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Cumulative Mild Head Injury (CMHI) in Contact Sports

Kathryn Nel and Saraswathie Govender

Abstract

The effect of cumulative mild head injury (CMHI) in contact sports such as rugby union and football (soccer) is seen at all levels as more concussive injuries are reported each year globally and in South Africa. This is problematic as repeated concussions may lead to cognitive deficits in attention and poorer overall cognitive profiles both in the short and long term. The aim of this chapter is to present a brief review of research on CMHI in football and rugby and other sports (briefly) both international and South African underpinned by an overview of the anatomy and neuroanatomy of the brain to illustrate the mechanisms involved in head injuries. Risk factors for all types of MHI are also given.

Keywords: rugby (union), football (soccer), concussion

1. Background

Concussions, described as a traumatically induced disturbance of brain function involving complex pathophysiological processes, are a major concern in a number of contact and collision sports. Multiple concussions can be referred to as cumulative mild head injury (CMHI), and are problematic particularly if athletes have competed from a very young age. There is a concern about the sequelae of repeated concussions which has gained prominence in sports psychology [1].

Rugby union and football are examples of designated contact sports where this occurs as a result of collision injuries and in football because of repeated heading of the ball [2]. Rugby union (hereafter referred to as rugby) players (both backs and forwards) are involved in tackling where head to head, head to body or head to ground impact occurs also resulting in concussive injury [3].

Various kinds of head injury occur ranging from severe to mild. Traumatic Brain Injury (TBI) refers to a catastrophic event in which a closed or open head-injury results in serious neural damage which causes permanent cognitive damage [4]. Lack of oxygen to the brain (anoxia) and Cerebrovascular Accidents (CVAs), commonly called 'strokes,' also fall under TBIs.

This chapter focuses on CMHI in the contact sports soccer (hereafter referred to as football) and rugby union (rugby). A description of CMHI will be undertaken however, in order to contextualise this properly TBI and MHI (sometimes called Mild Traumatic Brain Injury—MTBI) will be described. After this a brief review of research, referring to international studies generally and South African studies in particular will be undertaken with specific reference to those involving the author(s).

Nonetheless, international studies on rugby, football and other contact sports are included. It must be noted that this is a complex topic which, cannot be covered comprehensively in a chapter, thus it is a contextual overview of the subject.

1.1 Head injury

Traumatic Brain Injury (TBI) is a catastrophic brain injury. The vast majority of TBIs are closed meaning that the brain is not exposed (the skull is not opened). Closed head injuries (CHIs) are usually called blunt head trauma injuries. This means that the skull can have a fracture but the injury is still closed. Penetrating head injuries (PHIs) are referred to as open head injuries. PHI can include injuries from any source in which the skull and dura are penetrated. The term TBI also encompasses other aetiologies for instance, CVAs (cerebrovascular accidents or strokes) and lack of oxygen to the brain (anoxia) which can be catastrophic [4].

1.2 Frontal lobes of the brain

The frontal lobes of the brain are very vulnerable to damage because of their position (at the front of the head = forehead). Damage to these lobes can be caused by illness (for instance, viral meningitis) or any kind of head injury from a blow to the head caused by a fall, being hit with an object or repeated blows to the head (for instance, in a sport such as boxing). A blow to another part of the head can also cause damage to the frontal lobes of the brain. This happens because the brain is not attached to the inside of the skull and moves around when a head injury is incurred. When the skull hits the back of the head the brain moves and hits the bony protuberances in the skull. This causes bruising (or bleeding) in the brain from slight to catastrophic, depending on the force of the blow to the skull [4, 5]. **Figure 1** is used to illustrate where the frontal lobes of the brain lie illustrating their larger size, which makes them vulnerable to damage from multiple contexts for instance, MVAs (Motor Vehicle Accidents), to illness (Meningitis) and/or sporting injuries (Concussion and CMHIs).

1.3 Open head injury

An open head injury occurs when the skull is penetrated by force which results in a perforated skull [5]. Damage that is incurred is usually in the pathway of the foreign object which often results in the exposure of the intra-dural contents of the brain [6]. Damage may occur because of tangential injuries when something strikes the skull and bone fragments are driven into the brain. These objects may pass through the brain or become embedded in it for instance, bullets causing either/or entrance and exit wounds [5]. There is specific neurological symptomology associated with different types of wounds in this regard [7]. For instance, severe scalp wounds resulting in loss-of-blood may cause low blood pressure (hypotension) and gunshot wounds can cause severe bruises in the brain (contusions) particularly where the enter and leave the skull (countercoup sites). The brain swells and fill with blood and intracranial haematomas can occur [4–6] (**Figure 2**).

1.4 Closed head injury

There are various causes of closed-head injury however, the most common cause is when the skull is injured and the brain suffers acceleration or deceleration and/or both [8]. This can happen in sport for instance, when the skull is hit by something moving quickly such as ball or bat of some kind. This type of accident commonly

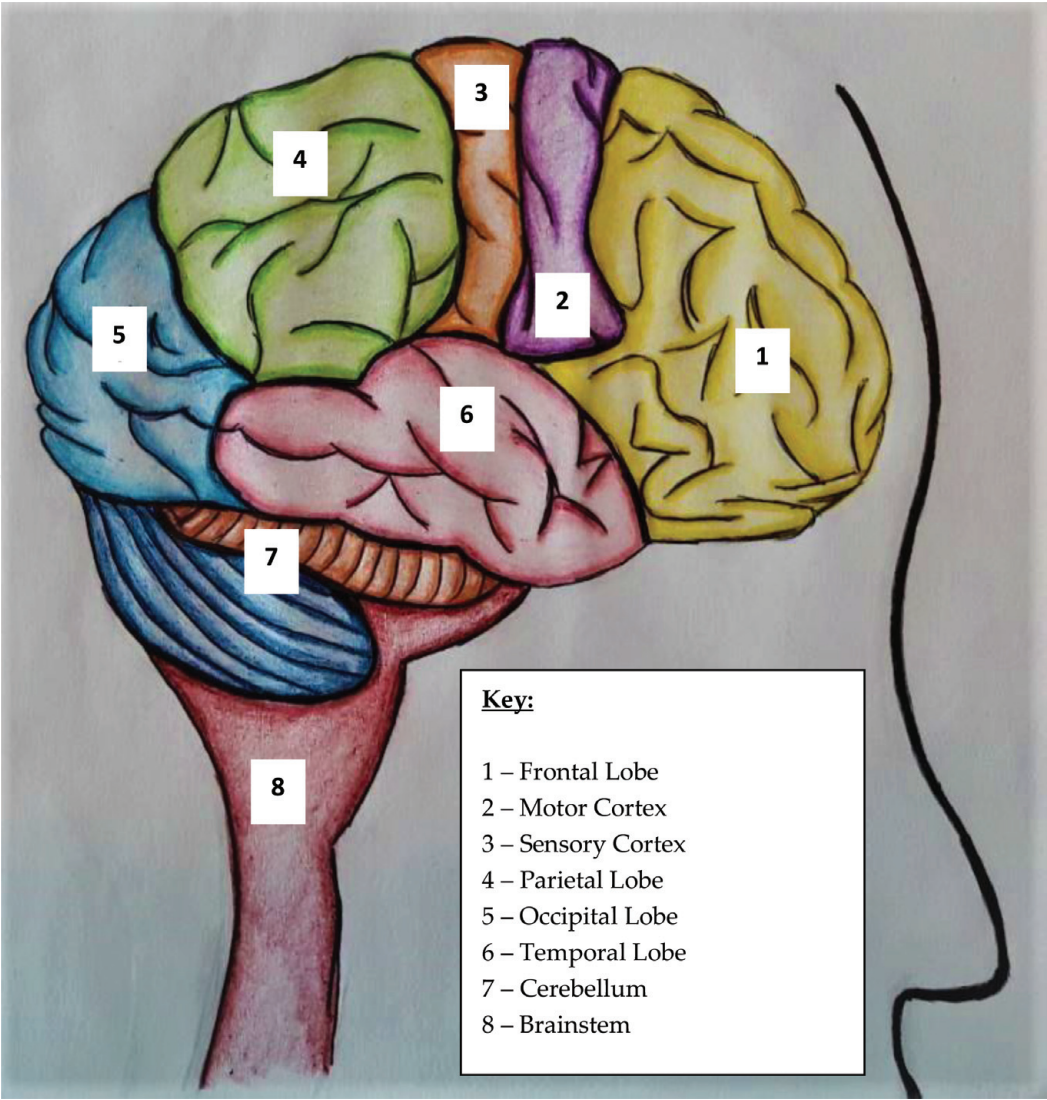


Figure 1.
Lobes of the brain.

occurs in MVAs when a fast moving vehicle stops suddenly which can cause anything from mild to massive brain trauma. However, repeated blows to the head in sport can cause mild brain injury (CMHI) which can be chronic rather than acute and, as a result, the injury effects may not be noticed [9, 10] (Figure 3).

1.5 Mild head injury (MHI)

Mild head injury (MHI) and its effects are controversial particularly with regard to definition and classification. For instance, the classification of mild to moderate head injuries is problematic in the research field [11]. MHI refers to an injury in which loss of consciousness (LOC) and/or Post Traumatic Amnesia (PTA) is quite brief and where there is no pathology (or noticeable injury) to the skull [12]. Criterion used to define MHI is underpinned by states of consciousness defined by the Glasgow Coma Scale (GCS) [13]. Problematically, when these are used to define CMHI or MHIs they are unreliable. A classification of MHI in terms of a LOC lasting 30 minutes or less, which is not linked to more neurological symptomology, is often used [14].

A broader definition of MHI includes different grades of injury and: (a) any period of LOC for less than 30 minutes, with GCS of 13–15 following the LOC; (b) any loss of memory for events immediately before or after the accident with

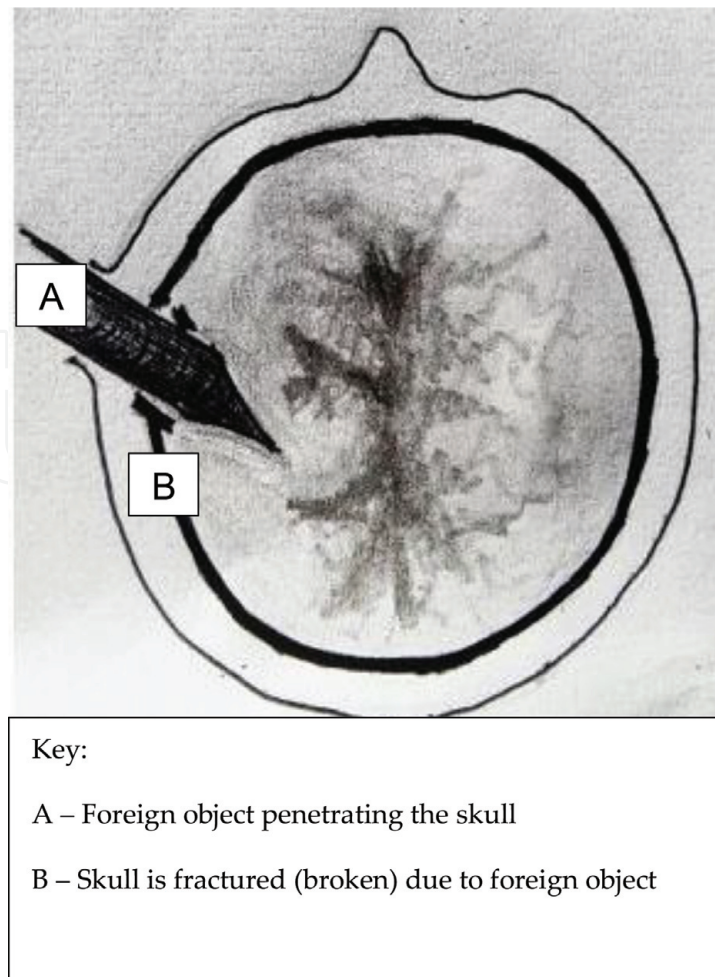


Figure 2.
Schematic diagram of an open head injury.

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 [11].

1.6 Cumulative mild head injury (CMHI)

There is increasing evidence that CMHI can cause more neuropsychological impairment as a result of neural attrition, which can cause athletes problems later in life [15]. Cumulative damage to hippocampal cells can cause cognitive damage [16]. Moreover, CMHI which occurs over months or years is likely to cause neurological and cognitive deficits [17]. The effects of MHI and concussions in the sporting arena are likely cumulative which has significant implications for athletes who play contact sports where concussion and CMHI occur frequently [18, 19]. Research suggests that permanent cognitive deficits are increasing as a result of CMHI [20].

1.7 Concussive injuries (Concussion)

Concussion is any head injury which causes headaches and/or changed levels of consciousness. Fundamentally, an immediate change in neurological functioning because of a blow or injury to the head which results in diffuse axonal injuries (DAI) in the brain structures [20]. This suggests that even short-lived impairment to neural function, after a head injury resulting in a LOC or alteration of consciousness, disturbance of vision and/or equilibrium, is referred to as a concussion. Concerns about the various concussion categories making medical and other research

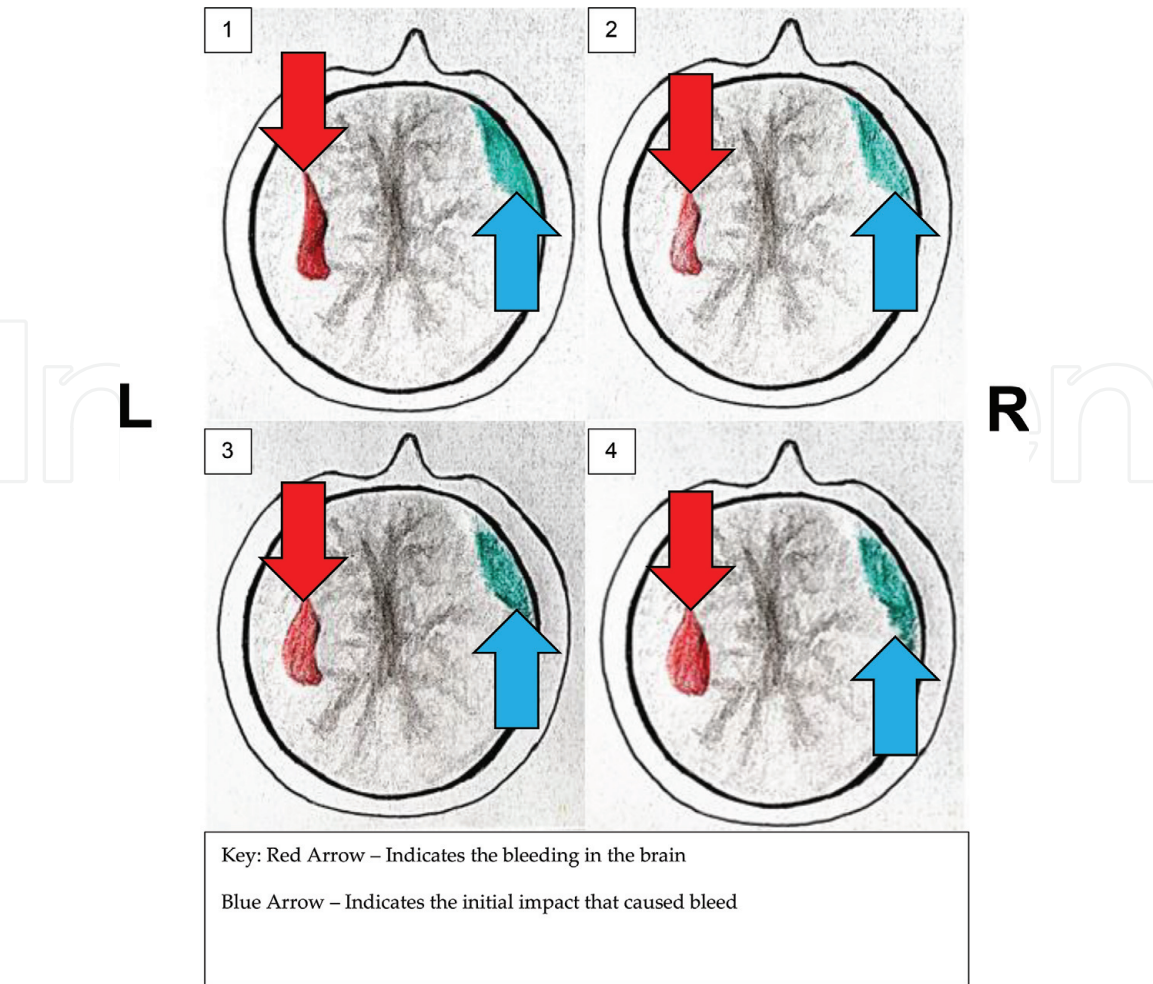


Figure 3.
Schematic representation of a closed head injury.

difficult resulted in guidelines for cerebral concussion [21]. Grade 1 (mild) = transient confusion; no loss of consciousness with symptoms resolving in 15 minutes or less; Grade 2 (moderate) = transient confusion, no loss of consciousness and symptoms lasting longer than 15 minutes and lastly, Grade 3 (severe): any loss of consciousness (brief or prolonged) [22].

The authors in this chapter often use terms like concussion, mild head injury (MHI), mild traumatic brain injury (MTBI) and cumulative mild head injury (CMHI) interchangeably as in the sporting arena to all intent and purpose they often refer to the same thing depending on the sporting code and/or country the injury.

At the first International Conference of Concussion it was stated that concussion was a complex pathophysiological caused by biochemical forces impacting on the brain [23]. The definition included the following concussion: (1) may be caused by a blow to the head face, neck, or elsewhere with force that is transferred to the brain; (2) characteristically causes the speedy onset of brief impairment of neurological functions that resolve spontaneously; (3) results in neuropathological changes however, acute clinical symptomology reflects functional disturbances as opposed to structural injury; (4) is a set of clinical syndromes that sometimes (but not always) involves a LOC. Symptomology is resolved following a specific sequence and (5) it is characteristically associated with fundamentally normal structural neuroimaging. A later addition to this was that in some cases post-concussive symptoms can be protracted and persistent [19]. It is used to refer to a closed, MHI such as those incurred by athletes who play contact sports. It falls within the ambit

of MHI and CMHI and can be difficult to detect as symptoms can last from a few seconds to minutes [5]. In this chapter this type of head injury denotes any impact to the head which often go un-reported (**Figure 4**).

1.8 Occurrence of mild head injury (MHI)

Reliable statistics about the occurrence (prevalence) of MHIs that are closed are quite difficult to ascertain. This is as a result of different names for instance, mild, minor, moderate, and minimal being applied to this type of head injury.

The incidence of MHI is difficult to determine because the majority of country health surveys only look at patients who have been hospitalised. This means that patients who have suffered a MHI or CMHI are not included in survey data. The International Classification of Diseases (ICD 10, 2010) has specific terms of reference for instance, for maxillofacial injuries and scalp lacerations but do not specify CMHI or MHI. Individuals admitted to hospitals who have multiple injuries are usually classified in terms of their most severe injuries (thus an MHI goes unreported [24]).

In South Africa there are few statistics on MHI however, an average of 316 per 100,000 incidents of brain injuries per year was reported in the early nineties [25]. It is estimated for instance, that up to 89,000 brain injuries (mostly TBIs) are seen per year in the country [26]. Moreover, rugby has the highest incidence of concussion amongst contact sport with up to 50% of athletes suffering from a concussion in their playing careers [11].

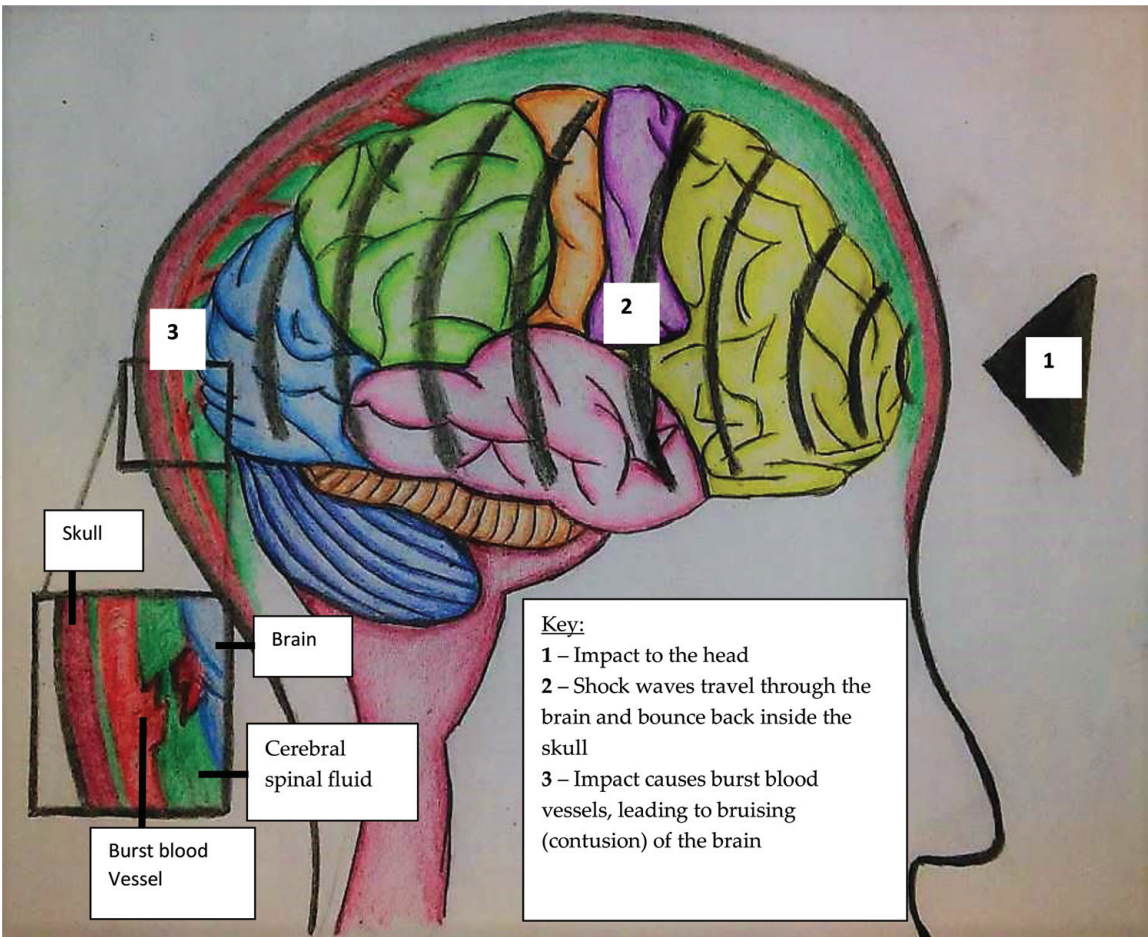


Figure 4.
Schematic representation of how a concussion occurs.

1.9 The mechanics of mild head injury (MHI)

Mild head injury (MHI) involves primary and secondary brain injury. Primary injury occurs on impact and secondary injury occurs after the impact. Secondary brain injury can stem from complications arising out of a primary brain injury. The time-span in which secondary damage can be from seconds, minutes to hours and/or days after the first injury [27].

1.10 Primary brain injury

The contact force is the major origin of brain damage in so-called still injuries where an immobile victim receives a blow to the head. The knock to the head results in movement of the head and neck on impact and causes angular acceleration, a combination of translational and rotational acceleration [5]. Cerebral bruises or contusions are made up of focal damage to the brain tissue which can result in a tear or laceration as a result of head trauma. The coup is an injury that results from a direct blow to the head and appears below the site of impact. A countercoup is when the brain sustains contusion(s) in an area opposite the blow which occurs mostly in the frontal and temporal lobes [5].

The major theories which explain countercoup injuries are: (1) vibration or Echo theory which states that the traumatic impact sets up vibrations which are reflected in damage to the opposite pole of the brain; (2) Transmitted Force Theory which suggests that traumatic impact results in a transmission of applied force through brain tissue which causes the contralateral structures of the brain to be pushed against the inside of the skull; (3) Brain Displacement Theory which posits that countercoup injuries are a result of avulsion of the cerebral cortex from the overlying meninges; (4) Pressure Gradient Theory suggests that when there is a sudden fall in intracranial pressure, opposite to the point of impact, blood vessels rupture; and (5) Rotational Theory posits that after a blow to the skull the brain is set in a centrifugal motion in line with the direction of the original force or impact. The brain is then thrust against the bony protuberances on the interior of the skull [4].

1.11 Secondary brain injury

Secondary brain injury occurs at different lengths of time after head trauma. It is important to recognise that Mild Traumatic Brain Injury (MTBI) is a dynamic process as the symptomology and pathology evolves hourly and sometimes days after an injury occurs [28]. In fact, much of the brain damage which eventually ensues is as a result of the secondary injury [1]. Hypoxia or low oxygen to the brain and/or insufficient blood supply (ischemia) are the mechanisms through which it suffers an insult [29]. 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 are other conditions which cause secondary brain injury [28]. Furthermore, although far less common, the risk of Second Impact Syndrome (SIS), a very serious and even fatal brain injury may occur even after a relatively mild impact, which can be significant in young rugby players. SIS occurs when an athlete suffers a concussion and before the first injury has recovered suffers another injury to the head. In SIS it is possible for rapid deterioration and even death. This happens because the brain has not recovered from the first injury and the second injury results in rapid swelling and pressure within the skull. This intracranial pressure, if uncontrolled, can lead to death [30].

1.12 The pathophysiology of mild head injury (MHI) and CMHI

1.12.1 Diffuse Axonal Injury (DAI)

Diffuse Axonal Injury (DAI) is caused through acceleration-deceleration trauma when the brain twists or rotates inside the skull (rotational acceleration). Focally diffuse axonal strain and tensile stress results in one of the most compromising types of injury in brain trauma [31]. Fundamentally, after a serious head injury it is probably the major cause of unconsciousness and persistent vegetative states. It was first described in the late 1950s after post-mortems conducted on individuals who had died as a result of severe head trauma [32]. This kind of injury has a serious impact on the executive functioning of the brain and alters for instance, the speed of information processing, working memory and attention span [4]. It is postulated that DAI is involved in persistent post-concussive symptomology and attentional deficits following MTBI [33] (Figure 5).

1.13 Post-concussive syndrome (PCS)

Minor impacts to the head cause a pattern of self-reported symptomology referred to as post-concussive syndrome (PCS). These symptoms can persist long after the original injury, and can be both acute and/or chronic. They are categorised into three main symptom areas: cognitive, physical and psychological [34]. Although symptoms generally resolve within a period of a week to 3 months there can be chronic symptomology which occurs from months to years after the initial trauma [5]. These reactions to MHI are facilitated by various issues and are based on an interaction between organic and psychological factors basically, they begin at an organic level and sometimes persist and are experienced at a psychological level. Somatic symptomology includes dizziness, tiredness and headaches whereas psychological symptomology is related to: poor concentration and memory; irritability, emotional lability and depression and anxiety [4].

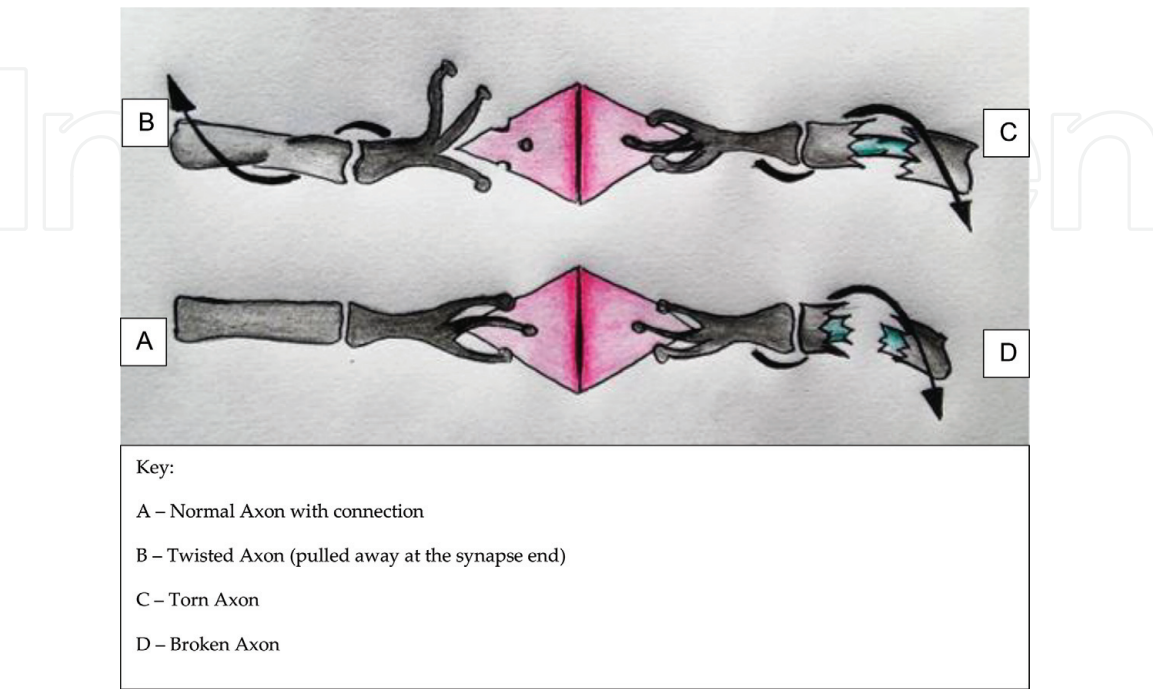


Figure 5. Schematic representation of a diffuse axonal injury.

In the sporting arena there are various physical and neurological symptoms experienced by athletes for instance, headaches and dizziness, impaired concentration and memory plus poor problem solving ability. This type of symptomology may be based on personality characteristics in athletes or be related to malingering and/or the possibility of financial gain [5].

1.13.1 Post-concussive syndrome (PCS) diagnostic criteria

The two most commonly cited systems for defining and diagnosing PCS are the 10th edition of the International Classification of Disease [35] and the Diagnostic and Statistical Manual of Mental Disorders—DSM-5 [36]. In this chapter we present the ICD-10 (2010) diagnostic criteria as it is more universally applied and many individuals do not meet the DSM-5 criteria pertaining to cognitive deficits and clinically significant criteria (which can also be problematic in terms of finding incidence and prevalence of PCS): (a) history of head trauma with loss of consciousness precedes symptoms onset by maximum of 4 weeks and (b) symptoms in three or more of the following 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 grading of concussions is also important in this regard see **Table 1** [22].

1.13.2 Recovery and symptomology related to post-concussive syndrome (PCS)

Diagnosis of PCS is based on the subjective symptomology reported by individuals as is recovery (based on symptom resolution) [5]. Adults’ cognitive deficits and symptoms in terms of PCS are commonly found in the acute stage and resolve within 3–12 months [37]. The duration of amnesia related to any LOC is also very important [4] (**Table 2**).

Grade of Concussion	Severity of Concussion	Symptoms
Grade 1	Mild	Temporary Confusion No Loss of Consciousness (LOC) Symptoms resolve in less than 15 minutes
Grade 2	Moderate	Temporary Confusion No LOC Symptoms last longer than 15 minutes
Grade 3	Severe	Any LOC (transitory or protracted)

Table 1.
Concussion grading.

Time of Amnesia	Severity
Less than 5 minutes	Very Mild
Five (5) to sixty (60) minutes	Mild
One (1) to twenty-four (24) hours	Moderate
One (1) to seven (7) days	Severe
One (1) to four (4) weeks	Very Severe

Table 2.
Post traumatic amnesia.

MHI symptomology are often non-specific and may be the same as those reported after for instance, orthopaedic injuries. The most frequently reported symptoms are headaches, blurred vision, dizziness, subjective memory problems and sleep disturbance [35] (**Table 3**).

Return to play protocols for professional athletes in football and rugby are the norm and well-defined (see **Tables 4** and **5**) but this is not the case in the amateur spheres of the game where injuries may be more severe because of the poorer skill levels of the athletes [38].

1.14 Neuropsychological sequelae of mild head injuries (MHIs)

Individuals who sustain MTBI often report symptomology comparative with PCS. It has been reported that 10–20% of MTBI patients report PCS that go beyond a recovery period of 6–12 months [39]. Severe tiredness (up to 50% of individuals who report PCS) is often reported which impacts on an individual’s cognitive ability and can cause day-to-day problems in living relating to work, exercise and sports participation as well as social interactions. Psychological symptomology for instance, depression is also associated with MHIs as well as anxiety and irritability [40]. Children who experience MHI are more likely to experience impulse control problems and, as a result, have poorer planning ability. They are at a higher risk of difficulty with high-level cognitive functions [41]. As many children and adolescents play rugby and football this is a problematic finding.

1.14.1 Changes to the neurochemical make-up of the brain after a head injury

Neurochemical change as a result of head injury is facilitated by damaged brain cells and occurs within an hour and up to 10 days post-injury [18]. This creates a metabolic dysfunction which means there is an imbalance between the demand of

	Criteria
1.	History of Head trauma with loss of consciousness precedes symptoms onset by maximum of four weeks
2.	Symptoms in three or more of the following symptom categories: <ul style="list-style-type: none">• 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 adaption of sick role.

Table 3.
ICD10 post concussive criteria.

When a player shows any symptoms or signs of concussion.		The following shall be applied for Acute Injury:	
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.	
NB: A player should never return to play while symptomatic		"When in doubt, sit them out"	
Level	Return to play protocols Activity undertaken		Time Post-Concussion (Approx. 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.	Sport specific training, such as 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

Table 4.
Management of concussion and return to play protocols.

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 activity	Walking, swimming or stationary cycling keeping intensity <70% of maximum. 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	

Table 5.
Graduated return to play protocol.

the brain for energy (to heal itself) and for it to work at its usual capacity. This may be one of the reasons for SIS. The protein S-100 has been found at higher than normal levels for around a year after MHIs which impairs neurological functioning in the brain [42].

1.14.2 Imbalances in hormones after a head injury

Damage to the hypothalamus and/or the pituitary gland can cause hormonal problems as these glands regulate hormones in the body [4]. Changes in sexual function, depression, headaches and tiredness may occur. As these are also linked to concussive injury, hormonal damage can be overlooked. In one study after severe

TBIs abnormal hormone levels were found in 60% of patients [43]. This could occur in some cases of MHI and CMHI (Figure 6).

1.15 Neuropsychological recovery following mild and cumulative mild head injuries (CMHI)

Some pundits suggest that acute sequelae resolution of any MHI neuropsychological deficits takes from 4 to 5 weeks post the initial head trauma but that there still might be problems with psychosocial capabilities. These can be mild cognitive insufficiencies related to slower information processing and slower visuomotor speed. Deficits pertaining to tiredness, dizziness and headaches often reduce after a period of 2 months post-injury but some patients still report PCS 3 months post-injury. These symptoms are often mild and may go unnoticed at medical follow-ups [34]. Return to play protocols are thus very important [38].

1.16 Neuropsychological assessment of deficits related to CMHI

In the mid-1980s medical and allied health professionals started neuropsychological testing [22] because head injuries in professional sports (especially contact sports) were noted as potentially keeping the athlete of the field of play (and sport requires its sporting heroes in order to make money). A number of high profile professional athletes who played American Football incurred head injuries and did

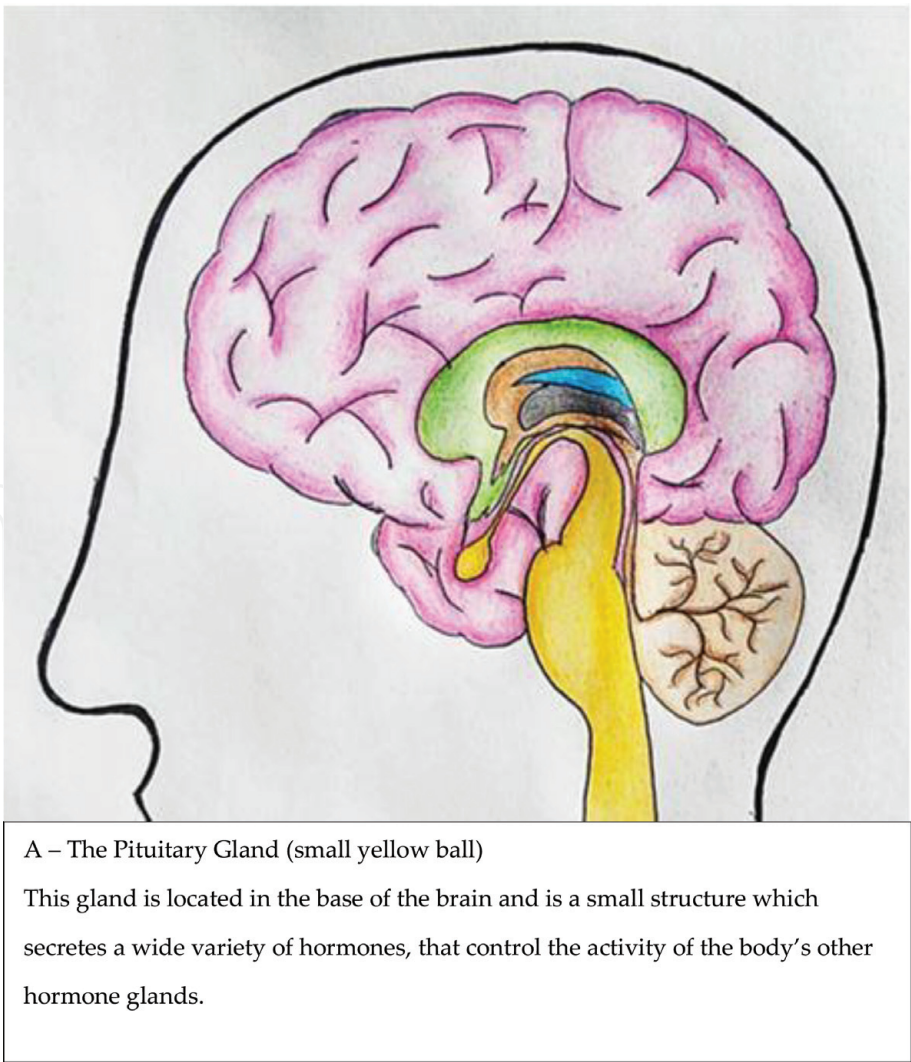


Figure 6.
The pituitary gland.

not recover timeously, costing their franchises much money. As a result baseline neuropsychological testing was used by several major American Football franchises. The National Hockey League (NHL) in the United States of America (USA) authorised this type of testing for comparable reasons. Baseline neuropsychological testing has, since then, become the norm in some countries in collegiate and professional sport and has allowed post injury evaluation of subtle cognitive functions linked to CMHIS. In turn, this data has supported intervention (and treatment) protocols for various professional sporting codes [4].

This suggests that the assessment of cognitive functions is critical in terms of amateur and professional sport particularly in contact sports, and should include baseline cognitive and postural stability testing for athletes in high-risk contact sports [44, 45].

Different types of attention can be assessed using various neuropsychological tests for instance, arousal and alertness can be evaluated using an electroencephalography spectral analysis because the hippocampal theta rhythm is linked to heightened attention [5]. Selective attention can be assessed by using different neuropsychological measures such as hemi-spatial inattention using Line Bisection, Letter cancellation, and the drawing of symmetrical figures. Focused attention can be assessed using the Stroop Color and Word Test. Fundamentally, focused attention is usually evaluated in the visual and auditory areas by utilising dichotic listening tasks. In this type of test an individual listens to various kinds of auditory stimuli and is asked to make specific choices. Other tests of focused attention are the Letter Cancellation Task, the Trail Making Tests and Reaction Time with Distraction tests. Divided attention deficits are usually seen by lower speeds in performance tasks and assessed using for instance, the Paced Auditory Serial Addition Test where the degree of deficit compares positively with the severity of injury. Individuals suffering mild concussion have been found to be up to three times slower than control group individuals with no concussive injury. Athletes who experienced severe concussion were found to be up to five times slower than control group members with no concussive injury. Sustained attention deficits can be seen as time-on-task deficits. In other words, an individual takes longer than the norm to complete a task. Other neuropsychological tests for sustained attention are for instance, the Letter cancellation test, Vigilance tests and Perceptual Speed tests [4, 5].

Computerised cognitive tests (CCTS) are available that evaluate changes in cognition [46]. For instance, the Post-Concussion Assessment and Cognitive Test (ImPACT). These can be more accurate than pen-paper tests but qualitative data from interviewing patients must also be used to give a complete report on any head injured athlete.

1.17 Reaction time related to head injuries

Reaction time (RT) is the period in milliseconds from when a test stimulus is presented to the time the individual reacts. In simple RT testing (using a computerised programme) there is only one stimulus and one response which measures psychomotor skills [46]. In choice RT testing the testee gives a response when presented with a stimulus on the computer screen [4]. Computerised assessment is more accurate than older pen-paper tests and, because of high overall use of smart phones and computers, all populations are able to complete these tests [45]. The two critical measurements taken during this type of assessment are reaction time (RT) and movement time (MT). RT, according to some pundits, reflects decision time which is defined as the length of time for stimulus evaluation and response programming. Conversely, movement time (MT), is the measure of the time it takes to complete a response. RT is reflective of cognitive processes while

MT is linked to the motor component of the RT. RT can be defined as the sum of RT and MT which equals Total Reaction Time. Good RT is needed by athletes in order to perform well in any given sporting code and a head injury, which results in poorer RT, is a challenge (caused by for instance, CMHI, MHI). Factors that influence good RT are high stimulation, tiredness, alcohol consumption and any type of brain injury [45].

1.18 Use of neuroimaging techniques in diagnosing MHI deficits

Traditional neuroimaging devices for instance, Magnetic Resonance Imaging (MRI) and Computerised Tomography (CT) scans are not appropriate for MHI and CMHI as they do not pick up the pathophysiological processes in this type of head injury [18]. However, newer structural MRIs which include gradient echo perfusion and diffusion imaging are more sensitive to structural abnormalities so may be more useful [19]. Traditional neuropsychological assessments (pen and paper and computerised tests) have proven their usefulness in diagnosing MHI deficits and are very sensitive to diffuse axonal damage thus are used successfully in the sporting environment.

1.19 Cross-cultural neurological assessment

The culture and ethnicity of an athlete must be looked at carefully when interpreting data from any kind of neuropsychological/neurological assessments. Many of these tests have not been standardised on people from non-westernised Caucasian backgrounds which could prove problematic. Construct equivalence for the assessment of individuals who are not from the culture that a test has been standardised and validated on usually does not exist. The assessment of an athlete's responses on a neurological/neuropsychological evaluation must take into account their socio-cultural context and experiences [4, 5]. If these are overlooked there may be a culturally inappropriate analysis which can result in false positive or false negative results.

1.20 Examples of research on CMHI in the contact sports rugby union, rugby 7s and football

Cumulative mild head injury (CMHI) research, to a large extent, has been conducted on rugby union players. Players are fit, the forwards are heavy (up to 140 kg) and it is described as a very physical sport [4]. Although the sport originated in, and was initially played, in Europe it is a very popular sport in the southern hemisphere (for instance in Australia (AUS), South Africa (SA) and New Zealand (NZ)). It is becoming increasingly popular in Japan and the USA which has prompted more research in the field.

In 2000 an investigation looked at the cumulative effects of concussion and CMHI on professional rugby players. A group of 26 professional rugby players and a control group of non-contact sports (professional cricketers) athletes was used. In the rugby group forward and backline players were compared over a neuropsychological test-battery. Results indicated that rugby players, particularly the forwards, had deficits in verbal, working and visual memory as well as visuoperceptual tracking skills as compared to the cricket playing controls. It was also found that within the rugby group mean score test comparisons indicated that the forwards displayed greater cognitive deficits than the backline players [47]. Conversely, research into intellectual deficits incurred by CMHI in high school rugby players, revealed no

significant association between the actual reported MHIs and intellectual or cognitive dysfunction [48].

In 2001 an investigation into CMHI amongst schoolboy rugby players and a hockey playing control group revealed more variability, on a battery of neuropsychological tests, amongst the forward rugby playing group (as compared to the backline rugby players) as well as the hockey playing group. Working memory and visuospatial processing skills were more impaired in the rugby forwards than the backline and hockey players and the entire rugby playing sample showed more of these deficits than the hockey playing control group [49].

Enduring PCS amongst school boys and adult players (at the national level) was looked at using visuomotor processing speed tests. Results suggested that the rugby playing group had less capability on various tests post-season. It was postulated that this was probably due to unreported concussions and/or the cumulative effect of mild head injuries. It was also reported that the rugby forwards (who engage in more scrummaging and heavy tackling) showed more cognitive deficits than the rugby backline players [50].

In 2010 an investigation, using a neuropsychological test battery, was carried out into the effects of three or more concussive injuries in adult male rugby players. It was found that rugby players who had suffered multiple concussive injuries performed lower on the test-battery than those who had no previous history of concussion. The results suggested that rugby players who had incurred three or more concussions were likely to suffer cognitive dysfunction [3].

On the other hand, an investigation into CMHI using computerised testing, on a sample of high school rugby players, did not support research which indicates that concussive injury and/or CMHI results in cognitive deficits. The major body of research in SA indicates that CMHI results in some cognitive deficits particularly in rugby forwards. In this research neither rugby forwards or backs showed cognitive dysfunction post-season relative to the hockey controls. It was anticipated that, as the computerised test was very sensitive to diffuse brain injury, some cognitive dysfunction would be found. The research concluded that perhaps MHIs in this group (adolescent boys) does not have a cumulative effect as previously postulated. It was also suggested that factors such as education and age may mitigate against CMHI [9] however, as this was a small sample results were considered provisional [51].

Conversely, a small 2017 study looking at CMHI in college rugby union players found that there was significant variability on mean scores between rugby frontline and backline players on verbal memory, concept formation, cognitive flexibility, working memory and visual-motor processing speed on a pen-paper neuropsychological test battery. In this research it was postulated that poor scores on PCS might also indicate depression in the rugby playing group as insomnia and anger were frequently reported [52].

An Australian study found that in a sample of 104 amateur rugby sevens players (males and females) in one season thirty-one injuries occurred. These were mostly caused by contact at speed and tackling. In the investigation it was noted that head, neck and shoulder injuries made up 50% of all injuries however, CMHI was not investigated. It was reported that athletes that were slower and less agile were at more risk of injury as were female 7s players. The study concluded that there are limited studies on risks factors associated with rugby sevens player and that more pre and post season assessment was required [53].

A study on school level rugby union players in Australia looked at 332 injuries in different age ranges over a season (10–18 years). It was found that the incidence of supposed concussion injuries was 4.3/1000 players and that the most usual way of incurring this injury was tackling. Risk factors were that the game itself is a

physically challenging contact sport where many tackles are made at high speed. Over a third of injuries in this sample were to the head and face and overall there were 61 reported concussion. As this was a high incidence it was recommended that prevention programmes need to be put in place [54].

A recent study in Ireland looking at recurrent injuries in teenage rugby in 15–18 year olds (15–18 years) found that recurrent injuries numbered 426. Eighty-one concussions were reported of these, 5% were recurrent (in the season under investigation). Although these were the lowest number of recurrent injuries it was noted that any concussive injury that occurs on multiple occasions could be potentially disastrous [55]. In this regard the evaluation and management of concussive injury in young contact sports players is very important because they may have a long-term effect on the athletes to heal [56, 57] for instance, the development of chronic traumatic encephalopathy [55].

There has been very little research into CMHI in football (soccer) players in South Africa. Originally the sport was a non-contact sport but the contemporary game is a designated contact sport [4]. It has been reported that concussive injuries in football are often not reported (players do not wish to leave the field as they do not want to be ‘benched’ for 6 weeks or until recovery) and, as a result, are under-diagnosed [4]. Athletes involved in football may incur head-to-head, head-to-ground (or post) and head-to-ball injuries thus there is the likelihood of CMHI. Although head-to-ball injuries may seem unlikely a ball kicked at half-speed travels between 22 and 83 km an hour and can hit the skull with a force of 116 km an hour [59]. A full powered kick could hit the head at around 200 km an hour. As there are about five possibilities a match for any team member to head the ball [60], there is the possibility of CMHI. As early as 1989 research concluded that 12 of 37 football players in a study had slightly abnormal or abnormal EEG (Electroencephalograph) results compared to only 4 of 37 controls who had never played football [58].

In 1989 a study in Norway found that football players self-reported symptoms of irritability, inability to sleep, poor working memory, dizziness, neck pain and headaches after they had repeatedly headed the ball [58]. This supported another 1993 study using male and female Olympic football players. In this research 55% of female and 54% of male football players in the sample reported concussive symptomology after repeated headings of the ball [59]. These investigations underpin earlier findings in 1983 where it was reported that out of every 10 football players 2 had abnormal EEGs when they had trained for 15 minutes in heading the ball [60]. There is concern that youngsters who play football from an early age, and repeatedly head the ball (or are involved in collisions), can have cognitive deficits in later life, possibly as a result of CMHI and concussive injury in the game [4, 61].

Conversely, a study in 2000 reported no acute cognitive deficits in a sample of male and female football players and any significant differences were reported as due to practice effects [62]. However, a 2001 study did find cognitive dysfunction relating to memory and planning abilities in amateur football players in an American college sample [18]. On the other hand, it was reported that concussion from head to ground injuries and collisions were more likely to cause this type of brain injury than repeated heading of the ball [63].

A 2016 study in South Africa looking at sample football players and a control group of non-contact sport athletes (volleyball) using a computerised assessment package (measuring reaction time) and looking at PCS found the following: pre-season volleyball players actually had a better (or faster) reaction time than the football players (not significant). Post-season on a test of simple reaction time there were no significant differences. Both groups reaction times improved in relation to their pre-season results. This may suggest that playing these sports and engaging in

reaction time training actually improves and develops athletes reaction times. As this was computerised testing any diffuse brain damage should have been seen in the results, this was not the case as no significant deficits in football players were found. Conversely, on PCS over a quarter of the football players and only 6% of the control group experienced symptoms such as headaches, attention problems, memory problems, irritation, nervousness and anxiety. This may suggest that athletes involved in football do sustain some CMHI and/or concussive injury which is not reflected in their reaction time scores. This was a small sample of 15 footballers and 15 volleyball controls thus results are provisional [64].

A 2016 study looking at concussion in elite male football players found that this type of injury was a risk factor for incurring another such injury within a year of the first injury. Interestingly, the athletes who had a previous concussive injury also had more other injuries than the non-concussed football players in the study. It was suggested that this may be caused by the type of behaviour these athletes engage in (on the field of play) and due to their inherent personality characteristics [65].

The aforementioned literature indicates why there is a debate in football as to whether young players should be allowed to head the ball [66] particularly if tiredness has an impact on performance [67].

1.21 Recent literature concussive injuries in other sporting codes

This section will present several studies related to head injuries in other sporting codes so that the reader is made aware of the extent of these injuries in the sporting arena today.

Combat sports such as wrestling, mixed martial arts, taekwondo incur many injuries which are mostly soft-tissue and contusion injuries, however, a meta-review found that 15% of injuries in these sports are concussive in nature (90% classified as mild to moderate). Risk factors include age, weight category, experience, training and gender, females more likely to incur this type of injury [68].

Cricket players, although the sport is usually designated a non-contact sport, can also receive craniofacial and head injuries if they are hit by a cricket ball (usually when batting). Although using a helmet helped with neck and head injuries it is possible for possible head injuries to occur [69]. Furthermore, another review which looked at craniofacial injuries between 1870 and 2015 found a relatively small number (36). However, 5 resulted in a fatal injury and 9 resulted in the cricketer no longer being able to play the game. In this study it was also reported that in some instance concussion was difficult to diagnosed. It was concluded that all cricket clubs should have medical professionals available and that concussive injuries needed early identification and appropriate management [70].

Boxers generally suffer more repetitive chronic traumatic brain injury (CTBI) which is not mild or moderate in nature. However, all repetitive injuries to the brain are cumulative in nature and, in the case of boxing, where many blows to the head are received there is the possibility of SIS. As a result since the early twentieth century boxing careers were, on average, 19 years in length but are now around 5 years long. There are still deaths in the sport however, recent research suggest that CBTI caused by repetitive blows to the head will become fewer because of medical interventions such as neuroimaging and the early discovery of these injuries [71].

American football is another sport where there are many concussive injuries as it is a contact sport that is considered both violent and dangerous. In a review of head injuries in the game it was revealed that athletes, who have many concussions are, as they age, at risk of non-resolving cognitive deficits, dementia and depression. Retirement age is not prescribed in the sport however, it was suggested that risk

Risk factors for MHI (CMHI & MTBI)	Impact and/or outcomes
Age	<ul style="list-style-type: none">• Young contact sport players as brain development not complete;• Older players of contact sports because of:<ul style="list-style-type: none">➢ Likelihood of repeated MHI;➢ Less agile and slower reaction times than younger contact sports players.
Gender	<ul style="list-style-type: none">• Studies suggest that female contact sport players are more at risk of MHI and concussive injury.
Duration of concussion	<ul style="list-style-type: none">• Mild• Moderate• Severe (see table 1)
Duration of Post-Traumatic Amnesia (PTA)	<ul style="list-style-type: none">• Very mild• Mild• Moderate• Severe• Very severe (see table 2)
General conditioning of the athlete	<ul style="list-style-type: none">• If athlete not fit and overweight high risk factor;• If athlete fit but moving a fast speed and making contact with/or tackling other physically fit players;• Position played within the contact sport for instance, rugby forwards because of tackling and scrummaging may be more at risk than rugby backline players.
Un-reported illness	<ul style="list-style-type: none">• Players who have had influenza or other illnesses and who have taken medication are often allowed to play during important games (particularly in the amateur arena). This is a risk factor for injury and MHI.
Overuse of alcohol and/or substances	<ul style="list-style-type: none">• These (even over the counter or prescribed substances) put athletes at risk;• In both professional, but particularly amateur, contact sporting codes much alcohol is consumed which is also a risk factor.
Poor skills	<ul style="list-style-type: none">• Amateur and young players do not have a full skill-set thus may be more prone to MHI;• Players with poor skill sets may be inclined to be more reckless in their sport of choice facilitating MHI.
Poor or inappropriate training	<ul style="list-style-type: none">• Related to the abovementioned bullet – lack of proper or incorrect training techniques in the sport of choice leads to vulnerability to MHI.
Poor concussion management	<ul style="list-style-type: none">• Poor concussion management protocols are a high risk factor for repeated injuries and SIS (see table 4).
Poor return to play protocol	<ul style="list-style-type: none">• Return to play protocols must be in place and strictly adhered to otherwise the athlete is at risk of CMHI and SIS (see table 5).
The ‘blind eye’ syndrome	<ul style="list-style-type: none">• Coaches and management turning a ‘blind eye’ to concussive injuries and either allowing or making athletes return to their sport too quickly.
Athlete under-reporting of concussive injury	<ul style="list-style-type: none">• Pressure from team management, coaches and possible loss of earnings or losing place in the team facilitates under-reporting of these injuries by athletes.
Non- availability of medical staff	<ul style="list-style-type: none">• Professional games are required to have medics however; these are not always available during practices;• In contact sports at amateur level there are often either no trained medical professionals or poor trained allied health professionals;• The abovementioned is the same for school contact sports and during practices there are usually no health professionals present.
No pre and post season testing cognitive (or reaction time) tessting	<ul style="list-style-type: none">• In professional sport this is usually carried out on all players (though not always);• In amateur and school sports pre and post season testing for MHI is not usually undertaken. There are many head-impact measurement devices available which may be too expensive for all stakeholders however, pen-paper testing is available.
Unsuitable protective clothing/gear	<ul style="list-style-type: none">• Protective clothing of any kind (including helmets) is often not the correct size or incorrectly worn, which is a risk factor;• Second hand protective clothing/gear should not be used as it has lost much of its efficacy;• Many sporting codes do not have rules that protective clothing/gear is worn by a) all players or b) by amateur or young players (for instance, skull caps in rugby and helmets when batting in cricket).
Family history of specific illnesses	<ul style="list-style-type: none">• Players of contact sport who have familial histories of:<ul style="list-style-type: none">➢ Depression;➢ Senile Dementia➢ And other degenerative diseases, are
	are likely more at risk of early onset of the last two bullets and, at any stage during their sport career, of becoming depressed.

Table 6.
Risk factors in contact sports for cumulative mild head injury (CMI) and concussive injury.

factors for neurocognitive impairment should be taken into account for instance, age, number of concussive injuries, length of time taken to recover and any non-resolving functional deficits [72].

Ice-hockey players are also at risk of concussive head injuries. It was found that young and older players are more at risk due to possible lack of skills and in the latter case tiredness causing them to be more careless in their playing style. Overall, it was concluded that this type of MTBI is serious in nature and more research needs undertaken in this sporting code [73].

There are many risk factors for CMHI and concussive injury in rugby union, rugby 7s, football and other sporting codes. A summary of these is provided in **Table 6**. **Table 6** is not exhaustive but based on the authors reading of the literature.

2. Conclusion

Recently in South Africa (as well as internationally) there has been much interest in research into the neuropsychological sequelae following concussive injuries [1, 50, 51, 61, 63–67]. This type of research is vital in contributing to the field of sports psychology and sports medicine in terms of understanding the clinical features and assessment techniques, clinical management, rehabilitation, education of athletes and their health care providers, return-to play guidelines and long-term outcomes of concussive injuries. Although it is widely believed that athletes are fit to return to play when observed symptoms resolve, researchers continue to investigate the prolonged effects of concussion and repeated concussion on cognitive difficulties, emotional disturbances and behavioural issues.

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