (McCrory et al., 2009), is given as many athletes today are very young when they start their professional careers, and many enter training programmes in their early teens. For instance, Manchester United Football Club which is in England and is one of the so-called glamour clubs of football has always developed young

athletes. In recent years (from the 1990ôs) the franchise developed the Manchester United Soccer School which takes in youngsters in their early teens. The objective is to develop home-grown talent (from the United Kingdom) so that chosen adolescents have a chance to develop optimal football skills. The coaching is carried out in programmes developed according to the developmental age of the participants. Although these adolescents attend school, the focus of the programme is to develop football players not academics, which is underpinned by the way the school is advertised, *Live, Train, and Play the United Way* (Manchester United Website, 2012). This means that many youngsters who incur MHI do not have one of the protective factors (the level and years of education) that help protect against any lasting cognitive damage (Shuttleworth-Jordan, 1999).

McCrory et al., (2009) report that the management recommendations for concussion made by the International Concussion Conference (McCrory et al., 2009), can also be applied to children and adolescents after the age of ten. Children below the age of ten report different concussion symptoms from adults and need age appropriate symptom checklists as a part of their assessment. When a child or adolescent athlete with concussion is assessed, the health professional usually needs to include the coach and the parent as well as the teacher and patient him or herself, to gain the correct details. The neuropsychological testing of children is, to a large extent, the same as for the adult. However, the timing and testing may differ in order to assist planning in the school and home management care whilst the patient is still symptomatic. When children are cognitively tested, the test must be developmentally sensitive because of their on-going cognitive development during this period. It is strongly recommend that children should not return to practice or play until completely clinically symptom free, which might require longer time frames than adults. Because of the different physiological responses, longer recovery after concussion and specific risks (for example, diffuse cerebral swelling) related to head impact during childhood and adolescence, a more conservative return to play approach is recommended.

3.16 Summary

The chapter discussed at some length the theoretical context and indications for this study. There are approximately 70% to 90% of treated MHIôs and many of them occur as a result of sports related injuries. However, because of habitual under-reporting it is difficult to determine how common the condition is. The epidemiology and pathophysiology of MHI in sport was discussed. The neurological effects which increased susceptibility to SIS and Dementia Pugilistica are also highlighted. Different contact-sports where athletes incur MHI were also discussed namely, American Football, Boxing, Australian Football, Rugby League, Rugby Union and Football. Recommendations on how to manage concussion in athletes and their return-to-play was stated as outlined by the 3rd International conference on concussion in sports.

CHAPTER 4: RESEARCH METHODOLOGY

4.1 Introduction

This chapter focuses on the methodological procedures which were used in the study. The study is a quasi-experimental survey design (multiple measures = pre and post ï testing with different tools). Participantsô exclusion and inclusion criteria will be discussed first, sample characteristics are described, including age, education and number of prior concussions. The research procedure and research materials used, including demographic questionnaires, the CALCAP (California Computerised Assessment Package) a computerised neurocognitive measure (Miller (1993a), the Rivermead Post-Concussion Symptom Checklist (King et al., 1995) are discussed and then the data processing and analysis procedures.

As indicated in the literature review and theoretical framework a number of sport-related MTBI studies, including football studies, have methodological limitations which must be considered in the present research findings. It appears pertinent, therefore, to provide an account of possible methodological limitations that need to be taken into account for this study. These are discussed in Methodological Limitations in Chapter 6 (See 6.5.2).

4.2 Selection Criteria

- Football participants (experimental group)

A purposive sample of football players was invited to participate in this study. The professional football club is based at the University of Pretoria (AmaTuks) in Gauteng, South Africa (n=33). All football players were post-school sportsmen who were playing for the First Division Football League. In 2012 the team was promoted to the Premier Soccer League (PSL).

- Control group (volleyball players)

Initially a non-contact sport regional hockey team was approached to participate in the study as a control group. However, in October 2010, the management of the regional hockey team indicated that because of their practice and match schedule they would be unable to participate. The management and coach of non-professional volleyball playing team (at the University of Limpopo (Medunsa Campus) were approached to participate in this study and a purposive

sample of twenty two males participated in the research (n=22). As volleyball is a designated non-contact sport, where dangerous bodily contact seldom occurs, it was deemed appropriate to use volleyball players as a control group when the initial participants withdrew from the study.

4.2.1 Inclusion criteria

Inclusion criteria included all football and volleyball players in the sample who met the criteria for the study and who did not report any of the exclusion criteria.

4.2.2 Exclusion criteria

To establish which participants should be excluded from the study, information was elicited from a pencil and paper questionnaire that each participant had to complete. It elicited comprehensive historical and current information with regard to each participantôs health, education, sport and concussion history (see Appendix A). Any participants with a history of the following were excluded from the study.

- **History of neurological disorder**

This included seizures, weakness in limbs and tremors. All football players or control participants were excluded on these grounds. However, none reported any history of this type of neurological disorders.

- **History of alcohol or substance abuse**

This included a diagnosed history of alcohol or substance abuse. No football player or control participants were excluded on these grounds as none reported a history of alcohol or substance abuse.

- **Current psychiatric disorder**

This included depression, anxiety, and sleep disorder. No football player or control participants were excluded on these grounds as none of the participants reported any current psychiatric disorder.

- **History of recent concussions**

This included any participant who had suffered any non-penetrative or traumatic force to the brain resulting in an alteration of consciousness, which included LOC for a period of less than thirty minutes, or Post Traumatic Amnesia (PTA) of less than 24 hours was excluded from the study. One control participant (n=1) who reported a history of LOC (as noted above) from an MVA, more than ten years ago was included in the study. He was included because he had recovered from his injury and was not experiencing any post-concussion symptoms at present.

- **History of moderate to severe Traumatic Brain Injury(TBI)**

This included moderate to severe TBI that a football or control participant had sustained at any time in the past, and constituted a TBI incident that resulted in hospitalisation and was defined as a non-penetrative (or penetrative) traumatic force to the brain, with a LOC exceeding thirty minutes.

- **Test-taking issues**

Potential confounding test-taking issues included the potentially confounding criteria of extreme tiredness (veisalgia) any symptoms resulting from excessive alcohol consumption that might confound the results. Five football players were excluded at the end of the season (n=5) and also three control group volleyball participants were excluded at the end of the season (n=3) for this reason.

- **Additional miscellaneous exclusions**

Additional miscellaneous exclusions included the fact that seven football players withdrew from the research due to injury (n=7) and two control participants also withdrew from the research (n=2). Six football players did not return for end of season testing (n=6) and two control participants did not return for end of season testing (n=2). This, to some degree may have confounded the study results as the final sample was smaller than anticipated (See Table 7).

Table 8: Exclusions from the experimental Football and Volleyball control groups

	Football	Control Group
	Original Pool=33	Original Pool=22
Exclusion Categories	Final Pool= 15	Final Pool= 15
Neurological Disorder	0	0
Psychiatric Disorder	0	0
Alcohol and substance abuse	0	0
Moderate to severe TBI	0	0
Test taking issues	5	3
Absent from post -.test (end of season testing)	6	2
Miscellaneous exclusions	7	2

4.2.3 Final sample

In total, the final sample for analysis of the experimental group (Football players) was fifteen participants (n = 15). The final sample of the control group (Volleyball players) consisted of fifteen participants (n = 15). The total sample was made up of thirty participants (n = 30).

4.2.4 Sample characteristics

Football players and control groups were compared on common demographic variables that might affect neurocognitive test performance, namely age, educational level, race and language.

4.2.5 Age, language, education and race

The age of each participant was documented in years. The educational level of each participant was calculated in years according to the number of grades successfully completed at school (12 years being assumed as the minimum) because all participants were enrolled at a tertiary institution) either for a degree, diploma or certificate course. Shuttleworth-Edwards et al. (2004) state that researchers should be aware that, in addition to level of education, quality of education also has an effect on neurocognitive test performance. They found that the differences in neurocognitive test performance were minimised between White English first language and

Black African second language English speakers when they are exposed to a relatively advantaged quality of education. Language bias was controlled in terms of the ability of the sample to speak and understand English as the participants are students and/or team members (with 12 years of schooling) at the University of Pretoria or the University of Limpopo (Medunsa Campus). At these tertiary institutions the language used in team management and team coaching for football and volleyball is English. The football playing sample consisted of one White participant and fourteen Black participants. While all participants were fluent in English, there were Tswana, Pedi, Venda, Zulu and Xhosa first language speakers. The white participantôs first language was Portuguese and English his second language. The volleyball control group, were all Black African, and spoke the same selection of languages as the football group. However, as all participantsô had 12 years of schooling, and were fluent in English, the questionnaires and instructions were administered in English

4.3 Data administration

The research team consisted of the researcher, promoter and one assistant. Due to time constraints associated with the assessment of a professional football team, the service of one assistant researcher was needed to assess the University of Pretoriaôs professional football team. The assistant had previously used CALCAP and was trained to administer the test and questionnaires under the supervision of the research promoter. Inter-rater reliability was maintained by the promoter training both the researcher and the assistant researcher in how to administer the CALCAP and other questionnaires.

4.3.1 Pre-season (Baseline) testing

The football players were assessed at a room at University of Pretoriaôs High Performance Centre and the control group at the University for Limpopo (Medunsa campus), Psychology Department in the research room. Players were assessed individually. However, three laptop computers were loaded with the CALCAP programme in case of any computer malfunction. The room in which the CALCAP was administered was cool, there was no noise, the lights were dimmed and there were no outside stimuli to distract the participants. Football and volleyball

control participants were first required to sign written consent forms. Pre-season assessment took longer, as the participants had to first complete two questionnaires, the neurological symptom check list and the Post-Concussion Symptoms checklist (See Appendix C for copies of the consent forms and questionnaires). The participants were then assessed on CALCAP.

The back of the computer monitor was slightly elevated to reduce glare from any overhead lights. Identification numbers and demographic information of the control and experimental groups were entered. Instructions to the participants were given by the researcher. Each participant was asked to sit at the computer and comfortably position himself to see the screen and was also told how to use the space bar for all responses on the CALCAP. The baseline testing took approximately 45 minutes for each participant.

At the time of the baseline testing the questionnaires and CALCAP were administered in the following order. First to be administered was the demographic questionnaire. The demographic questionnaire was completed with all participants, on the occasion of baseline testing, to provide the researcher with the required demographic information and the different exclusion criteria. This questionnaire comprised of biographical details, educational history, medical history including history of any neurological disorders, sports related injuries, alcohol, substance or nicotine abuse, any current psychiatric disorder or previous psychiatric disorder and/or any history of recent concussion or history of TBI. The PCS checklist was the second to be filled in and the CALCAP was the last test to be administered. Participantsô performances were automatically recorded by and the computer which produced a report in seconds. The report included the mean and median reaction times and total numbers of true and false positive responses. Participants found the computerised tasks stimulating, non-threatening and many reported that they enjoyed the experience.

The football playersô pre-season testing took place at the beginning August 2010 before their season started. The volleyball controls were tested at the beginning of the year January, 2011 before the volleyball season started. It took approximately two weeks to complete testing for both the experimental and control groups, thus four weeks in all.

4.3.2 Post-season testing

Participants had to first complete the PCS checklist questionnaire and were then assessed on the CALCAP. Post-season testing for the football players took place in May 2011, after the football season had ended. The volleyball controls were tested at the end of the year, November 2011 when they had finished playing league matches. The average time between baseline and post-season test for both football the football group and volleyball controls was eleven months. The same venues and same data administration procedures were used for post-season testing. The same researchers tested the groups again to obviate administration bias. As the demographic questionnaire did not have to be filled in again testing time was shorter, approximately 25 minutes per participant.

4.4 Instruments used in the study

The instrument, CALCAP (California Computerised Assessment Package) was used to assess specific cognitive deficits. CALCAP was developed by Eric Miller (1990). It was updated and validated in 1990, 1991, 1992 and 1993. CALCAP measures cognitive functions such as attention and reaction time. This measure was used to assess slowed cognitive function, focused and divided attention, sustained attention and rapid visual scanning. It was ideal for longitudinal assessment of cognitive changes due to disease, medication and cognitive rehabilitation. The CALCAP test battery was used to study changes in reaction time and speed of information processing in multiple sclerosis, hyperbaric nitrogen narcotics, HIV infection, dementia, drug abuse and (TBI). Findings at present suggested that the CALCAP was a practical and inexpensive screening tool for detecting early cognitive decline. Preliminary data suggested that the CALCAP would eventually prove to be more sensitive than conventional neuropsychological procedures (pen and paper tests) for detecting cognitive changes over time (Miller, 1993a). The abbreviated CALCAP was used in the study because the test battery was ideal for collecting reliable information on reaction (psychomotor) functioning in a brief period of time, and can be used effectively for assessing change over time. The CALCAP programme accurately measured and recorded the responses to the stimuli, including the range, mean, median, z-score, percentile of reaction time and the total numbers of true and false positive responses. It also fitted with the

purpose of this study as it was sensitive to diffuse brain injury. The Abbreviated CALCAP Test Battery includes the following as outlined by Miller (1993a).

- Simple Reaction Time. Participants are asked to press a key as soon as they see anything at all on the screen. This procedure provides a basal measure of reaction time.

- Choice Reaction Time for Single Digits. Participants were asked to press a key as soon as they saw a specific number such as ó7ô; otherwise they were to do nothing. This procedure added a simple element of memory to the task.

- Serial Pattern Matching #1-Sequential Reaction Time #1. Participants were asked to press a key only when they saw two of the same numbers in sequence, for example, if they saw the number ó3ô followed by a second occurrence of the numberõ3ô. This procedure added a more complex element of memory (and focused attention) since the subject had to keep in mind the last number that was seen.

- Serial Pattern Matching #2 - Sequential Reaction Time #2. Participants were asked to press a key only when they saw two numbers in sequence (increasing order). For example, if they see the number ó3ô followed by the number ó4ô, the number ó6ô followed by the number ó7ô and so on. This measured the same cognitive functions as above.

4.4.1 Validity and Reliability of The California Computerised Assessment Package (CALCAP)

- **Validity**

The CALCAP has repeatedly showed to have discriminated cognitively impaired index cases from matched controls, as well or better than conventional neuropsychological tests. These findings have been established both cross-sectionally (Miller, 2001; Worth, Savage, Baer & Esty, 1993) and longitudinally (Miller, 1992). These results have demonstrated the sensitivity of reaction time measures for perceiving changes in motor functioning and supported the use of

reaction time procedures for assessment and monitoring of symptoms of dementia and other cognitive slowing (Miller, 1993a).

- **Internal consistency reliability of CALCAP**

The CALCAP Reaction Time measures have very high internal consistency reliability (.77-.96), thereby indicating that the constructs measured are assessed in a uniform manner across the multiple trials of each reaction time (Miller, 1995).

- **Test-retest reliability**

The choice reaction time measures show 6-month test-retest reliability (.43-.68) that is equivalent to that seen in conventional neuropsychological procedures (.47-.77). In general, the simple reaction time measures had low test-retest reliability (.20-.29), but had very high internal consistency reliability (.77-.95), suggesting that the psychomotor skills measured by the simple reaction time tasks differ considerably depending on state variables such as mood, attention, fatigue and time of day. This hypothesis is also supported by the modest inter-correlations observed between the first, second and third iterations of the simple reaction time task (.41-.68) during the standard CALCAP test battery (Miller, 1995).

- **Inter-subtest correlations**

Multiple iterations of the simple reaction time task, administered at four separate times during the standard CALCAP procedures, correlate from .41 to .68 with each other. Choice reaction time measures correlates from .31 to .60. According to Miller (1995) Form Discrimination shows the lowest inter-correlations with the other choice reaction time measures. Inter-correlations between simple and choice reaction time are very small (from .11 to .29).

- **CALCAP'S correlation with conventional neuropsychological tests**

Inter-correlations of reaction time measures with conventional neuropsychological procedures are small (.02 to .37). The conventional procedures that correlate most highly with reaction time are Symbol Digit Substitution (.19 to .37), Verbal Fluency (.13 to .25), and Trail-Making, Part B (.17 to .18). Surprisingly, the Grooved Pegboard, a relatively pure motor measure, had negligible correlations with the reaction time tasks (.07 to .18). A factor analysis of the measures showed

independent clustering of the computerised and conventional neuropsychological measures. Simple reaction time measure and choice reaction time measures form distinct factors (Miller, 2001; Miller, 1995).

- **Validity studies**

The CALCAP has been shown repeatedly to discriminate cognitively impaired index cases from matched controlsô as well as, or better than, conventional neuropsychological tests. These findings have been established both cross-sectionally (Miller, 2001; Miller, Satz, Van Gorp, Visscher & Dudley, 1989; Worth et al., 1993) and longitudinally (Miller, 1992; Miller et al., 1989). These data demonstrate the sensitivity of reaction time measures for detecting changes in motor functioning and support the use of reaction time procedures for assessment and monitoring of symptoms of dementia and other reasons for cognitive slowing.

4.4.2 Additional questionnaires

The first questionnaire, the demographic questionnaire was adapted from a non-copyright version developed by Rhodes University Psychology Clinic (2000). This questionnaire has been used and adapted by researchers into CMHI for both PhD theses and Masters Dissertations since 1993, thus was deemed fit-for the purpose for the present study. It was used to collect information on educational qualifications, occupational history and sport playing history, previous head injuries and exclusion type criteria (See Appendix A). In order to assess the frequency of residual PCS suffered by players, a second questionnaire was also administered (King et al., 1995).

The first questionnaire consists of two parts, part A and part B. Part A of the questionnaire elicited demographic, and historical information about all the participants in the study. (i) Biographical details like name, educational history, date of birth and age; ii) medical history, including any history of neurological disorder, alcohol or substance abuse; iii) any current psychiatric disorder, history of recent concussion(s), including TBI; iv) psychological history including history of depression, anxiety and Attention Deficit/ Hyperactivity Disorder (ADHD and v) recreational history including present use of alcohol and nicotine (smoking). Part B was

only completed by football players who self-reported PCS from earlier injuries both on the field of play and non-sports related PCS.

The second questionnaire consists of 31 items on the neurological/neuropsychological Post-Concussion Symptom Checklist (Hereto referred to as the Post-Concussion Checklist or PCS) that participants rated on a scale ranging from *never, sometimes* or *often* (See Appendix B). This questionnaire, the Rivermead Post Concussion Symptoms Checklist, was adapted from a non-copyright version developed by King et al., (1995) and has been used in on-going head injury studies, mostly by Rhodes University, Grahamstown, South Africa (Shuttleworth-Edwards, Border, Reid & Radloff, 2004).

4.4.2.1 Reliability and validity of the Rivermead Post Concussion Symptoms Checklist Questionnaire (RPQ)

According to King et al., (1995) the questionnaire consisted of 31 questions derived from published material and its reliability was investigated. The questionnaire reliability was investigated under two experimental conditions. The first study examined its test-retest reliability when used as a self-report questionnaire at seven to ten days after injury. Forty one (41) head injured patients completed a RPQ (questionnaire) at seven to ten days following their head injury and again approximately twenty four hours later. The second study examined the questionnaire inter-rater reliability when used as a measure administered by an investigator at six months after injury. A second investigator re-administered the questionnaire approximately seven days later. Spearman rank correlation coefficients were calculated for ratings and total symptoms scores and for individual items. High reliability was found for the total PCS scores, under these experimental conditions (King et al., 1995**)**.

4.4.2.2 Threats to internal validity of the study

The following threats to the internal validity of the study are noted.

- **History**

According to Cook and Campbell (1979) this type of threat occurs when an observed effect for the football players (and volleyball) players might be due to the different events that take place between pre-season testing (baseline) and the post-season testing (end of season), when this event was not the treatment of research interest. As the demographic questionnaire was not re-administered, it is possible that for instance, a player might have been involved in an MVA in the interval between pre season and post season testing.

- **Maturation**

Although the gap between pre-and post-season testing was relatively short, this type of threat can be a threat when an observed effect was due to the participantôs growing older, wiser, or more experienced between pre-season testing and post-season testing and when maturation was not the treatment of the research interest. For instance, participants may have stopped drinking alcohol due to becoming older and wiser (or conversely, started taking some form of substance to enhance their on-field performance due to pressure from peers or coaches). This type of threat occurs when an observed effect, due to maturation, is not in the interests of the research (Cook & Campbell, 1979).

- **Mortality**

This type of threat occurred when six participants from the experimental group dropped out by post-season testing. This resulted in a selection artefact since the experimental group composed of fewer participants at post-season testing (Cook & Campbell, 1979).

- **Interaction with selection**

The above mentioned threats to internal validity act together with selection to produce forces that might have falsely appeared as treatment effects. Amongst this type of threat is selection-maturation, and selection-history. Selection-maturation occurs where participants amongst the football group (and volleyball controls) mature at different speeds. Selection-history (local

history) was related to the sample participantsô different environmental contexts, which is noted as their unique local history that may have had an impact on the research outcomes (Cook & Campbell, 1979).

4.5 Data Analysis

The collected data were analysed using the following methods. For the CALCAP, a two sample t-test was used in this cross-sectional study to compare the means of football players and the volleyball control group at baseline (pre) and post season assessments. The t-test was interpreted as exploratory and used experimentally (Schoeman, 2011). Analyses of variance (ANOVA) was performed on the group means for the football sample and the volleyball controls at post-season (end of season) testing. The ANOVA is a statistical model which makes specific assumptions about errors, specifically that they are random variables which are ñnormally and independently distributed with a mean of zero and constant variance, It should be noted that pre-test scores do not enter into the model (Cook & Campbell, 1979, p. 151)ò. An analysis of covariance (ANCOVA) is an extension of the ANOVA, which includes the pre-test means as co-variance. ñIn this way it provides an adjustment for the initial differences between groups even in a non-equivalent group design (Cook & Campbell, 1979 p. 153).ò The ANCOVA was performed between the football and volleyball playing control group. The ANCOVA used the pre-test outcome scores which were compared to the post-test scores. This helped with regard to comparing participants at entry level (baseline testing) to ascertain equivalence between the groups on the variables of age and educational levels (Cook & Campbell, 1979).

The Fisher's Exact Test (1954) is a statistical test used to determine if there are non-random associations between two categorical variables. It is used as a statistical significance, test which is normally used for small samples. It was employed to compare the percentages of the PCS checklist for the experimental and control groups. It was expected that the PCS symptoms of the football playing control group would shift from pre-season testing to post-season testing. Therefore, it was expected that predominantly ñneverò response would be given during the (Baseline) beginning of the season testing. It was expected that this would occur in the volleyball controls as well. If, in the experimental group (Football players) a shift towards ñsometimesò or

ñoftenò was noted the implication was that PCS symptomology had worsened. However, there was no expectation of this type of shift in the control group. A two sample t-test analysis (Schoeman, 2011) compared the group means between the football sample and volleyball controls at baseline and end of season testing on four CALCAP neurocognitive composite scores.

4.5.1 Significance level

Hypothesis testing was conducted to calculate the probability that the sample data could have occurred under the assumption that the null hypothesis is true. Due to sampling problems, data was never in complete agreement with the null hypothesis and thus the probability (p-value) of data was evaluated. This p-value or attained significance level was the criterion used for rejecting the null hypothesis, as it was the smallest value of alpha for which the null hypothesis can be rejected. If the null hypothesis was rejected, when it actually should not be rejected, it was concluded that the difference between the means exist when it does not exist which is a Type I error. To help avoid such errors, a cut-off value, termed Alpha or level of significance, was used against which the p-value was evaluated. The smaller the Alpha value the less likely the risk of falsely rejecting the null hypothesis. Most commonly used is the 5% level of significance (Ŭ =.05). The 5% level of significance can be considered to be too lenient, thus the 1% level of significance (Ŭ=.01) can be used which indicates that there is a 1% probability that the observed differences in mean scores could have occurred by chance. A Type II error occurs where the null hypothesis is not rejected, when it actually should have been rejected. The 5% level of significance was thus considered appropriate to use in this study.

4.6 Hypotheses

Pre-season testing and post-season testing was conducted with the objective of analysing the effects of Cumulative Mild Head Injuries (CMHI) and Post-Concussive Symptomatology (PCS) amongst football players with non-contact sport control participants, as a result of CMHI and concussive injury sustained during the footballôs first division league season. As it is understood that CMHI or concussive injury may not be reported on purpose by football players or go unrecognised it was expected that some CMHI or concussion would go unreported. It was expected that the football playersô outcomes would be different (in a negative manner) to the controls participants at pre-pre-season (or baseline) testing. It was further expected that the

outcome for the football players would be more evident at post- season testing than at baseline testing due to them sustaining head injuries or PCS during the season. In view of the indications from empirical research reviewed above and Satzôs Brain Reserve Capacity theory (1993), the following hypotheses were generated. Generally, it was hypothesised that on a measure that are sensitive to diffuse head injuries the overall performance of the football players (as reflected in mean scores) would be significantly slower than that of the volleyball controls due to long term CMHI (concussive injuries). The following hypotheses were generated from the reviewed literature and theoretical framework used by the study.

Neurocognitive measures:

- football players who sustained Cumulative Mild Head Injuries (CMHI or concussion) would be slower in simple reaction time tasks of the California Computerised Assessment Package or CALCAP) than the volleyball control group;

- football players who sustained CMHI (or concussion) would take significantly longer to respond, on Choice Reaction Time for Single Digits (Task II of the CALCAP) than the volleyball control group;

- football players who sustained CMHI (or concussion) would take more time processing information on Serial Pattern Matching 1 (Sequential Reaction Time 1 ï task III of CALCAP) than the volleyball control group;

- football players who have sustained CMHI (or concussion) would take more time processing information on Serial Pattern Matching 2 (Sequential Reaction Time 2 ï task IV of CALCAP) than the volleyball control group.

PCS Symptom Measure:

- football players who have sustained CMHI (or concussion) would experience a higher frequency rating of symptom change on the PCS check list than the volleyball control group in terms of the frequency and intensity of symptom count.

4.7 Ethical considerations

Approval for this research was obtained from the University of Limpopo (Medunsa campus) Ethics committee, the management of the University of Pretoriaôs football club and the management of the UL (Medunsa campus) Volleyball club. Participation in this study was voluntary and participants were free to withdraw at any time. In introductory meetings the nature and the purpose of the assessment was explained to the participants. Participants were verbally briefed about the research and each participant was provided with a document that outlined the statement concerning the participation in the research (Appendix C) before they gave written and informed consent. If any problems were reported that might have interfered with either the experimental or control groups ability to perform was highlighted permission was asked (from the participant) to discuss this with the team manager or coach. If any participant reported problems either physical or psychological they were referred to appropriate professionals, this was also the case if the participant did not want to reveal any physical, psychological or psychosocial problems to management or coaching staff. However, it was explained to the both the experimental and control group that the research was entirely confidential, unless any condition reported by an individual (medical, psychological or psychosocial) was considered by the researcher (in consultation with the supervisor) to be a danger to self or others.

4.8 Summary

This chapter dealt with methodological (research) procedures used in this study. Participantsô consent, selection criteria for football and control groups were highlighted. Exclusion and inclusion criteria, sample characteristics and biographical details were briefly discussed. Research procedures, measures and their administration, instruments used in the study, California Computerised Assessment Package (Miller, 1993a), a self-report questionnaire and the Rivermead neurological/neuropsychological PCS checklist (King et al., 1995) were described. Each of the measuresô validity, reliability, and threats to internal validity were discussed. Data analysis and the studyôs hypotheses were outlined. Lastly, ethical considerations were discussed.

CHAPTER 5: ANALYSIS OF RESULTS

5.1 Introduction

The football players and the volleyball control groupsô results are presented in this chapter. The demographic results are presented first followed by t-test results, comparisons of the group meansô, and then the ANOVA and ANCOVA results are discussed. The chapter will also highlight the significant results and trends of each statistical analysis. The results will also be presented in the form of tables, graphs and figures at the end of each subsection.

5.2 Demographic characteristics of the sample

5.2.1 Age

The results for the age distribution means for the football and the volleyball control participants reveal no significant difference (See table 9). The football playersô age mean is = 22.07 years and the control group controlsô age mean is 21.80 years (p= 0.786).

Table 9: Age distribution for football and the control group

Team	N	Mean (years)	Median	Std Dev	Minimum	Maximum
Total Football Group	15	22.07	22.00	2.31	19.00	29.00
Total volleyball Controlsô	15	21.80	21.00	2.98	18.00	27.00

5.2.2 Education

The distribution of the number of education years for both football players and the volleyball control groups are shown in tables 10 and 11. The education distribution for both football and the control groups were compared, the results revealed that the volleyball control group had significantly higher years of education than the football players (p = 0.002).

Analysis conducted on the demographic data consequently revealed that there is a significant difference between the football participants and control group with respect to education (p = 0.002) and no significant difference with regard to age (p = 0.786). The result indicates that the football players and the control group are not equal with regard to minimum educational qualifications. This may influence the studyôs results in terms of specific risk factors, in this case, lower education-levels, which according to Shuttleworth-Jordan (1999) may lower brain capacity (threshold) and increase an individualôs vulnerability to functional impairment (cognitive deficits) over-time.

Table 10: Education distribution for football playing group

Education(years) Football group	**Frequency**	**Percentage %**	**Cumulative Frequency**	**Cumulative Percentage %**
9	1	6.67	1	6.67
11	2	13.33	3	20.00
12	7	46.67	10	66.67
13	3	20.00	13	86.67
14	1	6.67	14	93.33
16	1	6.67	15	100.00

Table 11: Education distribution for the volleyball playing control group

Education(years)	Frequency	Percentage %	Cumulative Frequency	Cumulative Percent %
13	3	20.00	3	20.00
14	7	46.67	10	66.67
15	2	13.33	12	80.00
16	2	13.33	14	93.33
17	1	6.67	15	100.00

5.2.3 Pre-season (Baseline) testing: Two sample t-test comparison on CALCAP

Two sample t-test comparisons of the two group means between the football group and the volleyball control group on the CALCAP, neurocognitive measures at baseline testing (See Table 12). There is no significant difference at baseline testing for both the football and volleyball controls. Furthermore, there is no overall trend towards changes on the CALCAP, neurocognitive measures at baseline in both groups.

Table 12: Pre-Season two sample t-test mean scores comparison on CALCAP for the football groupandthevolleyballcontrols(markedeffectsignificantifpÒ0.05)

	Soccer (n=15)		**Controls (n=15)**			
	Pre-Season (Baseline)		**Pre-Season (Baseline)**			
	Mean	**SD**	**Mean**	**SD**	**t-test**	**p-value**
Simple RT	386.3	(±101.7)	335.5	(±68.9)	1.60	0.120
Choice RT	444.2	(±41.0)	446.9	(±36.9)	-0.19	0.849
Sequential RT 1	562.7	(±98.0)	541.5	(±86.3)	0.63	0.536
Sequential RT 2	565.7	(±104.3)	642.1	(±121.2)	-1.85	0.075

5.2.4 Post-season assessment: Two sample t-test comparison for the experimental and control groups on CALCAP

Two sample t-tests comparisons of the two group means between the football group and the volleyball control group on the overall CALCAP, neurocognitive measures at post-season (end of season) testing (see Table 13). There is a significant difference between the football and volleyball control group only at Sequential RT 1 measure, (p Ò 0.05) at the end of the season. However, there is no significant difference in the other three measures, Simple, Choice and sequential RT 2. There is also no overall trend of one group having changed more than the other group in all four measures of the CALCAP, even though there is a small difference on the Sequential RT 1 measure at the end of season.

Table 13: Post-season two sample t-test mean scores comparison on CALCAP for the football groupandthevolleyballcontrolgroup(markedeffectsignificantifpÒ0.05)

	Soccer (n=15)		**Controls (n=15)**			
	Post-Season		**Post-Season**			
	Mean	**SD**	**Mean**	**SD**	**t-test**	**p-value**
Simple RT	342.0	(±42.1)	343.2	(±58.4)	-0.06	0.949
Choice RT	445.8	(±66.5)	459.3	(±52.0)	-0.62	0.541
Sequential RT 1	542.7	(±76.6)	486.0	(±63.1)	2.21	0.035*
Sequential RT 2	553.4	(±91.6)	601.0	(±92.2)	-1.42	0.167

Note: *denotes statistical significance

5.2.5 Pre and post-season assessment: Two sample t-test comparison on CALCAP

Two sample t-tests comparisons of the two group means between the football group and the volleyball control group on CALCAP, neurocognitive measures at baseline and end of season reveal that there is no significant difference (See Table 14), except on Sequential RT 1 (p value = 0.3830).

Table 14: Pre-season versus post-season comparison of means for the football and control groups on the CALCAP using a two sample t-test(markedeffectsignificantifpÒ0.05)

Test	Football			Volleyball			t-test	p-value
	n	mean	SD	n	mean	SD		
Simple RT	15	7.7333	68.5549	15	-44.333	103.2	-1.63	0.1149
Choice RT	15	12.3333	53.4879	15	1.6000	82.1921	-0.42	0.6749
Sequential RT 1	15	-55.5333	110.0	15	-19.9333	110.0	0.89	* 0.3830
Sequential RT 2	15	-41.1333	87.5197	15	-12.2667	111.9	0.79	0.4380

Note: * denotes statistical significance

5.2.6 Repeated measure analysis of variance (ANOVA) for post-test mean scores for the football and volleyball groups on CALCAP

Repeated measure analyses of variance (ANOVA) for the post test mean scores for both football and the control groups on the CALCAP neurocognitive measure (Table 15) revealed only a small difference on the Sequential RT 1 (p value = 0.351). No other significant difference was revealed in the other three CALCAP measures, Simple RT, Choice RT, and Sequential RT 2.

Table 15: Post-season CALCAP repeated measure analysis of variance (ANOVA) for the football players and volleyball controls (marked effectsignificantifpÒ0.05)

Test	Football			Control			p-value
	n	post mean	Std Dev	n	post mean	Std Dev	
Simple RT	15	343.20	58.41	15	342.00	42.14	0.9490
Choice RT	15	459..27	51.97	15	445.80	66.46	0.5414
Sequential RT 1	15	486.00	486.00	15	542.73	542.73	0.0351 *
Sequential RT 2	15	601.00	601.00	15	553.40	553.40	0.1670

Note: * denotes statistical significance

5.2.7 Analysis of Covariance (ANCOVA) with a single covariance comparison between the football and volleyball control groups

The ANCOVA is an extension of the ANOVA with the inclusion of the pre-test means as a covariance to provide adjustment for initial differences between the groups. The ANCOVA is a general linear model which blends ANOVA and regression. It looks at whether population means of a dependent variable are equal across levels of a categorical variable, while statistically controlling for the effects of other continuous variables that are not of prime interest. The ANCOVA analyses does not reveal any significant differences in the three CALCAP neurocognitive measures, Simple RT, Choice RT and Sequential 2 RT except for the Sequential RT 1 (p value = 0.0445), which is significantly different (See table 16).

Table 16: Analysis of covariance (ANCOVA) between football players and the volleyball control group (markedeffectsignificantifpÒ0.05)

Test Variables	Football					Control					p-value
	n	Post mean	Std Dev	Pre mean	Std Dev	n	Post mean	Std Dev	Pre mean	Std Dev	
Simple RT	15	343.20	58.41	335.47	68.87	15	342.00	42.14	386.33	101.69	0.6219
Choice RT	15	459.27	51.97	446.93	36.89	15	445.80	66.46	444.20	40.97	0.5557
Sequential RT 1	15	486.00	63.13	541.53	86.30	15	542.73	76.56	562.67	98.04	0.0445*
Sequential RT 2	15	601.00	92.20	642.13	121.17	15	553.40	91.58	565.67	104.29	0.6435

Note: * denotes statistical significance

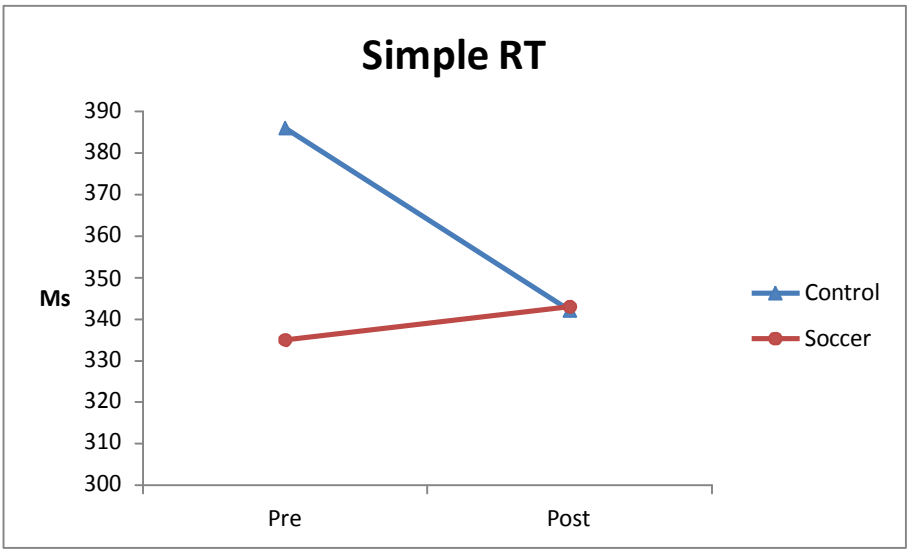
5.2.8 Summary of ANOVA and ANCOVA results on CALCAP

After the two sample t-test analysis, the repeated measures ANOVA was introduced to calculate the post-season test outcomes of the football and control groups on the CALCAPôs four neurocognitive measures (Table 15). The ANOVA did not reveal any significant difference for the three neurocognitive measures, Simple RT (p value = 0.9490), Choice RT (p = 0.5414) and Sequential 2 RT (p = 0.1670) except for one measure, Sequential 1 RT (p = 0.0351), whose results revealed a significant difference. Subsequently an ANCOVA was introduced by adding pre-season test outcomes as a covariant. The football players and controlsô post-season test and pre-season test outcomes were compared. Again the results yielded the same outcome as the ANOVA (Table 15) and the two sample t-tests (Table 13), revealing no significant difference for Simple RT (p = 0.6219), Choice RT (p = 0.5557) and Sequential 2 RT (p = 0.6435) except for Sequential 1 RT (p = 0.0445) which revealed a significant difference.

5.2.9 Figure representation of the mean reaction times of the football and volleyball groups

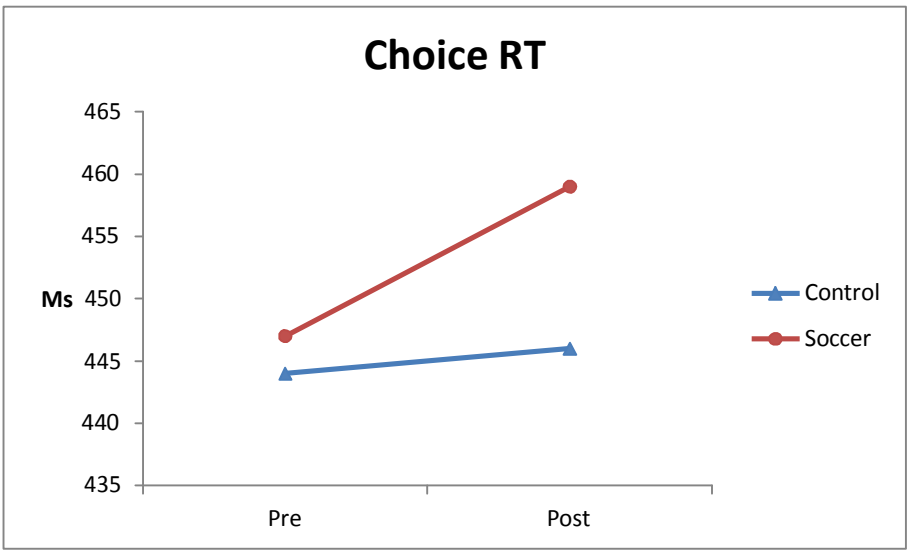
A graphical representation of the mean reaction times for the experimental football and volleyball control groups on Simple Reaction Time, Choice Reaction Time and Sequential Reaction Times 1 and 2 of CALCAP are provided to illustrate the trend of the relationships between the football playing group and the volleyball controls.

Figure1: Pre-Test mean scores on Simple RT measure on CALCAP for football and the controlsô



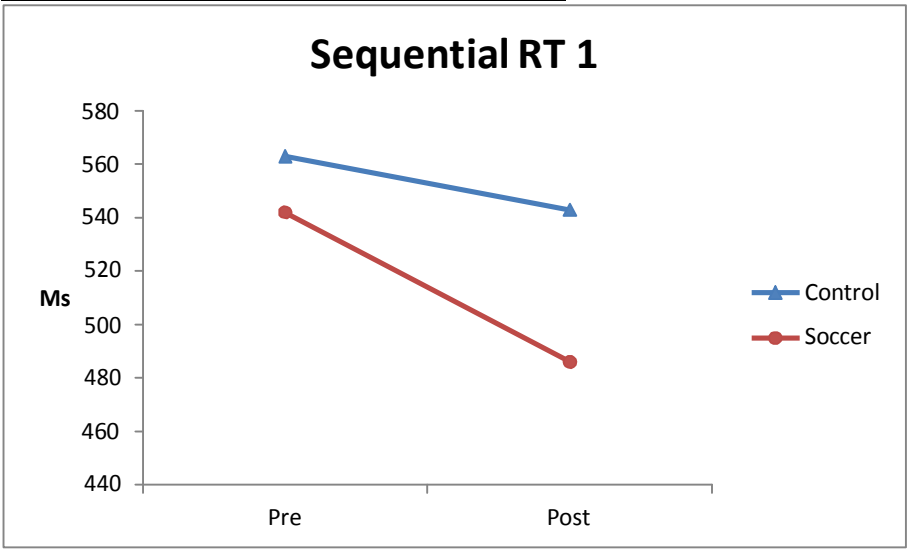
At pre-season testing, volleyball control participants had a higher mean reaction time than the football participants on Simple Reaction time. At the end of the season both the football and the volleyball control group did not differ. The Simple RT provides a basal measure of reaction time and indicates an outcome where the pre-season superiority of the control group is diminished and the football groupsô scores are slightly increased by post-season testing (see Figure 1). There could be a pattern of selection maturation that may have been a threat, as a result of the football players being more tired/ fatigued, due to the rigorous football league season and commensurate training than the control group. However, the First Simple Reaction time task can be considered as practice trials (Miller, 1993a). This is because although each test has a practice component many participants scores do not stabilize until after the first task. This means that interpretations of the difference between the control and experimental group should not be considered noteworthy.

Figure 2: Pre-Test mean scores on Choice RT measure on CALCAP for football and the controlsô



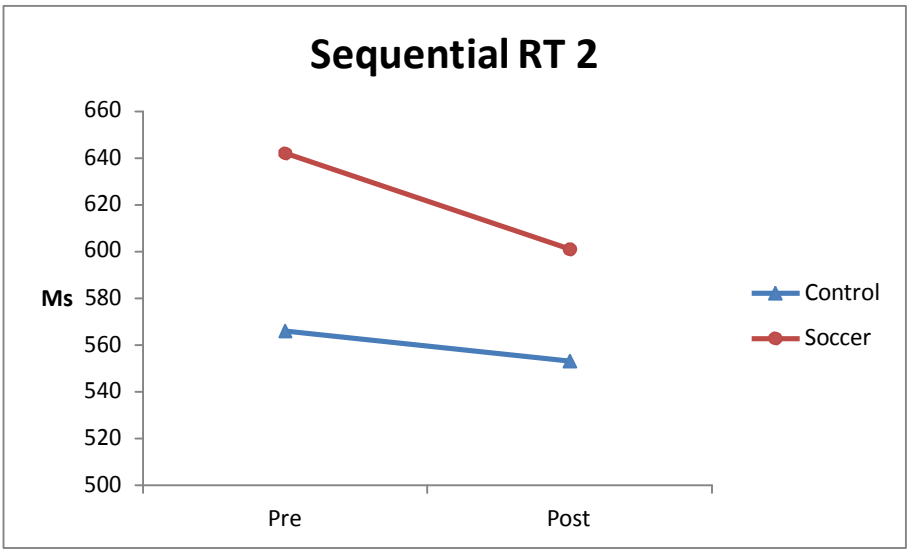
The Choice Reaction time, figure 2 above, depicts the results that reveal larger post-season testing than pre-season testing differences between football players and the volleyball control participants. This difference, it is postulated, is not due to treatment effects but due to selection maturation which is masquerading as treatment effect. According to Miller (1993a) one should consider both the first simple and choice reaction time tests to be practice trials (unless very significant results appear). He notes that although each test has a practice component most scores do not stabilize until after the first tasks.

Figure 3: Pre-Test mean scores on Serial Pattern Matching 1 (Sequential Reaction Time 1) measureonCALCAPforfootballandthecontrolsô



Sequential RT 1ôs figure above indicates a significant difference where the footballôs pre-season test superiority is diminished by post-season testing. The volleyball controlôs results have also slightly diminished in post-season testing (see Figure 3 above). The overall results reveal a statistical significant difference between the experimental football playing group and the volleyball playing control group. Serial Pattern Matching 1 (Sequential Reaction Time 1) is a more complex memory task, and it may be indicative of the experimental group, experiencing attentional difficulties as a result of MHI, specifically a more functional deficit in the area of fluctuating attention (through repeated heading of the football), as CALCAP is very sensitive to diffuse brain damage (Waterloo, Ingebrigstein & Romner, 1997).

Figure 4: Pre-Test mean scores on Serial Pattern Matching 2 (Sequential Reaction Time 2) measureonCALCAPforfootballandthecontrolsô



Sequential RT 2ôs figure also indicates an outcome where the football playersô pre-season testing has a higher reaction time than the control group by the post-season testing (see Figure 4 above). The football players at pre-season testing had a higher Sequential Reaction time 2 than the control group. At post-season testing, the football playersô reaction time diminished, implying that there was no treatment effect. The control group reveals that there was almost no change as reaction time was only slightly diminished. This outcome could be attributed to football players being tired after experiencing fatigue due to the rigorous physical sport/ league matches at post-season testing, thus masquerading as treatment effect, whereas it is selection maturation or mortality. Since Sequential Reaction Time 2 is a more complex element of memory than Sequential Reaction Time 1. There is a possibility that this could be an indication of MHI due to repeated on-field collisions and heading of the ball, suggestive of more specific deficits, specifically in the area of fluctuating attention. This, because CALCAP results seem to be more sensitive for detecting abnormal neurocognitive signs after an MHI than the more conventional measures of testing (Waterloo et al., 1997).

5.3 Post-Concussive Symptom checklist comparisons for the experimental football group and volleyball controls

Cross tabulation comparisons of the summary ratings of symptoms frequencies of the football and the volleyball control group on the PCS checklist measures at pre and post season were performed (Tables 17 & 18). The football and the control group were also compared on frequency ratings of *sometimes, often* or *never* on the PCS checklist (Tables 21 and 22).

The tables 23, 24 and 25 below depict a comparison of rating of symptoms frequencies of the football and control participants at pre-season and post-season testing. An exact test that is appropriate for computing percentages of small counts was used for cross tabulations to establish the distribution of the symptom frequency rating for ñ*worse"*, ñ*no change"*, and ñ*improved"* counts. The ñ*worse count"* percentages (6% and 9.3%) for the football group and the control group (Table 23), do not differ significantly (p = 0.62). The total number of frequencies for the football players is 453 and the control group is 460. Football players had 12 ambiguous frequencies and the volleyball controls had 5. Table 24 depicts the ñ*unchanged"* symptom counts percentages used to compare the football and the controlsô percentages (92.7% and 73% respectively. The two percentages differ significantly with (p=<0.001) for the football group. Table 25 depicts the *"improved"* symptom counts percentages used to compare the football and the controlsô percentages (1.3% and 17.6%) respectively. The two improved counts percentages also differed significantly with (p=0.001).

It was predicted that football players who had sustained CMHI or concussion would experience a higher frequency (more) symptoms on the PCS checklist than the control group. Further, it was predicted that the football group would experience a higher intensity (change) of symptoms on the PCS checklist than the control participants. The results are presented for both pre and post-season symptoms checklists (See Tables 21 and 22) and are then cross tabulated to indicate the distribution of the symptom frequencies (See Tables 17-18).

Table 17: Summary of pre and post-assessments of Post-Concussion Symptoms: experimental group

Symptoms	Pre-assessment	Post-assessment	Frequency	Percent %	Cumulative Frequency	Cumulative Percentage %
Headaches	Never	Never	4	26.67	4	26.67
	Never	Sometimes	1	6.67	5	33.33
	Sometimes	Sometimes	10	66.67	15	100.00
Poor eyesight	Never	Never	13	86.67	13	86.67
	Often	Never	1	6.67	14	93.33
	Sometimes	Sometimes	1	6.67	15	100.00
Hearing difficulty	Never	Never	13	86.67	13	86.67
	Sometimes	Sometimes	2	13.33`	15	100.00
Weakness in	Never	Never	12	80.00	12	80.00
limbs	Sometimes	Sometimes	3	20.00	15	100.00
Clumsiness	Never	Never	11	78.57	11	78.57
	Sometimes	* Often	1	.	.	.
	Sometimes	Sometimes	1	7.14	12	85.71
	Sometimes		2	14.29	14	100.00
Fits/ seizures	Never	Never	13	86.67	13	86.67
	Sometimes	Sometimes	2	13.33	15	100.00
Dizziness	Never	Never	8	53.33	8	53.33
	Never	Sometimes	1	6.67	9	60.00
	Sometimes	Often	3	20.00	12	80.00
	Sometimes	Sometimes	3	20.00	15	100.00
Easily tired	Never	Never	7	50.00	7	50.00
	Never	Sometimes	1	7.14	8	57.14
	Often	Often	1	7.14	9	64.29
	Sometimes	*	1	.	.	.
	Sometimes	Sometimes	5	35.71	14	100.00
Sensitivity to	never	*	1	.	.	.
noise	Never	Never	7	53.85	7	53.85
	Sometimes	* Never	1	.	.	.
	Sometimes	Often	1	7.69	8	61.54
	Sometimes	Sometimes	1	7.69	9	69.23
	Sometimes		4	30.77	13	100.00
Seeing/hearing	Never	Never	10	66.67	10	66.67
Feeling unusual	Never	Sometimes	1	6.67	11	73.33
things	Sometimes	Sometimes	4	26.67	15	100.00
Sexual problems	Never	Never	12	80.00	12	80.00

	Never	Sometimes	1	6.67	13	86.67
	Sometimes	Often	1	6.67	14	93.33
	sometimes	Sometimes	1	6.67	15	100.00
Speech problems	Never	Never	11	73.33	11	73.33
	Sometimes	Sometimes	4	26,67	15	100.00
Stumble over	Never	Never	9	60.00	9	60.00
words	Often	Often	1	6.67	10	66.67
	Sometimes	Never	1	6.67	11	73.33
	Sometimes	Sometimes	4	26.67	15	100.00
Stutter/ stammer	Never	Never	14	93.33	14	93.33
	Sometimes	Sometimes	1	6.67	15	100.00
Slur of words	Never	*	1	.	.	.
	Never	Never	11	78.57	11	78.57
	Never	Sometimes	1	7.14	12	85.71
	Sometimes	Sometimes	2	14.29	14	100.00
Memory	Never	Never	10	66.67	10	66.67
difficulties	Never	Sometimes	1	6.67	11	73.33
	Sometimes	Never	1	6.67	12	80.00
	sometimes	Sometimes	3	20.00	15	100.00
Attention and	Never	Never	6	42.86	6	42.86
concentration	Never	Sometimes	1	7.14	7	50.00
problems	Sometimes	*	1	.	.	.
	Sometimes	Sometimes	7	50.00	14	100.00
Attention	Never	*	2	.	.	.
wondering when	Never	Never	4	30.77	4	30.77
conversing or	Never	Sometimes	2	15.38	6	46.15
watching TV/	Often	Often	1	7.69	7	53.85
reading	Sometimes	Sometimes	6	46.15	13	100.00
	*	.	.	.	.	.
Impatient	Never	Never	9	60.00	9	60.00
	Never	Sometimes	1	6.67	10	66.67
	Sometimes	Sometimes	5	33.33	15	100.00
Irritability	Never	Never	12	80.00	12	80.00
	Never	Sometimes	3	20.00	15	100.00
Easily angry/ hurt	*	Often	1	.	.	.
	Never	Never	5	35.71	5	35.71
	Often	Often	1	7.14	6	42.86
	Sometimes	Sometimes	8	57.14	14	100.00
Sadness/	*	Sometimes	1	.	.	.

depressed	Never	Never	3	21.43	3	21.43
	Never	Sometimes	1	7.14	4	28.57
	Sometimes	Often	1	7.14	5	35.71
	Sometimes	Sometimes	9	64.29	14	100.00
Enjoy seeing	Never	Never	1	6.67	1	6.67
friends or social	Never	Sometimes	1	6.67	2	13.33
contacts	Often	Often	5	33.33	7	46.67
	Sometimes	Sometimes	8	53.33	15	100.00
Restlessness	*	Sometimes	1	.	.	.
	Never	Never	8	57.14	8	57.14
	Never	Sometimes	2	14.29	10	71.43
	Sometimes	Never	1	7.14	11	78.57
	Sometimes	Sometimes	3	21.43	14	100.00
Sleeping	Never	Never	9	60.00	9	60.00
problems	Never	Sometimes	1	6.67	10	66.67
	Often	Often	1	6.67	11	73.33
	Sometimes	Sometimes	4	26.67	15	100.00
Appetite	Never	Never	12	80.00	12	80.00
problems	Sometimes	Sometimes	3	20.00	15	100.00
Nervousness/	Never	Never	5	35.71	5	35.71
anxious	Often	Often	1	7.14	6	42.86
	Sometimes	*	1	.	.	.
	Sometimes	Sometimes	8	57.14	14	100.00
Worried/on edge	Never	Never	5	33.33	5	33.33
	Never	Sometimes	1	6.67	6	40.00
	Often	Often	1	6.67	7	46.67
	Sometimes	Sometimes	8	53.33	15	100.00
Argumentative	Never	Never	7	46.67	7	46.67
	Never	Sometimes	2	13.33	9	60.00
	Often	Often	1	6.67	10	66.67
	Often	Sometimes	1	6.67	11	73.33
	Sometimes	Sometimes	4	26.67	15	100.00
Short tempered	Never	Never	7	46.67	7	46.67
	Often	Often	1	6,67	8	53.33
	Sometimes	Sometimes	7	46.67	15	100.00
Aggressiveness	Never	Never	8	53.33	8	53.33
	Never	Sometimes	1	6.67	9	60.00
	Sometimes	Sometimes	6	40.00	15	100.00

Key to table: * = Ambiguous frequencies (not filled in).

A complete summary of the pre and post-assessments of the entire experimental football playing group, in respect to all symptoms as reflected in Table 17 above. An observation of the PCS summary table, indicates that 26.67% of football playing control group (which is over a quarter of the sample), reported that they ñ*sometimes*ò experienced headaches (both at pre and post assessment) on the summary of PCS symptoms. According to McCrory et al., (2005) headaches are a pointer toward neurological problems which can indicate signs and symptoms of head injury, particularly in the acute phase.

Table 18: Summary of pre and post-assessments of Post-Concussion Symptoms: control group

Symptoms	Pre-assessment	Post-assessment	Frequency	Percent	Cumulative Frequency	Cumulative Frequency
Headaches	Never	Never	1	6.67	1	6.67
	Never	Sometimes	1	6.67	2	13.33
	Sometimes	Never	2	13.33	4	26.67
	Sometimes	Often	1	6.67	5	33.33
	Sometimes	Sometimes	10	66.67	15	100.00
Poor eyesight	Never	Never	10	66.67	10	66.67
	Never	Sometimes	1	6.67	11	73.33
	Often	Often	1	6.67	12	80.00
	Often	Sometimes	1	6.67	13	86.67
	Sometimes	Sometimes	2	13.33	15	100.00
Hearing difficulty	Never	Never	11	73.33	11	73.33
	Never	Sometimes	1	6.67	12	80.00
	Often	Often	1	6.67	13	86.67
	Sometimes	Never	1	6.67	14	93.33
	Sometimes	Sometimes	1	6.67	15	100.00
Weakness in	Never	Never	10	66.67	10	66.67
limbs	Sometimes	Never	4	26.67	14	93.33
	Sometimes	Sometimes	1	6.67	15	100.00
Clumsiness	Never	Never	10	66.67	10	66.67
	Never	Sometimes	1	6.67	11	73.33
	Sometimes	Never	2	13.33	13	83.67
	Sometimes	Often	1	6.67	14	93.33
	Sometimes	Sometimes	1	6.67	15	100.00
Fits/ seizures	Never	Never	14	100.00	14	100.00
	Sometimes	*	1	.	.	.
Dizziness	Never	Never	5	33.33	5	33.33
	Never	Sometimes	3	20.00	8	53.33

	Sometimes	Never	5	22.22	13	86.67
	Sometimes	Sometimes	2	13.33	15	100.00
Easily tired	Never	Never	8	53.33	8	53.33
	Never	Sometimes	1	6.67	9	60.00
	Sometimes	Never	2	13.33	11	73.33
	Sometimes	Often	1	6.67	12	80.00
	Sometimes	Sometimes	3	20.00	15	100.00
Sensitivity to	Never	Never	8	53.33	8	53.33
noise	Never	Sometimes	3	20.00	11	73.33
	Often	Never	1	6.67	12	80,00
	Often	Often	1	6.67	13	86.67
	Sometimes	Sometimes	2	13.33	15	100.00
Seeing/hearing	Never	Never	13	86.67	13	86.67
Feeling unusual	Sometimes	Never	1	6.67	14	93.33
things	Sometimes	Sometimes	1	6.67	15	100.00
Sexual problems	Never	Never	14	93.33	14	93.33
	Never	Sometimes	1	6.67	15	100.00
Speech problems	Never	Never	10	66.67	10	66.67
	Never	Sometimes	1	6,67	11	73.33
	Often	Sometimes	1	6,67	12	80.00
	Sometimes	Never	2	13.33	14	93.33
	Sometimes	Sometimes	1	6.67	15	100.00
Stumble over	Never	Never	8	53.33	8	53.33
words	Never	Sometimes	1	6.67	9	60.00
	Often	Often	1	6.67	10	66.67
	Sometimes	Never	1	6.67	11	73.33
	Sometimes	Sometimes	4	26.67	15	100.00
Stutter/ stammer	Never	Never	10	66.67	10	66.67
	Never	Sometimes	2	13.33	12	80.00
	Often	Never	1	6.67	13	86.67
	Often	Often	1	6.67	14	93.33
	Sometimes	Sometimes	1	6.67	15	100.00
Slur of words	*	Never	2	.	.	.
	Never	Never	6	46.15	6	46.15
	Never	Sometimes	1	7.69	7	53.85
	Sometimes	Never	2	15.38	9	69.23
	Sometimes	Sometimes	4	30.77	13	100.00
Memory	Never	Never	8	53.33	8	53.33
difficulties	Never	Sometimes	1	6.67	9	60.00

	Sometimes	Never	1	6.67	10	66.67
	Sometimes	Sometimes	5	33.33	15	100.00
Attention and	Never	*	1	.	.	.
concentration	Never	Never	3	21.43	3	21.43
problems	Never	Sometimes	2	14.29	5	35.71
	Often	Often	2	14.29	7	50.00
	Sometimes	Never	2	14.29	9	64.29
	sometimes	Sometimes	5	35.71	14	100.00
Attention	Never	Never	1	6.67	1	6.67
wondering when	Often	Often	1	6.67	2	13.33
conversing or	Often	Sometimes	3	20.00	5	46.67
watching TV/	Sometimes	Never	2	13.33	7	60.00
reading	Sometimes	Often	1	6.67	8	66.67
	Sometimes	Sometimes	7	46.67	15	100.00
Impatient	Never	Never	3	20,00	3	20.00
	Often	Often	2	13.33	5	33.33
	Often	Sometimes	2	13.33	7	46.67
	Sometimes	Never	2	13.33	9	60.00
	Sometimes	Often	1	6.67	10	66.67
	Sometimes	Sometimes	5	33.33	15	100.00
Irritability	Never	Never	4	26.67	4	26.67
	Often	Never	1	6.67	5	33.33
	Sometimes	Never	1	6.67	6	40.00
	Sometimes	Often	1	6.67	7	46.67
	Sometimes	Sometimes	8	53.33	15	100.00
Easily angry/ hurt	Never	Never	4	26.67	4	26.67
	Often	Sometimes	4	26.67	8	53.33
	Sometimes	Never	4	26.67	12	80.00
	Sometimes	Sometimes	3	20.00	15	100.00
Sadness/	Never	Never	5	33.33	5	33.33
depressed	Never	Sometimes	1	6.67	6	40.00
	Often	Never	1	6.67	7	46.67
	Often	Sometimes	1	6.67	8	53.33
	Sometimes	Never	2	13.33	10	66.67
	Sometimes	Sometimes	5	33.33	15	100.00
Enjoy seeing	Never	Often	1	6.67	1	6.67
friends or social	Often	Often	10	66.67	11	73.33
contacts	Often	Sometimes	1	6.67	12	80.00
	Sometimes	Sometimes	3	20.00	15	100.00
Restlessness	Never	Never	7	46.67	7	46.67

	Never	Sometimes	11	6.67	8	53.33
	Often	Sometimes	1	6.67	9	60.00
	Sometimes	Never	4	26.67	13	86.67
	sometimes	Sometimes	2	13.33	15	100.00
Sleeping	*	Never	1	.	.	.
problems	Never	Never	6	42.86	6	42.86
	Never	Sometimes	3	21.43	9	64.29
	Often	Often	1	7.14	10	71.43
	Often	Sometimes	1	7.14	11	78.57
	Sometimes	Never	2	14.29	13	92.86
	sometimes	sometimes	1	7.14	14	100.00
Appetite	Never	Never	7	46.67	7	46.67
problems	Never	Sometimes	3	20.00	10	66.67
	Sometimes	Never	3	20.00	13	86.67
	Sometimes	Sometimes	2	13.33	15	100.00
Nervousness/	Never	Never	2	13.33	2	13.33
anxious	Never often	Sometimes	2	13.33	4	26.67
	Sometimes	sometimes	2	13.33	6	40.00
	Sometimes	Never	2	13.33	8	53.33
		Sometimes	7	46.67	15	100.00
Worried/on edge	Never	Never	4	26.67	4	26.67
	Never	Sometimes	2	13.33	6	40.00
	Often	Sometimes	1	6.67	7	46.67
	Sometimes	Sometimes	8	53.33	15	100.00
Argumentative	Never	Never	1	6,67	1	6,67
	Never	Sometimes	1	6,67	2	13.33
	Often	Never	2	13.33	4	26.67
	Often	Often	3	20.00	7	46.67
	Often	Sometimes	2	13.33	9	60.00
	Sometimes	Never	2	13.33	11	73.33
	Sometimes	Sometimes	4	26.67	15	100.00
Short tempered	Never	Never	5	33.33	5	33.33
	Often	Never	1	6.67	6	40.00
	Sometimes	Never	3	20.00	9	60.00
	Sometimes	Often	1	6.67	10	66.67
	Sometimes	Sometimes	5	33.33	15	100.00
Aggressiveness	Never	Never	9	60.00	9	60.00
	Never	Sometimes	1	6.67	10	66.67
	Sometimes	Never	2	13.33	12	80.00
	Sometimes	Often	1	6.67	13	86.67

	Sometimes	Sometimes	2	13.33	15	100.00

Key to table: * = Ambiguous frequencies (not filled in)

Table 18 is a complete summary of pre and post-assessment of the control group in respect of PCS. An observation of the PCS summary indicates that just over 6% of the entire sample, as opposed to 26.67% of the football playing experimental group (which is over a quarter of the sample), reported that they ñsometimesò experienced headaches (both at pre and post assessment) on the PCS Check list.

Table 19: Summary of all pre and post-assessment of Post-Concussion Symptoms change: football playing experimental group

Post-concussion symptoms	Difference between pre and post assessment	Frequency	Percent %	Cumulative Frequency	Cumulative Percentage %
Headache	-1	1	6.7	1	6.7
	0	14	93.33	15	100.00
Poor eyesight		14	93.33	14	93.33
		1	6.67	15	100.00
Hearing difficulty	0	15	100	15	100.00
	2	15	100	15	100.00
Weakness in limbs	0	1	7.4	1	7.4
	0	13	92.86	14	100.00
Clumsy					
ambiguous	-1				
frequency ==1	0	15	100.00	15	100.00
Fits/ seizures	0	4	26.67	4	26.67
		11	73.33	15	100.00
Dizziness	-1				
	0	1	7.14	1	7.4
		13	92.86	14	100.00
Tire easily	-1				
	0				
ambiguous					
Frequency ==1		1	7.69	1	7.69
Sensitivity to		11	84.62	12	92.31
noise		1	7.69	13	100.00
	-1				
ambiguous	0				
frequency ==2	1				
		1	6.67	1	6.67
Seeing, hearing or	-1	14	93.33	15	100.00
feeling unusual	0				
things					
		2	13.33	2	13.33
Sexual problems	-1	13	86.67	15	100.00

	0				
Speech problems		15	100.00	15	100.00
	0				
Stumble over words		14	93.33	14	93.33
	0	1	6.67	15	100.00
Stutter/ stammer	1				
		15	100.00	15	100.00
Slur words	0				
ambiguous		1	7.4	1	7.14
frequency = 1	-1	13	92.86	14	100.00
	0				
Memory difficulties					
		1	6.67	1	6.7
	-1	13	86.67	14	93.33
Attention &	0	1	6.67	15	100.00
concentration	1				
problems		1	7.14	1	7.14
	-1	13	92.86	14	100.00
ambiguous	0				
frequency = 1					
Wandering attention					
		2	15.38	2	15.38
ambiguous		11	84.62	13	100.00
frequency = 2	-1				
	0				
Impatient					
		1	6.67	1	
Irritability		14	93.33	15	6.67
Easily angry/ hurt	-1				100.00
	0	15	100.00	15	
ambiguous		14	100.00	14	100.00
frequency = 1	0				100.00
	0				
Feel sad/ depressed					

ambiguous		2	14.29	2	
frequency = 1		12	85.71	14	14.29
	-1				100.00
Friends & social	0				
contact					
Restlessness		1	6.67	1	
		14	93.33	15	6.67
ambiguous	-1				93.33
frequency = 1	0	2	14.29	2	
		11	78.57	13	14-29
	-1	1	7.14	14	92.86
Sleeping	0				100.00
problems	1				
Appetite		1	6.67	1	
problems		14	93.33	15	6.67
	-1				100.00
Feel nervous/	0	15	100.00	15	
anxious					100.00
ambiguous	0				
frequency = 1		14	100.00	14	
					100.00
Feel worried/ on	0				
edge					
	-1				
	0	1	6.67	1	
	-1	14	93.33	15	6.67
Argumentative	0				100.00
	1				
Feeling short	0	2	13.33	2	
tempered		12	80.00	14	13.33
		1	6.67	15	93.33
Aggressiveness	-1	15	100.00	15	100.00
	0				100.00
		1	6.67	1	
		14	93.33	15	6.67
					100.00

Key to table: -1 and -2 = Symptoms changed and became worse: 0 = symptoms did not change: 1 and 2 = symptoms changed and became better: * = Ambiguous frequencies (not filled in).

Table 19 lists a summary of symptoms change of the pre and post-assessments as reported by the football playing experimental group. A perusal of the list indicates that 50% of the football group reported that ñsometimesò they experience problems with attention and concentration and 57.14 % easily get angry and hurt. They also experience being nervous or anxious. Further, 53.33% get worried and are on the edge. These results can also be signs of either emotional or behavioural problems, in the acute phase of a head injury, but are often not reported to the coaching or medical staff for fear of being subjected to testing and/or being dropped from the team (McCrory et al., 2005; Ruchinskas et al., 1997).

Table 20: Summary of all pre and post-assessment of Post-Concussion Symptoms change: volleyball control group

Post- concussion symptoms	Difference between pre and post assessment	Frequency	Percent %	Cumulative Frequency	Cumulative Percentage %
Headache	-1	2	13.33	2	13.33
	0	11	73.33	13	86.67
	1	2	13.33	15	100.00
Poor eyesight	-1	1	6.67	1	6.67
	0	13	86.67	14	93.33
	1	1	6.67	15	100.00
Hearing difficulty	-1	1	6.67	1	6.67
	0	13	86.67	14	93.33
	1	1	6.67	15	100.00
Weakness in limbs					
	0	11	73.33	11	73.33
Clumsy	1	4	26.67	15	100.00
	-1	2	13.33	2	13.33
	0	11	73.33	13	86.67
Fits/ seizures	1	2	13.33	15	100.00
Ambiguous frequency = 1	0	14	100.00	14	100.00
Dizziness					20.00
	-1	3	20.00	3	66.67
Easily tired	0	7	46.67	10	100.00
	1	5	33.33	15	13.33
	-1	2	13.33	2	86.67
Sensitivity to	0	11	73.33	13	100.00
noise	1	2	13.33	15	20.00
	-1	3	20.00	3	93.33
Seeing, hearing or	0	11	73.33	14	100.00
feeling unusual	2	1	6.67	15	

things					93.33
	0	14	93.33	14	100.00
Sexual problems	1	1	6.67	15	
Speech problems					6.67
	-1	1	6.67	1	100.00
	0	14	93.33	15	
Stumble over					6.67
words	-1	1	6.67	1	80.00
	0	11	73.33	12	100.00
	1	3	20.00	15	
Stutter/ stammer					6.67
	-1	1	6.67	1	93.33
	0	13	86.67	14	100.00
Slur of words	1	1	6.67	15	
Ambiguous					13.33
frequency = 2	-1	2	13.33	2	93.33
	0	12	80.00	14	100.00
Memory	2	1	6.67	15	
difficulties					7.69
	-1	1	7.69	1	84.62
	0	10	76.92	11	100.00
	1	2	15.38	13	
Attention and					6.67
concentration	-1	1	6.67	1	93.33
problems	0	13	86.67	14	100.00
Ambiguous	1	1	6.67	15	
frequency = 1					14,29
	-1	2	14.29	2	85.71
Wandering	0	10	71.43	12	100.00
attention	1	2	14.29	14	
Impatient					
	-1	1	6.67	1	6.67
	0	9	60.00	10	66.67
	1	5	33.33	15	100.00
Irritability					
	-1	1	6.67	1	6.67
	0	10	66.67	11	73.33

	1	4	26.67	15	100.00
Easily angry/ hurt	-1	1	6.67	1	6.67
	0	12	80.00	13	86.7
Feel sad/	1	1	6.67	14	93.33
depressed	2	1	6.67	15	100.00
	0	7	46.67	7	46.67
	1	8	53.33	15	100.00
Seeing friends/					
social contact	-1	1	6.67	1	6.67
	0	10	66.67	11	73.33
	1	3	20.00	14	93.33
Restlessness	2	1	6.67	15	100.00
	-2	1	6.67	1	6,67
	0	13	86.67	14	93.33
Sleeping	1	1	6.67	15	100.00
problems					
	-1	1	6.67	1	6.67
ambiguous	0	9	60.00	10	66.67
frequency = 1	1	5	33.33	15	100.00
Appetite problems	-1	3	21.43	3	21.43
	0	8	57.14	11	78.57
	1	3	21.43	14	100.00
Feeling nervous/					
anxious					
Feeling worried/	-1	3	20.00	3	20.00
on edge	0	9	60.00	12	80.00
	1	3	20.00	15	100.0
Argumentative					
	-1	2	13.33	2	13.33
	0	9	60.00	11	73.33
	1	4	26,67	15	100.00
Feeling short	-1	2	13.33	2	13.33
tempered	0	12	80.00	14	93.33
	1	1	6.67	15	100.00

	-1	1	6.67	1	6.67
Aggressiveness	0	8	53.33	9	60.00
	1	4	26.67	13	86.67
	2	2	13.33	15	100.00
	-1	1	6.67	1	6.67
	0	10	66.67	11	73.33
	1	3	20.00	14	93.33
	2	1	6.67	15	100.00
	-1	2	13.33	2	13.33
	0	11	73.33	13	86.67
	1	2	13.33	15	100.00

Key to table: -1 and -2 = Symptoms changed and became worse: 0 = symptoms did not change: 1 and 2 = symptoms changed and became better: * = Ambiguous frequencies (not filled in).

The volleyball control group reported an overall ñworsenedò PCS, a change from *never t*o *sometimes* or *often,* from pre to post assessment (See summary of symptoms in tables 18 and 19). The total number of PCS checklist questions for both the football playing group and control group was 465. However, the football group did not fill in 12 questions on the questionnaire while the control group did not fill in 5. The results revealed no significant difference between the two groups.

A comparison of the rating of symptom frequencies of the football players and the controls is presented in tables 21 and 22. Cross tabulations between the football and the control groups to establish the distribution of the symptom frequency ratings were performed. The experimental or football playing group, and the volleyball control group, were compared on frequency ratings of ñsometimesò, *"often"* or ñ*never"* on the PCS checklist questionnaire. The mean for the total football group is 6.9% and the mean for the control group is 9.3% with $p < 0.062$. The results reveal that there is no significant difference between the football and control groups regarding the frequency of PCS.

For frequency (incidence), the football group and the control group were compared on frequency of symptoms on the PCS Checklist. Frequency of symptoms as reported by the football group in pre and post-season assessment (See Tables 23, 24 and 25) are 30.9% for ñ*often"* while 5.2% of the control group reported ñ*often"*. Almost 31% of the football players and 22.83% of the control group reported ñ*sometimes"* on the PCS checklist frequency ratings. Over 58% of the football group reported that they had *never* experienced most of the symptoms on the PCS checklist. This implies that they never experienced the following symptoms, which frequently point to some cognitive impairment, hearing difficulty, weakness of the limbs, clumsiness, dizziness, speech problems, short temper and/or aggressiveness. However, only 45% of the control group ñ*never"* experienced most of the symptoms on the checklist. This is an anomaly as it is expected that the football playing group would have experienced more PCS symptoms than the volleyball controls due to CMHI.

Table 21: Frequency of all pre and post-assessment of Post-Concussion Symptom change: football playing group

Total pre-season	Total post-season	Frequency	Percentage %
.	Often	1*	.
.	Sometimes	2*	.
Never	.	4*	.
Never	Never	206	58.72
Never	Sometimes	20	4.42
Often	Never	1	0.22
Often	Often	14	3.09
Often	Sometimes	1	0.22
Sometimes	.	5*	.
Sometimes	Never	4	0.88
Sometimes	Often	7	1.55
Sometimes	Sometimes	140	30.91

Note: * = ambiguous frequencies (ambiguous frequencies= 12)

Table 22: Frequency of all pre and post-assessment of Post-Concussion Symptom change: volleyball control group

Total pre-season	Total post-season	Frequency	Percentage %
.	Never	3*	.
Never	.	1*	.
Never	Never	207	45.00
Never	Often	1	0.22
Never	Sometimes	34	7.39
Often	Never	7	1.52
Often	Often	24	5.22
Often	Sometimes	20	4.35
Sometimes	.	1*	.
Sometimes	Never	54	11.74
Sometimes	Often	8	1.74
Sometimes	Sometimes	105	22.83

Note: * = ambiguous frequencies (ambiguous frequencies = 5)

The tables 23, 24 and 25 below depict a comparison of rating of symptoms frequencies of the football and control participants at pre-season and post-season testing. An exact test that is appropriate for computing percentages of small counts was used for cross tabulations to establish the distribution of the symptom frequency rating for ñ*worse"*, ñ*unchanged"*, and ñ*improved"* counts. The ñ*worse count"* percentages (6% and 9.3%) for the football group

and the control group (Table 23), do not differ significantly (p = 0.62). The total number of frequencies for the football players is 453 and the control group is 460. Football players had 12 ambiguous frequencies and the volleyball controls had 5. Table 24 depicts the ñ*unchanged"* symptom counts percentages used to compare the football and the controlsô percentages (92.7% and 73%) respectively. The two percentages differ significantly with (p Ò 0.001).

Table: 23 Footballandvolleyballcontrolgroupsôcountonsymptomintensity(worsened)at pre-season versus post-season

Post-concussion symptoms	**Football**		**Volleyball**	
	Frequency	Percentage %	Frequency	Percentage %
Worsened	27	6.0	43	9.3
Not Worse	426		417	
Total	453		460	

Table 24: Footballandvolleyballcontrolgroupsôcountonsymptomintensity(unchanged)at pre-season versus post-season

Post-concussion symptoms	**Football**		**Volleyball**	
	Frequency	Percentage %	Frequency	Percentage %
Unchanged	420	92.7	336	73.0
Changed	33		124	
Total	453		460	

Table 25: Footballandvolleyballcontrolgroupsôcountonsymptomsintensity(improved) counts pre-season versus post-season

Post-concussion symptoms	**Football**		**Volleyball**	
	Frequency	Percentage %	Frequency	Percentage %
Improved	6	1.3	81	17.6
Not improved	447		379	
Total	453		460	

Bar Charts 1, 2 and 3 give a graphic representation of ñworsened,ò ñunchangedò and *"improved"* symptom count percentages pre ï versus post season testing.

Figure 5: Football and volleyball control groupsô count on symptom intensity (worsened) counts pre-season testing versus post-season testing

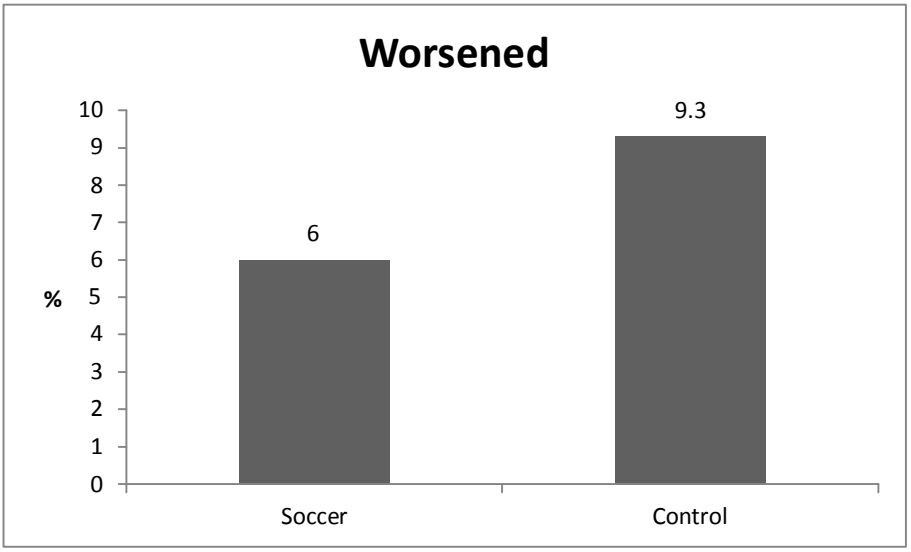


Figure 6: Football and volleyball control groupsô count on symptom intensity (no change) counts pre-season testing versus post-season testing

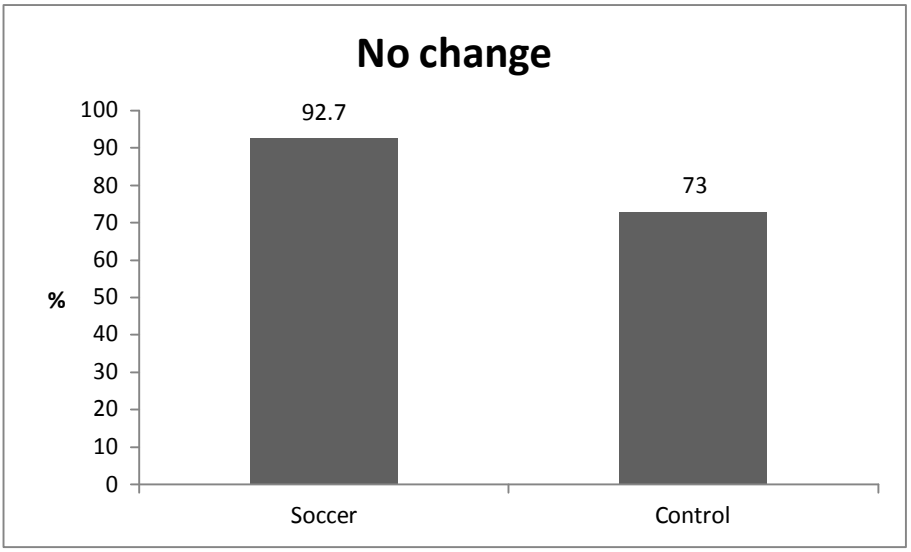
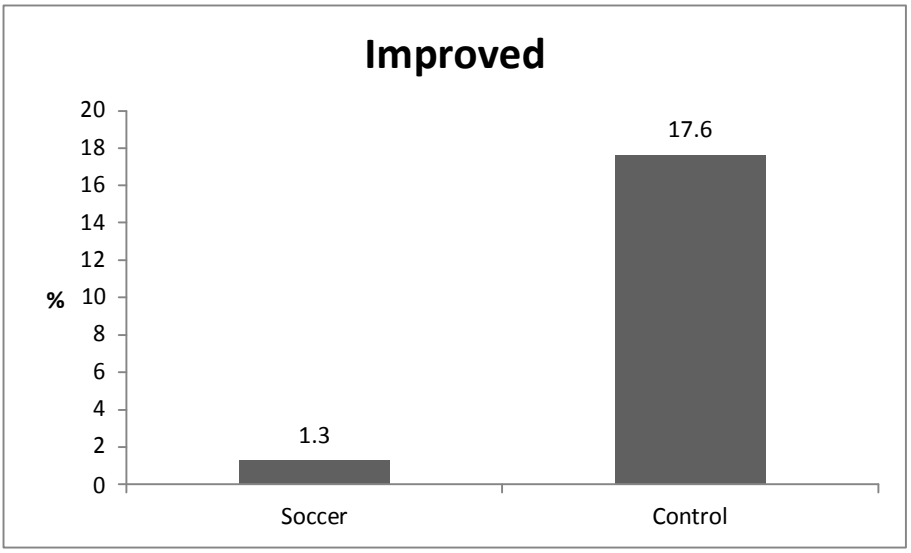


Figure 7: Football and volleyball control groupsô count on symptom intensity (improved) counts pre-season testing versus post-season testing



5.4 The combination of results for the football experimental group and the volleyball control group

5.4.1 Neurocognitive measures, CALCAP

On the two sample analyses between the football and control groups, no significant differences were revealed on the CALCAP neurocognitive measures at pre season but at post season testing there was a small difference for Sequential Reaction Time 1 .There were no significant findings for any of the other CALCAP tests for the football and volleyball control groups at pre and post season. The results indicated that there were no significant differences between the two groups when compared for Simple, Choice, and Sequential 2 reaction times on the CALCAP. According to Waterloo et al., (1997) as Serial Pattern Matching (Sequential Reaction time 1) is largely a measure of divided attention skills, it maybe that the CALCAP which is sensitive to mild diffuse brain damage, could indicate fluctuating attention in the football group as a result of MHI or CMHI. However, this must be interpreted with caution as in general, poor performance on one measure does not indicate a specific type of cognitive impairment, although certain types of tasks do seem related to deficits in specific skills (Miller, 1993b).

On the two sample t-test analyses for the football group and the volleyball control group, the repeated measures ANOVA revealed a small difference on one CALCAP measure, Sequential RT 1 only; and for the three remaining measures, Simple, Choice and Sequential 2 Reaction times, the ANOVA did not reveal any significant differences. On the ANCOVA analyses of the football group and the volleyball control group confirmed the results yielded by the t-test and ANOVA on the Sequential 1 Reaction time. The ANCOVA also revealed a small difference on one CALCAP measure, Sequential RT 1 and no significant differences are revealed for the other measures, Simple, Choice and Sequential 2 reaction times. The overall trend indicates that the t-test, ANOVA and ANCOVA suggests a small difference on the Sequential RT 1 which may indicate that the football group did experience some signs of CMHI or concussion during the course of the football season.

5.4.2 Post-Concussion Symptom checklist

The Fisher exact test was used for the comparison of the summary of PCS between the football and volleyball control participants at pre-season (baseline) test and post-season (end of season) testing to establish the frequency and distribution of the intensity symptoms frequency ratings. The results reveal that there is no significant difference between the football and control groups regarding the frequency of PCS.

The second part of the hypotheses for intensity (or symptom change) symptoms for ñ*worse"*, ñ*unchanged*ò, and ñ*improved"* counts reveal that there is no significant difference for ñ*worse"* count percentages for the football group and the control group. But there is a significant difference for both ñ*no change"* and ñ*improved"* symptoms frequency count for the football group and the control group. The total number of symptoms frequencies filled in by the football playing group is 453 and the control group is 460. Football participants have 12 ambiguous frequencies and the controlsô has 5. Taking into consideration the small difference on the ñ*unchanged*ò and ñ*improved"* symptoms frequency count for the football group and the volleyball control group, these results revealed that statistically, the exact test analyses cannot be generalised, as the differences may have happened by chance**.** However, as the sample was small any interpretations must be interpreted with caution.

A two sample t - test was used for the comparison of the summary of post-concussion symptoms between the football and control participants at pre-season (baseline) test and post-season (end of season) testing to establish the frequency and distribution of the intensity symptoms frequency ratings. The results reveal that there is no significant difference between the football and control groups regarding the frequency of post-concussion symptoms.

6. Summary

In summary, taking into consideration the small differences in the results that are revealed across the statistical tests the football group, as a whole, do not take significantly more time to process information as compared to the volleyball control group, except on Sequential RT1, where significant difference was found on three tests however, as the sample was small results must be interpreted with caution. It cannot be discounted however, that these results are indicative of CMHI in the football playing experimental group as the CALCAP is very sensitive to diffuse brain damage (Waterloo et al., 1997).

CHAPTER 6: DISCUSSION OF RESULTS, EVALUATION AND RECOMENDATIONS OF THE RESEARCH

6.1 Introduction

This chapter will discuss the aims and significance of the research questions as an introduction. Then neurocognitive measures will be evaluated in respect of two-sample t-test comparison of group means, analysis of variance (ANOVA) and analysis of covariance (ANCOVA). This is followed an exact test used for computing percentages of small counts which was used for cross tabulations to establish the distribution of the symptom frequency ratings on the PCS. These are followed by an evaluation of the study, followed by a discussion of the overall implications of the research.

6.2 Broad summary of the study

There is only one known unpublished sport-related CMHI study investigating the correlation between symptoms of MHI and cognitive impairment among adult athletes. This study used computerised testing (CALCAP) which is sensitive to diffuse brain injury however the study was not related to football (Nel, 2009). There are however, published studies that investigate the effect of CMHI or concussion and Post-Concussive Symptoms of adult athletes that have used the traditional pen and paper tests that relate to football (Abreau etal., 1990; Ancer, 2000; Barnes et al., 1998; Boulind, 2005; Enchemendia & Julian, 2001; Putukian et al., 2000; Tysvaer, Storli & Bachen, 1989). However, three of the studies did not use control groups of non-contact sports athletes for comparative purposes (Barnes et al., 1998; Putukian et al., 2000; Tysvaer, Storli & Bachen, 1989). This provided the motivation for the present study in using a quasi-experimental non-equivalent design that incorporated non-contact sport controls and also in using a computerised test to measure neurocognitive symptoms as well as a pen and paper PCS checklist questionnaire. The present study investigated neurocognitive and post-concussion symptoms profiles of professional football players as compared to non-contact sport volleyball playing controls over one season, utilising the CALCAP and a Post-Concussion Symptom checklist questionnaire. The objective of this research design was to investigate the correlation between symptoms of MHI and cognitive impairment amongst professional football players, over one football season.

The following was carried out in order to achieve this objective: a sample of 15 football players and 15 non-contact sportsmen participated in the study subsequent to the exclusion criteria (as described in Chapter 4, section 4.1.3), which resulted in the final sample groups being football Players (n= 15) and Non-equivalent Controls (n = 15). Age is the only demographic variable in which the comparison of football and control groups was matched. There is a small difference in t-test, ANOVA and ANCOVA in the Sequential RT 1 at the end of the season. However, there is no significant difference between the football players and control group on the overall pre and post-season testing for neurocognitive outcomes. The results cannot therefore be generalised as the sample for this study is small. However, as there is a significant difference on three of the tests on Sequential RT 1 the results cannot be totally discounted and further testing, using a larger experimental and control group should be undertaken. This because the CALCAP is very sensitive to diffuse brain damage and Sequential RT 1 is a measure of focused attention which may suggest that football players are susceptible to CMHI.

On the symptoms frequency ratings (using the Fisher Exact test) for ñ*worse"*, ñ*no change"*, and ñ*improved"* counts, there is no significant difference for worse count percentages between the football and the control groups. But there is a significant difference for both ñ*no change",* (p=0.001) and ñ*improved*, (p=0.001) symptoms frequency count for the football group and the control group. Football participants have 12 frequencies not filled in (ambiguous) and the controlsô have 5. Both Football and the Control participants were tested at baseline (pre) season and again at the end of the season. CALCAP was used as a computerised neurocognitve test measure and a traditional pen and paper, PCS checklist questionnaire, was used to elicit symptoms experienced.

All measures were administered at pre and post-season. The two sample t-test, ANOVA and ANCOVA were conducted on the group means to investigate potential differences in neurocognitive functioning. An exact test was used on the PCS to compute small count percentages for cross tabulations to establish the distribution of symptom frequency ratings for ñ*worse"*, ñ*no change"*, and ñ*improved"* counts. The level of significance used in the study on all statistical tests was 5%.

The findings of the study are evaluated against the background of the research hypotheses (See 4.5).

Neurocognitive measures:

1. Football players who sustained Cumulative Mild Head Injuries (CMHI or concussion) would be slower in simple reaction time tasks of the California Computerised Assessment Package or CALCAP) than the volleyball control group.

2. Football players who sustained CMHI (or concussion) would take significantly longer to respond, on Choice Reaction Time for Single Digits (Task 11 of the CALCAP) than the volleyball control group.

3. Football players who sustained CMHI (or concussion) would take more time processing information on Serial Pattern Matching 1 (Sequential Reaction Time 1 ï task 111 of CALCAP) than the volleyball control group.

4. Football players who have sustained CMHI (or concussion) would take more time processing information on Serial Pattern Matching 2 (Sequential Reaction Time 2 ï task 1V of CALCAP) than the volleyball control group.

5. Football players who have sustained CMHI (or concussion) would experience a higher frequency rating of symptom change on the PCS checklist than the volleyball control group in terms of frequency and intensity of symptom count.

6.3 Interpretation of findings for the experimental football group and the volleyball control group

Pre-season testing (baseline) and post-season testing (end of the season) across the neurocognitive measures are discussed first and will be followed by pre-season (baseline) and post-season (end of season) findings for the PCS measure.

6.3.1 Interpretation of findings on the neurocognitive measures

- **Two sample t-test comparisons**

Two sample t-test analyses on the CALCAP neurocognitive test measure at pre- season testing revealed no significant difference between the football and control groups on all four CALCAP measures (Simple RT, Choice RT, Sequential 1 RT and Sequential 2 RT). Two sample t-test analyses on the CALCAP neurocognitive test measure at post-season testing revealed no significant difference between the football and Control group on three CALCAP measures (Simple RT, Choice RT, and Sequential 2 RT), except for Sequential 1 RT ($p=0.3830$) which revealed a small significant difference between the football playing group and volleyball controls.

The overall results for the two sample t-test analyses between the football and the control groups, at pre-season and post-season testing viewed in conjunction with relevant literature do not support hypotheses 1, 2 and 4. Results for post-season testing were confirmed for hypotheses 3, which indicate a small significant difference for the football playing group which may be due to the CALCAP being very sensitive to diffuse brain damage. However, this statistic must be interpreted with caution because of the small sample size and other threats to validity. A comprehensive evaluation of the study limitations is undertaken in Chapter 6 and some are reviewed in Chapter 5.

- **Repeated measures, ANOVA**

The repeated measures ANOVA for the CALCAP neurocognitive test measure, revealed no significant difference between the football and control groups on three CALCAP measures (Simple RT, Choice RT, and Sequential 2 RT), except for Sequential 1 RT (p=0.0351) which revealed a small significant difference in that the football group which was slightly diminished from pre-season testing (Figure 3). Pre-season testing superiority diminished by a large margin at post-season testing on the Sequential 1 RT on repeated measures. This outcome confirmed the post-season (baseline) analysis of the two sample t-test results.

- **Analysis of Covariance (ANCOVA) with a Single Covariance Comparison**

With regards to the ANCOVA analysis, the CALCAP neurocognitive test measure at post-season interval revealed no significant difference between the football and control groups on three CALCAP measures (Simple RT, Choice RT, and Sequential 2 RT), but a small difference for Sequential 1 RT (p=0.0445) was also noted. The results of the ANCOVA highlighted and revealed a trend of a small significant difference outcome across all three analyses conducted in this study on Sequential 1 RT. These findings, although small, appear to support hypothesis 3.

- **Summary of neurocognitive findings for the two sample t-test analysis, ANOVA and ANCOVA**

The overall findings for the football and the control groups, on the neurocognitive measures, highlight consistency on the CALCAP neurocognitive outcome. Most of the results did not support hypotheses one, two and four (in chapter 4 section 4.7) that broadly suggests that football players who have sustained CMHI or concussion will take significantly longer time to respond to all CALCAP measures. There may be evidence of treatment effect on Sequential 1 RT (Table 13; Figure 3), as the football playersô reaction time is evidently slower than the baseline task reaction time. If the reaction time was faster than the baseline task, this would have suggested that the baseline measure was spoiled due to attentional problems, lack of motivation or environmental distractions. Even though there is a small significant difference in the third measure of the CALCAP (Sequential 1RT) it is difficult to confirm this finding in terms of CMHI because of the small sample size and threats to validity. However, this finding cannot be completely discounted due to the measure being

extremely sensitive to diffuse brain damage over time. Another possibility, according to Cook and Campbell (1979) is that the findings are an indication of sample maturation masquerading as treatment effect.

6.3.2 Interpretation of findings on the Rivermead Post – Concussion Symptom Measure

This section discusses the results of the football and the control groupsô symptoms measure. The first section discusses the findings for the percentage frequency of all post-concussion symptoms reported by the football and the control groups. It will be followed by the football and control groupsô pre- and post-season frequency of Post-Concussion Symptom changes. Lastly, the summary count on symptom change, ñ*worse"*, ñ*unchanged"* and ñ*improved"* counts on comparison between the football and control groups will be discussed.

The results of percentage frequency ratings comparison of ñ*sometimes", "often"* or ñ*never"* on the post-concussion symptom measure reveal that there is no significant difference between the football and control groups. But with regards to specific symptoms, the frequency symptoms reported by football and the control groups at both pre and post-season testing (Tables 17 and 18) are that they sometimes experienced headaches. An observation of the total frequency list of the controlsô revealed that 6% of the control participants as opposed to over a quarter of the football participants experienced headaches. According to McCrory et al., (2000) headaches are the most common MHI symptoms after sustaining head injury particularly in the acute phase. Further perusal of the frequency list indicates that half of the football players reported that they sometimes experienced problems with attention and concentration, over half of the football players easily get angry and hurt and also experience being nervous or anxious. The specific symptoms reported above by the football players indicate that there is a possibility that treatment had an effect on this group. However, the specific symptoms are commensurate with PCS in the chronic phase which, to an extent, backs up the significant finding on CALCAP Sequential 1 RT, but again because of small sample size and internal threats to validity the finding cannot be definitive. The results also highlight the signs of either emotional or behavioural problems in the acute phase of a head injury. These are often not reported to the coaching staff as football playersô fear that they will be subjected to neuropsychological testing and/or be dropped from the team (McCrory et al., 2005; Ruchinskas et al., 1997).

Just over a quarter of the football group reported ñ*often"* on the summary of all pre and post-season frequency symptoms while a fifth of the controlsô reported ñ*often"* at pre and post-season assessment. Over a quarter of the football group and less than a quarter of the controlsô reported ñ*sometimes"* on the symptom checklist frequency ratings. The majority of (58.72%) the football group reported that they ñ*never"* experienced most of the symptoms on the checklist. Thus the ñ*never"* experienced symptoms imply that there was no treatment effect at post-season testing. The symptoms that are ñ*never"* experienced are the following: hearing difficulty, weakness in the limbs, clumsiness, dizziness, speech problems, short temper and/or aggressiveness, which frequently point to some cognitive impairment. However, almost half of the control group also ñ*never"* experienced most of the symptoms on the checklist. The results for the football group is an anomaly as it was expected that the football playing group would have experienced more Post-Concussive Symptomology than the controls due to CMHI or concussion, (See Tables 17 and 18).

The results for ñ*worse"* symptoms counts reveal that there is no significant difference between football and the control groups. These results do not support hypothesis 5 where it is expected that the football players will experience a higher symptom frequency rating change due to sustaining CMHI or concussion. However, the results for ñ*improved"* symptoms count reveal that there is a significant difference, even though it is small difference, between the football group and volleyball controls. Again the significant outcome might imply that there is a probability that the football players experienced treatment effect (concussion or CMHI). As small samples are prone to Type II error, it can be argued that the study failed to find significant effects even when such effects do exist, thus failing to be statistically relevant.

- **Summary of PCS findings**

The frequency symptoms overall findings for the football group and the control group, on the symptom measure, revealed that all the results were not in support of hypothesis 5 which states that the football playing group would experience more intensity of symptoms than the controls due to CMHI or concussion and the football playing group would experience a higher rating of symptom change than the controls due to CMHI or concussion. The control group had improved symptomology on the PCS. Even though the difference for ñ*improved"* symptoms between football and control groups is small, it may reveal an apparent treatment

effect for the football group. The treatment effect, in this instance, might be due to selection maturation difference that is masquerading as a treatment effect for the football players.

6.4 Combination of findings for the neurocognitive and PCS measures

The overall consistency direction and the trend of not significant results for the three neurocognitive measures between the football and the control groups, amongst the two sample t-test, ANOVA and ANCOVA provides cross validation of the outcome of the results. The results do not support hypotheses 1, 2 and 4. There is also a trend and consistency of significant results which is small, for only one measure of the CALCAP on hypothesis 3 between the football and control groups. Overall, there is no difference between football and the control group except for a small significant difference on the divided attention and short-term memory on the neurocognitive measure Sequential RT 1**.** The small difference in the results may be due to interaction with selection, specifically selection maturation (football players became more experienced between pre and post-season testing) and selection history (due to the different events that took place between pre and post-season testing).The overwhelming majority of football groupôs post-concussion symptoms did not improve. This provides a probability of evidence that AmaTuks football players may sustain concussion or MHI effects (treatment effect) that become evident from only one season of playing football. Post-concussion symptoms reported by football players are headaches, attention and concentration problems, easily getting hurt, experiencing being nervous and anxious and getting worried and being on the edge. Of the 31 symptoms questionnaires, the football group did not fill in 12 while the control group did not fill in 5. The findings of this study reveal that there is a probability that football players are vulnerable to sustaining concussion or CMHI during play, as revealed by the small, but significant, differences in the analysis of the neurocognitive testing and the PCS test.

6.5 Critical evaluation of the study

A number of studies on the neuropsychological effects of sport-related MHI state that there are many methodological limitations as stated in Chapter 4. The following presents a critical evaluation of the present study.

6.5.1 Methodological strengths of the study

Two earlier studies of football players and the control groups did not include biographical details, in particular the age and educational level of both the football and control groups, and did not investigate the effect of CMHI but investigated MHI (Abreau et al., 1990; Echemendia & Julian, 2001). The present study included a non-contact sport control group for comparison purposes. Age was controlled between the football players and control group. However, the control group had more educational years than the football players (12+ years).

Race or ethnicity is traditionally considered to account for a proportion of variance in cognitive test performance (Miller, 1993a). In this study however, there was equivalence for racial distribution between the comparative groups, with the majority of the sample being Black African. There White participant's first language is Portuguese and English was his second language.

A significant strength of the present study is that it evaluated both neurocognitive functioning and symptoms report. Furthermore, this study used an abbreviated version of the CALCAP, a computerised neurological/neuropsychological test battery, because it has been proven to be sensitive to diffuse brain damage. This study is also one of the first in South Africa to focus on the change of symptoms (intensity) of Post-Concussion Symptoms from (baseline) pre to post-season assessment. To date there are no known studies that have assessed pre and post-season Post-Concussive Symptomology in research on CMHI in football players in South Africa.

6.5.2 Limitations of the Study

A quasi-experimental cross-sectional design is used in this study which involved a designated contact sport (football) experimental group and a volleyball non-contact sport control group for comparison purposes. This cross-sectional design was implemented to evaluate the effects of CMHI and Post-Concussive Symptomology between the contact sport (football), as opposed to non-contact sport controls. The control group formed a non-equivalent comparison group (they were non-equivalent on concussion and/or MHI history). They were well matched on the age demographic variable to establish demographic equivalence. However, this is partially eliminated in this study because although the comparative sample groups were statistically matched on the variable of age they were not well matched in terms of years of education. It was anticipated that the two groups would be well matched in terms of a minimum of 12 years of education, as they were attached to a tertiary institution. However, the results revealed a significant difference.

The study was conducted in English and both the football players and the non-contact sports controlsô were second language English speakers. Nell (1999) notes that second language speakers understand nuances in language (for instance, English) differently which could be problematic when interpreting test results when a test or checklist has not been normed or validated against that population. It is possible that the absence of construct equivalence may be an issue in this study.

6.6 Comparative Groups

The experimental group was not randomly selected by the researcher and comprised of a purposive sample of contact sport group of professional football players, who fell under the management of the AmaTuks football administration. The management of the football team acceded to the request for a study to take place. As the sample met the requirements of the study needs it was deemed appropriate, by the professional judgement of the researcher. In order to investigate the neurological effects of concussion injuries in a contact sport groups, it requires non-contact participants who are less susceptible to concussion injuries (Shuttleworth-Edwards & Radloff, 2008). The non-contact volleyball control group were also

not randomly selected. Management of the volleyball team at the University of Limpopo (Medunsa campus) were contacted and consented to take part in the study. The purposive sample was drawn on an ad hoc voluntary basis, from the volleyball playing group thus effects noted in the study might be due to a volunteer sampling bias (selection-maturation).

A small difference, for example, might exist between contact and non-contact sports. Impulsive athletes might choose to play contact over non-contact sport, and the test performance might be the consequence of this variable, rather than the effects of concussion or MHI (Shuttleworth-Edwards & Whitefield, 2007). The inherent differences between contact and non-contact sport groups, irrespective of MHI histories, are often concealed. According to Shuttleworth-Edwards and Radloff (2008), research outcomes based on comparisons between contact sport and control groups in the final analysis remain somewhat speculative. To help negate such effects, an attempt was made to match the comparative group on extraneous demographic variables and the groups were statistically equivalent for age. A limitation in the demographic variables was, as previously noted, the significant difference in education scores between the football players versus the control group. However, this difference was not evident for the overall football group and control groupôs outcome on the neurocognitive assessment. This could be a reason for the general trend of small significant differences in divided attention and short-term memory outcomes for the football group in this study.

Shuttleworth-Edwards and Radloff (2008), note that sport-related concussive studies have usually had small sample sizes, with 20-30 participants. In this study, the sample size was small for both football and the control groups (n = 15 for each group). Despite this, some significant and consistent analysis trends in the one hypothesis direction and in terms of sport-related concussive literature was still identified, which might have been strengthened were the study sample numbers increased.

The football players and control participants in this study were predominantly in their early twenties. Not all of the participants were equal in terms of education. The control group had significantly higher years of education than the football players. However, this difference does not influence the football group negatively in the overall results as there is little overall

difference between the football and the control groups. African languages were the first languages for the majority of the participants rather than the English language, and the distribution of race skewed towards an African Black population. The sample consisted of sportsmen exclusively, but the results cannot be generalised to all sportsmen. The sample is not representative of the South African population, and the findings cannot be generalised to all South African cultural and socio-economic groups. The reasons for this are revealed by the following statistics. According to mid - 2011 estimates from Statistics South Africa, the countryôs population was 50 million of which 52% were females and 48% were male. Africans are in majority at 79.5% while White and Coloured people make up 9, 0% and Indians/ Asians 2.5% of the population. However, according to 2011 census, nearly a quarter of the populationôs home language is isiZulu; Xhosa is 17.6%, Afrikaans 13.3%, Sepedi 9.4%, English and Setswana at 8.2%, Sesotho 7.9% and Tshivenda 2.85%. The average number of formal years of Education years was not available at Statistics South Africa (2011) and the different sports played among the population groups were also not available.

6.7 Research Measures

This study used only two tests, the CALCAP, a computerised neurocognitive test measure and pen and paper Post-Concussive Symptom checklist questionnaire. The majority of sport-related MHI studies used a number of traditional tests (Collins, et al., 1999; Hatfield, Bieliauskas, Begloff, Steinberg & Kauszler, 2004 & Rutherford et al., 2005). This study can be criticised for not tapping into several other neurocognitive functions, by not using traditional tests. According to Waterloo et al., (1997) however, computerised tests are much more sensitive to diffuse brain damage than traditional test. Essentially, this study provided a focused approach to test selection in targeting specific neurocognitive functions, thus decreasing the chances of incurring a Type I error when fewer tests are used. When studies use a large number of tests, the results are considered ñexploratoryò, because this increases the likelihood of significant findings being found as a result of chance (Rutherford et al., 2005). The present study only used four CALCAP test comparisons. On the grounds of using relatively fewer tests, the present study can be considered to be less exploratory and there is a possibility that significant findings are thus not a result of chance.

6.8 Other methodological and statistical concerns for research on MTBI in sport

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

The use of different neuropsychological measures used to assess specific neurocognitive function also makes it difficult to draw comparisons between studies. Furthermore, authors frequently give different interpretations of what functions a specific test measures. Participants who are involved in sports, particularly contact-sports, who are studying, who suffer from work fatigue or have personal or family problems, may show symptomology which suggests MBTI on neurocognitive measures. This makes it is difficult to draw comparisons between the different studies (Shuttleworth-Edwards, Smith & Radloff, 2008).

Below is a description of methodological issues that pertain to sports-related MTBI and thus making it difficult in drawing general inferences.

6.8.1 Practice effects

Practice effects are limitations that are associated with neuropsychological test-retest condition used in the assessment of sports-related MTBI (Bernstein, 1999; Erlanger et al., 2003). Comparisons of studies are difficult because of the practice effects that differ across participantsô personal characteristics and assessments characteristics. Significant practice effects have been evidenced on traditional neuropsychological testing (Erlanger et al., 2003).

6.8.2 Small sample size

Small sample sizes are problematic in sport-related MTBI research. Two studies which both included control groups concluded that there is no significant difference with regard to MTBI sequelae, also used small samples (Putukian, et al., 2000; Stephens, Rutherford, Potter & Ferni, 2005). Small samples are prone to the Type II error, and methodologically it can be argued that these studies failed to find significant effects, when such effects do exist. Generally, statistical power is less in small samples, and therefore even if differences do exist, they fail to be revealed statistically (Shuttleworth-Edwards & Whitefield, 2007). Inferences are difficult to make when small samples are used (Maddocks & Sailing, 1996; Putukian et al., 2000; Rutherford et al., 2005; Shuttleworth-Jordan et al., 1993).

6.8.3 Lack of control for extraneous variables

A small number of studies that included sizeable control groups which disproved the presence of MTBI sequelae did not provide convincing evidence that there was no MTBI (Shuttleworth-Edwards et al., 2004). For example, the studies by Barr (2003) and Guskiewiz et al. (2003) lacked control for the extraneous variables education, intelligence and gender. Studies need to control for confounding factors and match comparative groups on extraneous variables, particularly education and vocabulary that are known to affect test performance (Shuttleworth-Edwards et al., 2004).

Shuttleworth-Jordan (1996) indicates that drastic effects on cognitive tests is highly contentious, and that differences on test performance often attributed to ethnic or cultural differences, are actually the result of differences in educational level. As the socio-cultural gap diminishes, so does differing test performance between races, revealing a basically common neuro-behavioural function on cognitive test attainment. Thus, comparative sample groups should, to a certain extent be matched on educational level, which implies socio-cultural equivalence rather than being matched on race or first language.

6.8.4 Under-reporting of MTBI

The under-reporting of concussion events has major implications for the study of sport-related MTBI for comparative purposes. Contact sport studies investigating the cumulative effects of concussive injuries require non-contact sports controls that are less susceptible to these injuries (Shuttleworth-Edwards & Radloff, 2008). However, these authors note that studies involving comparative contact and non-contact sport controls have used sample sizes of 20 to 30 participants such as (Rutherford et al., 2005; Shuttleworth-Edwards et al., 2004) which may not be sufficient. In these groups it was also thought likely that there was under-reporting of MTBI because of peer, coach and/or parent pressure to perform.

6.9 Implications of the results

6.9.1 General implications

The main findings of this study are as follows: There is no significant difference on cognitive changes among football players and the control group. However, there is a small significant difference for Sequential Reaction Time 1 (on divided attention and short-term memory). The general implication of this study is that there is no significant correlation between the symptoms of Cumulative MHI and cognitive impairment among football players and the non-contact sport controls except for the probability that football players seemed to have a problem with divided attention and short-term memory.

There is also a significant difference on improved PCS between the football players and the control group. The football participantsô symptoms did not improve, and it therefore suggests that there is a probability that football players can experience Post-Concussion Symptoms for example, this study has indicated that football players experienced headaches, attention and concentration problems at post-season testing. These results highlight the effects of selection interaction, where football players indicated a probability that they might have experienced the acute phase of PCS as they reported headaches and attention and concentration problems. The ambiguous (not filled in) frequencies on the PCS questionnaire by both football players and the control group may have impacted on the study results and the conclusions.

The problem of a small sample size would be the failure to identify differences when there are differences (Type II error), and in spite of the small sample size in the present study, the ñimprovedò symptoms and sequential 1 RTôs, neurocognitive measure, results were significant and trends are demonstrated by the statistical analysis, neurocognitive measures of divided attention and short-term memory and also on the improved (intensity) symptoms count. Even though the two groups were matched for gender and age on the reduced sample, a significant difference was still found in education levels. These results did have an influence on the study because the control groupôs PCS improved, whereas football players indicated a probability that treatment did have an effect (Post-Concussive Symptoms) as revealed by their symptom count not improving. This also implies that a higher BRC is likely to act as a protective factor and would therefore decrease the risk of functional impairment and the likelihood of individuals exhibiting symptoms of neurological impairment. This supposition which is underpinned by Satzôs BRC theory is supported by Binder, (1986), Dicker and Maddocks, (1988), Kibby and Long, (1996), Maddocks and Dicker, 1989; Shuttleworth-Jordan, (1999) who noted that education is one of the factors that act as a protective factor against cognitive deficit and mediate the recovery process.

6.9.2 Implications of Satz's (1993) Brain Reserve Capacity Theory (BRC)

The findings of this study give support to the Brain Reserve theory linked to Shuttleworth-Jordanôs (1999), *Shuttle theory,* which proposes that even in the absence of observable or testable negative functional outcomes, mild brain injury can result in a reduction of BRC. The results revealed a probability of the football players having sustained concussion or CMHI. A likely contributing reason for this, is that the football group, overall had 12 years or less of formal education, which suggests they have a lower cognitive reserve than the control group in this study. The education distribution of the football participants was a minimum of 9 years and a maximum of 16 years of educational study, whereas the control groupôs educational minimum years of study was 13 years with a maximum of 17 years. Thus it implies that the football players have comparatively lower cognitive reserve as compared with this studyôs relatively highly educated control group. Therefore, with the sample of football players with lower education, it would appear that their scores became worse because their BRC was not substantial enough to benefit from practice effects.

In spite of the small sample size in the present study, the symptoms getting *better* revealed significant results and trends as reported in the statistical analysis. A significant difference was found for education implying that the results did have an influence on the study because the control groupôs post-concussive symptoms *improved*, whereas football players did not have a marked an improvement in their Post-Concussive Symptoms. This implies that a higher BRC is likely to act as a protective factor and would therefore decrease the risk of functional impairment and the likelihood of individualôs exhibiting symptoms of neurological impairment.

6.10 Implications for future research

Longitudinal neuropsychological studies of football players are needed, using more neuropsychological measures, increased sample number and samples matched demographically for race, premorbid IQ, language and education to properly evaluate the effects of CMHI or concussion and PCS as a result of being involved in a designated contact sport (football).

The findings from the present study were limited to a professional lower educated male contact sports population (football) and therefore cannot be generalised to wider sport populations or generalised to other football populations. Therefore football studies should be carried out amongst educationally diverse populations as well as among female football players and primary and high school football players. Finally, future research should also b required to develop sport-related norms for neuropsychological measures that are based on playersô estimated cognitive reserves or estimated IQ scores that takes practice effects into account.

6.11 Summary

This study utilised a quasi-experimental non-equivalent cross sectional design, using a computerised test and a pen and paper symptoms measure which do not provide evidence that both football players and non-contact sports controls differ on cognitive changes even though, the results suggest, that there is a probability that the football players experienced problems on divided attention and short-term memory. According to BRC theory, the professional football sample was a lower functioning group of sportsmen, in terms of education, which renders them vulnerable to exhibiting symptoms of neurological impairment. Thus, sports management should assess football players at baseline (pre-season) and withdraw a football player from the game whenever he experiences a head injury. Before returning to play players should be assessed again to ascertain if they are asymptomatic. Neuropsychological follow-up and good medical care should be provided to players who experience concussion or head injury during practice or actual matches.

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Biographical and pre assessment questionnaire **APPENDIX A**

This questionnaire is entirely confidential and will be used by Ms. P. Maite to access your potential risk for neurological injury from concussive injury in soccer. Please make a cross (X) next to the correct answer (Yes or No) or answer the question in the space as required.

Football Research

Pre-assessment questionnaire

NAME:_____FIRST LANGUAGE:_____

AGE:_____D.O.B:_____

UNIVERSITY YEAR OR SCHOOL STD PASSED:_____SPORT: FOOTBALL/VOLLEYBALL

OTHER:_____

TEAM:_____ POSITION CURRENT:_____

MOST FREQUENTLY PLAYED PAST POSITION:_____

GENERAL HISTORY

Question 1

Have you ever failed a standard at school OR UNIVERSITY? Yes No

If **Yes**, when? _____For what reason?_____

If you did not complete matric why? _____

What was your overall grade in when you completed matric? _____

How many of your subjects did you take on Standard Grade _____/ Higher Grade?_____

Question 2

Have you ever experienced learning difficulties or required remedial classes? Yes No

If **Yes**, what was the problem?_____

Question 3

Have you ever experienced neurological problem (e.g. seizures, tremors, stroke)? Yes No

If **yes**, what was the problem?_____

Question 4

Have you ever suffered from a psychological/ psychiatric disorder? (e.g. Depression, Anxiety, Attention Deficit or

Hyperactivity)? Yes No

If **Yes**, what was the problem?_____

Question 5

Are you currently taking any form of medication? Yes No

If **Yes**, please specify?_____

Question 6

Do you smoke? Yes No

If **Yes**, how much?_____

Question 7

Do you drink alcohol? Yes No

If **Yes**, how much?_____

Have you felt that you should cut down on your drinking? Yes No

Question 8

Do you use any other substances? Yes No

If **Yes**, specify type and frequency of use?_____

Question 9

Have you ever sustained a head injury or concussion that was not related to sport (e.g. motor vehicle accident)

Note to examiner: **DO NOT INCLUDE SPORTS-RELATED INJURIES HERE**.

Yes No

If **Yes**, date/s? Injury 1

_____**2**_____

Injury 1

- What caused the injury/concussion?_____

_____

_____

- Did you lose consciousness? Yes No

If **Yes**, for how long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

Were you hospitalized? Yes No

If Yes, for how long?_____

Injury 2

What caused the injury/ concussion?_____

_____

_____

Did you lose consciousness? Yes No

If Yes, for how long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

Were you hospitalized? Yes No

If Yes, for how long?_____

SPORTS HISTORY

Question 10

Football Players

- At what age did you first start playing football?
- Have you ever played any other sport for a length period of time?

(For football players, check whether they have participated in BOXING and RUGBY)
(For volleyball players, check whether they have participated in BOXING and RUGBY)

Yes No

If Yes, specify sport and time period played?_____

Question 11 (football players only)

How many times can you remember sustaining a head injury or concussion during a game of soccer, including occasions when you were knocked or ñdingedò so hard that you felt dazed, confused and or disoriented, even though you continued to play in the game? (Note to examiners: Try to ascertain the specific incidences of injury, beginning with the most recent, followed by other incidences in as consecutive an order as the subject can recall.)

If **Yes**, specific date/s? Injury 1_____Injury 2_____

Injury 3_____Injury 4_____

Injury 5_____

Injury 1

What caused the injury/
concussion?_____

_____

___

_____

___

Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how
long?_____

Did you lose consciousness? Yes No

If Yes, for how
long?_____

Did you lose your memory? Yes No

If **Yes**, for how
long?_____

Were you taken off the field? Yes No

If **Yes**, for how
long?_____

Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify?_____

Injury 2

What caused the injury/ concussion?_____

- Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how long?_____

- Did you lose consciousness? Yes No

If Yes, for how long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

- Were you taken off the field ? Yes No

If **Yes**, for how long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify?_____

Injury 3

What caused the injury/ concussion?_____

_____

_____

- Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how long?_____

- Did you lose consciousness? Yes No

If Yes, for how long?_____

- Did you lose your memory? Yes No

If **Yes**, for how long?_____

- Were you taken off the field? Yes No

If **Yes**, for how long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify?_____

Injury 4

What caused the injury/ concussion?_____

_____

_____

- Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how long?_____

- Did you lose consciousness? Yes No

If Yes, for how long?_____

- Did you lose your memory? Yes No

If **Yes**, for how long?_____

- Were you taken off the field? Yes No

If **Yes**, for how long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

- Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify?_____

Injury 5

What caused the injury/ concussion?_____

- Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how long?_____

- Did you lose consciousness? Yes No

If Yes, for how long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

- Were you taken off the field ? Yes No

If **Yes**, for how long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify?_____

Question 12

What other injuries have you sustained while playing soccer (e.g. facial injuries, sprains, fractures)?

Please specify._____

_____

Question 13

Have you ever sustained a head injury or concussion while playing a sport other than soccer?

Yes No

If **Yes**, specify which sport/s and date/s_____

Injury1_____ Injury 2_____

Injury 3_____ Sport_____

Sport_____ Sport_____

Injury 1

What caused the injury/
concussion?_____

- Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how
long?_____

- Did you lose consciousness? Yes No

If Yes, for how
long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

- Were you taken off the field ? Yes No

If **Yes**, for how long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify?_____

Injury 2

What caused the injury/ concussion?_____

- Where you dazed, confused and/ or disoriented? Yes No

If Yes, for how long?_____

- Did you lose consciousness? Yes No

If Yes, for how long?_____

Did you lose your memory? Yes No

If **Yes**, for how long?_____

- Were you taken off the field? Yes No

If **Yes**, for how long?_____

- Were you hospitalized? Yes No

If **Yes**, for how long?_____

Did you have any other neurological symptoms (e.g. seizures, weakness of limbs, tremors)?

Yes No

If **Yes**, please specify_____

Rivermead Post-Concussion Questionnaire (King et al., 1995)

Appendix B

PLEASE ANSWER EACH OF THE FOLLOWING QUESTIONS INDICATING THE DEGREE TO WHICH THE QUESTION APPLIES TO YOU NOW

NAME:_____

1	Do you suffer from headaches?	0 Never	0 Sometimes	0 Often
2	Do you have poor eyesight?	0 Never	0 Sometimes	0 Often
3	Do you have difficulty hearing?	0 Never	0 Sometimes	0 Often
4	Do you experience weakness in your Limbs?	0 Never	0 Sometimes	0 Often
5	Are you Clumsy?	0 Never	0 Sometimes	0 Often
6	Do you have fits or seizures	0 Never	0 Sometimes	0 Often
7	Do you become dizzy?	0 Never	0 Sometimes	0 Often
8	Do you become tired easily?	0 Never	0 Sometimes	0 Often
9	Are you sensitive to noise?	0 Never	0 Sometimes	0 Often
10	Are you ever felt that you were seeing, hearing or feeling unusual things?	0 Never	0 Sometimes	0 Often
11	Are you experiencing any sexual problems?	0 Never	0 Sometimes	0 Often
12	Do you have any problems with your speech?	0 Never	0 Sometimes	0 Often
13	Do you stumble over your words when you speak?	0 Never	0 Sometimes	0 Often
14	Do you stutter or stammer?	0 Never	0 Sometimes	0 Often
15	Do you slur your words?	0 Never	0 Sometimes	0 Often
16	Do you have memory difficulties?	0 Never	0 Sometimes	0 Often
17	Do you have problems with attention and concentration?	0 Never	0 Sometimes	0 Often
18	Does your attention wander while following a conversation or when you are watching TV or reading?	0 Never	0 Sometimes	0 Often
19	Are you impatient?	0 Never	0 Sometimes	0 Often

20	Are you irritable?	0 Never	0 Sometimes	0 Often
21	Do you become easily angry or hurt?	0 Never	0 Sometimes	0 Often
22	Do you feel sad or down in the dumpsĉ or depressed?	0 Never	0 Sometimes	0 Often
23	Do you enjoy seeing your friends and having social contact?	0 Never	0 Sometimes	0 Often
24	Do you suffer from restlessness?	0 Never	0 Sometimes	0 Often
25	Do you have problems sleeping?	0 Never	0 Sometimes	0 Often
26	Is there a problem with your appetite?	0 Never	0 Sometimes	0 Often
27	Do you feel nervous or anxious?	0 Never	0 Sometimes	0 Often
28	Do you feel worried or on edge?	0 Never	0 Sometimes	0 Often
29	Are you argumentative?	0 Never	0 Sometimes	0 Often
30	Are you feeling short tempered?	0 Never	0 Sometimes	0 Often
31	Do you become aggressive for no apparent reason?	0 Never	0 Sometimes	0 Often

APPENDIX C

UNIVERSITY OF LIMPOPO (Medunsa Campus) ENGLISH CONSENT FORM

Statement concerning participation in the Research Project.

Name of Research Study:

Cumulative mild head injuries in football players: a comparison of cognitive and post-concussive symptomatology between the University of Pretoria football players (AmaTuks) and a University of Limpopo volleyball playing control group.

I have read the information on the aims and objectives of the proposed study and was provided the opportunity to ask questions and given adequate time to rethink the issue. The aim and objectives of the study are sufficiently clear to me. I have not been pressurized to participate in any way.

I understand that participation in this Clinical Study is completely voluntary and that I may withdraw from it at any time and without supplying reasons. This will have no influence on the regular treatment that holds for my condition neither will it influence the care that I receive from my regular doctor.

I know that this Study has been approved by the Medunsa Campus Research and Ethics (MCREC), University of Limpopo (Medunsa Campus). I am fully aware that the results of this Study will be used for scientific purposes and may be published. I agree to this, provided my privacy is guaranteed. Please contact me via my Promoter at knel@ul.ac.za if you have further queries.

I hereby give consent to participate in this study

....

Name of patient/volunteer Signature of patient or guardian.

....

Place. Date. Witness

Statement by the Researcher

I provided verbal and written information regarding this study

I agree to answer any future questions concerning the Study as best as I am able.

I will adhere to the approved protocol.

....é é .....

Name of Researcher Signature Date Place

Name of Promoter Signature Date Place

Approval Letter from University of Pretoria APPENDIX D

APPENDIX E

List of radiological assessments

Computerised Tomography (CT)

Is a specialised X-ray that is also known as (CAT) Computer Axial Tomography. It is an x-ray procedure that combines many x-rays images with the help of a computer to generate cross-sectional views.

Magnetic Resonance Imaging (MRI)

It is a non-invasive medical test used for looking at soft tissue. It is mostly used to study the brain and the spinal cord.

Electroencephalography (EEG)

It is a test that measures and records the electrical activity of the brain by using sensors (electrodes) attached to the head and hooked by wires to the computer

List of Psychological/neuropsychological tests **APPENDIX F**

Cambridge Neuropsychological Test Automated Battery (CANTAB)

CANTAB was originally developed at the University of Cambridge in the 1980s. It is a cognitive computerised battery of neuropsychological tests, administered to subjects using a touch screen computer. It measures motor skills, visual attention, spatial memory, working memory and nonverbal memory span. It also assesses executive function, which is the highest level of cognitive function that manages planning and allows flexible thought and action. CANTAB consists of twenty two (22) tests.

Symbol Digits Modalities (SDMT)

The Sub-Test primarily assesses complex scanning and visual tracking, with the added advantage of providing a comparison between visual motor and oral responses. The SDMT is a five minute assessment measure that quickly screens participants for any kind of cerebral dysfunction by using a simple substitution task. It is easy to administer and accurate when detecting the presence of brain damage and other changes in a patientôs cognitive function ing. It is effective for those with cerebral dysfunction as they will always perform poorly due to deficiencies in attention span, scanning abilities and motor skills.

Trail Making Test (TMT)

The test was originally developed in 1938 as the Divided Attention Test. It was adapted in 1955 by Reitan and it added to the Halstead Battery. It requires the subject to connect , by making pencil lines, 25 en circled numbers randomly arranged on a page in a proper order (Part A) and twenty five (25)encircled numbers and letters in alternating order (Part B). It is a test of complex visual scanning that has a motor component such that motor speed and agility make a strong contribution to success and itôs vulnerable to the effects of brain injury. The test consists of two forms: the child (Intermediate) Form for ages 9 to 14 years and the Adult form for ages 15 years and older.

Finger Tapping

Finger Tapping measures are included in neuropsychological examinations in order to assess subtle motor and other cognitive impairments. The finger Tapping was originally called The Finger Oscillation Test and was part of Halsteadôs (1947) test battery. It is a timed speed test of manipulative agility. It consists of a tapping key with a device for recording the number of taps. Each hand makes 5-10 seconds trials with short rest periods between the trials. It tends to have a slowing effect on finger tapping if subjects have brain disorder.

Purdue Pegboard Test

It assesses manual dexterity for employment selection and it can help in identifying lateralised impairment. The pegboard is designed to test hand dexterity. It specifically tests the gross hand movement and fingertip dexterity. It consists of a pegboard and a collection of pins, washers and collars. The participant manipulates the pins and collars and inserts them into the boardôs holes according to the test routine.

Digit Supra Test

It measures verbal learning and memory including immediate and delayed recall. It is rote memorisation in sequence of an eight or nine digit number exceeding immediate memory span.

Paced Auditory Serial Addition Test (PASAT)

The test was devised to provide an estimate of speed of information processing in head injured individuals. In all versions of the PASAT, random series of numbers from 1 to 9 are presented and the subject is instructed to consecutively add pairs of numbers such that each number is added to the first, the third number to the second number the fourth number to the third number etc. The response required is sustained over the numerous items until the end of the trial the interstimulus is then decreased and the same process is repeated.

Stroop Word Colour Test

It is based on the findings that it takes longer to call out the colour names of coloured patches than to read words and even longer to call out the colour of the ink in which a colour name is pointed when the print ink is a different colour than the colour name.

Alphabet backwards

It is a three-item test of mental tracking that requires the subject to (1) count backwards from 20 in 30 seconds; (2) repeat the alphabet in 30 seconds.

Continuous Performance Test (CPT)

It is a computerised vigilance test that normally presents brief stimuli and provides reaction time as well as accuracy data. It measures sustained or waning attention over a relatively long time. Letters of the alphabet appear briefly in random order in the centre of the screen. In the simple condition, participants are asked to respond to every X and in the more difficult version, X only if it follows A. Even though CPT is meant to measure sustained attention, a failure may occur due to different reasons, namely impulsivity, anxiety and environmental noise.

Perceptual Speed Test

The main objective of this test is to identify how fast and accurate an individual can check thing is his/ her head.

Example:

Part 1	Part 2	Part 3	Part 4
E e	Q y	D d	K k

1 2 3 4

In this case, you will see four pairs of letters. Each pair has been put in its own box. You must decide how many pairs contain letters that are the same.

The Weschler Adult Intelligence Scale-Third Edition (WAIS-R)

The Weschler Adult Intelligence Scale-Third Edition (WAIS-III) evolved from the Weschler-Bellevue Intelligence Scale in 1939. It was then revised and renamed Weschler Adult Intelligence Scale (WAIS) in 1955, which was revised in 1981 as WAIS-R). It is an instrument used for assessing intellectual ability and it is also appropriate for a number of purposes. It can be used as a psycho-educational test for secondary and post-secondary school planning. It is also useful for differential diagnosis of neurological and psychiatric disorders affecting mental disorders. Digit Span, The Digit Symbol Substitute Test and Picture Completion Subtest are the WAIS-R III subtests.

Digit Span

Digit Span is composed of two tasks administered independently of each other, namely Digits Forward and Digits Backward. For both tasks,, the examiner read a series of number sequences to the participant, for each digit forward item, the participants is required to repeat the number sequence in the same order as presented. For digits backwards, the participant is required to repeat the number sequence in the reverse. It assesses immediate verbal recall and involves auditory attention.

Digit Symbol Substitution Test

It is also contained in the WAIS-R and it is called Digit Symbol. It explores attention and psychomotor speed. It has a code table that displays the correspondence between pairs of digits (1-9) and symbols. Subjects have to fill in blank squares with the symbol that is paired to the digit displayed above the square. The subjects have to fill in as many squares as possible in 90 seconds. For administration see Digits Symbol above.

Picture Completion Subtest

Picture Completion test is a visual reasoning test which involves both visuo-perceptual and verbal abilities. It is potentially good indicator of premorbid ability as it consistently demonstrates resilience to the effects of diffuse cerebral damage. The test consists of twenty five (25) Picture Completion Items in the stimulus Booklet. For each item in the subtest, the examinee views a picture and then points to or names the important part that is missing from the picture. The participant should respond to each item within a twenty (20) second time limit.

Weschler Intelligence Scale for Children Revised (WISC-R)

WISC-R is a revised edition that was published in 1974 as the WISC-R (Wechsler, 1974), featuring the same subtests however the age range was changed from 5-15 to 6-16. The third edition was published in 1991 as WISC-III; and it brought with it a new subtest as a measure of processing speed. The original WISC, Wechsler, 1949 was an adaption of several of the subtests which made up the Wechslerï Bellevue Intelligence Scale in 1939 but it also featured several subtests that were designed specifically for it. The subtests were organized into Verbal and Performance scales, and provided scores for Verbal IQ (VIQ), Performance IQ (PIQ), and Full Scale IQ (FSIQ). The WISC is one of a family of Wechsler intelligence scales.

Denkla Finger Tapping

As brain damage tends to have a slowing effect on finger tapping rate, bilateral slowing would indicate diffuse brain damage in the absence of any physical impairment. The participant is instructed to place both elbows on the table (researcher demonstrates what is required) and with one hand at a time, to touch each finger to the thumb beginning with the index finger as quickly as they can. The score is the number of seconds taken by the participant to do five sets of sequential taps.

Four-Choice Reaction Time

It measures psychomotor performances. There are four basic means of measuring RT that give different operational conditions during which a participant has to provide a desired response, namely Simple reaction time is the time required for an observer to respond to the presence of a stimulus. Recognition reaction time tasks require that the subject press a button when one stimulus type appears and withhold a response when another stimulus type appears. For example, the subject may have to press the button when a green light appears and not respond when a blue light appears. Choice reaction time (CRT) tasks require distinct

responses for each possible class of stimulus. For example, the subject might be asked to press one button if a red light appears and a different button if a yellow light appears. Discrimination reaction time involves comparing pairs of simultaneously presented visual displays and then pressing one of two buttons according to which display appears brighter, longer, heavier, or greater in magnitude on some dimension of interest.

Immediate Post-Concussion Assessment and Cognitive Test (ImPACT)

Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) is the first most-widely used and scientifically validated computerized concussion evaluation system. ImPACT was developed in the early 1990's by Drs. Mark Lovell and Joseph Maroon. It is a twenty (20) minutes test that has become a standard tool used in a comprehensive clinical management of concussion for athletes of all ages. ImPACT can be administered by an athletic trainer, school nurse, athletic director, team doctor or psychologist, provided that they have completed training in the administration of the test.

CogState Sport

It is a computerised concussion test and management system for use by professional, elite and amateur athletes used in fifty (50) countries; It is used to assess sensitive to mild cognitive changes and helps guide medical decisions about return to activity and rehabilitation. Early research and development for CogState Sport occurred in one of the toughest sports in the world - Australian Football. It is presently used by many of the world's elite contact sporting organisations.

The Attentional Network test (ANT)

ANT was developed by Fan and his colleagues in 2002. It is designed to measure the function of three distinct attentional networks, namely alerting, orienting and executive control. The attention network test (ANT) is a brief computerized battery that measures three independent

behavioural components of attention, namely conflict resolution (ability to overcome distracting stimuli), spatial Orienting (the benefit of valid spatial pre-cues), and Alerting (the benefit of temporal pre-cues). Imaging, clinical, and behavioural evidence demonstrate hemispheric asymmetries in these attentional networks.

Line Bisection

Line Bisection Test is a quick measure to detect the presence of unilateral spatial neglect. The purpose of this test is to look for improvements in the right hemisphere activation. To complete the test, one must place a mark with a pencil through the center of a series of horizontal lines. The closer the patient ticks to the center the line, the better the functioning. If the ticking of the center is greatly displaced to the right of the line, this would be an indication of poor right lobe functioning. Normally a displacement of the bisection mark towards the side of the brain lesion is interpreted as a symptom of neglect.

Letter cancellation

It is a pencil and paper tests that is widely used in clinical and research settings as a quick measure of attention/ concentration, visual-spatial dysfunctions such as spatial neglect.

Motor Free Visual Perception

It is an individually administered test designed to assess overall visual perception ability. Perceptual tasks include spatial relationship, visual discrimination, figure-ground, visual closure and visual memory. Performance in these areas provides a single score that represents the individualôs general visual perceptual ability. Administration and scoring of the test can be completed in approximately twenty minutes. The norms are based on children and adults living in the United States.

Reaction Time (RT) with distraction

Studies reviewed by Welford in 1980 and Broadbent in 1971 showed that distractions increase reaction time. Trimmel and Poelzl (2006) found that background noise lengthened reaction time by inhibiting parts of the cerebral cortex. Richard et al. (2002) and Lee et al. (2001) found that college students given a simulated driving task had longer reaction times when given a simultaneous auditory task. They drew conclusions about the safety effects of driving while using a cellular phone or voice-based e-mail. Horrey and Wickens (2006) and Hendrick and Switzer (2007) had similar conclusions about cell phone use while driving, and said that hands-free phones did not improve reaction time performance.

Sport Concussion Assessment Tool (SCAT)

The first Sport Concussion Assessment Tool (SCAT) for sports clinicians designed to assess concussion.

Sport Concussion Assessment Tool 2 (SCAT 2)

It was designed as a practical assessment tool pertaining particularly to the clinical evaluation and evolution of the concussed athlete as it was discussed at the Zurich Consensus Meeting in 2008. It has now become an international template for clinicians and is widely distributed in its original format.

The Sports Concussion Office Assessment Tool (SCOAT)

It is a clinical evaluation tool that evolved from the SCAT 2 by retaining the important features relevant to acute, sub-acute and subsequent presentations. It is designed to be used by clinicians in a consulting room environment and it excludes those aspects of SCAT 2 only relevant to the field side assessment of concussion.

Rey-Osterrieth Complex Figure Test

It is a neuropsychological assessment in which participants are asked to reproduce a complicated line drawing, first by copying and then from memory. Many cognitive abilities are needed for correct performance. The test therefore permits the evaluation of different

functions, like visuo-spatial abilities, memory, and attention, planning and working memory (executive function).

Wisconsin Card Sorting Test (WCST)

The WCST was originally developed to assess abstract reasoning and ability to shift cognitive strategies in response to environmental changes. Over the years, it has gained popularity as a clinical neuropsychological instrument. It provides objective measures for overall ability and particular sources of difficulty for instance, inefficient initial conceptualization, perseveration, failure to maintain set, and inefficient learning across several stages of the test. It is also one of the few tests that show specific sensitivity to brain lesions involving the frontal lobes. The test uses stimulus and response cards that show different forms in various colours and numbers. Individually administered, it requires the participant to sort the cards according to different principles (that is, by colour, form or number). As the test progresses there are unannounced shifts in the sorting principles which require the client to change his or her approach.

Wechsler Memory Scale

It is a neuropsychological test designed to measure different memory functions. it is made up of seven (7) subtests namely, Spatial Addition, Symbol Span, Design Memory, General Cognitive Screener, Logical Memory, Verbal Paired Associates and Visual Reproduction. Participantôs performance is reported as five index score, namely Auditory Memory, Visual Memory, Visual Working memory, Immediate Memory and Delayed Memory.