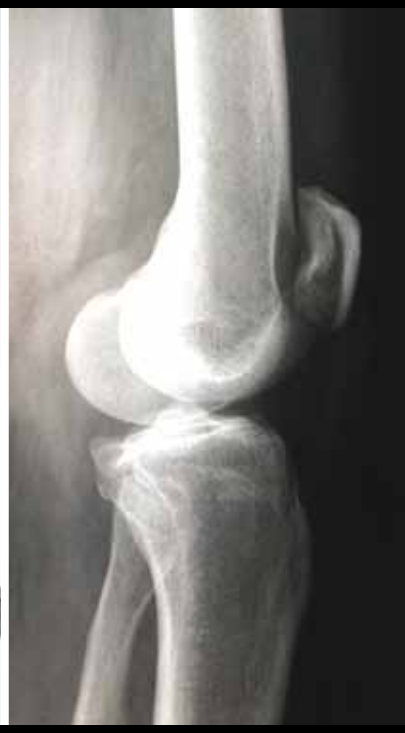
Rehabilitation Research and Practice

Mild Brain Injury

Guest Editors: Brian D. Greenwald, Anne Felicia Ambrose, and Gina P. Armstrong



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Editorial Board

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Hindawi Publishing Corporation
Rehabilitation Research and Practice
Volume 2012, Article ID 469475, 3 pages
doi:10.1155/2012/469475

Editorial
Mild Brain Injury

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Received 11 July 2012; Accepted 11 July 2012

Mild traumatic brain injury (mTBI), commonly known as concussion, is one of the most common neurologic disorders [1]. Based on emergency room visits, hospitalizations, and deaths, an estimated 1.7 million people sustain a traumatic brain injury (TBI) annually in the United States [2]. About 80% of TBIs that occur each year are mild TBI [3]. Since many people are not evaluated in a medical setting, the true annual incidence of mTBI is thought to be much higher. Estimates of the incidence of sport-related mTBI range from 1.6–3.8 million per year in the United States [4]. Research indicates that up to 15% of persons who suffer an mTBI may have persistent disabling problems [5, 6].

Despite the high incidence, mTBI has only entered the consciousness of the medical community and lay press in the last decade. This interest has been fueled by combat-related injuries as well as high-profile athletes. There has been increased public awareness regarding the need for evaluation and treatment of mTBI as well as possible long-term health effects. Chronic traumatic encephalopathy (CTE) is a neurodegenerative disease thought to be caused, at least in part, by repetitive brain trauma that can occur during contact sports and military participation [7, 8]. CTE has also become a popular topic due to its close association with sports like American football, hockey, soccer, boxing, and professional wrestling. Only small uncontrolled studies currently exist regarding the prevalence, risk factors, and natural history of CTE.

Recent reports in the lay press about professional athletes, in particular in the National Football League, have led to fears about the cumulative effects of mTBI on children, adolescents, and college athletes and risk of CTE. State governments have created laws in response to these fears. As of May 2012, 38 states (plus the District of Columbia and the city of Chicago) had adopted youth concussion laws. The main tenets of the laws include the following: inform and educate youth athletes, their parents, and guardians and require them to sign a concussion information form; removal of a youth athlete who appears to have suffered a concussion from play or practice at the time of the suspected concussion; require a youth athlete to be cleared by a licensed health care professional trained in evaluation and management of concussions before returning to play or practice. To promote the prevention of, recognition of, and appropriate responses to TBI, the CDC has developed the Heads Up initiative, a program that provides concussion and mild TBI education to specific audiences such as healthcare providers, coaches, athletic trainers, school nurses, teachers, counselors, parents, and student athletes [9].

The Centers for Disease Control and the World Health Organization have promoted that the term "concussion" be replaced by the term "mild traumatic brain injury." There is ongoing debate over whether mTBI is synonymous with concussion or not. mTBI and concussion have been used interchangeably, although the latter is more commonly used in sports and mTBI in general medical contexts [10, 11].

In comparison with moderate and severe traumatic brain injuries, mTBIs are often more challenging to diagnose. Often there is swift resolution of signs and symptoms and typical absence of evidence of injury on standard neuroimaging. Because of this, in 1993 The American Congress of Rehabilitation Medicine (ACRM) (Mild Traumatic Brain Injury Committee, 1993) was the first organized interdisciplinary group to advocate for specific criteria for the diagnosis of a mild TBI (Table 1) [12]. This ACRM definition has been widely used, especially in the field of rehabilitation and neuropsychology. Centers for Disease Control (CDC) and

Table 1: Diagnostic criteria (Mild Traumatic Brain Injury Committee, Head Injury Interdisciplinary Special Interest Group, American Congress of Rehabilitation Medicine) [12].

A traumatically induced physiological disruption of brain function, as manifested by at least one of the following
Any loss of consciousness
Any loss of memory for events immediately before or after the accident
Any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, or confused)
Focal neurologic deficit(s) that may or may not be transient
But where the severity of the injury does not exceed the following
loss of consciousness of approximately 30 min or less
after 30 min, an initial Glasgow Coma Scale score of 13–15 and
post-traumatic amnesia not greater than 24 hr

the World Health Organization (WHO) have advanced a standardized definition that was derived from the ACRM definition [13, 14].

Recently an expert consensus relating to soldiers returning from Iraq and Afghanistan used the same clinical signs to define mTBI [15]. For these soldiers, the most frequent combat-related injury incurred is TBI, in particular mTBI [15]. Approximately 22% of the wounded soldiers arriving at Landstuhl Regional Medical Center in Germany have head, neck, or face injuries. TBIs result primarily from improvised explosive devices (IEDs), landmines, high-pressure waves from blasts, blunt force injury to the head from objects in motion, and motor vehicle accidents [16, 17]. mTBI characterizes most of the blast-induced traumatic brain injuries seen in service members returning from Iraq and Afghanistan, with reports of 300,000 service members sustaining at least one mTBI as of 2008 [18]. The frequency of these injuries has led to mTBI being called the "signature injury of war" [16].

mTBI is a major health problem that affects both civilians and military populations. This edition of the journal Rehabilitation, Research and Practice reviews the up-to-date literature on the diagnosis, management, and outcomes of MTBI in both populations. Screening for mTBI and return to play after sports concussion are also reviewed. It is our desire that readers gain further insight into the complexities of this disorder, useful tools for diagnosis, and further treatment options.

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Hindawi Publishing Corporation
Rehabilitation Research and Practice
Volume 2012, Article ID 659652, 10 pages
doi:10.1155/2012/659652

Review Article

Traumatic Brain Injury in Sports: A Review

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Received 5 November 2011; Revised 25 January 2012; Accepted 6 February 2012

Academic Editor: Anne Felicia Ambrose

Traumatic brain injury (TBI) is a clinical diagnosis of neurological dysfunction following head trauma, typically presenting with acute symptoms of some degree of cognitive impairment. There are an estimated 1.7 to 3.8 million TBIs each year in the United States, approximately 10 percent of which are due to sports and recreational activities. Most brain injuries are self-limited with symptom resolution within one week, however, a growing amount of data is now establishing significant sequelae from even minor impacts such as headaches, prolonged cognitive impairments, or even death. Appropriate diagnosis and treatment according to standardized guidelines are crucial when treating athletes who may be subjected to future head trauma, possibly increasing their likelihood of long-term impairments.

1. Introduction

Traumatic brain injury has received increased attention, both in the medical literature and social media, particularly in the field of sports. There are 1.7 million documented TBIs annually, with estimates closer to around 3.8 million [1], 173,285 of which are sports- and recreation-related TBIs among children and adolescents [2]. As the number of participants in youth sports continues to grow, the incidence of brain injury is proportionally increasing as well [2]. There is a greater awareness of potential short- and long-term sequelae of athletes who suffer brain injuries such as increased propensity for reinjury, cognitive slowing, early onset Alzheimer's, second impact syndrome, and chronic traumatic encephalopathy [3–23]. Federal and State governments, along with many sport's governing bodies are implementing rule and policy changes designed to increase protection of athletes and to standardize medical care. There is an inherent risk in many sports for repetitive head trauma that athletes subject themselves to and it may be up to the physician to protect their well-being. It is important to understand that athletes are a unique demographic of patients who have many behaviors that may differ from the "normal" office patient.

The evaluation and management of an athlete with TBI includes symptoms assessment, medical examination, and neurocognitive testing with serial evaluations over the following days, weeks, to months of recovery. An initial cognitive and physical rest period followed by a gradual increase in physiologic and cognitive stress in asymptomatic athletes is the hallmark of management and change in the paradigm of management. Proper treatment includes accurate assessment and management using current guidelines in an attempt to minimize potential future deleterious effects from TBI. The purpose of this paper is to provide a review of contemporary views of mild traumatic brain injury in sports including definition, epidemiology, pathophysiology, diagnosis, and management including return to play. The timeliness of this paper is apparent now that 37 States have established laws requiring youths who sustain a sporting related brain injury be required to see a physician prior to returning to play; as of August 2011, to the best of the authors' knowledge. Schools, communities, and athletic leagues must be aware of these legislations and follow them appropriately.

2. Materials and Methods

Key articles from major sources that are considered the gold standard of knowledge within this topic were reviewed to give a comprehensive up-to-date review of this topic. Pubmed was used to search and identify supplementary articles for supporting data and topics. There were sources and articles used in this paper including epidemiology were found using the Centers for Disease Control and Prevention (CDC) website on traumatic brain injury.

3. Definition

The term mild TBI (mTBI) is now used in place of concussion in the nomenclature according to the Centers for Disease Control and Prevention CDC and the World Health Organization (WHO). Traumatic brain injury is a clinical diagnosis of neurological dysfunction following head trauma. Multiple definitions exist however the CDC defines a mTBI as a complex pathophysiologic process affecting the brain, induced by traumatic biomechanical forces secondary to direct or indirect forces to the head. The American Academy of Neurology (AAN) defines mTBI as a biomechanically induced brain injury resulting in neurologic dysfunction [24]. MTBI results in a constellation of physical, cognitive, emotional, and/or sleep-related symptoms and may or may not involve a loss of consciousness (LOC). Duration of symptoms is highly variable and may last from several minutes to days, weeks, months, or even longer in some cases [25]. There is inherent weakness in all the definitions of mTBI as they are based on clinical evaluation and may be biased by the examiner or examinee.

4. Epidemiology

According to the CDC, there are 1.7 million documented TBIs each year, with estimates closer to around 3.8 million [1]. Direct medical costs and indirect costs such as lost productivity of TBI totaled an estimated $76.5 billion in the United States in 2000 [26, 27]. Annually, US emergency departments (EDs) treat an estimated 173,285 sports- and recreation-related TBIs among children and adolescents, ages ranging from birth to 19 years averaged over 10 years [2]. During the same 10-year period, ED visits for sports- and recreation-related mTBIs among children and adolescents increased by 60% annually [2], from 153,375 to 248,418 in 2009 [28]. ED visits for mTBI occurring in organized team sports almost doubled in children aged 8 to 13 years and more than tripled among youths aged 14 to 19 years from 1997 to 2007 [29]. Breakdown of these numbers show that 71.0% of all sports- and recreation-related TBI emergency department visits were males and 70.5% of total visits were among persons aged 10–19 years [2]. Overall, the activities most commonly associated with TBI-related ED visits included bicycling and football; followed by playground activities, basketball, and soccer [2, 28].

CDC data is not available for non-ED visits which include primary care and specialist office visits because the collection of brain injury data is easier to collect and quantify in ED patients. Increase incidence is multifactorial, and is in part due to the increase in participation of our youth in athletic activities [31]. There is also increased awareness by the general public including parents and coaches to report and refer patients with concussive symptoms for physician evaluation.

5. Pathophysiology

TBIs are a result of dysfunction in neuronal metabolism and the microscopic anatomy of the brain that occurs in two distinct phases. Diffuse axonal injury (DAI) is the hallmark injury of TBI and occurs during an initial phase of neuronal and parenchymal as the direct result of the traumatic force. DAI is a result of a rotational forces, and is important to distinguished from cortical contusions or other hemorrhages due to linear acceleration/deceleration injury [32]. A secondary delayed phase of the brain injury model includes inflammatory cascade activation, edema, ischemia, effects of free radicals, excitatory amino acids, ion release, and programmed cell death [33]. Disruption of axonal neurofilament organization occurs and impairs axonal transport leading to axonal swelling, Wallerian degeneration, and transection [34]. Release of excitatory neurotransmitters acetylcholine, glutamate and aspartate, and the generation of free radicals may also contribute to secondary injury [35].

6. Clinical Presentation of TBI in Athletes

The clinical signs and symptoms of mTBIs may range from subtle mood changes to obvious loss of consciousness. The onset of symptoms may be immediately following the injury, or several minutes later [36]. The AAN identifies signs of mTBI to be amnesia, behavior or personality changes, confabulation, delayed verbal and motor responses, disequilibrium, orientation, emotional labiality, loss of consciousness, slurred/incoherent speech, or a vacant stare. Symptoms of mTBI may include blurry/double vision, confusion, dizziness, excessive drowsiness, sleep difficulties, feeling hazy, foggy, or groggy, headache, inability to focus or concentrate, nausea, vomiting, and photo- or phonophobia [24]. Mood changes, emotional outburst, and behavioral changes also may be the principle manifesting symptoms of mTBI. Mild TBI should also be only a part of a broader differential diagnosis of the previously mentioned signs and symptoms of other common sports-related conditions such as poorly fitting helmet, dehydration, migraine headache, heat exhaustion/stroke, metabolic disturbances, and cardiac or other medical conditions.

Clinicians need consider athletes a unique population subset during evaluations. The sporting world has a culture and mentality that is predicated on pushing athletes beyond their perceived physical and mental abilities. This includes participating in adverse conditions and through a multitude of injuries. Athlete's desire to better themselves and help their team succeed will frequently supersede all other considerations, even at risk to their own bodily harm. Athletes are well known to underreport symptoms that may exclude them from participation. Long standing philosophies such

as "getting their bell rung" is often just an accepted part of athletic competition. Other considerations are signs or symptoms of TBI may only be presents under stressful, high exertion game-like conditions. There may also be other incentives and outside motivators to perform well in athletic arena such as the presence of professional scouts, possible scholarships, advancement to a higher-level team, or even money. Coaches may not fully disclose all information attempting to keep key players on the playing field. Parents who desire to see their children perform well may not wish to have their son or daughter pulled from the sporting event. These unique circumstances and conditions must be taken into account by physicians when evaluating athletes.

7. On the Field Assessment and Management

Sports-related mTBI is a common and challenging injury to diagnose, with a constellation of signs and symptoms that can evolve over hours or days after a concussive episode. Evaluation of mTBI should begin with cervical spine evaluation given the similar mechanism of action in both processes. It is important to note that players who sustain severe head trauma causing a loss of consciousness require prompt, on the field assessment of airway, breathing, circulation, and immediate stabilization of the neck with helmet and shoulder pads left on. Those athletes who have persistent loss of consciousness (LOC) or alteration of consciousness should be kept in a stable position and rapidly transported on a backboard and ambulance to an emergency room. However, most athletes will not suffer LOC and may be evaluated on the sidelines.

Any player suspected of sustaining a mTBI should be immediately removed from the playing field for proper evaluation. If a player has a suspected brain injury and a physician is not present at the venue, the player must be removed from practice or play and referred for proper evaluation before being able to return to play. The point should be made again that most concussions to do involve loss of consciousness. There is also the possibility of delayed symptoms or neurologic decline in these patients, which makes it imperative to perform serial examinations. Multiple studies have shown that collegiate and high school level athletes may demonstrate delayed onset of neuropsychological deficits and symptoms post-injury [37–42]. If the diagnosis of TBI is made, the athlete is required to sit out the remainder of the game or competition. Initial treatment should begin with symptomatic management by reducing the physical and cognitive stressors that may be profound in the sporting arena. The bright lights and loud noises should be minimized which may require removing the athlete completely from the sporting complex. The player initially may require mild analgesics for persistent headache for which Tylenol or NSAIDs may be prescribed. Relative cognitive and complete physical rest should be maintained for at least 24 hours or until follow up evaluation with a physician can be made to begin the return to play protocol.

The paradigm shift in recent years has moved the focus of the initial assessment from grading the severity of the TBI to injury detection and characterization [43]. The scales previously used for grading TBIs have been the Cantu and Colorado guidelines. These guidelines stratify the severity of the TBI based on presence/duration of loss of consciousness (LOC) and presence/duration of amnesia or confusion. Management of that athlete and the RTP is then based on the grade of mTBI they receive at the time of initial assessment, however, this is no longer the current recommended practice.

The primary assessment used today by sports medicine physicians is the SCAT-2 (Sport Concussion Assessment Tool-2), which is a product of the consensus guidelines established in Zurich in 2008 during the 3rd International Conference on Concussion in Sport. Although no prospective studies exist establishing its efficacy, it is believed to be the best screening tool as it incorporates the key components from other scales, and was constructed by the leaders in the field of mTBI in sports in the form of consensus guidelines. Components include review of subjective symptoms, the Glasgow coma scale, the standardized assessment of concussion (SAC) cognitive assessment, Maddocks score, and an evaluation of balance and coordination. Scores of the SCAT-2 can be summated, however clinicians should be mindful that there is not a "normal score" or score cut off to allow RTP (Supplementary material available online at doi:10.1155/2012/659652). The SCAT-2 is most effective when it is compared to a baseline screen, as well as serial examinations following a TBI. Athletes seen in the office setting undergo detailed evaluation including history and past medical history, neurologic examination focusing on coordination and balance, and cognitive functioning.

In addition to having properly trained medical professionals performing TBI assessments, it is important to ensure coaches, trainers, players, and family are also educated about the possible signs and symptoms to ensure early recognition. Physicians are not present at all the athletic venues in which TBIs may occur such as practices or training sessions. The CDC has an initiative termed "Heads Up" to educate not only physicians, but also coaches, parents, schools, and athletes on preventing, recognizing, and responding to TBIs. Information includes statistics, fundamentals of TBI, sign and symptom lists, prevention techniques, and treatment protocols with wording that is directed for their respective audience. Also available are pocket size cards with condensed information on recognition, assessment, and management that is available for non-medical professionals to take out in the field. Studies have found that the coaches' version of the toolkit helped them to better identify signs and symptoms of mTBI, increased their awareness of the requirement of health care professional evaluation, and provided helpful information about possible length of recovery [44]. Chrisman et al found physicians were more likely to be aware of and to follow recommended guidelines for RTP activity after reading the Heads Up toolkit than those who did not [45].

8. Neurocognitive Evaluations and the Role of Baseline Testing

Available baseline cognitive screening tools include neurocognitive testing, Immediate Measurement of Performance

and Cognitive Testing (ImPACT), Brain Injury Screening Questionnaire (BISQ), Automated Neuropsychological Assessment Metrics (ANAM), CogSport (formerly Concussion Sentinel), Concussion Resolution Index (CRI), and the Standardized Assessment of Concussion (SAC). Evaluation of many scales including the SCAT-PCSS, IMPACT-PCSS, Signs and Symptoms checklist, Pittsburgh Steelers Post Concussion Scale, Concussion Symptom Inventory, and the Head Injury Scale did not find one particular scale statistically superior to the rest in screening for TBI, however, neurocognitive evaluation was not included [43]. Computerized and traditional neurocognitive testing of verbal and visual memory, complex attention, reaction time, and processing speed is a useful tool to diagnose and to track athletes when baseline testing is performed and compared with post-injury scores [46]. The Zurich consensus guidelines state that neurocognitive testing is the cornerstone to TBI identification and management [47]. Resolution of post-concussive symptoms and return to baseline cognitive status typically are thought to occur on similar timelines [48]. However, when comparison of baseline and post-injury results in a group of collegiate athletes, 83% of athletes with concussions had significantly lower neurocognitive test scores when compared with their baseline scores demonstrating that neurocognitive testing was nearly 20% more sensitive for detecting injury than symptom reporting alone [49]. None of the athletes in the control group had symptoms or lower scores on neurocognitive testing demonstrating a high sensitivity and specificity for neurocognitive testing in identifying concussion. Similar studies have also confirmed these findings demonstrating the "added value" of computerized neurocognitive testing [50]. This clearly identifies the integral role of neurocognitive testing in the management of TBI in the athletic venue. Administration of traditional neuro-psychologic testing to this point has not been available for all athletes mainly due to the financial cost and resources required to administer the examination, specifically a trained neuropsychologist. One solution is through the use of computerized testing which presents many advantages. Computerized neurocognitive testing has been shown to provide sensitive and specific objective data to quantify injury and track recovery [49–51]. Advantages include screening of athletes at a lower financial cost and with only minimal human resource. Also, preseason testing of large numbers of athletes can be now be quickly and efficiently accomplished at most levels of competition. Large databases of information may also be constructed allowing researchers more data for analysis to continue advancing our knowledge in the management of TBI in sports.

Baseline neurocognitive testing is recommended when possible. Cognitive function should be evaluated and tracked following a TBI in an athlete and used as a component in the decision-making management of that player, but never as a sole factor.

9. Neuroimaging

Urgent Neurologic imaging does not play a primary role in evaluation or management of an athlete who has sustained a TBI, but is used to rule out significant structural pathology such as intracerebral hemorrhage. Prolonged unconsciousness, persistent mental status alterations, or abnormalities on neurologic examination require urgent neuroimaging [24]. Other commonly used criteria for urgent head computerized tomography (CT) scan in the acute setting include the Canadian CT head rule which require a CT scan if concussed patients have any one of the following: GCS <15 two hours after injury, suspected open or depressed skull fracture, any sign of basilar skull fracture (hemotympanum, periorbital bruising or raccoon eyes, retroauricular bruising or battle's sign, cerebrospinal fluid leak, oto- or rhinorrhea), two or more episodes of vomiting, 65 years of age or older, amnesia before impact of 30 or more minutes, or dangerous mechanism (pedestrian struck by motor vehicle, occupant ejected from motor vehicle, fall from =3 feet or =5 stairs) [52]. Using this criteria has a 100 percent sensitivity and 88 to 40 percent specificity in detecting neurosurgical and clinically important brain injury abnormalities [53, 54]. Imaging may also be considered in patients who have worsening symptoms, severe acute headaches, or failure of resolution of symptoms within a few weeks. Alternative imaging modalities such as MR (sequences including gradient echo, perfusion, diffusion tensor imaging), functional MRI, and PET scans are all informative into the pathophysiology of TBI, but are not currently recommended as a component of clinical management [47]. Limitations of these modalities include financial cost, limited equipment availability, and lack of evidence guiding changes in management. Future work also includes the use of transcranial Doppler to evaluate cerebrovascular reactivity abnormalities in TBI. Asymptomatic TBI patients at rest had physiologically stress-induced impairments of cerebrovascular reactivity when compared to control subjects representing brains which have not fully healed [55]. In the future, addition cerebrovascular flow studies would add another component to determine whether athletes have recovered physiologically prior to returning to play.

10. Return to Play Criteria

The key feature of TBI management in sports is physical and cognitive rest until symptoms resolve. A graduating program of exertion and cognitive workload prior to medical clearance and return to play. The basis behind the RTP criteria is that a concussed brain has a lower threshold of reinjury in the first few days or weeks following the initial injury [56]. Recovery times may be longer in adolescents and children [57]. An athlete who returns to play within this vulnerable time period risks permanent disability or even death [58, 59]. Athletes are unique in particular regarding the desire to quickly return to the same venue in which the brain injury was sustained. The RTP guidelines are established to protect the health of the athletes.

Previously, the Cantu and Colorado guidelines were used basing the RTP criteria on severity of mTBI and number of mTBIs that season. Although they take into account the athletes symptoms when returning to play, there were not established guidelines for a graduating stepwise addition of physical and cognitive workload prior to return to play.

The current standard of care is based on the consensus guidelines established at the 3rd International Conference on Concussion in Sport in 2008 when determining RTP (Table 1). The guidelines allow for an initial phase of physical and cognitive rest, with slow reintroduction of physical and cognitive activity in a stepwise fashion, providing the patient remains asymptomatic at each step. There are 6 phases in the protocol starting with complete physical and cognitive rest, then advancing to light aerobic exercise, sport-specific exercise, noncontact training drills, full contact practice, and finally RTP. The initial rest period should not only include complete physical rest, but the athlete's academic work also requires modification. This may include, but is not limited to, a reduced number of work assignments, more time to complete class work and tests, breaking down complex tasks into simple steps, and providing a distraction free area for work. A comprehensive medical examination, incorporating SCAT-2, as well as computerized neurocognitive testing should also be conducted at this point. If the athlete is asymptomatic, they may advance to light aerobic exercise (e.g., walking, swimming, stationary cycle) and may continue to progress through the protocol if they remain asymptomatic. If the athlete becomes symptomatic at any point, they must return to the previous level of activity until symptoms resolve for at least 24 hours.

Unrestricted return to play is permitted when the athlete has progressed through the protocol, is asymptomatic, and has returned to baseline or normal values on neurocognitive testing. Given that 90% of mTBI symptoms resolve within one week [41], this protocol can usually be completed in one week as the athlete advances each step in 24 hours. It is recommended to take a more conservative approach to children and adolescents when evaluating for RTP due to particular risks of this age group (i.e., diffuse cerebral swelling) [47]. The guidelines recommend allowing for an extended amount of time of asymptomatic rest and/or the length of graduated exertion in this population. High school athletes had prolonged impairments on neurocognitive testing when compared to professional football players [60] or collegiate athletes [61, 62]. There is evidence that adult brains may be less susceptible to mTBIs and may be able to RTP sooner. Pelman et al. states that some professional American football players are able to RTP more quickly, with even same day RTP supported by National Football League studies without a risk of recurrence or sequelae [63].

It is an important consideration in advancing athletes through the RTP protocol that they remain symptom-free without the use of any pharmacological agents/medications that may mask or modify the symptoms of mTBI [11]. The Zurich consensus guidelines also list modifying factors that require special consideration for RTP criteria and obtaining additional testing such as neuroimaging (Table 2). These include prolonged duration of symptoms, prolonged LOC, seizures, multiple mTBIs especially in the recent past, or change in mental health. Currently, there are no recommendations on the total number of TBIs that are "allowable" for an athlete to sustain before recommending them to sit out the remainder of the season or retiring from the sport. Elite athletes are also recommended to follow the same treatment plan and RTP protocol.

The goal of the guideline is to allow full physical, cognitive, and metabolic recovery to the concussed brain before subjecting it to forces that may cause reinjury. Additional brain trauma within the metabolic recovery window may have both potential short-term and long-term consequences. Even when neuropsychological testing is normal, physiologic, and metabolic dysfunction still may persist for some time. Currently, there are no recommended laboratory testing or imaging modalities that are readily available and reliable to evaluate and follow the microcellular dysfunction. In the future, additional tools may be added into the RTP guidelines such as the previously discussed transcranial Doppler to determine full physiologic recovery.

The evaluation and management of an athlete with TBI is multifactorial assessing symptoms, medical examination, and neurocognitive testing ensuring to catch the variability in presentations of injured athletes. Initial, followed by serial medical and neurocognitive examinations as the patient progresses through the RTP protocol is warranted. Athletes are required to remain fully asymptomatic and returned to baseline cognitive functioning before returning to their sport.

11. Sequelae of TBI in Sports

The increase in media attention, legislation, and constant revision of medical guidelines with respect to TBI is due to the increased awareness of short- and long-term consequences. The obvious immediate impact on the athlete is dealing with the symptoms of a TBI including most commonly headaches, but also poor sleep, excessive drowsiness, poor concentration, and poorer cognitive aptitude. It is estimated that 1.8 million individuals develop acute PTHA each year and 400,000 individuals develop chronic PTHA [64]. Considering most athletes are student-athletes, these symptoms will have an obvious impact on their academic performances as well. In studies of high school and collegiate athletes with a history of three or more concussions had a more severe presentation of concussion, [13] were more likely to have baseline headaches [21], were more vulnerable to brain injury than those without concussion history [4], and were three times more likely to sustain an additional injury [65]. Also, repeated mild TBIs occurring within a short period of time (i.e., hours, days, or weeks) may be catastrophic or fatal [3].

A growing body of evidence exists linking brain injuries of all severity with long-term sequelae.

Repeated mild TBIs occurring over an extended period of time (i.e., months, years) may result in cumulative neurological and cognitive deficits. Retired American professional football players with a history of three or more TBIs were 5 times more likely to have mild cognitive impairment [12]. Professional boxers are well known to have a risk of significant cognitive decline and alterations in brain function [7]. However, there is increasing concern that cumulative effects may also be occurring in athletes who

Table 1: Graduated return to play protocol established at the 3rd international conference on concussion in sport.

Rehabilitation state	Functional exercise at each stage of rehabilitation	Objective of each stage
(1) No activity	Complete physical and cognitive rest	Recovery
(2) Light aerobic exercise	Walking, swimming, or stationary cycle keeping intensity <70% MPHR; no resistance training	Increase HR
(3) Sport-specific exercise	Skating drills in ice hockey, running drills in soccer, no head impact activates	Add movement
(4) Non-contact training drills	Progression to more complex training drills, for example, passing drills in football and ice hockey, may start progressive resistance training	Exercise, coordination, and cognitive load
(5) Full contact practice	Following medical clearance; participate in normal training activates	Restore confidence and asses functional skills by coaching staff
(6) Return to play	Normal game play	

Table 2: Modifying factors in concussion management from consensus guidelines.

Factors	Modifier
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
	"Recency"—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 premorbidities	Migraine, depression or other mental health disorders, attention deficit hyperactivity disorder, learning disabilities, sleep disorders
Medication	Psychoactive drugs, anticoagulants
Behavior	Dangerous style of play
Sport	High risk activity, contact and collision sport, high sporting level

sustain more "routine" injuries as a function of playing a contact sport such as football or ice hockey [8, 9]. Long term effects of repeated concussions include chronic motor and neuropsychological deficits [10, 11]. Collins et al. found that among 400 collegiate football players with two or more previous TBIs independently predicted long-term deficits of executive function, processing speed, and self-reported symptom severity [8]. The nature, burden, and duration of the clinical postconcussive symptoms may be more important than the presence or duration of amnesia alone [14–16]. A telephone-based survey performed by the University of Michigan Institute of Social Research in association of the National Football League of 1,063 retired NFL players found a 19-fold increase rate of memory-related diseases such as Alzheimer's in the 35–49-year-old age group and a 5-year-old increase in ages 50+ when compared to national control groups. Chronic Traumatic Encephalopathy is an entity classically described in former boxers [20, 66], however, there are increasing numbers of case reports described in the literature of athletes in other sports who have a significant history of TBIs [17]. McKee et al. reviewed the autopsy findings of three professional athletes in addition to published reports of 48 cases of suspected CTE and concluded that it is a neuropathologically distinct, slowly progressive tauopathy with a clear environmental etiology [19]. A full discussion is not within the scope of this paper, however, the point should be understood that an association between CTE and TBI is evident within the literature and warrants consideration and future study.

12. Second Impact Syndrome

Second-impact syndrome (SIS) is a rare form of reinjury that occurs prior to the complete resolution of a previous TBI [5]. SIS may result in serious permanent neurologic injury or even death, even if the second impact is only considered to be a minor force. According to the AAN, SIS is a diffuse cerebral dysregulation leading to massive cerebral edema and subsequent herniation. Typically athletes diagnosed with SIS are children or adolescents rather than adults.

Fourteen of the 17 case reports of SIS have occurred in persons less than 20 years old, the others were in a 21 y/o and two 24 years old [6]. This is due to the physiologic differences of children and adolescents compared to adults who have prolonged and diffuse cerebral edema after traumatic brain injury with increased sensitivity to glutamate, increasing their risk to secondary injury [22, 23]. Although rare, SIS has a high associated morbidity and mortality and therefore must be considered.

13. Clinical Training of Clinicians in Sports

As important as having a physician conduct the appropriate brain injury evaluation of an athlete is ensuring the appropriate training of that medical professional conducting the examination. Many studies have concluded that most

physicians have little to no knowledge on the accurate diagnosis or management of patients with TBI. Powel et al. found in their study that over 50 percent of patients who presented to the emergency department with TBIs were not accurately identified by ED physicians [67]. Surveys to determine the knowledge of TBI guidelines in primary care physicians found that less than half were up to date with current medical management [45]. Of patients admitted to the hospital for TBI, 9% were allowed to RTP too quickly and 60% were given no advice in regards to RTP [68]. In a survey of the members of the American Society of Sports Medicine, only 30% of physicians treated their patients per the current established guidelines [69]. As the incidence of brain injuries continue to increase, there must also be a concurrent increase and improvement of physician knowledge and training regarding assessment and management of TBI in sports.

14. Prevention of TBI in Sports

Prevention of TBI is paramount and should be the focus of sporting governing bodies, the athletes, coaching staff, and medical professionals. Two main avenues to accomplish this are through improved protective equipment and rule changes. It has long been understood with literature dating back to the 1960s that hard helmets in sports reduce the incidence of skull fractures and bony head trauma, however, they do not reduce the risk of brain injury [70]. Biomechanical studies which show a reduction in impact forces to the brain with the use of head gear and helmets, but these findings have not been translated to show a reduction in TBI incidence [47]. The use of helmets has been argued to increase brain injury rates through behavioral changes in the athletes who are able to assume a more dangerous playing style and use their helmet as a "weapon" when contacting another player [71]. Clinical evidence that current available protective equipment helps to prevent TBI is not established. Helmets protect against head and facial injury and hence should be recommended for participants in alpine sports [72]. This failure to reduce mTBIs is a product of the biomechanical forces needed to generate the primary neuronal pathology in TBI, diffuse axonal injury (DAI). Helmets are primarily designed to reduce linear accelerative/decelerative forces, not the rotational forces which cause the DAI and in fact may increase rotation forces experienced. Mouth guards have a definite role in preventing dental and oro-facial injuries [47].

The primary means in which rates of TBI incidence in sports will reduce is through rule changes to minimize head impacts moving forward. Penalizing, fining, or suspending athletes who intentionally impact another players head are means to discourage brain trauma. No longer allowing football (soccer) players to head the ball removes a large risk factor as it has been shown that heading accounts for around 50% of brain injuries in sport [73].

15. Discussion

Traumatic brain injury continues to be a popular topic in the medical community and social media, especially in youth sports. We have seen double to triple the number of ED visits by children and adolescents for evaluation of sports related TBI in the past ten to fifteen years [74]. It is important to understand that athlete should be considered a unique population with its own culture and risk factors. Maximizing performance is often the primary objective, even if that is at the cost of bodily harm. The prior thinking that mTBI only occurred in contact sports is not correct as demonstrated by the incidence of brain injury in soccer players [2, 28, 30, 73]. Cognitive impairments as well as long-term consequences such as early dementia have been linked to recurrent mTBIs and even repetitive subconcussive impacts [8, 9]. Chronic traumatic encephalopathy or second impact syndrome are seen mostly in children and adolescents and are rare but devastating potential sequelae of repetitive brain injuries as well. Protecting athletes using our current understanding of the brain injury model is the primary goal of the medical community serving this group. Under the advisement of the medical community, legislatures have passed laws mandating that youths who are suspected of sustaining a TBI during a game or practice, must be removed from competition and sat out until cleared for RTP by a physician. It is imperative that these athletes are evaluated by a physician trained in sports medicine and familiar with the culture of this subset population. Consensus statement guidelines establish clear management of athletes with TBI and have been outlined in this paper. The evaluation of an athlete with TBI is multifactorial assessing symptoms, medical examination, and neurocognitive testing followed by serial evaluations over the days, weeks, to months of recovery. An initial cognitive and physical rest period, followed by a gradual increase in physiologic and cognitive stress in asymptomatic athletes is the hallmark of management. Athletes are not permitted to return to play until asymptomatic under physiologic stress. Continued education of the general public who may interact with athletes is essential to correctly identify concussed individuals and direct them to appropriate medical care.

Future work should focus on providing evidence to support using the SCAT2 assessment format. Continuing work to improve imaging modalities such as the discussed transcranial Doppler or using serum biomarkers as means to assess and follow physiologic dysfunction and recovery would be excellent additional tools for managing athletes with brain injuries. A better understanding of what and why there appear to be differences in brain injuries in adults compared to children or adolescents and how that would affect RTP management. Presently, the focus should continue to prioritize proper assessment and management by medical professionals based on the current guidelines reviewed in this article, and continued rule changes to minimize head trauma and incidence of brain injury.

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Hindawi Publishing Corporation
Rehabilitation Research and Practice
Volume 2012, Article ID 528265, 5 pages
doi:10.1155/2012/528265

Research Article

Postconcussion Symptoms in Patients with Injury-Related Chronic Pain

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Received 4 December 2011; Revised 19 March 2012; Accepted 19 March 2012

Academic Editor: Brian D. Greenwald

Background. Postconcussion symptoms (PCSs)—such as fatigue, headache, irritability, dizziness, and impaired memory—are commonly reported in patients who have mild traumatic brain injuries (MTBIs). Evaluation of PCS after MTBI is proposed to have a diagnostic value although it is unclear whether PCS are specific to MTBI. After whiplash injuries, patients most often complain of headaches and neck pain; the other PCS are not as closely evaluated. In patients with chronic pain because of other injuries, the presence of PCS is unclear. This study aimed to describe the frequency of PCS in patients with injury-related pain and to examine the relationships between PCS, pain, and psychological factors. *Methods*. This study collected data using questionnaires addressing PCS (Rivermead Postconcussion Questionnaire, RPQ), pain intensity (Visual Analogue Scale), depression, anxiety (Hospital, Anxiety, and Depression Scale), and posttraumatic stress (Impact of Event Scale). *Results*. Fatigue (90.7%), sleep disturbance (84.9%), headache (73.5%), poor concentration (88.2%), and poor memory (67.1%) were some of the most commonly reported PCS. Significant relationships were found between PCS and posttraumatic stress, depression, and anxiety. *Conclusion*. To optimize treatment, it is important to assess each patient's PCS, the mechanism of injury, and factors such as posttraumatic stress and depression.

1. Introduction

The great majority (80–90%) of traumatic brain injuries is classified as mild traumatic brain injury (MTBI)/concussion [1, 2]. These injuries are a matter for general concern because of their potential long-term consequences—persistent post-concussion symptoms (PCSs). PCS include headache, fatigue, dizziness, and impaired memory. These symptoms can affect both work and leisure time [3–5]. Although the natural course of recovery after MTBI consists of restitution of many symptoms within three months after injury [6], a significant number of persons report PCSs that last for many months to even years after injury. Evaluation of these symptoms after MTBI can have diagnostic value. For patients with longterm symptoms, a diagnosis of post-concussion syndrome is sometimes used. According to the ICD10 criteria, at least three symptoms, which may include headache, dizziness, fatigue, depression, irritability, difficulty in concentration, and memory problems, are required for a diagnosis of postconcussion syndrome [7]. The DSM IV criteria for postconcussion disorder include evidence of three or more of these symptoms present for at least three months combined with signs of impaired cognitive function and social disability [8].

Whether PCS are specific to MTBI is unclear since symptoms commonly reported after MTBI have been reported in the general population and by patients with chronic pain. Some of the factors that have been found to contribute to the persistence of symptoms are depression and posttraumatic stress [9, 10].

Chronic pain is an acute and/or intermittent pain that persists more than three months [11], and the great majority of chronic pain is musculoskeletal pain. Apart from individual suffering, chronic pain is costly for society. Some of the most common causes of musculoskeletal pain are injuries related to traffic accidents, falls, and sports.

In western countries, whiplash as the result of traffic accidents has a very high annual incidence: 1.0 to 3.2/1000 per year [12]. Whiplash is an acceleration-deceleration mechanism of energy transferred to the neck that may result in soft tissue injury/distortion of the neck. Most studies of the long-term outcome of whiplash-associated disorder (WAD) have focused on neck pain and headache since these are the dominating complaints, while other PCSs are less investigated. Cognitive symptoms such as memory and concentration difficulties have been reported in patients with whiplash injuries, but the relevance of these symptoms has not been fully examined. Although some studies have shown neuropsychological dysfunction in persons with long-term cognitive symptoms after whiplash injury [13, 14], other studies have suggested neurotic development or preexistent stress as the underlying cause [15].

Although most studies suggest that female gender is a potential prognostic factor related to poor recovery both after MTBI [16] and whiplash injuries [17], some studies fail to show any gender differences regarding chronic symptoms as the result of MTBI and whiplash injuries [6, 18]. For MTBI patients with PCS, some studies have divided the symptoms into three subgroups: emotional, cognitive, and physical [19, 20]. For example, when comparing patients with chronic pain and patients with MTBI, Smith-Seemlier et al. found that cognitive difficulties were more often reported by the MTBI patients [19]. No group differences were found for total scores of postconcussion symptoms. Since their study focused on chronic pain patients regardless of the cause of pain, it may be of interest to study symptoms in patients with injury-related chronic pain.

The aims of the present study were (i) to describe the frequency of postconcussion symptoms in patients with injury-related chronic pain, (ii) to study the relationships between postconcussion symptoms and pain intensity, posttraumatic stress, and depression, (iii) and to examine gender differences regarding these variables.

2. Patients and Methods

A cross-sectional study design was used to study patients with chronic pain caused by an injury and referred by regional general practitioners to the Pain Rehabilitation Clinic at the Umeå University Hospital (Umeå, Sweden). The study included 86 patients—59 women and 27 men (aged 41.1 ± 10.3 years). The participants suffered from pain caused by falls (14.0%), whiplash injuries (44.4%), other nonwhiplash traffic injuries sustained as the result of bicycle and motorcycle accidents (8.1%), horseback riding (8.1%), sports (1.2%), assaults (5.8%), and other injuries such as work-related injuries (18.4%). For all patients, the time between injury and assessment was more than one year.

2.1. Assessments. During assessment in the clinic, the patients answered a set of questionnaires. Information about each participant's trauma history was collected from hospital records. Symptoms (pain intensity and whiplash-related symptoms) were assessed using the Visual Analogue Scale [21], the Rivermead Postconcussion Symptoms Questionnaire [22], the Impact of Event Scale [23], and the Hospital Anxiety and Depression Scale [24].

2.2. The Visual Analogue Scale. Pain intensity was rated using the Visual Analogue Scale (VAS) [21]. The scale consists of a 100 mm straight line with defined end points ("no pain" and "worst pain imaginable") on which the patients were asked to mark their experienced pain (results in mm). The VAS is considered to have a high degree of reliability and validity.

2.3. Rivermead Postconcussion Symptoms Questionnaire. The Rivermead Postconcussion Symptoms Questionnaire (RPQ) [22] is a validated instrument that was used to assess the frequency and severity of 16 symptoms that are commonly encountered postconcussion symptoms. The RPQ asks patients to rate the extent to which their symptoms (compared with their premorbid levels) have become more problematic over the previous 24 hours. The RPQ uses a rating scale with values 0–4, from no problem at all to a severe problem. A total symptom score can be calculated as a sum of all scores (possible score 0–72) [22].

2.4. The Impact of Event Scale. The Impact of Event Scale (IES) is a widely used self-report scale [23]. It is a valid measure of posttraumatic stress reactions and has been suggested as a screening tool for posttraumatic stress disorder (PTSD). The IES comprises 15 statements: seven questions about intrusive symptoms and eight questions about avoidance symptoms. The patients answer the questionnaire regarding their symptoms during the last week. The total score, which varies between 0 and 80, is divided into four stress reaction grades: subclinical (0–8), mild (9–25), moderate (26–43), and severe (44–80) [23].

2.5. HAD. To measure anxiety and depression, the Hospital Anxiety and Depression Scale (HAD) was developed and validated on nonpsychiatric medical patients [24]. The questionnaire comprises of 14 items divided in two parts, for rating of depression and anxiety. Each item has a four response category ranging between 0 and 3. The scale ranges between 0 and 21 for both depression and anxiety. According to Zigmond and Snaith, the cut-off level for possible cases of anxiety disorder and depression is a score ≥ 8 on each subscale [24].

2.6. Statistical Analyses. All statistical analyses were performed with SPSS for Windows (version 19.0). Data are reported as means ± standard deviations unless indicated otherwise. Comparisons of populations were made using the Mann-Whitney U test. Pearson's correlation coefficient was calculated for the analysis of bivariate correlations. Statistical significant level was set at 0.05.

Table 1: Frequency of occurrence of postconcussion symptoms (Rivermead Postconcussion Symptoms Questionnaire).

	All patients ($n = 86$) (%)	Mean	Women ($n = 59$) (%)	Mean	Men ($n = 27$) (%)	Mean	P
Headache	73.5	2.25	51.0	2.21	40.72	2.33	n.s.
Dizziness	61.7	1.70	40.0	1.65	34.9	1.81	n.s.
Nausea/vomiting	29.1	1.01	30.0	1.09	16.3	0.85	n.s.
Sleep disturbance	84.9	2.86	42.0	2.84	45.3	2.88	0.024
Fatigue	90.7	3.01	57.0	3.18	53.5	2.67	n.s.
Irritability	66.3	2.11	54.0	2.14	50.0	2.04	n.s.
Feeling depressed	55.8	1.82	41.0	2.02	40.7	1.41	n.s.
Feeling frustrated	55.8	1.93	40.0	2.03	44.5	1.70	0.046
Poor memory	67.1	1.81	51.0	1.90	53.5	1.63	n.s.
Poor concentration	88.2	2.01	50.0	2.21	52.3	1.59	n.s.
Noise sensitivity	59.5	1.52	45.0	1.87	34.9	1.87	n.s.
Blurred vision	55.8	1.05	31.0	0.96	32.6	1.22	0.001
Sensitivity to light	51.2	1.31	41.0	1.35	44.2	1.22	n.s.
Double vision	26.2	0.44	13.0	0.40	16.3	0.52	n.s.
Restlessness	69.0	1.37	35.0	1.37	48.8	1.37	n.s.
Taking longer to think	74.1	1.05	41.0	1.83	48.8	0.85	0.005
Total score (mean)		27.32		28.33		25.15	n.s.

Table 2: Proportion of patients reporting symptoms and symptoms scores (%).

Symptom scores	0 (not experienced symptom)	1 (no longer a problem)	2 (mild problem)	3 (moderate problem)	4 (severe problem)	2–4 (problem)
Headache	14.5	16.9	21.7	22.9	24.1	68.7
Dizziness	23.5	14.8	34.6	22.2	4.9	61.7
Nausea/vomiting	44.4	25.9	17.3	8.6	3.7	29.6
Sleep disturbance	7.1	6.0	19.0	29.8	38.1	86.9
Fatigue	4.8	4.8	14.3	36.9	39.3	90.5
Irritability	9.4	23.5	27.1	27.1	12.9	67.1
Feeling depressed	18.8	25.9	18.8	27.1	9.4	
Feeling frustrated	15.3	28.2	15.3	30.6	10.6	55.3
Poor memory	21.2	20.0	23.5	27.1	8.2	58.8
Poor concentration	11.8	21.2	32.9	22.4	11.8	67.1
Noise sensitivity	40.5	22.6	13.1	13.1	10.7	36.9
Blurred vision	42.2	30.1	13.3	9.6	4.8	27.7
Sensitivity to light	30.5	17.1	23.2	18.3	11.0	52.5
Double vision	75.0	10.7	10.7	2.4	1.2	14.3
Restlessness	32.1	27.4	20.2	11.9	8.3	20.4
Taking longer to think	25.9	30.6	16.5	20.0	7.1	43.6

The study was approved by the ethics committee of Umeå University.

3. Results

3.1. Pain Intensity. For all patients, the pain intensity on the VAS was 65.8 ± 20.2 mm. No statistically significant difference was found between women (65.5 ± 21.1 mm) and men (66.8 ± 18.2 mm).

3.2. Postconcussion Symptoms. The most common PCSs reported were fatigue, sleep disturbance, and poor concentration (Table 1). The most common symptoms rated as a problem were fatigue, sleep disturbance, and headache (Table 2). Statistically significant differences between women

and men were only found for the symptoms sleep disturbance, feeling frustrated, blurred vision, and taking longer to think (Table 1). No statistically significant difference between men and women was found for the total RPQ score.

3.3. Posttraumatic Stress. The total score of the IES for all patients was 19.3 ± 15.0, and the scores for the subscales were avoidance 9.8 ± 9.1, and intrusion 9.5 ± 7.3. Mild level of posttraumatic stress was reported by 37.2%, moderate stress by 22.1%, and severe stress by 8.1%. No statistically significant differences between men and women were found with respect to total IES (men: 19.7 ± 13.3; women: 19.1 ± 15.8), avoidance (men: 10.2 ± 8.8; women: 9.6 ± 9.3), and intrusion (men: 9.5 ± 5.9; women: 9.5 ± 7.8).

3.4. Depression. Depression scores on the HAD for all patients were 6.9 ± 4.4 mm (women: 7.4 ± 4.3; men: 5.9 ± 3.9). A significantly higher proportion of women (47.5%) reported possible-probable depression (HAD score ≥ 8) compared to men (22.2%) ($P = 0.038$).

3.5. Correlations. Total score of RPQ was significantly correlated to posttraumatic stress ($r = 0.375$, $P < 0.001$), HAD anxiety ($r = 0.455$, $P < 0.001$), and HAD depression ($r = 0.560$, $P < 0.001$). No significant correlation was found between RPQ and VAS ($r = 0.150$, $P = 0.183$).

4. Discussion

The present study shows that patients with injury-related pain often reported postconcussion symptoms several years after injury. Although more women than men participated in the study, few differences between genders were found. A significant relationship was found between postconcussion symptoms and posttraumatic stress and between postconcussion symptoms and depression and anxiety.

As whiplash is reported as the most common traffic injury, it was not surprising that most patients related their chronic pain condition to a previous whiplash trauma. Previous studies have used several different constructions of questionnaires to ask about postconcussion-like symptoms after whiplash [14, 15]. Neck pain and headache are the most often reported complaints after whiplash injury, but other symptoms such as dizziness and visual impairments have also been reported [15]. In comparison with a previous follow-up study that also used the RPQ to examine persons five years after whiplash trauma [25], the proportion of the separate symptoms, except for poor concentration, was clearly higher in the present study. The differences may be due to different study populations. Patients in the present study represent a selected group of patients with chronic pain who exhibited severe consequences after their injury and who were referred to a specialist clinic.

The frequencies of symptoms were high and clearly higher than that for MTBI patients who reported on the RPQ from our hospital one year after the trauma [5]. The most common problematic symptoms were headache, fatigue, and sleep disturbance. In a previous study from Canada of MTBI-patients that also used the RPQ, these symptoms were also the most frequently cited both ten days and six weeks after injury [26]. In patients with MTBI and chronic pain, sleep dysfunction is common. Sleep dysfunction is important to assess since sleep dysfunction and fatigue have been shown to aggravate pain and other symptoms [27]. The frequency of cognitive symptoms in the present study was surprisingly high; more than half of the patients described memory and concentration difficulties. Cognitive difficulties are often related to neuropsychological impairments after MTBI, but not all patients are investigated using neuropsychological tests. These patients are screened using self-reported symptoms of memory and concentration dysfunction [6]. In addition, pain has been associated with worse cognitive functioning in persons with a traumatic brain injury (TBI). Pain, posttraumatic stress, and depression all could cause prolonged cognitive impairment after MTBI [15]. In the present study, these factors were also significantly correlated to post-concussion symptoms.

Some limitations of this study should be noted. General practitioners referred patients to a pain rehabilitation clinic because they reported injury-related chronic pain due to an injury sustained more than twelve months since the referral. Thus the results represent a selected group of patients with chronic pain and with severe consequences after the injury.

Although patients reported high frequencies of symptoms, these are seldom assessed in patients with chronic injury-related pain. The results in the present study agree with Smith-Seemiller et al.; they demonstrated that post-concussion symptoms were common in patients with chronic pain [19]. Since several studies have shown a lack of specificity of PCS [15, 28], the challenge is to establish a causal link between MTBI and PCS and to the diagnosis post-concussion disorder. According to our findings, the optimization of treatment for PCS requires clinicians to assess postconcussion symptoms, to investigate causes for each patient, and to account for factors such as posttraumatic stress and depression.

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Hindawi Publishing Corporation
Rehabilitation Research and Practice
Volume 2012, Article ID 174579, 6 pages
doi:10.1155/2012/174579

Review Article

Substance Use and Mild Traumatic Brain Injury Risk Reduction and Prevention: A Novel Model for Treatment

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Received 5 December 2011; Revised 15 February 2012; Accepted 21 February 2012

Academic Editor: Brian D. Greenwald

Traumatic brain injury (TBI) and substance use disorders (SUDs) frequently co-occur. Individuals with histories of alcohol or other drug use are at greater risk for sustaining TBI, and individuals with TBI frequently misuse substances before and after injury. Further, a growing body of literature supports the relationship between comorbid histories of mild TBI (mTBI) and SUDs and negative outcomes. Alcohol and other drug use are strongly associated with risk taking. Disinhibition, impaired executive function, and/or impulsivity as a result of mTBI also contribute to an individual's proclivity towards risk-taking. Risk-taking behavior may therefore, be a direct result of SUD and/or history of mTBI, and risky behaviors may predispose individuals for subsequent injury or continued use of substances. Based on these findings, evaluation of risk-taking behavior associated with the co-occurrence of SUD and mTBI should be a standard clinical practice. Interventions aimed at reducing risky behavior among members of this population may assist in decreasing negative outcomes. A novel intervention (Substance Use and Traumatic Brain Injury Risk Reduction and Prevention (STRRP)) for reducing and preventing risky behaviors among individuals with co-occurring mTBI and SUD is presented. Areas for further research are discussed.

1. Introduction

Traumatic brain injury (TBI) and substance use disorders (SUDs) frequently co-occur. Individuals with histories of alcohol or other drug use are at greater risk for sustaining TBI, and individuals with TBI frequently misuse substances pre- and post-injury [1–6]. Research suggests that members of general population who consume alcohol are at four times the risk of sustaining a TBI than those who do not [2]. Up to 75% of TBIs are incurred when individuals are intoxicated [2, 7]. These figures are not surprising given that alcohol use is implicated as a risk factor for injury resulting from motor vehicle accidents, falls, and/or violence. Further support for the link between intoxication and serious injury exists due to factors such as poor motor control, impaired decision making, vulnerability to victimization, or propensity toward belligerent/aggressive behaviors secondary to substance use. Moreover, prior history of a SUD, regardless of the presence of intoxication at time of injury, is a risk factor for morbidity and excessive use following injury [8].

Postinjury substance using behaviors are also problematic [9–11]. While a decrease in alcohol and other drug use and higher rates of abstinence have been observed immediately after TBI [12, 13], return to preinjury levels of consumption [12, 13] or increased use at one year after injury have been reported [14, 15]. Long-term substance abuse may increase as the time postinjury increases [9, 14–16], particularly among individuals whose use is not restricted by external factors (e.g., living in an institution where access to substances is limited, under consistent supervision by a caregiver who does not use substances). In the Veteran population, Brenner and colleagues [17] found that among individuals with TBI, the likelihood of problematic postinjury drug and alcohol use, given a preinjury history, was significantly higher than the probability given no history. Similar results were observed for members of the general TBI population with a history of alcohol abuse [13].

A growing literature supports the relationship between comorbid histories of TBI and SUDs and negative outcomes [5, 8, 15]. Those with co-occurring histories are at greater risk for subsequent injury and/or psychosocial and psychiatric problems than those without such misuse [1, 3, 18–21]. In comparison to those who sustained a TBI but did not have substance use problems, individuals with histories of co-occurring misuse/abuse and TBI report (1) lower subjective well being and life satisfaction; (2) unmet psychological needs; and (3) increased perceived barriers to mental healthcare [22–24]. Alcohol use prior to sustaining a TBI has been found to increase the risk of an individual developing mood disorders after injury [22], and individuals with substance abuse (SA) after TBI exhibit more severe psychiatric symptoms than those who have SA problems without a history of TBI. Additionally, in comparison to the general population, the risk of death by suicide has been reported as being four times higher for those with TBI and co-morbid SUD [25]. Olson-Madden and colleagues [26] found that, among SA treatment-seeking Veterans with a history of TBI, individuals at risk for TBI are also at risk for mental illness and vice versa, providing further support for the hypothesis that the cumulative impact of co-occurring conditions would be expected to culminate in poorer outcomes [26].

Risk taking refers to the tendency to engage in behaviors that have the potential to be harmful or dangerous, but which may be perceived by the person engaging in the behavior as an opportunity to obtain a positive outcome (e.g., short-term pleasure). Such risk taking is present in a variety of behaviors, including substance use, gambling, unprotected sex, or dangerous pasttimes such as skydiving. Risky behavior can be conceptualized as an expression of a personality trait that Zuckerman [27] identifies as "sensation seeking." This trait embodies four components: thrill/adventure seeking, experience seeking, disinhibition, and susceptibility for boredom. Zuckerman [28] conceptualizes this trait as having a strong biological influence, claiming that a tendency toward sensation seeking and risk taking may be genetic.

Another factor that can contribute to risky behavior is an individual's appraisal of risk. Work by Gilman and colleagues [29] shows that after consuming alcohol, social drinkers have decreased sensitivity in brain regions involved in detecting threats and increased activity in brain regions involved in reward [29]. This suggests that after alcohol exposure, threat-detecting brain circuits are less able to differentiate between threatening and nonthreatening social stimulus. One outcome could be failure to avoid risky situations (e.g., an argument and a fight). Dulled reactions also could be translated into potentially dangerous situations such as drunk driving.

Studies show that there is a strong association between risk taking and alcohol and other drug use [27]. The search for novel experiences sets off the same brain reward system as substance use, providing a biological explanation for alcohol and drug abuse among people who constantly seek out new and exciting experiences. The hypothesis is that the inhibitory response of these personality types is also diminished, adding to the individual's tendency to take risks. Disinhibition, impaired executive functioning, and/or impulsivity (e.g., lack of premeditation and sense of urgency) [29–31] as a result of TBI could also contribute to an individual's proclivity towards risk taking. Risk-taking behavior may be a direct result of SA and/or TBI, and risky behaviors may predispose individuals for subsequent injury or continued use of substances. As such, evaluation of risk-taking behavior associated with the co-occurrence of SA and TBI seems to be a salient area for further study.

2. Implications for Treatment

Critical gaps exist with regard to how best improve TBI-related outcomes for individuals who continue to engage in risky behaviors like misusing substances. According to the Centers for Disease Control Injury Research Agenda [32], priorities regarding TBI intervention should focus on understanding and preventing the development of secondary conditions following TBI, and identifying strategies to ensure care for those with TBI. Some findings suggest that meeting the needs of individuals with SUDs and TBI may require clinicians to modify current practices to address cognitive dysfunction, poor emotional regulation, and/or limited social skills. For those with mild TBI (mTBI), few evidence-based treatments are available. Generally, comprehensive neuropsychological rehabilitation therapies, which involve a combination of therapies targeting cognitive, emotional, interpersonal, and motivational deficits associated with TBI, are focused on, and more effective for, those with moderate to-severe TBI. While there is some evidence that rehabilitation increases community integration and engagement in work in persons with moderate/severe TBI, the same has not been demonstrated in individuals with mTBI [33]. Given the unique needs of individual with mTBI and co-occurring SUDs, treatments focusing on providing education regarding symptoms of both conditions, coping strategies to minimize their impact, and reducing risky behaviors may be more successful than existing treatments [34].

There is also some evidence that motivational interviewing (MI) techniques and motivational counseling [35] may be potentially efficacious for preventing use or return to substance abuse postinjury. Similarly, structured motivational

counseling [36], a community model using consumer and professional education, case management and consultation to address SUDs in adults with mTBI, also may improve outcomes. Quasiexperimental studies have provided modest support for the efficacy of MI and case management among those with history of TBI. Furthermore, there is some evidence to suggest that increasing negative outcome expectancies could lead to reductions in drinking [37]. As such intervention strategies which increase negative substance expectancies may contribute to risk reduction. Specifically, motivational enhancement therapy (MET) [36] may assist clients in exploring the pros and cons of substance abuse, or the pros and cons of thrill seeking or other risky behavior.

Employing such strategies may have particular relevance for individuals at elevated-risk for mTBI and SUDs, such as military personnel returning from conflicts. In particular, among Operation Enduring Freedom/Operation Iraqi Freedom Soldiers (OEF/OIF), TBI has been described as the signature wound, with rates of mTBI being reported as high as 23% [38]. The RAND Corporation [39] reported a "probable" TBI prevalence of 19.5%, which is equated with approximately 320,000 Veterans who served in Iraq or Afghanistan having sustained probable TBIs. Furthermore, a recent study by Olson-Madden and colleagues [26] indicated that 55% of a sample of Veterans seeking SA treatment in a metropolitan VA hospital had a positive history of TBI. Significant rates of SUDs among returning Military Personnel have also been identified (e.g., 11% acknowledge having an alcohol problem) [40].

3. A Potential Intervention to Address Risky Behavior

Individuals with mTBI and SUD may warrant a treatment targeting their potentially unique needs. The Substance Use and Traumatic Brain Injury Risk Reduction and Prevention (STRRP), seeks to address the tension between preventing a behavior and reducing the harmfulness of that behavior (e.g., harm reduction and risk reduction). It is an integrative model that incorporates aspects of motivational enhancement treatments as well as more educational approaches to treating individuals with mTBI and SUD. Specifically, individuals are provided with information and then asked to explore the relevance of this information using ME strategies. One example of this is providing information regarding common difficulties after mTBI which is offered at the beginning of one session, and group members are invited to consider how such problems might manifest in their own lives. They are then invited to consider the implications of reducing or avoiding behaviors that may contribute to, or be exacerbated by, those problems. The underlying therapeutic principles in STRRP include empathic listening, developing and emphasizing the discrepancy between the individual's present behavior and his/her goals and values, and supporting self-efficacy and confidence to change risky behaviors. A consistent finding in the literature involves the impact of therapist characteristics [41], with therapist empathy as a significant predictor of favorable treatment outcomes [42, 43]. Given the lack of knowledge concerning common manifestations and consequences of mTBI, educational components include a review of the common manifestations of mTBI, as well as a focus on symptom management, including physical, cognitive, and behavioral/emotional sequelae.

Specifically, it combines evidence-based guidelines with an orientation toward recovery that values clients' personal experiences and choices [44]. While this intervention and accompanying manual was designed to be used within the high-risk population of those with a history of mTBI seeking SA treatment, reducing alcohol and other drug use behaviors are not the only targets for intervention. The intervention emphasizes the importance of decreasing all behaviors that are harmful and increase an individual's risk for poor outcomes including risk for reinjury and psychiatric/psychosocial symptoms. It was developed based on the premise that changing risky behaviors in general is likely to result in positive SUDs treatment gains and reduce the risk for future TBI and other negative outcomes.

STRRP is based largely on the premise that people need to be ready for change [45]. This model suggests that individuals move through five stages of change, from not thinking about change to maintaining long-term change. This approach requires that the clinician continually assesses the client's readiness to change and promotes motivation through a series of techniques based on that individual's level of readiness. In particular, MI [19] and group-based ME [36] strategies are integral to each STRRP session for the purpose of facilitating clients' movement toward decreasing risky behaviors. MI is a well-documented approach informed by the Stage Theory of Behavioral Change of Addictive Behavior [45]. Emphasis is placed initially on feedback, future planning, and motivation for change, followed by reinforcement of progress and providing an objective perspective on the process of change. Collaboration with the client, versus confrontation or maintaining an authoritative approach, is a key strategy to this modality, providing opportunity for exploration as well. It is believed that change is motivated by a perceived discrepancy between present behavior and important personal goals and/or values, and so assisting the individual to identify such discrepancy is likely to facilitate change. Finally, supporting the individual to achieve self-efficacy around changing behaviors is a necessary principle of MI/ME.

In addition to incorporating MI/ME strategies throughout STRRP, psychoeducational information regarding mTBI and the impact of it when combined with SUDS and psychiatric disorders are provided. Psychoeducational materials were created from health and wellness models, and TBI-specific materials were adapted from a community-based intervention called the Substance Abuse and Traumatic Brain Injury Toolbox [46]. The toolbox was designed for healthcare facilities to address SA in clients receiving TBI rehabilitation in part by utilizing group media-based psychoeducation (e.g., a video) to address and facilitate substance use prevention through willingness to change behavior. Topics discussed include prevention, intervention, and treatment resources.

As currently conceptualized, STRRP is a manualized, 13-week, group-based intervention. Ideally, patients would

be referred to treatment upon identification of positive mTBI history and/or persistent complications related to mTBI. Treatment topics emphasize recovery, resumption of work/social/interpersonal obligations, and intervention and prevention regarding risk-taking behaviors (e.g., excessive substance use, behaviors that may lead to reinjury or poor psychiatric outcomes). The intervention is highly experiential, and clients practice and consolidate their learning via weekly assignments.

4. Conclusions

Substance use and abuse frequently occurs with other risk-taking behaviors. While some interventions specifically may help decrease substance misuse, other interventions may be better suited to address additional risk-taking behaviors. Further, considerations for treatment regarding potential complications associated with history of mTBI are warranted, for example, when behavior or cognitive ability conflicts with ability to engage in or gain from the current treatment [47]. Strategies which accommodate for limitations in thinking (e.g., repetition of material and assignments in-between session for consolidation) may be indicated. Regardless of history of mTBI, however, the aim of treatment in any setting is likely to optimize functioning and quality of life. It would be expected that some benefit would result from addressing risk factors and possible sequelae associated with co-occurring mTBI and SUDs.

In any case, rigorous evaluation of potentially efficacious treatments addressing these co-occurring behaviors is missing in the literature. As such, the authors of this paper seek to evaluate the efficacy and effectiveness of STRRP via a sequential and orderly research plan starting with assessment of feasibility and establishing an evidence base. In addition to collecting data regarding outcomes of interest (e.g., patient readiness to change) practical feasibility-related questions such as the following must be addressed: (1) Will the intervention work given the structure and rules of an existing SUDs treatment program? (2) Will changes need to be made to the manual to better tailor the intervention to the target population? and (3) Will activities of the intervention interfere or conflict with the established services? Areas of interest include patient comprehension of outlined procedures and clinicians' ability to implement practices. Such procedures for this evaluation fall under "Stage 1" of Onken et al.'s model, [48, 49] which consists of pilot/feasibility testing, manual and training program development, and adherence/competence measurement for new and untested treatments. Areas of interest include patient comprehension of outlined procedures and clinicians' ability to implement practices. In line with the model [48], the proposed next steps may include modifications to content and/or procedures specified in the original treatment manual as a result of feedback and expert consultation throughout the duration of the proposed feasibility study. The result of feasibility testing ultimately will be a treatment and accompanying manual sufficiently prepared for future randomized clinical trials, and dissemination, and implementation research.

In conclusion, further study is needed to clarify the mental health needs of individuals with co-occurring mTBI and SUDS to identify best practices. This proposed model may be a viable treatment approach that will contribute to the literature and evidence base. It may also offer significant impact in its attempt to address and improve significant clinical issues in military personnel and Veterans (co-occurring mTBI, SUD, and other risky behaviors).

Disclaimer

The views in this paper are those of the authors and do not necessarily represent the official policy or position of the Department of Veterans Affairs or the United States Government. This material is the result of work supported with resources and the use of facilities at the Eastern Colorado Health Care Center VA in Denver, CO, USA. The development of the novel intervention, STRRP, was made possible via a project funded by the Department of Defense Congressionally Directed Medical Research Program (CDMRP) Hypothesis Development Award (HDA): DR0 80125.

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Hindawi Publishing Corporation
Rehabilitation Research and Practice
Volume 2012, Article ID 924692, 10 pages
doi:10.1155/2012/924692

Research Article

The Association between Pain-Related Variables, Emotional Factors, and Attentional Functioning following Mild Traumatic Brain Injury

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Received 28 October 2011; Accepted 6 February 2012

Academic Editor: Gina P. Armstrong

This study examined how MTBI concomitants such as pain variables, depression, and anxiety were related to attentional functioning at different stages of recovery. Participants having sustained a MTBI who were in the earlier phase of recovery showed, compared to controls, slower reaction times and larger intra-individual variability on a Computerized Pictorial Stroop Task (CPST). They also reported more post-concussion symptoms, pain intensity and disability, whereas MTBI participants who were in the later phase of recovery presented a higher rate of post-concussive symptoms and somewhat higher pain intensity/disability. MTBI participants' scores on the cognitive items of the post-concussion symptoms scale were positively correlated with reaction times on the CPST, while pain intensity/disability levels were negatively correlated with standard attention measures. Results indicate that obtaining response times and intra-individual variability measures using tests such as the CPST represents an effective means for measuring recovery of attentional function, and that pain intensity/disability should be systematically assessed after a MTBI.

1. Introduction

Individuals who have sustained a mild traumatic brain injury (MTBI) may manifest postconcussional symptoms of a physical, cognitive, or emotional nature [1, 2]. Possible cognitive symptoms include difficulties with concentration, attention, memory, executive functioning [3], word finding, and information processing [4]. Therefore, the cognitive impact of MTBI can be extensive and wide ranging [5]. It is well known that of the reported symptoms, attention is especially problematic for many individuals having had a MTBI. In fact, meta-analytical studies indicate that attention deficits are the most persistent neuropsychological complaint following closed-head injury [6, 7]. Understanding these attentional difficulties is important in planning management and rehabilitation of persons suffering from the consequences of MTBI.

Multiple studies have demonstrated that MTBI produces attentional deficits [8, 9]. In fact, there appears to be a large variety of attentional deficits found within this group of patients in terms of both reported symptoms and neuropsychological performance [5]. Divided attention deficits and sustained attention deficits have been identified in the MTBI population [10–12]. As well, results of some experimental studies suggest that MTBI may produce difficulty in effectively filtering relevant sensory information from irrelevant information, or of selective attention [9, 13].

Pathophysiologically, acceleration, deceleration, and rotation forces are involved in traumatically induced brain injuries [14]. These forces cause microscopic shearing and diffuse damage to neurones [14, 15] in the cortex as well as in subcortical white-matter. As well, these forces also lead to a complex neurometabolic/neurochemical cascade that interferes with axonal transport [16, 17] and can result in axonal

blebbing and eventual disconnection [18]. Certain areas of the brain are more likely to be impacted by such processes, particularly the frontal lobes [15, 19]. The frontal cortex accounts for executive functioning, attention processes, working memory, and self-regulation [20]. Hence, deficits of attention identified following a MTBI are compatible with the pathophysiological changes that may occur with this type of injury.

Postconcussional symptoms usually resolve within 3 months [21]. Nonetheless, in approximately 5% to 15% of cases, individuals continue to show symptoms (including cognitive and attentional complaints) that are sometimes but not always identified on neuropsychological testing [2]. Of the many variables that are reportedly associated with persisting symptomatology are female gender, older age, poor academic achievement, lack of social support, and previous head injury [22]. Psychological factors have also been associated with persisting symptomatology and specifically, with persisting cognitive symptoms [23]. For example, Sherman et al. [24] found evidence that the depression status of persons having sustained a mild head injury 2 years before was related to scores on visual attention tasks and on psychomotor measures. Also, Gass [25] found that closed-head injury patients at 2.6 years after injury obtained scores on measures of attention that were related to scores obtained on the MMPI-2 anxiety scale. It is unclear if these associations between depression symptoms, anxiety symptoms, and performance on attention tasks are also present in the earlier stages of recovery that follow MTBI.

In trying to understand the attentional complaints of those having suffered a MTBI, one must consider the fact that acute pain and chronic pain problems may also be involved. It is in fact quite common for those having had a head injury to also present with comorbid pain issues. A study examining the prevalence of chronic pain (i.e., still present after 3 months) following TBI found that it was a very common complication [26]. It is especially common in the MTBI population, as indicated by the findings of Uomoto and Esselman [27], who reported that 95% of individuals that had sustained a MTBI reported chronic pain while only 22% of those having suffered a moderate- to-severe TBI did so. As for the type of pain presented by patients, Guérin et al. [28] found that 70% of individuals receiving out-patient holistic rehabilitation services following a MTBI presented posttraumatic headaches, while over 85% reported significant pain symptomatology in other parts of the body.

Chronic pain in itself is known to impact cognitive functioning. A review examining the impact of chronic pain on neuropsychological functioning [29] found that impairments on measures of attention, processing speed, and psychomotor speed were often present in the chronic pain population. There has also been some interest for the study of the stimulus-driven effects of pain-related information, on the attention of pain patients. Some research groups have in fact found evidence, using modified Stroop tasks and dot-probe paradigms [30] that chronic pain patients may develop a hypervigilance to pain, pain-associated information and environmental stimuli representative of pain, hence, having difficulty disengaging from this type of information and manifesting an attentional bias for this type of information [31]. Consequently, pain may compete with other attentional demands, leading to difficulties with attention [32].

There has been little research examining the cognitive/neuropsychological performance of TBI patients suffering from comorbid pain, as many studies have excluded this group that presents potential confounds. Furthermore, the existing studies have often specifically examined the effect of head and neck pain, which is frequent in the TBI population, but has also been suggested as being a particular type of pain which may increase vulnerability to cognitive impairment [29]. There are no published articles that have examined the impact of nonheadache/neck pain on the cognitive functioning and more specifically on attentional function of persons having sustained a MTBI, but some information may be provided by taking a closer look at a study performed with TBI patients with injuries of a variety of severity levels. In an abstract, Vernon-Wilkinson and Tuokko [33] describe how they examined the records of patients referred for assessment of head injury. Records were divided into those with and those without pain, regardless of the severity of brain injury. Although the brain injured patients with comorbid pain had less severe head injuries in terms of Glasgow Coma Scale scores as well as in terms of duration of posttraumatic amnesia and measures of ventricular enlargement, they did poorer than the brain injured patients without pain on many neuropsychological tests.

Hence, the assessment of attention following a MTBI can be influenced by a number of comorbidities, such as pain, symptoms of depression, as well as symptoms of anxiety. Each of these is frequently present in those having sustained a MTBI [26, 34, 35], may impact results obtained on measures of attention, and may also complicate or delay recovery, and must be better understood in order to provide appropriate rehabilitation interventions. The aim of the present study was to examine selective attention using both established neuropsychological measures and a newly designed Computerized Pictorial Stroop Task and to identify associations with MTBI concomitants using measures of depression, anxiety, pain variables, and postconcussive symptoms, in individuals having suffered a MTBI and who were in different stages of recovery (at 1–3 months after injury and at 5–7 months post-injury). Our hypotheses were that persons with MTBI perform worse than normal controls on selective attention measures, and that more important pain-related, cognitive and affective symptomatology is related to worse attentional performance, irrespective of stage of recovery.

2. Methods

2.1. Participants. Our study involved three groups of participants. These included two groups of individuals who had sustained a MTBI: 15 who were in the earlier phase of recovery, MTBI-early (mean months after injury = 2.2 ± 0.5; 10 men and 5 women, mean years of age: 39 ± 13, mean years of education: 13 ± 3), and 15 who were in the later phase of recovery, MTBI-late (i.e., mean months after injury = 5.6 ± 1.2; 13 men and 2 women, mean years of age: 38 ± 13, mean years of education: 13 ± 3).

The third group (normal controls) was made up of 17 neurologically unimpaired adults (10 men and 7 women, mean years of age: 31 ± 11, mean years of education: 14 ± 2). One-way ANOVAs showed that there were no significant differences between the three groups for age ($F(2, 44) = 2.29$, $P = .11$) and years of education ($F(2, 44) = 0.99$, $P = .38$). Normal controls were recruited from the community through local advertisement. Some patient participants were recruited from a neurotrauma unit and the majority were from an out-patient intervention program offered in a major rehabilitation center. All subjects were French-speaking and they all provided written informed consent. The study was approved by the institutional ethical review board.

All participants in the MTBI groups had sustained a MTBI based on the criteria of the American Congress of Rehabilitation Medicine Brain Injury-Interdisciplinary Special Interest Group [36]. According to these criteria, MTBI results from traumatically induced physiological disruption of brain function, manifested by *at least one* of the following: (1) any period of loss of consciousness; (2) any loss of memory for events immediately before or after the accident; (3) any alteration of mental state at time of the accident (e.g., feeling dazed, disoriented, or confused); (4) focal neurologic deficit(s) that may or may not be transient; but where the severity of the injury does not exceed a loss of consciousness of approximately 30 minutes or less; after 30 minutes, an initial Glasgow Coma Scale of 13–15, and posttraumatic amnesia not greater than 24 hours. TBI severity was identified in patient medical files and was confirmed by reviewing medical records related to neurological indices and via comprehensive retrospective patient interviews. The majority of participants had uncomplicated MTBI, that is, had negative clinical brain imaging (complicated MTBI: 3 in the early group; 2 in the late group). The causes of injury in the MTBI-early group were motor vehicle accidents ($n = 9$), falls ($n = 3$), and physical assaults ($n = 3$). In the MTBI-late group, causes of injury were motor vehicle accidents ($n = 8$), falls ($n = 5$), physical assault ($n = 1$), and a work-related accident ($n = 1$). Potential participants were excluded from the study for the following reasons: previous TBI (except that for which MTBI participants were referred), headache or neck pain, alcohol or substance abuse within 6 months prior to testing, intake of medication which can affect cognition (ex. opioids, anticonvulsants), or uncorrected visual impairment.

2.2. Materials

2.2.1. Self-Report Questionnaires

(1) *French Version of the Beck Depression Inventory: Second Edition.* The Beck Depression Inventory-II [37] is a 21-item questionnaire that is used to assess symptoms of depression. Each item is rated on a Likert scale ranging from 0 to 3. A score of <10 means no or minimal depression, 10 to 17 is mild to moderate depression, and 18 to 29 is moderate to severe depression, and severe depression is from 30 to 63.

(2) *French Version of the Beck Anxiety Inventory.* The Beck Anxiety Inventory [38] is made up of 21 items, each corresponding to a symptom that presents in anxiety disorders. Respondents must rate each symptom on a scale ranging from 0 to 3, based on how frequently they have suffered from that symptom in the past 7 days. A score of <7 represents a minimal level of anxiety, 8–15 represents mild anxiety, 16–25 is interpreted as moderate anxiety, and 26–63 represents severe anxiety.

(3) *French Version of the Postconcussion Scale.* The Post-concussion Scale [39] questionnaire was filled out by all participants. This scale was developed to provide a formal method of documenting postconcussion symptoms [40]. The scale is made up of 22 commonly reported physical, cognitive, and affective symptoms of TBI (e.g., dizziness, difficulty concentrating, sadness) and is commonly used with the MTBI population. Patients are asked to rate the intensity of every symptom on a Likert scale ranging from 0 to 6.

(4) *Present Pain Intensity from the French Version of the McGill Pain Questionnaire.* The Present Pain Intensity of the McGill Pain Questionnaire [41] is a ten-word intensity rating scale. Pain is rated from 0, which signifies "no pain," to 10, which is "intolerable" or "excruciating."

(5) *French-Canadian Version of the Pain Disability Index.* The Pain Disability Index [42] is designed to help patients measure the degree to which their daily lives are disrupted by persistent pain. It is made up of 7 items (each corresponds to a category of life activity, e.g., recreation, occupation) that the patient must rate on a Likert scale ranging from 0 to 10. For each of the categories, a score of 0 means no disability at all, and a score of 10 is equivalent to complete disability.

All questionnaires are widely used and both their English and French versions have been found to have excellent validity and reliability.

2.2.2. Assessment of Attention

(1) *Map Search from the Test of Everyday Attention (TEA) [43].* Participants each had 2 minutes to identify as many target stimuli as they could on a map. The obtained score was the number of targets identified out of a maximum of 80. Raw scores were used for statistical analyses.

(2) *Telephone Search from the TEA [43].* Participants had to look for designated key symbols and ignore other symbols, while searching entries in a simulated classified telephone directory. The score was calculated by dividing the total time taken to complete the search, by the number of symbols detected. The maximum number of symbols that could be detected was 20. Raw scores were used for statistical analyses.

(3) *Ruff 2 and 7 [44].* Participants completed the 20 trials (10 letter trials and 10 digit trials) of this visual search and cancellation task. In each of the trials, participants were required to detect and mark through as many as possible of the two target digits: "2" and "7," within a 15 s time limit. Automatic Detection Accuracy raw score and Controlled Search Accuracy raw score were used for statistical analyses.

Automatic Detection Accuracy raw score is calculated by dividing the Automatic Detection Speed raw score by the sum of the Automatic Detection speed and Error raw scores and by multiplying this number by 100. Controlled Search Accuracy is calculated by dividing the Controlled Search Speed raw score by the sum of the Controlled Search Speed and Controlled Search Errors raw scores and by multiplying this number by 100.

(4) *Computerized Pictorial Stroop Task.* The Computerized Pictorial Stroop Task is a selective attention task which involves a patient/participant naming the colour of the frame surrounding a picture, as quickly as possible. Hence, the patient must process the relevant information (colour of the frame) while trying to filter the irrelevant information (the picture). The development of this task stems from studies utilizing the modified Stroop paradigm, which found evidence that chronic pain patients selectively attend to both sensory and affective pain words (in comparison to neutral words) [45]. It was thought that replacing the words by pictures would add ecological validity to the task, as this aspect of the task had before been criticized [46, 47].

The task involved participants completing 2 blocs comprised of 80 trials (or pictures) each. The task included three categories of pictures: pictures representing pain, pictures representing anger, and neutral pictures. Pain pictures consisted of pictures of human faces expressing pain, and pictures of hands and feet in pain-evoking situations stemming from previously published picture databases in which the intensity of emotion was considered and controlled during the development of the database [48, 49]. Pictures representative of anger consisted of pictures of human faces presenting facial expressions of anger, and stemmed from the same database as pictures of human faces presenting expressions of pain [48]. Each of the experimental pictures was matched with a neutral picture (i.e., the same human face presenting a neutral facial expression or hand/foot in a non-pain-evoking situation). Pictures measured 256×256 pixels. Frames surrounding the presented pictures measured 8 pixels and the picture-frame distance varied in random fashion. Frames were presented in each of the following four equiluminant colours: red, yellow, green, and blue. The stimuli were presented in the center of the screen of a laptop computer. Patients were instructed to say out loud the colour names of each frame surrounding the presented pictures, as quickly as possible, without sacrificing accuracy. Latency in naming the colour of the frame was recorded with a Plantronics USB DSP V2 microphone. Stimuli were presented for 1000 ms and the interstimulus interval varied between 500–750 ms.

Measures obtained from the Computerized Pictorial Stroop Task included mean reaction time and intraindividual variability of reaction time. Mean reaction time was calculated by adding a particular patient's reaction times to all trials involving a certain type of picture (pain, anger, or neutral). This total was then divided by the number or trials that were summed up. The intraindividual variability of reaction time was calculated by computing the difference between the reaction time to each of the trials involving a certain type of picture (either pain, anger, neutral) and the mean reaction time to that type of picture. Each of these differences from the mean was then squared and the average of these values was calculated. The square root of this average is the intraindividual variability of reaction time. Examining intraindividual standard deviation as a measure of variability/consistency of performance has been used in prior head injury studies [50].

2.3. Procedure. All subjects participated in an individual testing session. They first underwent a screening of medical and psychiatric history, which included questions regarding pre- and postmorbid functioning, self-reported symptomatology, and psychiatric history. Following this, anxiety, depression, pain disability, and postconcussion symptoms as well as current pain intensity were assessed by means of the self-report questionnaires. Patients were randomly assigned to first complete either the standardized attention tasks, or the Computerized Pictorial Stroop Task. During the Computerized Pictorial Stroop Task, patients were seated at a distance of 57 cm from the computer screen and they were asked to focus on the centre of the screen at all times. Patients practised the colour-naming task with 10 pictures, which were not included in the experimental set, until they understood the task. The pictures were presented in a random sequence.

2.4. Statistical Analyses. Descriptive statistics were first computed to determine group means and standard deviations obtained on the standardized attention tests, on the Computerized Pictorial Stroop Task and on self-report inventories. Three separate univariate ANOVAs using planned contrasts (MTBI-early versus normal controls; MTBI-late versus normal controls; MTBI-early versus MTBI-late) were then calculated for the TEA subtests (Telephone Search and Map Search) and the Ruff 2 and 7 Selective Attention Test, in order to determine if there were differences in results between the three groups on these measures. Two-way repeated-measures ANOVAs, with image type as the within-subject factor (3 levels: pain, anger, neutral) and with group as the between-subject factor, were used to determine whether there were differences in reaction times, intraindividual standard deviation of reaction times and error rates for each condition of the Computerized Pictorial Stroop Task. Univariate ANOVAs using planned contrasts were then run to examine potential differences between the three groups on scores obtained on self-report questionnaires assessing pain, pain-related impairment, depression, anxiety, and TBI-related symptomatology. A series of Pearson correlations were then computed between scores obtained by participants with MTBI on each of the self-report inventories, and performance on the standardized measures of attention and the Computerized Pictorial Stroop Task conditions. Population variances were systematically assessed using Levene's test and variances were found to be homogenous in all cases. All data were analysed using SPSS version 17.0. The level used for statistical significance was .05.

3. Results

3.1. Attentional Tasks (Map Search from the TEA, Telephone Search from the TEA and Ruff 2 and 7 Selective Attention Test). Mean scores and standard deviations obtained by the three groups (MTBI-early, MTBI-late, and normal controls) on the standardized attentional tests are found in Table 1. According to the TEA manual, all three groups obtained scores that fell within the normal-range (i.e., above the clinical cut-off) on the Map Search and the Telephone Search. Separate three-group ANOVAs revealed that the groups did not differ significantly in their performance on the Telephone Search ($F(2, 44) = 1.53, P = .23$) nor on the Map Search ($F(2, 44) = 1.42, P = .25$). As well, separate three-group ANOVAs revealed that the groups did not differ significantly in their performance on the Ruff 2 and 7 Selective Attention Test, on both automatic detection accuracy ($F(2, 44) = 2.62, P = .08$) and controlled search accuracy ($F(2, 44) = 2.25, P = .12$).

3.2. Computerized Pictorial Stroop Task

Generalized Slowing. Mean reaction times to images (pain, anger, neutral) of the three groups (MTBI-early, MTBI-late, normal controls), as well as their respective standard deviations, are found in Table 2. The repeated-measures ANOVA indicated no significant interaction between image type and group ($F(4, 88) = 1.41, P = .24$). It did yield a significant group effect ($F(2, 44) = 4.09, P = .02$). Post hoc Tukey's tests showed that the MTBI-early group had significantly slower reaction times than those of normal controls, as well as those of the MTBI-late group. All other comparisons were not significant. For the impact of image type (pain, anger, neutral) on reaction time, there was no difference among the different conditions ($F(2, 88) = .016, P = .98$).

Variability in Response Times between Trials. Intraindividual standard deviation of reaction times to images (pain, anger, neutral) obtained by the three groups are found in Table 3, along with the standard deviations for each group. The two-way repeated measures ANOVA performed on the intraindividual standard deviation of response times showed no significant interaction between image type and group ($F(4, 88) = 1.05, P = .39$). It did yield a significant main effect of group ($F(2, 44) = 3.49, P = .04$). Post hoc tests demonstrated that individuals of the MTBI-early group had significantly more variable response times than did normal controls. Other comparisons were not significant.

The ANOVA also revealed a significant effect of image type on intraindividual variability of response time amongst trials ($F(2, 88) = 11.87, P = .00$). All of three pairwise comparisons that were conducted were significant at the .05 level. These demonstrated that intraindividual variability in response times for images of pain was significantly greater than that of images of anger and of that of neutral images. In addition, variability in response times for neutral images was significantly greater than that of images of anger.

Errors. The ANOVA indicated no significant interaction between image type and group ($F(4, 88) = 0.70, P = .59$). As well, no main effect of group was identified ($F(2, 44) = 2.32, P = .11$), nor was there a main effect of image type on errors made ($F(2, 88) = 2.60, P = .08$).

3.3. Self-Report Questionnaires Assessing Depression, Anxiety, TBI-Related Symptomatology, Pain, and Impairment

BDI-II and BAI. Scores obtained by the three groups on the self-report questionnaires can be found in Table 4. One-way ANOVAs did not reveal any significant group effects on scores obtained on the Beck Depression Inventory-II and on the Beck Anxiety Inventory ($F(2, 44) = 1.67, P = .20$ and $F(2, 44) = 0.47, P = .63$, resp.).

TBI-Related Symptomatology. A one-way ANOVA found the effect of group on the PostConcussion Scale to be significant ($F(2, 44) = 6.35, P = .00$). Posthoc Tukey's tests showed that the both MTBI-early and MTBI-late groups obtained significantly higher scores on the self-report inventory than did normal controls. All other comparisons were not significant.

Pain Intensity. A one-way ANOVA determined that the effect of group on the Present Pain Intensity Scale of the McGill Pain Questionnaire was significant ($F(2, 44) = 4.29, P = .02$). Pos thoc Tukey's tests found that the MTBI-early group endorsed significantly higher levels of pain than did normal controls. The MTBI-late group also showed higher pain intensity levels than to controls, but the difference did not reach significance.

Disability Associated with Pain. A one-way ANOVA was computed and found the effect of group on the Pain Disability Index to be significant ($F(2, 44) = 6.94, P = .00$). Post hoc Tukey's tests revealed that the MTBI-early group obtained significantly greater scores on the self-report inventory than did normal controls. Again, the MTBI-late group presented higher pain disability scores than controls, but the difference did not reach significance.

3.4. Correlations between Self-Report Questionnaires and Performance. In order to determine if performance was modulated by effect and by pain symptomatology and disability in the MTBI population, we computed Pearson correlations between scores obtained by all MTBI participants on each of the four self-report inventories (BDI-II, BAI, Present Pain Intensity Scale, Pain Disability Index), and results they obtained on both standard attention measures and the Computerized Pictorial Stroop Task. The correlation between scores obtained by participants with MTBI on the Present Pain Intensity Scale and scores obtained on the Map Search was significant ($r(28) = -.56, P = .001$), as was that between scores obtained by MTBI participants on the Present Pain Intensity Scale and those obtained on the Telephone Search ($r(28) = -.39, P = .03$). As well, the correlation between scores obtained by participants with MTBI on the

Table 1: Scores (mean ± SD) of the three groups (MTBI-early, MTBI-late, normal controls) on standardized measures of attention. F values and P values are given.

	Controls	MTBI-early	MTBI-late	Statistic	P value
Map search	77.53 (5.49)	72.53 (10.27)	73.87 (9.98)	$F = 1.42$	.22
Telephone search	2.44 (0.67)	2.88 (0.77)	2.94 (1.14)	$F = 1.53$	.23
Automatic detection accuracy	95.14 (3.51)	97.40 (2.62)	95.45 (2.55)	$F = 2.62$	.08
Controlled search accuracy	89.03 (7.60)	92.87 (4.51)	93.37 (3.53)	$F = 2.25$	.12

Table 2: Mean reaction times (in milliseconds) to images (pain, anger, neutral) for the three groups (MTBI-early, MTBI-late, normal controls). Standard deviations are in parentheses.

	Controls	MTBI-early	MTBI-late
Pain RT	475.3 (123.75)	620.0 (122.65)	478.7 (159.86)
Anger RT	489.4 (148.34)	596.7 (130.86)	486.0 (186.81)
Neutral RT	492.9 (131.42)	608.0 (111.18)	475.3 (161.24)

Table 3: Intraindividual standard deviation of reaction times (in milliseconds) to images (pain, anger, neutral) of the three groups (MTBI-early, MTBI-late, normal controls). Standard deviations are in parentheses.

	Controls	MTBIs-early	MTBIs-late
Pain	136.5 (43.87)	174.7 (41.90)	128.0 (42.63)
Anger	105.3 (61.45)	125.3 (28.00)	111.3 (55.14)
Neutral	115.9 (42.29)	148.7 (44.38)	122.7 (27.38)

Pain Disability Index and scores obtained on the Map Search was significant ($r(28) = -.4$, $P = .03$). No other correlation reached significance.

In order to evaluate if performance was modulated by TBI-related cognitive symptoms, we computed Pearson correlations between scores obtained by MTBI participants on the cognitive items of the Postconcussion Scale and results they obtained on both standard attention tasks and the Computerized Pictorial Stroop Task. There were significant correlations between this cognitive component of the Post-Concussion Scale and response times to images of pain ($r(28) = .56$, $P = .00$), anger ($r(28) = .5$, $P = .005$), and neutral images ($r(28) = .52$, $P = .003$). None of the correlations between intraindividual variability in response times and the cognitive component of the Post-Concussion Scale were significant. Also, correlations between standard tests of attention and the cognitive component of the Post-Concussion Scale did not reach the significance level.

4. Discussion

Findings from the present study indicate that individuals having had a MTBI may manifest reaction time deficits on a selective attention task. In fact, MTBI participants in earlier phases of recovery showed both slower reaction times as well as less consistent reaction times than healthy normal controls on the Computerized Pictorial Stroop Task. These findings are consistent with prior literature which has consistently revealed a generalized slowing of information processing following head injury [51]. Many authors have suggested that this deficit in information processing is related to diffuse axonal injury (DAI), which typically occurs with TBI [52]. Although to a lesser extent than in more severe TBI, diffuse injury to white matter tracts following MTBI could presumably reduce interconnections between neural networks, thereby reducing the speed at which information is transmitted.

The observed disturbance in the intraindividual variability in reaction time of individuals having sustained a MTBI and who were in the earlier phases of recovery is also consistent with results of previous studies. It has been observed for many years that brain damage causes increased intraindividual variability [53]. Numerous studies using various methods for measuring variability have in fact confirmed greater variability in patients with TBI at all levels of severity, with or without focal frontal lesions [50, 51, 54–57]. In TBI, the extent of intraindividual variability is closely associated with impaired maintenance of stable "top-down" attentional processes, where large intraindividual variability can be due to a general deficit in regulation of attention in any cognitive domain, and is most consistently described in patients with impairments in executive functions [53].

Our findings did not indicate a general slowing of responses (or attentional bias) specific to pain pictures; however we did obtain a main effect of image type on intraindividual variability. Potential explanations of this effect may lie in literature examining the impact of emotional stimuli on attention. In fact, it is well documented that emotional stimuli can interfere with ongoing activities [58]. While several theories exist as to how and why effect influences attention [59], the arousal theory has received much interest. According to this theory, responses to affective pictures vary with the intensity of emotion evoked by the picture. Pictures found to be more arousing, command more attention and hence can interfere with ongoing tasks [59]. Since the pictures used in this study were controlled for emotional intensity, it is likely that the higher intraindividual inconsistency of responses found for images of pain reflected an increased attentional load, resulting in the observed variability for this type of picture. The main effect found, where pain pictures produced more variable responses overall compared to other picture types, but not specifically in the MTBI groups, suggests that our study patients did not show a higher attentional bias toward pain pictures.

Table 4: Scores (mean ± SD) of the three groups (MTBI-early, MTBI-late, normal controls) on self-report questionnaires. F values and P values are given.

	Controls	MTBI-early	MTBI-late	Statistic	P value
BDI	5.29 (±4.93)	9.20 (±6.06)	9.53 (±10.27)	$F = 1.67$	.200
BAI	5.94 (±4.28)	7.80 (±6.81)	9.33 (±15.61)	$F = 0.47$	.630
ESPC-R	2.18 (±4.45)	20.80 (±14.49)	20.93 (±26.55)	$F = 6.35$	.004
Present pain Intensity	0.94 (±2.11)	3.40 (±3.02)	2.47 (±2.00)	$F = 4.29$	.020
Pain disability	1.47 (±4.00)	20.40 (±17.13)	12.07 (±18.49)	$F = 6.94$	.002

This is interesting in light of the fact that pain intensity and pain-related disability ratings also were not related to MTBI participants' performance on the Computerized Pictorial Stroop Task, contrary to standard measures of attention, suggesting that this task was resistant to pain-related variables.

Notwithstanding the above and noteworthy is the fact that these deficits in the speed and variability of reaction time were only apparent in the MTBI group in earlier phases of recovery and not in the MTBI group in later phases of recovery. This result corroborates previous findings that indicate that persons that sustain a MTBI generally recover in the 3-month period that follows their injury in terms of their ability to perform adequately on cognitive/neuropsychological measures [4, 60, 61]. Prior studies have also shown that the post-MTBI focal parenchymal lesions seen on MRI brain scans resolve within 1 to 3 months following injury and that these changes are paralleled by improvements on neuropsychological tests [60]. These last results likely indicate some parallel between neurological recovery, and recovery as seen on cognitive/neuropsychological measures. Important to mention, however, is that while objectively quantified results in our study indicated cognitive improvement in persons having had a MTBI who were in later phases of recovery, this was not reflective of subjective symptoms or of subjectively perceived status, as measured by the Post-Concussion Scale. In fact, MTBI participants in later phases of recovery also were reporting significantly greater amounts of symptoms than normal controls. As will be discussed in later paragraphs, existing literature supports the fact that the improvement of cognitive functions (assessed via neuropsychological measures) and subjective recovery (assessed via self-report symptom scales such as the Post-Concussion Scale) do not always follow the same course, with cognitive recovery either preceding or following the resolution of subjective symptoms [62].

In contrast to the above finding that participants with MTBI who were in earlier phases of recovery show impairment on a computerized reaction time task, analysis of results obtained by MTBI participants on conventional neuropsychological tests of selective attention did not show any impairment compared to normal controls. This is in line with existing evidence that reaction time procedures can reveal cognitive impairment even when normal performance is shown on traditional neuropsychological measures [51]. In fact, conventional neuropsychological tests were designed initially to detect quite severe impairments in patients with neurological and psychiatric illness, in patients with brain lesions and in people exposed to neurotoxic substances [63]. Detection of more subtle cognitive changes or the sensitivity of a particular measure is obviously of particular importance when conducting the neuropsychological assessment of persons who sustain a MTBI. A number of studies have shown the utility of computerized reaction time measures in detecting cognitive changes associated with MTBI [54]. This supports the recommendation made by several authors that clinical neuropsychological evaluations comprise reaction time measures [64]. There are in fact many advantages (both theoretical and practical) to computerized reaction time testing [63]. Among these is the fact that relatively high test-retest reliability coefficients and split half coefficients are reported for RT tests [65, 66]. As well, RT measures are not only useful in initial assessments of cognitive functioning following injury, but also as a tool to track its recovery, as they lack practice effects [67].

MTBI participants in earlier as well as in later phases of recovery were found to be reporting significantly greater levels of postconcussive symptoms than normal controls. This is in agreement with existing literature suggesting that long beyond the typical recovery (or the typical period of resolution of symptoms) interval of 1 to 3 months, it is common for persons having suffered a MTBI to report persisting difficulties [2, 68, 69]. Nonetheless, this finding does raise questions about the discrepancy between objective neuropsychological/neurobehavioural indicators and subjectively reported symptomatology. In fact, in our study, significant subjective symptoms were identified by the MTBI group in later phases of recovery, while no impairment was found in this group on any neuropsychological measure, including the Computerized Pictorial Stroop task. Possible explanations of this discrepancy may be found in the correlations between the Post-Concussion Scale and performance on attention measures. Specifically, we found no correlation between the Post-Concussion Scale total score and performance on standard tests of attention. We did however find that the total score obtained on cognitive items of the scale was significantly correlated to reaction times on the Computerized Pictorial Stroop Task. This finding seemingly illustrates that the self-report symptom scale used in this study assesses a variety of symptoms commonly reported by persons having had a MTBI (e.g., physical/somatic, psychological, and cognitive symptoms) and hence its total score provides a more global picture of recovery that is not restricted to the cognitive domain. Individuals that sustain a MTBI may thus continue to present a variety of symptoms in later phases of recovery (as seen on

the self-report symptom scale), reflecting general functional recovery. In fact, as an alternative to mean group results where it is often impossible to appreciate varying levels of clinical evolution, individual cognitive recovery could be best gauged by examining scores obtained on the Computerized Pictorial Stroop Task, a reaction time measure, which was associated with the cognitive score of the PostConcussion Scale.

Results of our study indicated that participants with MTBI who were in earlier phases of recovery had significantly greater levels of pain (other than head/neck pain) than normal controls at the time of assessment and also presented with significantly greater levels of disability associated with their pain in everyday life. The MTBI-late group also showed higher, although not significantly, pain intensity and pain disability levels compared to controls, indicating that the pain-related recovery process was probably not attained around 6 months after injury. This is not surprising as literature provides evidence of high rates of comorbid pain in the TBI population [26]. As to understand how this reported pain may have impacted performance on attention tasks, we looked to the results of correlations computed between pain questionnaires and attentional performance. Interestingly, mirroring depression and anxiety results, pain and disability levels were not related to performance on our reaction time measure, the Computerized Pictorial Stroop Task, but they did modulate performance on certain standardized neuropsychological measures of attention, while depression and anxiety scores did not. Pain intensity, at the time of the assessment, modulated performance on the Map Search and the Telephone Search of the TEA, while pain disability was linked to results on the Map Search. These findings indicate that even in the absence of head/neck pain, the level of other types of pain at the time of the assessment as well as pain disability should be considered and assessed systematically following MTBI, as they may impact performance on standardized attention tasks. This is consistent with results of studies indicating that pain may affect scores obtained on neuropsychological measures of attention [29]. However, the fact that these variables did not affect response times on the Computerized Pictorial Stroop Task suggests that this measure is robust and much less susceptible to the effects of psychological and pain-related variables. The latter points are particularly important in regards to providing adequate and individually-designed rehabilitation interventions to individuals within this clinical population.

Possible limitations of this study must be considered. This investigation involved a cross-sectional design and it is possible that a longitudinal design might have produced a slightly different representation of the relationship between neuropsychological performance and various psychological variables. In addition, the groups in our study were relatively small and results of this study should be replicated with larger sample sizes.

In conclusion, our results confirmed our study hypothesis and highlight the complex multifactorial nature of post-MTBI symptoms and deficits. Obtaining response times and intraindividual variability measures using sensitive tests such as the Computerized Pictorial Stroop Task described in the present study represent effective means for measuring attentional and cognitive recovery after a MTBI. Such tasks and metrics are more sensitive to subtle cognitive changes and less affected by pain-related variables than conventional neuropsychological tests of selective attention. Furthermore, since pain intensity and pain-associated disability can modulate performance on standard tests of attention, these variables should be systematically assessed in individuals having sustained a MTBI and related to results on standardized neuropsychological tasks to allow for their correct interpretation. These considerations can have a fundamental impact on the clinical evaluation, followup, and provision of adequate rehabilitation interventions for these patients.

Acknowledgments

This work was supported by the Social Sciences and Humanities Research Council of Canada (scholarship to M. Beaupré), as well as by grants (to M. McKerral) from the Quebec Rehabilitation Research Network and the Centre de Réadaptation Lucie-Bruneau.

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Hindawi Publishing Corporation
Rehabilitation Research and Practice
Volume 2012, Article ID 415740, 7 pages
doi:10.1155/2012/415740

Clinical Study

Outcome from Complicated versus Uncomplicated Mild Traumatic Brain Injury

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Received 4 November 2011; Revised 3 February 2012; Accepted 27 February 2012

Academic Editor: Anne Felicia Ambrose

Objective. To compare acute outcome following complicated versus uncomplicated mild traumatic brain injury (MTBI) using neurocognitive and self-report measures. *Method*. Participants were 47 patients who presented to the emergency department of Tampere University Hospital, Finland. All completed MRI scanning, self-report measures, and neurocognitive testing at 3-4 weeks after injury. Participants were classified into the complicated MTBI or uncomplicated MTBI group based on the presence/absence of intracranial abnormality on day-of-injury CT scan or 3-4 week MRI scan. *Results*. There was a large statistically significant difference in time to return to work between groups. The patients with uncomplicated MTBIs had a median of 6.0 days (IQR = 0.75–14.75, range = 0–77) off work compared to a median of 36 days (IQR = 13.5–53, range = 3–315) for the complicated group. There were no significant differences between groups for any of the neurocognitive or self-report measures. There were no differences in the proportion of patients who (a) met criteria for ICD-10 postconcussional disorder or (b) had multiple low scores on the neurocognitive measures. *Conclusion*. Patients with complicated MTBIs took considerably longer to return to work. They did not perform more poorly on neurocognitive measures or report more symptoms, at 3-4 weeks after injury compared to patients with uncomplicated MTBIs.

1. Introduction

Most mild traumatic brain injuries (MTBIs) are not associated with visible abnormalities on structural neuroimaging. A *complicated* MTBI, in the original definition [1], was differentiated from an *uncomplicated* mild TBI by the presence of (a) a depressed skull fracture and/or (b) a trauma-related intracranial abnormality (e.g., hemorrhage, contusion, or edema). Other researchers have dropped the depressed skull fracture from the criteria and simply retained the criterion for an intracranial abnormality. The rates of complicated MTBIs, based on cohorts of patients who underwent acute computed tomography following head trauma, are presented in Table 1. The rates of abnormalities vary considerably. In general, when examining details within these studies, patients with GCS scores of 13 or 14 are more likely to have an abnormality than patients with a GCS score of 15. Other possible reasons for differences in abnormality rates could relate to technology (e.g., older scanners versus newer scanners) and referral patterns for neuroimaging (i.e., more liberal versus more conservative use of imaging).

It is seems logical to assume that worse short-, medium-, and long-term neuropsychological and functional outcome would result from complicated versus uncomplicated MTBIs.

Table 1: Rates of complicated mild TBI in adults.

First author	Year	Country	Total *N*	Number Scanned	GCS scores	% Abnormal
Livingston [26]	1991	USA	111	111	14-15	14
Stein [27]	1992	USA	1,538	1,538	13–15	17.2
Jeret [28]	1993	USA	712	702	15	9.4
Moran [29]	1994	USA	200	96	13–15	8.3
Borczuk [30]	1995	USA	1,448	1,448	13–15	8.2
Iverson [31]	2000	USA	912	912	13–15	15.8
Thiruppathy [32]	2004	India	381	381	13–15	38.9
Stiell [33]	2005	Canada	2,707	2,171	13–15	12.1
Stiell [33]	2005	Canada	1,822	1,822	15	8.0
Ono [34]	2007	Japan	1,064	1,064	14-15	4.7
Saboori [35]	2007	Iran	682	682	15	6.7

However, the results from a series of studies are mixed. As a group, patients with complicated MTBIs perform more poorly on neuropsychological tests in the first two months following injury [1–6]. These differences appear to diminish by six months following injury [7, 8]. When differences occur between groups, the effect sizes of these differences are lower than expected (i.e., medium to medium-large effect sizes or lower on a small number of tests [1–5, 7, 9]; see Borgaro and colleagues [5] for an exception).

Some researchers have reported that patients with complicated MTBIs have worse 6–12-month functional outcome (i.e., Glasgow Outcome Scale) compared to patients who sustained uncomplicated MTBIs [1, 10, 11], and they have similar 3–5-year outcome (i.e., Functional Status Examination) as patients with a history of moderate and severe TBI [12]. There are some exceptions, however. McCauley and colleagues reported that CT abnormalities were not associated with increased risk for postconcussion syndrome at 3 months after injury [13]. Similarly, Lee and colleagues [14] reported that CT and conventional 3T MRI imaging findings do not predict neurocognitive functioning at 1 or 12 months after injury, nor functional outcome at one year after injury. It is becoming increasingly clear that complicated MTBIs represent a fairly broad spectrum of injury, with some people having very small abnormalities and excellent functional outcome and other people requiring inpatient rehabilitation and having poor outcome.

The purpose of this study is to compare the outcome of patients with complicated versus uncomplicated MTBIs. To date, few studies have compared both neurocognitive outcome and self-reported symptoms following uncomplicated and complicated MTBI. This is a prospective study, with patients identified from the emergency department undergoing MRI and a neuropsychological evaluation at approximately 3-4 weeks after injury. It was hypothesized that patients with complicated MTBIs would report more symptoms, perform more poorly on neurocognitive testing, and take longer to return to work than patients with uncomplicated MTBIs. We hypothesized worse outcome in this group because we assume that complicated MTBIs tend to be more serious brain injuries than uncomplicated MTBIs.

2. Methods

2.1. Participants. Participants were 47 patients with MTBIs who presented to the emergency department of Tampere University Hospital, Finland (age: M = 30.3 years, SD = 9.4, Range = 16–46; education: M = 13.0 years, SD = 2.3). The patients were selected from a larger cohort of head trauma patients enrolled in a longitudinal study, based on meeting inclusion criteria below, having complete data on all outcome measures, and having a known duration of time off work. The diagnostic criteria for MTBI used in this study were from the World Health Organization Collaborating Centre Task Force on MTBI. Inclusion criteria were as follows: (i) biomechanical force applied to the head resulting in loss or alteration of consciousness, confusion, and/or posttraumatic amnesia, (ii) loss of consciousness (LOC), if present, for less than 30 minutes, (iii) Glasgow Coma Scale (GCS) score 13–15 after 30 minutes following injury, and (iv) posttraumatic amnesia (PTA), if present, of less than 24 hours.

Patients underwent computed tomography (CT) scanning if deemed clinically indicated, an evaluation by an ED traumatologist, and other examinations as needed. CT scanning was performed within 24 hours of admission and is used liberally for head trauma patients. Magnetic resonance imaging (MRI) was conducted at approximately three weeks after injury for research purposes, although the information was available to the patient's healthcare providers (complicated MTBI group M = 19.3, SD = 15.0, range = 1–53 days and uncomplicated MTBI group M = 25.8, SD = 5.5, range = 16–36 days). The MRI protocol included sagittal T1-weighted 3D IR prepared gradient echo, axial T2 turbo spin echo, conventional axial, and high-resolution sagittal FLAIR (fluid-attenuated inversion recovery), axial T2*, and axial SWI (susceptibility weighted imaging) series. Only trauma-related findings on CT or MRI were counted as abnormal; minor incidental findings, such as isolated white

matter hyperintensities, were not considered as abnormal. Patients were excluded if significant non-trauma-related abnormalities were identified. Most were excluded due to small vessel ischemic disease, but there were also patients with multiple sclerosis, unusually large ventricles, and a history of neurosurgery.

This sample included patients ($N = 13$; 27.7%) who had an intracranial abnormality on day-of-injury CT or follow-up MRI (i.e., a complicated MTBI). None of the patients required inpatient rehabilitation. None of the patients were involved in litigation. All patients provided written informed consent according to the Declaration of Helsinki. The study protocol was approved by the Ethical Committee of the Tampere University Hospital. All patients completed self-report measures and neurocognitive testing at 3-4 weeks after injury (M = 25.8, SD = 2.9, Range 21–34 days).

2.2. Measures. Postconcussion symptoms were assessed using the Rivermead Post-Concussion Questionnaire (RPSQ) [15]. The RPSQ is a 16-item self-report questionnaire that measures the severity of common postoncussion symptoms on a 5-point Likert scale. The patients rated the presence of the symptoms over the past 24 hours on a scale from 0 to 4 (0 = not experienced at all after the injury, 1 = experienced but no more of a problem compared with before the injury, 2 = a mild problem, 3 = a moderate problem, and 4 = a severe problem). A total score was calculated by adding all items with a score greater than 1 (not present anymore).

Possible depressive symptoms were assessed using the Beck Depression Inventory-Second Edition (BDI-II) [16], a 21-item self-report questionnaire. Subjects were asked to rate each item on a four-point scale ranging from zero to three. In this study, we used the total score which is the sum of all 21 items, giving a range from zero to 63. It should be noted that many symptoms on this questionnaire overlap with postconcussion symptom measured by the RPSQ.

Self-reported fatigue was examined using the Barrow Neurological Institute Fatigue Scale (BNI-FS), an 11-item self-report questionnaire designed to assess fatigue during the early stages of recovery after brain injury [17]. Subjects were asked to rate the extent to which each of the 10 primary items has been a problem for them since the injury on a 7-point scale. Response options are as follows: 0-1 = rarely a problem; 2-3 = occasional problem, but not frequent; 4-5 = frequent problem; 6-7 = a problem most of the time. The final item (item 11) asks subjects to provide an overall rating of their level of fatigue on a scale from 0 (no problem) to 10 (severe problem). In this study the total BNI-FS score is used which is the sum of all 10 scores (min = 0, max = 70).

General verbal intelligence was assessed with the Wechsler Adult Intelligence Scale-Third Edition (WAIS III) information subtest [18]. Learning and memory was assessed with the Rey Auditory Verbal Learning Test (RAVLT) total score (total number of words recalled in trials 1 through 5) and delayed recall (number of words recalled after 30 minutes delay) [19]. Attention and executive functioning were assessed with Stroop Color Word Test (color-word interference score, Golden version) [19], Trail Making Test (TMT) A and B (time needed to finish the task) [20], and two verbal fluency tasks: animal naming (category fluency, total number of words in one minute) and single-letter-based word generation (phonemic fluency, total number of words produced across the 3 trials) [21]. Raw scores for the neurocognitive tests were analyzed unless otherwise stated.

3. Results

There were no significant differences between MTBI groups for age, education, gender, GCS score, mechanism of injury, days tested after injury, or duration of LOC, PTA, or retrograde amnesia (see Table 2). There was a large statistically significant difference in time to return to work between groups. The patients with uncomplicated MTBIs had a median of 6.0 days (mean = 12.9, SD = 18.8, IQR = .75–14.75, range = 0–77) off work compared to a median of 36 days (mean = 58.0, SD = 83.8, IQR = 13.5–53, range = 3–315) for the complicated MTBI group.

There were no significant differences between MTBI groups for self-reported depression (BDI-II) or postconcussion symptoms (RPSQ) (all $P > .05$). There were, however, medium effect sizes for the BDI-II total score (Cohen's $d = .52$) and RPSQ total score ($d = .43$) between groups. These effects sizes suggest that the complicated MTBI group reported fewer depression symptoms and postconcussion symptoms compared to the uncomplicated MTBI group.

There was not a statistically significant difference in the percentages of patients in the uncomplicated (44.1%) versus complicated (38.5%) MTBI groups who met ICD-10 criteria for postconcussional syndrome based on the reporting of symptoms on the RPSQ as "mild" or greater (i.e., score of 2 or higher on individual items). In contrast, 17.6% of the patients in the uncomplicated MTBI group met ICD-10 criteria for postconcussional syndrome based on the reporting of symptoms on the RPSQ as "moderate" or greater (i.e., score of 3 or higher on individual items); no patients in the complicated MTBI group met this criterion for the syndrome.

As seen in Table 3, there were no significant differences between MTBI groups on the neuropsychological tests (all $P > .05$). Although not significantly different, medium effect sizes were found for the RAVLT total score ($d = .39$), RAVLT delayed recall score ($d = .47$), Animal Naming test ($d = .43$), and Stroop Color-Word ($d = .47$). Paradoxically, the complicated MTBI group performed better on verbal learning and memory (RAVLT) and executive functioning (Stroop), but worse on a test of verbal fluency (Animal Naming).

The prevalence of low scores across the battery of cognitive tests was determined for each group (i.e., eight scores (not including the Rey Copy) derived from the six tests were considered simultaneously). A low score on the neurocognitive tests was defined as falling below the 10th percentile compared to published normative data. There was not a significant difference in the percentage of patients with uncomplicated (26.5%) versus complicated (23.1%) MTBIs who had two or more low scores, when eight test scores were considered simultaneously.

Table 2: Demographic and injury severity characteristics.

	Uncomplicated MTBI		Complicated MTBI			
	M	SD	M	SD	p	d
Age (in years)	30.8	9.0	29.2	10.9	0.601	.17
Education (in years)	13.1	2.3	12.9	2.3	0.859	.06
WAIS-III information (SS)	10.9	2.4	10.4	1.4	0.492	.23
GCS score in ED	14.9	0.3	14.9	0.3	0.693	.13
Days tested after Injury	26.0	2.9	25.4	3.0	0.523	.21
Duration of loss of consciousness (minutes)	0.6	1.5	1.0	1.5	0.514	.25
Duration of posttraumatic amnesia (minutes)	333.0	448.2	366.7	420.8	0.822	.08
Duration of retrograde amnesia (minutes)	9.2	23.6	21.5	66.6	0.354	.34
Number of days to return to work	12.9	18.8	58.0	83.8	0.002	1.2
	f	%	f	%	χ^2	
Gender						
Male	17	50.0	7	53.8	0.813	—
Mechanism of injury						
MVA	15	44.1	5	38.5	0.726	—
Other bodily injuries	9	26.5	4	30.8	0.768	—
CT: day of injury						
Abnormal	0	0	8	61.5	—	—
MRI: 3 weeks after						
Abnormal	0	0	12	92.3	—	—
Not available	0	0	1	7.7		

Note: $N = 47$ (uncomplicated MTBI, $n = 34$; complicated MTBI, $n = 13$). Cohen's effect size (d): small (.20), medium (.50), large (.80). CT= computed tomography; MRI: magnetic resonance imaging; GCS: Glasgow Coma Scale; MTBI: mild traumatic brain injury; MVA: motor vehicle accident. A Mann-Whitney U test was used for the number of days to return to work comparison.

Table 3: Descriptive statistics (raw scores) and effect sizes: self-report and neurocognitive tests.

	Uncomplicated MTBI		Complicated MTBI			
	M	SD	M	SD	p	d
Self-report Measures						
BDI-II total	7.6	6.7	4.5	4.0	.130	.52
RPSQ total	11.6	11.8	7.2	6.2	.358	.43
Barrow Fatigue Scale	16.4	15.3	13.0	9.7	.464	.25
Neurocognitive Tests						
RAVLT total	56.9	7.8	60.2	10.1	.237	.39
RAVLT delay	11.1	2.8	12.4	2.6	.159	.47
RCFT copy	35.7	0.7	35.6	0.8	.792	.09
RCFT Immediate	25.4	5.9	23.8	5.2	.402	.28
Phonemic Fluency total	38.7	9.4	39.8	13.4	.766	.10
Animal Naming total	25.1	6.4	22.5	5.0	.194	.43
Trails A (in seconds)	27.3	9.5	28.5	8.7	.685	.13
Trails B (in seconds)	62.5	24.8	57.5	13.3	.495	.23
Stroop Color-Word	42.1	8.2	45.9	7.2	.157	.47

Note: $N = 47$ (uncomplicated MTBI, $n = 34$; complicated MTBI, $n = 13$); Cohen's effect size (d): small (.20), medium (.50), large (.80). BDI-II: Beck Depression Inventory-Second Edition; RPSQ: Rivermead Postconcussion Scale; RCFT: Rey Complex Figure Test; RAVLT:Rey Auditory Verbal Learning Test; MTBI:mild traumatic brain injury.

4. Discussion

A substantial minority of patients who sustain an MTBI and are evaluated in an emergency department will have a visible abnormality on early CT scanning (Table 1). These patients are conceptualized as having a complicated MTBI. Researchers have reported that those with complicated MTBIs, as a group, are more likely to have early cognitive deficits [1–6] and worse medium [1, 10, 11] and long-term [12] functional outcome. In contrast, however, some researchers have not found important differences between those with complicated versus uncomplicated MTBIs. For example, in one study, patients with complicated MTBIs were not more likely to have a postconcussion syndrome at three months after injury [13], and another research group reported that neuroimaging findings did not predict cognitive functioning at one or 12 months after injury, or functional outcome at one year after injury [14].

In the present study, as hypothesized, patients with complicated MTBIs took longer to return to work. It has been noted that most patients return to work after injury despite having some symptoms [22]. In this study, we did not explicitly examine if the patients had symptoms prior to returning to work. Yet, the majority of the study population had returned to work by the time of the neuropsychological assessment. Therefore, their self-reported symptoms at the time of evaluation likely reflect their situation after returning to work, supporting the idea that it is common to return to work while still having some symptoms. One possible reason for why intracranial lesions were correlated with longer time off work may be that doctors are likely to grant longer sick leaves when there is objective evidence of brain injury; in that case the duration of the postinjury sick leave might reflect, in part, the behavior of doctors in the Finnish system. We have no way of determining whether, or not, doctors in the community are likely to grant longer sick leaves to patients with complicated MTBIs, but we have anecdotal evidence from emergency department physicians that they tend to prescribe longer initial periods of leave for patients with more serious injuries such as those with intracranial abnormalities.

In the present study, the patients with complicated versus uncomplicated MTBIs were compared on neurocognitive testing and symptoms ratings at approximately 3-4 weeks after injury. It was hypothesized that those with complicated MTBIs would perform more poorly on cognitive testing and report more symptoms than those who did not have imaging abnormalities. Contrary to these hypotheses, there were no differences between the two groups on neurocognitive testing or symptom reporting. Surprisingly, there were trends toward those with complicated MTBIs reporting fewer symptoms and performing somewhat better on cognitive testing.

There are methodological differences and limitations with the present study, in comparison to previous studies, that might have influenced the results. First, there were a small number of subjects in this study that were identified as having imaging abnormalities; thus, the statistical analyses were underpowered. However, when examining the means and SDs, there was not a trend toward greater symptoms or worse cognitive test performance in the complicated MTBI group. In fact, there were trends toward fewer symptoms and better performance in this group. This reduces the likelihood that the present findings represent a Type 2 statistical error. Second, most previous studies classified patients as having complicated MTBIs based on day-of-injury CT scanning only, and some of these studies are older and the CT technology might have been less refined. The present study identified subjects based on CT and MRI, with some of our sample not showing day-of-injury CT abnormalities—only abnormalities on MRI. Third, some previous studies have included subjects with complicated MTBIs who required inpatient rehabilitation. None of the present patients were injured that badly. Finally, some previous studies have included patients in litigation, whereas no patients in this study were involved in litigation. Therefore, it is possible that the present group of patients with complicated MTBIs were less severely injured than some of the samples from previous studies.

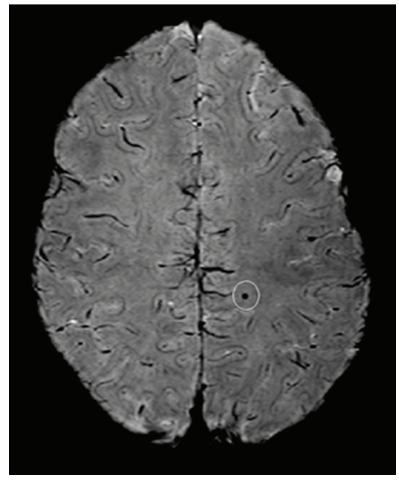

Figure 1: Hemosiderin detected with multiecho susceptibility weighted imaging using a 3 Tesla scanner. Multiecho SWI image (Philips Achieva 3T; 5 echoes; voxel size = 0.32 × 0.32 × 0.75 mm^3). Courtesy of Alexander Rauscher, Ph.D., UBC MRI Research Centre, Department of Radiology, University of British Columbia, Vancouver, Canada.

As technology evolves, some researchers will be tempted to broaden the criteria for complicated MTBI. In the past, intracranial abnormalities were identified using CT or conventional MRI. With advancements in technology, smaller and smaller abnormalities can be detected using structural imaging. For example, the area of hemosiderin (iron-rich staining of tissue from an area with past blood) shown in Figure 1 using multiecho susceptibility weighted imaging (SWI) with 5 echoes [23, 24] on a 3 Tesla MRI scanner would be undetectable with a modern CT scan and would likely be missed using 1.5 or 3.0 Tesla MRI conventional sequences [25]. Therefore, in past studies this subject would be classified as having an uncomplicated MTBI, but in future studies this abnormality might qualify for classification as a complicated MTBI. However, this subject was actually a healthy control subject in one of our studies. He had no known history of an injury to his brain.

Thus, not only might the criteria for a complicated MTBI evolve to include smaller and smaller abnormalities—but some of these abnormalities might not be related to the MTBI—thus resulting in misdiagnosis.

In conclusion, patients with complicated MTBIs took longer to return to work. They did not, however, perform more poorly on neurocognitive measures or report more symptoms, at 3-4 weeks after injury compared to those with uncomplicated MTBIs. As the literature evolves, it is becoming clear that complicated MTBIs represent a broad spectrum of injury, with some people having very small abnormalities and excellent functional outcome, other people requiring inpatient rehabilitation and having poor outcome, and a diverse set of outcomes in between.

Acknowledgments

This research was funded by Competitive Research Funding of the Pirkanmaa Hospital District, Tampere University Hospital. The authors thank Pasi Jolma (M.D., Ph.D.) for help with recruiting the patients and conducting neurological examinations. This study was presented at the annual meeting of the National Academy of Neuropsychology, October 2010, Vancouver, BC, Canada. The views expressed in this article are those of the authors and do not reflect the official policy of the Department of Army, Department of Defense, or US Government.

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Hindawi Publishing Corporation
Rehabilitation Research and Practice
Volume 2012, Article ID 816069, 9 pages
doi:10.1155/2012/816069

Review Article

Chronic Traumatic Encephalopathy: A Review

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Received 3 November 2011; Revised 24 January 2012; Accepted 6 February 2012

Academic Editor: Anne Felicia Ambrose

Chronic traumatic encephalopathy (CTE) is a progressive neurodegenerative disease that is a long-term consequence of single or repetitive closed head injuries for which there is no treatment and no definitive pre-mortem diagnosis. It has been closely tied to athletes who participate in contact sports like boxing, American football, soccer, professional wrestling and hockey. Risk factors include head trauma, presence of ApoE3 or ApoE4 allele, military service, and old age. It is histologically identified by the presence of tau-immunoreactive NFTs and NTs with some cases having a TDP-43 proteinopathy or beta-amyloid plaques. It has an insidious clinical presentation that begins with cognitive and emotional disturbances and can progress to Parkinsonian symptoms. The exact mechanism for CTE has not been precisely defined however, research suggest it is due to an ongoing metabolic and immunologic cascade called immunoexcitiotoxicity. Prevention and education are currently the most compelling way to combat CTE and will be an emphasis of both physicians and athletes. Further research is needed to aid in pre-mortem diagnosis, therapies, and support for individuals and their families living with CTE.

1. Introduction

Chronic traumatic encephalopathy (CTE) has been defined as a progressive neurodegenerative disease caused by repetitive head trauma [1]. CTE was first described in 1928 when Dr. Harrison Martland, a New Jersey medical examiner, began to note a constellation of symptoms in boxers. In an article he published in the Journal of the American Medical Association entitled Punch Drunk, he describes the boxers, "cuckoo," "goofy," "cutting paper dolls," or "slug nutty" [2]. Punch drunk was later termed dementia pugilistica, literally meaning dementia of a fighter. However, with the evolution of sports like American football, these symptoms were also being reported in athletes other than boxers and was renamed chronic traumatic encephalopathy in the 1960s.

CTE has become a popular topic due to its close association with American football, hockey, soccer, boxing, and professional wrestling. Many of these affected athletes, mostly retired, have struggled in their later years with depression, substance abuse, anger, memory/motor disturbances, and suicide [3]. Autopsy results from these athletes have suggested a link between these emotional, cognitive, and physical manifestations and CTE [3–5]. In addition to athletes, military soldiers have become another group of interest as many are returning from the battlefield with brain injuries from blast trauma causing closed head injury. In this paper we present a summary of the epidemiology, risk factors, clinical presentation, pathophysiology, neuropathological findings, treatment/prevention, and future research pertaining to CTE.

2. Epidemiology

Concussion or mild traumatic brain injury (mTBI) is one of the most common neurologic disorders accounting for approximately 90% of all brain injuries sustained [4]. Such injuries are a common occurrence in athletes with an estimated 1.6–3.8 million sport-related concussion annually in the USA [5]. This can be seen as a gross underrepresentation of the true number as many athletes do not seek medical attention or vocalize their symptoms. This may be due to head trauma being regarded as benign, or in some the injury

is not recognized at all. These behaviors are driven by the athletes' desire to return to play and the pressure to perform [6]. DeKosky et al. reported that each year more than 1.5 million Americans have mTBI with no loss of consciousness and no need for hospitalization as well as an equal number with conscious impairing trauma but insufficiently severe to require long-term hospitalization [7].

In a 2009 review of CTE, McKee et al. found that of 51 neuropathologically diagnosed cases of CTE 46 (90%) occurred in athletes. Specifically, athletes participating in American football, boxing, soccer, and hockey comprise the majority of cases. Many of these athletes began their respective sports at a young age between 11 and 19 years [5]. While it is not clear at what age CTE can begin, McKee has neuropathologically diagnosed CTE changes in asymptomatic 18-year-old high school football player with a history of concussion. While the exact incidence of CTE is unknown, it is thought to vary widely based on sport, position, length of career, number of head injuries, age of first head injury, and genetics [8].

Table 1: Risk factors associated with chronic traumatic encephalopathy.

(i) Head trauma: single or repetitive
(ii) History of head concussion
(iii) Participation in the following
Boxing
American football: offensive/defensive linemen/linebackers/running backs
Soccer
Professional wrestling
Hockey
Military service: blast injuries
(iv) Length of sport participation
(v) Epileptics
(vi) Victims of domestic abuse
(vii) Age of injuries: younger ages and older ages
(viii) Genetic variation: ApoE3 or ApoE4

3. Risk Factors

It has been well established that repetitive concussive or subconcussive blows to the head place individuals at risk for CTE [5, 6, 8]. CTE has been associated with athletes who participate in contact sports like American football, boxing, hockey, soccer, and professional wrestling. Other sports that are not directly associated with CTE, but have well-documented cases of concussion, include mixed martial arts, rugby, and horseback riding. Other groups at risk for repetitive head trauma and CTE are military veterans, epileptics, and victims of domestic abuse [5]. It has been reported that approximately 17% of professional retired boxers will exhibit CTE [3]. Although each group listed has a unifying factor of head trauma, they differ in particular aspects that may influence the severity or chronicity of their injury (see Table 1 for a summary of risk factors).

A recent study by Crisco et al. examined head impact exposure in collegiate football players and found that the average number of impacts received by an individual player during a single season was 420 with a maximum of 2,492 [9]. These impacts vary in severity based on their position. Offensive linemen, defensive linemen, and linebackers had the most frequent impacts while quarterbacks and running backs received the greatest magnitude of head impacts [9]. In the literature McKee and colleagues reported that in five football players with diagnosed CTE all played similar positions: 3 were offensive linemen, 1 defensive linemen, and one linebacker [5]. However, according to Boston University's Center for the Study of Chronic Traumatic Encephalopathy, CTE has also been found in other position players like safety and wide receiver dismissing the idea that only certain positions are at risk for developing CTE.

While it is clear that anyone who suffers repeated head trauma, regardless of the mechanism, may carry the risk of developing CTE, there is no clear consensus on how much or how little trauma is needed to cause CTE. While most feel CTE is a manifestation of repetitive trauma, the question still stands if it can be caused by a single TBI [10]. In a study by Johnson et al., widespread tau and beta-amyloid deposition was found in the brains of individuals who suffered a single traumatic brain injury. The study included the examination of postmortem brains from long-term survivors (1–47 years) of a single TBI ($N = 39$) versus uninjured age-matched controls ($N = 47$). Results showed NFTs to be exceptionally rare in young uninjured controls, yet were abundant and diffuse in one-third of TBI cases. This was also true of beta-amyloid deposition, which was found in greater density following TBI than controls [11]. While these brains showed classic changes associated with CTE, they did not have the symptomatic profile to accompany their neuropathologic findings [11]. If these subjects went on to have repeated brain injury, it would be reasonable to expect more extensive damage with more pronounced clinical symptoms.

In a study of repeated head trauma in mice, Kane et al. created an animal model where mice did not suffer severe TBI but rather mTBI to look for CTE-like changes. They reported that exposure to head trauma for 5 consecutive days showed increased expression of glial fibrillary acidic protein and phospho-tau 30 days (~160% increase) after the last injury when compared to controls. They also reported that with their mTBI model they did not find edema, cortical contusions, obvious loss of neuronal matter beneath the skull, disruption of the blood brain barrier, or microglial activation. However, they compared this to mice that were subject to a single traumatic injury and found substantial microglial activation in the hippocampus and overlying cortex 30 days after the initial impact [12].

Another high-risk group that has recently been studied are individuals in the military [12]. Operations in Iraq and Afghanistan are reporting that TBI accounts for roughly 28% of all combat casualties and approximately 88% of these are closed-head injuries [12]. While these numbers are significant, the US Defense and Veterans Brain Injury

Center has estimated that approximately 180,000 soldiers have been diagnosed with mTBI between 2001 and 2010 while others estimate the number to be more than 300,000. Additionally, soldiers may also be exposed to toxins like organophosphates, chemical nerve agents, and heavy metals like uranium increasing their risk for brain injury [13].

Age is another possible risk factor for the development of CTE. At younger ages, while the brain is developing traumatic injury may begin the cascade of destructive events and compounded through the years of continued play. Conversely, at younger ages the brain has more plasticity allowing greater ability to manage injury than that in the mature brain [8, 10]. Length of play is another risk factor where longer careers with prolonged exposures to injury may cause more severe CTE. Of the 51 cases reviewed by Dr. McKee, 39 boxers had an average career of 14.4 years (range 4–25) while the 5 football players averaged careers of 18.4 years (range 14–23 years). These athletes began their respective sports between 11 and 19 years of age [5].

Genetic factors have also been thought to play a role in the development of CTE specifically the apolipoprotein E gene (ApoE). The ApoE4 allele has been well described in its association with Alzheimer's disease (AD) where individuals with homozygous ApoE4/E4 genotype have a 19-fold increased risk of developing AD [14]. This same gene is now thought to possibly have a role in the development of CTE [5]. Studies have shown that ApoE4-positive individuals had poorer outcomes with head trauma. Teasdale et al. reported that that patients with ApoE4 allele are more than twice as likely than those without ApoE4 to have unfavorable outcomes 6 months after head injury [15]. Kutner et al. examined 53 professional American football players to see if their cognitive functioning differed based on age and ApoE4 genotype. They reported that older age and presence of ApoE4 scored lower on cognitive tests than did those without the allele or with less playing experience [16]. Jordan et al. looked at ApoE4 genotype in boxers in relation to chronic TBI. They found that all boxers with severe impairment, based on the chronic brain injury scale, had at least 1 ApoE4 allele. Therefore, they reported that ApoE4 may be associated with increased severity of chronic neurologic deficits in high-exposure boxers [17].

In McKee and colleagues' review of the 51 CTE cases, ApoE genotyping was reported in 10 cases where 50% carried at least one ApoE4 allele and one was homozygous for E4. While they did not report what the other 4 genotypes were, it raised their suspicions to believe that ApoE4 was the gene of interest. In animal studies ApoE4 transgenic mice had greater mortality from TBI than those with ApoE3 allele. Another study showed that transgenic mice that overexpress ApoE4 allele showed increased deposition of beta-amyloid after experimental TBI [5].

However, a study by Omalu et al. reported that 70% of their CTE cohort had the ApoE3 genotype. Of the 17 athletes they used in their study they were able to determine the ApoE genotype in 10 of 14 professional athletes and in 2 of 3 high school athletes. Of the 10 professional athletes 90% had at least one ApoE3 allele, and 7 of the 10 with confirmed ApoE genotype also had CTE. Of these 7 athletes with CTE 100% had at least one ApoE3 allele (5 E3/E3, 2 E3/E4). It should also be noted that the only professional athlete in their study that did not have the E3 allele (E2/E4) was negative for CTE. Additionally, the two other professional athletes that had the ApoE allele but did not have CTE were E2/E3 (24 years old) and E3/E3. The authors note that the one case of E3/E3 that did not have tauopathy was assessed from only select sections of the brain as they did not have access to the full brain. Of the two high school athletes both were E3/E3 genotypes and did not show any histological signs of CTE [18].

4. Clinical Presentation

It is important to define the timeline of CTE symptom development to distinguish it from concussive or postconcussive syndrome (PCS). The symptoms associated with acute concussion are headache, blurred vision, amnesia, tinnitus, fatigue, and slurred speech with resolution within days to weeks if managed properly. Although there is no strict timeline to the acute injury phase, it has typically been reported as three months, but 80–90% of patients show full recovery within the first 10 days [19]. When symptoms extend beyond three months, the individual is said to have PCS. These individuals will have additional symptoms of physical, emotional, cognitive, and behavioral problems [6]. PCS also has a variable timeline where symptoms typically improve within one year but may in some cases require several years for resolution. For those with persisting or permanent PCS symptoms, they would logically be considered to have CTE.

However, the typical clinical course of an individual who develops CTE is not as linear as direct progression from concussion to PCS to CTE. The onset of CTE symptoms typically starts later in the lives of certain athletes after the individual has removed themselves from competition. As reported by McKee, the first symptoms of CTE were noted at ages ranging from 25 to 76 years. McKee also reported that at the time of retirement one-third of these athletes were symptomatic with another half becoming symptomatic within 4 years of leaving sports [5]. In 14 cases (30%), mood disturbances were reported while movement abnormalities like Parkinsonism, ataxia, antalgic gait, and dysarthric speech were reported in 41% of subjects [5]. The course of symptom progression seems to follow a somewhat continuous path beginning with cognitive and emotional decline leading to eventual motor deterioration [5].

Initially, patients begin to have poor concentration, attention, and memory along with disorientation, dizziness, and headaches. They typically progress to experience irritability, outbursts of violent or aggressive behavior, confusion, and speech abnormalities. During this stage of the disease, there is a high frequency of suicide, drug overdose, and mood disorders, mainly major depressive disorder [5]. A study by Omalu and colleagues describes a similar clinical profile with a latent asymptomatic period between play and symptom onset. He reports worsening of cognitive and social functioning leading to poor money management, bankruptcy, social phobias, paranoid ideation,

insomnia, poor relationships, divorce, emotional/physical abuse, and substance abuse [18]. Family and friends of the affected individuals reported many of these symptoms to researchers through standard forensic interviews [18].

As the disease progresses in severity, there is a greater loss of motor functioning. Some patients may develop Parkinsonian symptoms of tremors, masked facieses, wide propulsive gait, poor speech, ocular abnormalities, vertigo, bradykinesia, deafness, and a small group developing dementia. Currently, the number of cases with confirmed dementia remains small. As more postmortem exams are done in the at risk group, it is expected more cases will be diagnosed. Some individuals with CTE have committed suicide, overdosed on drugs, or died from accidents preventing progression of the disease [5, 20].

5. Pathophysiology

The development of CTE is due to repetitive traumatic brain injury from the acceleration/deceleration forces of closed head impacts [5]. Damage to the brain generally occurs when the brain collides against the skull causing damage to the same side as the collision, coup, or to the opposite side of the impact, countercoup [6]. High-speed decelerations may also cause mechanical and chemical injury to the long axons resulting in traumatic/diffuse axonal injury. Crisco et al. report that impacts to the top of the head had the lowest rotational force but highest linear force leading more to cervical spine injuries. However, lateral blows cause rotational forces, which are the typical cause of mild traumatic brain injury [6]. While there is a firm understanding of head trauma being the general cause of the brain damage in CTE, researchers have not agreed upon a unifying mechanism of injury. Originally the associated axonal damage was thought to be due to shearing or mechanical forces at the time of injury. However, it is now reported that axonal shearing or tearing is a secondary event to the acute inflammation and neurodegeneration of axons [5]. In the acute setting there is rapid axonal swelling, perisomatic axotomy, and Wallerian degeneration [5].

Gavett et al. offered a general description of how the damage may occur through the repeated traumatic injury of axons. Damage to axons would cause changes in membrane permeability and ionic shifts causing a large influx of calcium. Subsequent release of caspases and calpains would trigger tau phosphorylation, misfolding, shortening, and aggregation as well as cytoskeleton failure with dissolution of neurofilaments and microtubules [8].

This idea was elaborated on in a recent paper by Blaylock and Maroon who describes the concept of immunoexcitotoxicity as a possible central mechanism for CTE. He describes a cascade of events that begin with an initial head trauma, which "primes" the microglia for subsequent injuries. When the homeostasis of the brain is disturbed, some of the microglia undergo changes to set them in a partially activated state. When these microglia become fully activated by continued head trauma, they release toxic levels of cytokines, chemokines, immune mediators, and excitotoxins like glutamate, aspartate, and quinolinic acid. These excitotoxins inhibit phosphatases, which results in hyperphosphorylated tau and eventually neurotubule dysfunction and neurofibrillary tangle deposition in particular areas of the brain [10]. There is also an apparent synergy between the proinflammatory cytokines and glutamate receptors that worsen neurodegeneration in injured brain tissue. This combination also increases the reactive oxygen and nitrogen intermediates that interfere with glutamate clearance keeping the injury response high. Priming can also occur from insults to the brain like systemic infections, environmental toxins, and latent viral infections in the brain (cytomegalovirus and herpes simplex virus) [10].

The microglia, however, have a dual function allowing them to switch between being neurodestructive and neuroreparative. During acute injury the microglia are responsible for containing the damage with inflammation, cleaning up debris, and repairing the surrounding damaged tissue [10]. However, if the individual experiences a second brain trauma or multiple continuous traumas, the microglia may never have the chance to switch from proinflammatory to reparative mode [10]. Such repetitive trauma may place the brain in a state of continuous hyperreactivity leading to progressive and prolonged neuronal injury. This would support the evidence that repeated mTBI results in a higher incidence of prolonged neurological damage than single-event injury [10].

The eventual neurodegeneration is also dependent on other factors like the age of the brain at the time of injury. Several studies have shown that older individuals have poorer outcomes when compared to younger subjects experiencing TBI. Streit et al. showed that as the microglia age, they become more dysfunctional, which may impair their ability to terminate immune activation. Therefore, as the brain grows older, it has more activation of microglia with weaker mitochondrial functioning, neuronal and glial dystrophy, higher levels of inflammation, and lifetime exposures to environmental toxins [10].

6. Gross Pathology Findings

As described by Corsellis et al., the most common gross pathological findings in CTE included reduced brain weight, enlargement of the lateral and third ventricles, thinning of the corpus callosum, cavum septum peallucidum with fenestrations, scarring, and neuronal loss of the cerebellar tonsils. Brain atrophy was most severe in the frontal lobes (36%), temporal lobes (31%), and parietal lobes (22%) with the occipital lobe rarely being affected [5]. McKee and colleagues reported that with increasing severity of disease marked atrophy is noted in the hippocampus, entorhinal cortex, and the amygdala [5]. Blaylock and Maroon reported that these areas showed the most severe atrophy and were noted to have the highest concentration of glutamate receptors and cytokine receptors [10].

7. Microscopic Pathology

According to Dr. Bennet Omalu, a forensic neuropathologist, the basic feature of CTE is the presence of sparse, moderate, or frequent band-shaped, flame-shaped small globose and large globose neurofibrillary tangles (NFTs) in the brain accompanied by sparse, moderate, or frequent neuropil threads (NTs) [18]. Similarly, McKee and colleagues described the core pathology of CTE to include tau-reactive NFTs, astrocytic tangles, and dot-like spindle-shaped NTs [10]. These changes are commonly noted in the dorsolateral frontal, subcallosal, insular, temporal, dorsolateral parietal, and inferior occipital cortices. Additionally, Dr. McKee also reported occasional tau immunoreactive neuritis and NFTs in the posterior, lateral, and/or anterior horns of the spinal cord (see Table 2) [10].

Beta-amyloid (Aβ) deposition is an inconsistent finding in CTE as Dr. McKee noted that of the 51 cases of confirmed CTE she reviewed that only 3 (6%) had amyloid angiopathy [5]. Animal studies by Iwata et al. used swine TBI models to show minimal Aβ accumulation in axons acutely after injury but saw greater accumulation one month following injury [20]. Similarly Chen et al., also using a swine TBI model, found evidence of axonal pathology 6 months following rotational brain injuries [21].

Although CTE and Alzheimer's disease (AD) both have NFTs and possibly beta-amyloid plaques, there are several unique features that distinguish the two [8]. First, beta-amyloid deposits are only found in 40% to 45% of patients with CTE while they are present in nearly all cases of AD. Secondly, the tau distribution in CTE is located more in the superficial cortical laminae whereas in AD they are found in large projection neurons in deeper layers. It is also important to note is the distribution of the NFTs in CTE that extremely irregular with uneven foci in the frontal temporal and insular cortices, while AD has a more uniform cortical NFT distribution. DeKosky et al. noted that the hippocampus is frequently spared by tauopathy in CTE, whereas it is the first location affected by tauopathy in AD [7]. Lastly, NFTs in CTE are most concentrated at the depths of the cortical sulci and are typically perivascular, which might indicate that there are disruptions of cerebral microvasculature and the blood-brain barrier at the time of injury leading to NFT formation [8].

A recent study by Omalu et al. described four histomorphologic phenotypes of CTE in American athletes (see Table 3). They examined specimens from 17 deceased athletes, 10 of which had histopathologically confirmed CTE. All were male, age range of 17–52, (8 were American football players, 4 professional wrestlers, 1 mixed martial arts fighter, 1 professional boxer, and 3 high school American football players). Omalu and colleagues created these histologic phenotypes based on the presences or absence of NFTs, NTs, and diffuse amyloid plaques as well as their quantitative distribution in the cerebral cortex, subcortical nuclei/basal ganglia, hippocampus, and cerebellum. These results are summarized in Tables 1 and 2. Phenotype one shows sparse to frequent NFT and NTs in the cerebral cortex and brainstem without involvement of the subcortical nuclei, basal ganglia, or cerebellum without any beta-amyloid. Phenotype two is the same as the first except they show diffuse beta-amyloid plaques. The third group had higher concentrations of NFTs and NTs only in the brainstem without involvement elsewhere with no beta-amyloid. The fourth group had sparse NFTs and NTs in the cerebral cortex, brainstem, subcortical nuclei, and basal ganglia with an unaffected cerebellum and no beta-amyloid [18].

Table 2: Areas of damage in the brain.

Gross areas of damage
(i) Reduced brain weight with atrophy of
Frontal lobe
Temporal lobe
Parietal lobe
Occipital lobe
(ii) Enlargement of lateral and third ventricles
(iii) Thinning of the corpus callosum
(iv) Cavum septum pellucidum with fenestrations
(v) Scarring and neuronal loss of cerebellar tonsils
(vi) Pallor of substantia nigra
Areas of tau NFTs and NT
(i) Superficial cortical layers
(ii) Dorsolateral frontal
(iii) Subcallosal
(iv) Insular
(v) Temporal
(vi) Dorsolateral parietal
(vii) Inferior occipital cortices
(viii) Thalamus
(ix) Hypothalamus
(x) Substantia nigra
(xi) Olfactory bulbs
(xii) Hippocampus
(xiii) Entorhinal cortex
(xiv) Amygdala
(xv) Brainstem

Trans-activator regulatory DNA-binding protein 43 or TDP-43 has been a recent addition to the growing neuropathologic findings associated with CTE. TDP-43 is a highly conserved protein that is found in many tissues including the CNS [22]. It plays a significant role in mediating the response of the neuronal cytoskeleton to axonal injury [8]. In a study by McKee et al., they reported widespread TDP-43 proteinopathy in 80% of their CTE cases. Until this study, TDP-43 was thought to be a unique finding to amyotrophic lateral sclerosis (ALS) and frontotemporal lobar degeneration (FTLD-TDP) but has now been found in other neurodegenerative diseases as a secondary pathology [22].

Table 3: Emerging histomorphologic phenotypes in American athletes.

Phenotype	Histological findings
1	Sparse to frequent NFT and NTs in the cerebral cortex and brainstem without involvement of the subcortical nuclei, basal ganglia, or cerebellum without any beta-amyloid
2	Sparse to frequent NFT and NTs in the cerebral cortex and brainstem with diffuse beta-amyloid deposition. No involvement of the subcortical nuclei, basal ganglia, or cerebellum
3	Higher concentrations of NFTs and NTs only in the brainstem. No involvement elsewhere or any beta-amyloid
4	Sparse NFTs and NTs in the cerebral cortex, brainstem, subcortical nuclei, and basal ganglia with an unaffected cerebellum and no beta-amyloid [18]

8. Clinicopathologic Correlations

The typical symptoms of CTE can be directly connected to the specific areas of the brain that are injured during the progression of disease. Based on these symptoms it is clear that there is damage to the hippocampal-septo-hypothalamic-mesencephalic circuitry (Papez circuit) also known as the emotional or visceral brain [5]. Damage to these areas correlate to the behavioral symptoms of emotional liability, aggression, and violence. Damage to the hippocampus, enteorhinal cortex, and medial thalamus conceivably causes the commonly reported complaint of memory disturbance. Destruction to the frontal cortex and white matter may result in the dysexecutive symptoms found throughout the many cases of CTE. Motor abnormalities may be due to degeneration of the substantia nigra and pars compacta along with symptoms of dysarthria, dysphagia, and ocular malfunction due to brainstem nuclei injury like the hypoglossal and oculomotor nuclei (see Table 4) [5].

9. Neurological Sequelae

Historically amyotrophic lateral sclerosis (ALS) has been thought to be a sporadic disease with no single causative factor. Literature has reported risk factors to include trauma to the brain or spinal cord, strenuous physical activity, exposure to heavy metals, cigarette smoking, radiation, electrical shocks, and pesticides [22]. Given these risk factors, the literature strongly correlates a history of head trauma with increased incidence of ALS. In a case control study, Chen et al. reported that having repeated head trauma within the 10 years prior to diagnosis had a 3-fold higher risk of ALS [23]. The same group also did a meta-analysis of 8 ALS studies and estimated a pooled odds ratio of 1.7 (95% CI: 1.3, 2.2) for at least one previous head injury. Another study reported increased ALS incidence and mortality in professional Italian soccer players when compared to the general population [24]. Additionally, an incidence study of 7,325 Italian professional soccer players showed an ALS incidence 6.5 times higher than expected [25]. ALS has also been seen in higher numbers among American and Canadian football players when compared to the general population [22]. The risk of ALS has also been reportedly high in war veterans. A study of Gulf War veterans reported that the risk of ALS was increased 2-fold during the 10 years following service [26]. A study by Schmidt et al. reported that veterans who received head trauma during war had an adjusted odds ratio for the development of ALS of 2.33 (95% CI: 1.18–4.61) [27].

A recent study by McKee and colleagues examined and compared the brains and spinal cords of 12 athletes with confirmed CTE to 12 cases of sporadic ALS to12-age matched controls. Of the 12 CTE cases, 3 also had a diagnosed motor neuron disease (MND) resembling ALS. The study found that those with CTE and the motor neuron disease not only had the typical neuropathologic presentation of CTE with tau-NFT, NT, and TDP-43 throughout the brain and brain stem, but they also had these changes in the anterior horns of the spinal cord in high concentrations. Of the 9 CTE patients that did not have the MND, they had similar CTE neuropathology, but it did not affect the spinal cord as significantly. When compared to the samples of sporadic ALS, they found TDP-43 immunoreactivity in all 12 cases with no tau immunoreactive NFTs. The age-matched controls showed no TDP-43 or tau reactivity. These results indicate that the widespread tauopathy and TDP-43 proteinopathy of CTE can in some cases extend beyond the brain and the brain stem to severely affect the spinal cord. The authors have labeled these cases as having chronic traumatic encephalomyelopathy (CTEM). While this has only been identified in three cases, it opens the floor to further discussion and research to see if CTEM is in fact a unique disease or just the coincidental occurrence of ALS and CTE. McKee has stated that the tau pathology in the three cases of CTEM is not only distinct from that of sporadic ALS, but the nature and distribution of the TDP-43 proteinopathy are also unique [22].

10. Diagnosis

Currently the only way to definitively diagnose CTE is through postmortem neuropathological autopsy [8]. Clinical diagnosis is difficult due to a lack of consensus on diagnostic criteria or large-scale longitudinal clinicopathologic correlation studies [8]. The differential diagnosis for CTE usually includes diseases like AD and frontotemporal dementia (FTD), which all share similar clinical symptoms, and all may have a history of head trauma making a clinical diagnosis difficult. Although age can help in distinguishing between AD and CTE, it does not help when deciding between FTD and CTE.

It is the hope of many that the advances in neuroimaging will aid in detecting chronic and acute changes associated with CTE. Diffusion tensor imaging (DTI) has been reported

Table 4: Clinicopathological correlations [5].

Damage area	Clinical presentation
Hippocampus Entorhinal cortex Medial thalamus	Early deficits in memory
Frontal cortex and underlying white matter	Dysexecutive symptoms
Dorsolateral parietal Posterior temporal Occipital cortices	Visuospatial difficulties
Substantia nigra Pars compacta	Parkinsonian motor features
Cortical and subcortical frontal damage Cerebellar tract injury in brainstem	Gait disorder: staggered, slowed, ataxic
Brainstem nuclei (hypoglossal/oculomotor)	Dysarthria, dysphagia, ocular abnormalities
Amygdala	Aggression and violent outbursts

to be sensitive enough to assess axonal integrity in the setting of mild, moderate, and severe TBI. DTI studies have shown their ability to show occult white matter damage after mTBI that was not visible on typical MRI scans. A study by Kumar et al. tracked serial changes in white matter using DTI techniques in mTBI and found that fractional anisotropy (FA) and mean diffusivity (MD) in the genu of the corpus callosum appear early and persisted at 6 months as a secondary injury to microgliosis [28]. Another study by Inglese et al. showed the abilities of DTI as they reported significant abnormalities in various regions of the brain after mTBI when compared to controls [29]. A third study by Henry and colleagues used DTI to detect changes in white matter by comparing a group of 10 nonconcussed athletes to 18 concussed athletes. They reported that at 1–6 days and at 6 months following concussion there was FA in dorsal regions of both cortical spinal tracts and the corpus callosum [30].

Although researchers are trying to identify biomarkers to aid in diagnosis, there currently are no makers identified in the literature that can be used to diagnosis CTE. However, there are several that are believed to help in identifying CTE like the use of magnetic resonance spectroscopy that can detect changes in glutamate/glutamine, N-acetyl aspartate, and mylo-inositol which have been shown to be abnormal in brain injury [8]. There has also been discussion of attempting to measure tau and phospho-tau in the cerebrospinal fluid of those suspected of having CTE [8].

11. Treatments/Prevention

Currently, the treatment methodologies for CTE are purely preventive. However, in sports like American football, prevention of head trauma is a seemingly difficult goal to attain. Hard hits and head collisions are more than simple aspects of the game; they are part of the sports identity. Therefore, prevention would require a multifaceted approach involving administrators, coaches, players, referees, team physicians, and even the fans who watch the games. The administrators create the policies that penalize athletes for reckless or dangerous hits as well as setting equipment standards for the various leagues. It is the role of the coaches to teach their players correct and safe technique for tackling, hitting, and personal protection while creating a team culture that encourages hard but controlled play. Coaches also need to be aware of the cumulative effect of repetitive mTBI and limit the amount of full contact during practice and drills. It is the role of the players to understand the potential dangers and consequences of head trauma beyond their playing years so they can protect themselves and limit the number of injuries during their career. It is also incumbent on the athlete to not downplay their injuries and to seek help or advice if they are suffering from signs or symptoms of head trauma. As for the referees, it is their role to create a safe playing environment and uphold the rules set forth to protect the players whether on the field, in the ring, or on the ice. As for the team physician, it is their task to remove players from play and appropriately manage their mTBI until they meet the return-to-play criteria. The decision to clear a player is challenging for the physician who has no baseline information of the player's cognitive function prior to the injury. Therefore, it has been suggested that players undergo neuropsychological testing prior to participation in sports as a tool to properly assess the athlete's cognitive deficits both acutely and chronically.

Another aspect of prevention is improving the protective equipment worn by the athletes. It has been shown that helmets and mouth guards function very well in protecting the player from severe head injury if the helmet fits correctly, is strapped in place, and lined with the appropriate padding [6]. A study by Viano and Halstead compared American football helmets from 1970 to 2010 and reported that the newer helmets are heavier, primarily from more padding, longer, higher, and wider then their 1970s counterparts. These larger helmets were better at absorbing forces and impacts associated with concussions in American football [31]. While helmets are important, they may also give some players a false sense of protection leading to a more reckless and violent style of play. Neck strength is another factor that can be important in minimizing head injury especially in younger populations of athletes and should be emphasized by trainers and strength coaches [6]. Some groups are looking for medical therapies to limit the damage after a head injury. Particularly, the use of beta-amyloid-lowering medications has been shown to improve the outcomes following TBI in rodent models [7].

12. Future Considerations for Research

Although there has been an exponential growth in research and interest in CTE over the last five years, the full understanding of it still remains in its infancy. Affected athletes have been the greatest supporters as Boston University's Center for the Study of CTE has more than 260 former athletes in their brain and spinal cord donation registry.

These donations will supply researchers with a wealth of information that will improve animal models, better define the mechanism of injury, as well as advance diagnosis and treatment. As the foundation of knowledge grows, we can better identify genetic variants that put individuals at risk for CTE. The CTE community will also benefit from ongoing concussion research as groups look for acute biomarkers to be used as a diagnostic test for brain injury. There is also a need for further research regarding the role of advanced neuroimaging like DTI and its ability to possibly detect early signs of acute injury. Additionally, more work must be done to quantify the magnitude and frequency of head impact that is needed to cause the neurodegeneration associated with CTE.

13. Conclusions

Chronic traumatic encephalopathy is a neurodegenerative disease that is a long-term consequence of single or repetitive closed head injuries for which there is no treatment and no definitive premortem diagnosis. It has been closely tied to athletes who participate in contact sports like boxing, American football, soccer, professional wrestling, and hockey. Aside from repeated head trauma, risk factors include presence of ApoE3 or ApoE4 allele, military service, and old age. It is histologically identified by the presence of tau-immunoreactive NFTs and NTs with some cases having a TDP-43 proteinopathy or beta-amyloid plaques. It has an insidious clinical presentation that begins with cognitive and emotional disturbances and can progress to Parkinsonian symptoms. The exact mechanism for CTE has not been precisely defined; however, research suggests it is due to an ongoing metabolic and immunologic cascade called immunoexcitotoxicity. Current research is attempting to identify specific biomarkers along with more sophisticated imaging techniques for the diagnosis of CTE. Future research should also be centered around how to manage CTE as suicide is a common fate for those battling the disease. Further efforts need to be made to educate players, coaches, and administrators of all levels of athletics to make them aware of the determents of mTBI and how to best protect themselves. There must also be further investigations into the possible link between CTE and motor neuron disease. Establishing such a causal link may open new doors in ALS research and hopefully lead to better treatments. Through the continued efforts of athletes, scientists, and physicians, our knowledge of CTE will advance and allow for the evolution of better diagnosis, treatment, and prevention.

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Hindawi Publishing Corporation
Rehabilitation Research and Practice
Volume 2012, Article ID 371970, 5 pages
doi:10.1155/2012/371970

Review Article

Mild Traumatic Brain Injury: Lessons Learned from Clinical, Sports, and Combat Concussions

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Received 21 December 2011; Accepted 1 February 2012

Academic Editor: Brian D. Greenwald

Over the past forty years, a tremendous amount of information has been gained on the mechanisms and consequences of mild traumatic brain injuries. Using sports as a laboratory to study this phenomenon, a natural recovery curve emerged, along with standards for managing concussions and returning athletes back to play. Although advances have been made in this area, investigation into recovery and return to play continues. With the increase in combat-related traumatic brain injuries in the military setting, lessons learned from sports concussion research are being applied by the Department of Defense to the assessment of blast concussions and return to duty decision making. Concussion management and treatment for military personnel can be complicated by additional combat related stressors not present in the civilian environment. Cognitive behavioral therapy is one of the interventions that has been successful in treating symptoms of postconcussion syndrome. While we are beginning to have an understanding of the impact of multiple concussions and subconcussive blows in the sports world, much is still unknown about the impact of multiple blast injuries.

1. Introduction

As early as 1962, Symonds made the observation that any concussion, no matter how mild, may result in permanent neurologic impairment [1, 2]. Almost a decade later, researchers in New Zealand first documented delayed return to work and complex processing speed deficits related to "minor head injury" [3]. By the early 1980's, neuroscientists at the University of Virginia were finding that patients who had sustained a concussion were slow to return to work and were continuing to experience deficits in attention, memory, new problem solving, mental flexibility, and cognitive processing speed three months postinjuries [4, 5].

Around this same time, animal studies were revealing neuropathological/histological changes in the brain stems of primates when subjected to acceleration deceleration, rotational injuries, or mild traumatic brain injuries (mTBIs) [2, 6]. These acceleration or torque forces were expressed as "shear-strain", or stretching and tearing of axonal fibers. Concurrent neurochemical cascades occur during any severity of traumatic brain injury (TBI). These reactions to trauma typically involve the rapid transfer of potassium and sodium to the extracellular space, the influx of calcium, and the release of oxygen free radicals, which can result in cell death (apoptosis). Returning to a homeostatic state requires energy (glucose) and time, which in the case of mild concussion is often 5 to 10 days.

To better understand the neurocognitive sequel of this concussion phenomenon in humans, a new method of study was initiated at the University of Virginia: the Sports as a Laboratory Assessment Model (SLAM) [2]. This research model involved the use of contact sports as a controlled laboratory to collect brief baseline neurocognitive test data that would later be compared to repeat postconcussion test data. In the first study of its kind, more than 2300 football players at 10 universities were assessed before the football season with brief neurocognitive tests [7]. When a concussion occurred in these players, the same preseason battery was administered 24 hours, 5 days, and 10 days postinjury. Scores on these measures were not only compared to the preseason performance of the individual athlete, but also to a matched control (redshirt) group (a student athlete who delays eligibility to play, typically during their freshman year of college) at the same time intervals. On neurocognitive tests,

significant differences were noted between the concussed players' baseline test scores and the concussed and control players' test scores during the 24 hour and 5 day assessments. By the 10 day reevaluation, players who had sustained a concussion were performing at nearly the same level as their matched controlled peers. This model suggested that there is a typical, and relatively quick, recovery curve that occurred in healthy, young, motivated athletes when they were allowed to rest. This is especially true when there are no complicating psychosocial or medical factors, which are more often seen in clinical populations.

The SLAM model was initially instituted as a controlled laboratory to more accurately determine the acute neurocognitive cost to any individual suffering a single mild concussion, and to plot the typical recovery curve for clinical applications. Today this model is a standard part of the sports medicine concussion assessment and management programs at many high schools, universities, and professional sports teams, to protect athletes from the dangers of returning to play before complete resolution of neurologic and cognitive concussion symptoms. By having a preseason baseline assessment, athletic trainers and team physicians can better evaluate and treat concussions when they occur. This helps to ensure that an injury, no matter how mild, can be properly assessed and treated appropriately in order to reduce the potential severe consequences of a second injury. A rare condition known as second impact syndrome (SIS) [8] can occur, especially in adolescents who have sustained two minor impacts in close proximity. It is hypothesized that these closely occurring injuries can result in a catastrophic increase in intracranial pressure due to dysfunction of autoregulation of the cerebrovascular system. Identifying concussions in order to allow full neurologic recovery is critical to avoid further injury and to facilitate prudent return-to-play (RTP) decisions.

2. Return to Play

Based on this line of research which has been expanded and refined by many other neuroscientists, specific recommendations have been created to guide safe return-to-play (RTP) decisions. In 2008, the Third International Conference on Concussion in Sport was held in Zurich which produced a consensus on managing sports concussions [9]. Based on these guidelines, a gradual RTP protocol was created. The protocol goes into effect once an athlete is symptom free while at rest. As the athlete completes each component and remains symptom free, he or she moves to the next step. First, light aerobic exercise, which keeps the heart rate at less than 70% of the maximum, predicted heart rate is recommended. If symptoms do not return, the second stage involves engaging in sport-specific exercises that do not involve any potential impact to the head. For example, these would be skating in ice hockey or running in soccer. Third, noncontact training drills are added, such as passing drills in ice hockey or football, along with beginning progressive resistance training. Fourth, the athlete can engage in a full contact practice and return to normal training activities. Once they have reached this stage without experiencing a return of symptoms, they are able to return to full play.

3. Application of SLAM to Military Concussions

Another area where concussions have gained significant attention is in the current conflicts in Iraq and Afghanistan. One of the signature injuries of Operation Iraqi Freedom (OIF), Operation Enduring Freedom (OEF), and now Operation New Dawn (OND) has been traumatic brain injury. Approximately 79% of combat-related TBIs are caused by improvised explosive devices, leading to blast concussive injuries [10]. Traumatic brain injuries that occur as a result of a blast can involve any aspect of the four blast phases [11].

The primary phase is characterized by atmospheric overpressurization immediately followed by a vacuum or atmospheric underpressurization as a function of the initial blast wave. Typical injuries resulting from this blast phase are damage to hollow organs such as rupture of the tympanic membranes, pulmonary barotrauma, and acute gas emboli (AGE) in the cerebrovasculature and cerebrospinal fluid [12, 13]. The secondary phase involves objects that are placed in motion due to the blast leading to blunt and penetrating injuries [12, 13]. For example, a secondary phase of injury can occur when blast debris (rocks, cement, etc.) hits a service member. The tertiary blast phase occurs when service members are thrown off of their feet and hit stationary objects, which often results in acceleration-deceleration and blunt head trauma injuries. Quaternary-phase injuries are sustained following a blast and may include burns, toxic inhalation, exposure to radiation, asphyxiation, dust inhalation, and crush trauma [12, 13]. The primary and quaternary phases of combat blast injury make it considerably more complex than the typical sports related concussion, which involves blunt trauma and acceleration-deceleration dynamics (similar to secondary and tertiary phases in combat blast).

4. Using SLAM in the Military: Return to Duty

The SLAM baseline methodology was recently engaged by the military to better assess and manage concussions. Concussions are identified in the field by a combination of description of blast exposure (being within 50 meters of a blast), symptom report, and use of a brief mental status/neurocognitive assessment measure, the Military Acute Concussion Evaluation (MACE) [14], which is based upon the standardized Assessment of Concussion (SAC) from the sports literature [15]. In May 2008, the military began using the Automated Neuropsychological Assessment Metric (ANAM) to assess neurocognitive functioning in all predeployers. This provided baseline neurocognitive data for military members to compare to postconcussive test results to determine the extent of injury.

Using the sports concussion literature, the military has created guidelines for returning military members to duty following a concussion. In 2010, a statement was issued that required designated periods of rest following a concussion (Deputy Secretary of Defense). Procedures were established that specify when a concussion evaluation is mandatory. Clinical practice guidelines are available to medical personnel in the deployed environment regarding the evaluation and treatment of concussions. These mandatory events are

tracked and reported to Department of Defense Executive Agent's Joint Trauma analysis and Prevention of Injury in Combat (JTAPIC) program office. Additionally, there are currently specific teams in the deployed environment, Concussion Care Centers that assess and treat traumatic brain injuries.

5. Treatment in CONUS

Once a TBI occurs, rehabilitation begins, whether on the battlefield or in the Continental United States (CONUS). The symptoms of most mild TBIs will resolve with rest shortly after the injury (typically 48 hours to 3 months). However, if symptoms do not resolve with rest in 10–14 days or if the service member has had multiple concussions, they are evacuated to a higher level of in-theater care [16] to be evaluated by a specialized TBI medical unit. If their symptoms are severe, they can be med-evaced out of theater and admitted to an Acute Care Unit at a military treatment facility (MTF) for medical stabilization and to begin undergoing assessments by psychiatry, neurology, neuropsychology, physical therapy, occupational therapy, and speech and language pathology [17].

As patients improve, they transition to acute inpatient rehabilitation [17] with a goal to "set the stage for optimal community reintegration post discharge and to restore functional independence" (Page 185). Many individuals with TBIs will make effective recoveries during acute rehabilitation programs. Those that do not may be referred to transitional rehabilitation programs with a goal of community reintegration or a return to the least restrictive environment. Transitional rehabilitation programs may be residential, community-based day treatment as part of a larger medical system, or a home-based model.

The U.S. Department of Veterans Affairs has designed a polytrauma system of care (PSC) to offer specialized rehabilitation to veterans in areas closer to their home than most major military treatment centers [17]. There are four polytrauma rehabilitation centers (PRCs), spread across the USA (Minnesota, California, Virginia, and Florida) with a goal of acting as a hub for acute medical and rehabilitation care, along with research and education in the fields of polytrauma and TBI.

The Defense Centers of Excellence (DCoE) houses the Defense and Veterans Brain Injury Center (DVBIC). DVBIC brings together the DoD, VA, and civilian providers to assess and treat TBIs, provide education, and conduct clinical research. In addition to active duty military members and veterans, DVBIC also provides care to military beneficiaries who have sustained a TBI. DVBIC is comprised of 19 centers including MTFs, VAMCs, and two civilian TBI community reentry partners spread across the USA and in Germany.

6. Traumatic Brain Injury and Posttraumatic Stress Disorder

One of the complicating factors in recovery from concussion that is seen in the military population that is typically not seen in athletes is that of psychosocial stressors. These include sleep deprivation or alternated sleep schedules and mental health concerns, including acute stress disorder and at times posttraumatic stress disorder. When working with returning veterans, it is important for providers to be able to determine whether an individual has a mild TBI that has not resolved which is referred to as Postconcussion Syndrome (PCS)/Postconcussion Disorder (PCD), Posttraumatic Stress Disorder (PTSD)/posttraumatic spectrum disorder, or both. While symptom management is key, knowing the root cause of a symptom such as irritability, can direct treatment that targets the root cause and improves the efficiency of the recovery process. While most mild TBI symptoms resolve within 1–3 months of the event, in rare cases symptoms may persist longer. When the neuropsychological symptoms last beyond the initial 1–3 month time period, PCS is diagnosed. Common symptoms of PCS are difficulty with memory, changes in mood, problems paying attention and concentrating, decreased energy, and mental slowness. Despite advances in our understanding of mTBI, it is still unclear why some individuals recover faster than others after a mild TBI. Undoubtedly, recovery is a multifactorial process based upon age, gender, genetic predisposition, premorbid neurocognitive abilities, severity of injury, number of previous concussions, substance abuse history, sleep disturbances, pain, depression, and psychosocial supports to name a few.

The sequelae of an mTBI are varied and have far reaching implications for the individual if not clinically addressed. Feelings of loss and reduced functioning may lead to depression, in conjunction with having ecological impacts, such as relationship issues and decreased work performance. Untreated depression and other psychiatric conditions that can be comorbid with mTBI not only impede psychosocial functioning but also reduce the amenability to treatment [18]. The increased propensity for an individual to experience psychiatric symptoms with mTBI requires treatment methodologies which address both the neurologic and emotional injury. Because of the variability of neurologic and emotional injury manifestations, standard approaches to treatment have proven ineffective in many cases.

As seen with treating depression, cognitive behavioral therapy (CBT) has also been helpful in ameliorating anxiety spectrum disorders such as Obsessive Compulsive (OCD) and Posttraumatic Stress Disorders (PTSD). Williams and Evans [19] postulated that anxiety spectrum disorders can be just as debilitating as depression and if left untreated often result in reduced quality of life and maladaptive coping mechanisms. Until recently, there has been a scarcity of research evaluating the treatment of depression and other psychiatric disorders related to mTBI [18]. The first line of therapy to help remediate depression and anxiety symptoms during the course of neurorehabilitation has been behavioral therapy (BT) [18]. As a result of an mTBI, often maladaptive behaviors emerge in those who have prolonged impairments. Schlund and Pace [20] suggest that BT has been effective enhancing social skills and cognitive rehabilitation.

Treatment protocols for CBT have been modified to apply to those less capable of participating in traditional CBT approaches for psychiatric conditions [19]. More often than

not, emotional and cognitive symptoms persist after mTBI and are maintained by dysfunctional thought processes and emotional impairment. Early research suggests addressing these emotional issues in the initial stages of treatment, simultaneously with the neurologic injury has been helpful in preventing postconcussion sequelae [21].

Tiersky and colleagues [22] noted that with a combination of cognitive remediation and CBT, participants reported less emotional distress and increased cognitive functioning after treatment. Additionally, the emotional improvements continued to increase at the 1-month and 3-month followups posttreatment. The cognitive remediation portion of the treatment focused on attention, information processing, and memory. The focus of CBT was to provide increased coping behaviors, teach skills to prevent relapse, and cope with the feelings of loss related to their decreased functioning. Although the treatment was not purely CBT, it provides additional evidence that CBT can be effective with patients who have sustained both mild and moderate TBIs.

A more recent study of an individualized treatment of CBT was also found effective in reducing emotional distress in patients with a TBI [23]. In this research, CBT was modified from its standard form to include repetition of important concepts and frequent breaks. Because cognitive assessments were completed prior to the study, the intervention was individualized to each patient based on their ability to learn and retain information and their cognitive processing speed. The intervention was delivered in both a group format and over the telephone. Those in the treatment groups had statistically significant less emotional distress following the study and at the 1-month followup. This suggests that patients who have sustained a TBI can benefit from CBT, even though some alterations may need to be made to the basic CBT concepts.

Insomnia is also commonly associated with mTBI and often prolongs the recovery process. Ouellet and Morin [24] conducted a case study of a man who sustained a brain injury and had failed pharmacotherapy for insomnia. Following eight weeks of treatment focusing on stimulus control, sleep restriction, cognitive therapy, and sleep hygiene education, the participant's sleep had improved. At the 3-month followup, the positive effects were still apparent. This case study showed that the use of CBT is promising for post-TBI insomnia. In 2007, Ouellet and Morin [25] expanded on their work. With a small group of participants ($n = 11$) who had sustained a TBI, they provided the same treatment as above and added a fatigue management component. Sleep efficiency had improved for each participant in the study at the end of treatment and the positive effects remained at the 3-month followup. This appears to be an effective way of managing insomnia in individuals with a TBI, however, more research with larger sample sizes continues to be needed in the field.

7. The Impact of Multiple Concussions

While some studies have shown little to no impact of 2 concussions [26], other research has shown that multiple concussions and subconcussive blows may lead to chronic traumatic encephalopathy (CTE) [27]. Chronic traumatic encephalopathy has been found on autopsy in boxers, football players, and hockey players who suffered multiple concussions, and many more subconcussive blows, eventually exhibiting early dementia and depression. Histologically, CTE is characterized by the expression of high concentrations of tau protein (neurofibrillary tangles) and Beta Amyloid plaques. These findings appear to be relevant to the military due to the high risk of multiple blast injuries. This offers further support for the importance of new guidelines for concussion identification, management, and long-term follow-up.

8. Conclusion

Almost four decades ago, mild head injury in clinical populations began to gain attention from the medical and neuropsychological communities. Much of the initial controlled data on mTBI and concussions was gleaned from athletes who were at high risk for mild head trauma. With the multiple combat tours and the nature of warfare in Iraq and Afghanistan that are putting service members at risk for multiple concussive injuries, the lessons learned from the sports concussion literature are being applied to the military. While there are differences in the mechanism of injury and additional challenges when TBIs occur in the combat setting, there are also similarities. Due to these similarities and the need for a graduated return to duty (or play), the military is now implementing clinical practice guidelines similar to the Zurich recommendations used in many civilian sports. Following a TBI, military personnel and veterans have services available to them though military medical treatment facilities and the Department of Veterans Affairs for continued treatment of symptoms.

There is continuing research on the impact of multiple concussions and subconcussive blows in boxing, football, soccer, and hockey that have led to a better understanding of the possible links to CTE. There are, however, no longitudinal bodies of data currently available from which to draw conclusions regarding the impact of multiple concussions during combat. Continued study of combat related blast concussions is important to our efforts to quickly and safely return service members to duty, treat persistent concussion symptoms, and avoid long-term sequel and CTE.

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Hindawi Publishing Corporation
Rehabilitation Research and Practice
Volume 2012, Article ID 316575, 7 pages
doi:10.1155/2012/316575

Research Article

Possible Lingering Effects of Multiple Past Concussions

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Received 30 October 2011; Revised 2 January 2012; Accepted 3 January 2012

Academic Editor: Anne Felicia Ambrose

Background. The literature on lingering or "cumulative" effects of multiple concussions is mixed. The purpose of this study was to examine whether athletes with a history of three or more concussions perform more poorly on neuropsychological testing or report more subjective symptoms during a baseline, preseason evaluation. *Hypothesis*. Athletes reporting three or more past concussions would perform more poorly on preseason neurocognitive testing. *Study Design*. Case-control study. *Methods*. An archival database including 786 male athletes who underwent preseason testing with a computerized battery (ImPACT) was used to select the participants. Twenty-six athletes, between the ages of 17 and 22 with a history of three or more concussions, were identified. Athletes with no history of concussion were matched, in a case-control fashion, on age, education, self-reported ADHD, school, sport, and, when possible, playing position and self-reported academic problems. *Results*. The two groups were compared on the four neuropsychological composite scores from ImPACT using multivariate analysis of variance followed by univariate ANOVAs. MANOVA revealed no overall significant effect. Exploratory ANOVAs were conducted using Verbal Memory, Visual Memory, Reaction Time, Processing Speed, and Postconcussion Scale composite scores as dependent variables. There was a significant effect for only the Verbal Memory composite. *Conclusions*. Although inconclusive, the results suggest that some athletes with multiple concussions could have lingering memory deficits.

1. Introduction

Sport-related concussions result in temporary neurocognitive deficits and subjectively experienced physical, emotional, and cognitive symptoms in the initial hours, days, and sometimes weeks after injury [1–17]. In group studies, athletes tend to recover in terms of perceived symptoms and neuropsychological test performance within 2–28 days, with most studies suggesting recovery occurs in 5–10 days [7, 10, 13, 15–17]. There is some evidence suggesting that high school athletes might recover more slowly than university or professional athletes [18–21].

Athletes with prior concussions are at statistically increased risk for a future concussion [22–25]. Even more concerning is whether athletes with previous concussions are at risk for long-term damage to the structure and/or function of their brains. Numerous studies have been published employing cross-sectional methodologies in an attempt to determine whether groups of previously concussed athletes appear to have lingering effects detectable using symptom rating scales, neuropsychological testing, and electrophysiology. Literature on this subject is somewhat mixed, partly due to methodological differences and limitations across studies. Moreover, some aspects of the research designs and

data analyses vary considerably across studies, and many researchers have not consistently reported effect sizes. Sample size is usually the limitation because the accrual of multiply concussed athletes is slow. For example, several published studies have sample sizes of multiply concussed athletes of fewer than 20 (e.g., [26–31]). Other studies do not define the injury severity characteristics of prior concussions (e.g., [9, 27, 32–40]) or when the prior concussions occurred (e.g., [9, 22, 27, 28, 30, 32, 34–38]). Most studies rely on a cross-sectional methodology of examining small [28, 29, 34, 41] or large [2, 22, 27, 32, 35, 38–40, 42] groups of athletes during baseline preseason testing. Some studies, however, have followed multiply concussed athletes prospectively [2, 22, 29, 32–34, 38, 43, 44].

Despite the methodological challenges associated with this area of research, there is an accumulation of evidence that some athletes with a history of multiple concussions might have lingering, long-lasting adverse effects. In a large scale NCAA study, Collins and colleagues [2] reported that athletes with a history of two or more concussions (i.e., 2–10) reported more symptoms and performed more poorly on two neuropsychological tests than athletes with no concussion history. Other studies have reported that athletes with a history of three or more concussions have changes in electrophysiology, [28, 31, 40] subjective symptoms, [28, 34] and neuropsychological test performance [34]. One large study involving jockeys ($N = 618$) reported that those with two or more previous concussions did not report more subjective symptoms or perform more poorly on neuropsychological testing than those with no previous concussion. When comparing jockeys with a history of one prior concussion to those with two or more prior concussions, those with more injuries performed more poorly on a single neuropsychological test (Stroop Color-Word Test) [39]. Stephens et al. [42] administered a battery of neuropsychological tests to a group of athletes and found that those with previous concussions performed more poorly on a test of attention and a visual memory test. In contrast, some large-scale studies have not found differences in neurocognitive functioning in multiply concussed athletes [27, 32, 45].

Due to concerns that the mixed results observed in the literature are due to differences in the type of neuropsychological tests employed, Bruce and Echemendia [46] investigated the relationship between self-reported history of concussion and neuropsychological test performance in a large multisport sample of college athletes using three testing approaches: (1) performance on a computerized neuropsychological test battery (ImPACT), (2) performance on traditional paper-pencil neurocognitive tests, and (3) the relationship between concussion history and performance on both traditional neuropsychological tests and computerized neuropsychological tests. None of the approaches yielded statistically significant relationship between history of multiple concussion and neurocognitive deficits. Finally, evaluations of mechanisms of cumulative subconcussive impacts have not shown a definitive link between repeated impacts such as exposure to heading a soccer ball [42, 47] or impact biomechanics in football to concussion [48]. Thus, recent studies have not clearly demonstrated whether a lingering effect of three or more previous concussions can be reliably detected. Moreover, given that most research designs are cross-sectional (not prospective), statements about causation must be made cautiously.

Given the contradictory findings, more research is needed. The purpose of this study was to examine whether athletes with a history of three or more concussions perform more poorly on neuropsychological testing, or report more subjective symptoms, during a baseline, preseason evaluation. A precise matching of athletes on numerous variables, in a case-control fashion, was used to improve the methodological rigor of the study.

2. Method

2.1. Participants. An archival database including 786 male athletes who underwent preseason testing with a computerized battery (ImPACT) was used to select the participants. Twenty-six athletes, between the ages of 17 and 22 (mean age = 19.7, SD = 1.4), with a self-reported history of three or more concussions, were identified (16 sustained three previous concussions, six four previous concussions, two five previous concussions, one six previous concussions, and one ten previous concussions). Athletes with no self-reported history of concussion were precisely matched, in a case-control fashion, on age ($M = 19.7$, SD = 1.4; $t(50) = 0.04$, $P = .97$; each person matched within 6–12 months of age) and education ($M = 12.8$, SD = 1.1; $t(50) = 0.23$, $P = .82$; each person matched within one year of education). Athletes with no previous history of concussion were precisely matched on self-reported ADHD (two subjects in each group). Matching was also attempted with self-reported learning problems (two subjects in the three or more concussions group and one subject in the no concussion group), the need for "special education" (one subject in the three or more concussions group and no subjects in the no concussion group), and repeated grades (three subjects in the three or more concussions group and two subjects in the no concussion group).

In addition to matching the above demographic and academic information, we also sought to match the participants on athletic variables. With the exception of one lacrosse player who was matched to a baseball player in the three or more group, we matched each of the athletes on the sport that they played (e.g., football, 30.8%; soccer, 23.1%; ice hockey, 15.4%; lacrosse, 15.4%; wrestling, 7.7%; and water polo, 3%). When possible, position played (e.g., forward in ice hockey, linebacker in football, goalkeeper in soccer, and weight class in wrestling) and school attended or the organization that they identified was also matched to controls.

2.2. Measures. ImPACT is a brief computer-administered neuropsychological test battery that consists of six individual test modules that measure aspects of cognitive functioning including attention, memory, reaction time, and processing speed. Each module contributes to the calculation of four composite scores, Verbal Memory, Visual Memory, Reaction Time, and Processing Speed.

(i) The Verbal Memory composite score represents the average percent correct for a word recognition paradigm, a symbol number match task, and a letter memory task with an accompanying interference task. These tests are conceptually similar to traditional verbal learning (word list) tasks and the auditory consonant trigrams test (i.e., the Brown-Peterson short-term memory paradigm), although the information is presented visually on the computer, not verbally by an examiner.

(ii) The Visual Memory composite score is comprised of the average percent correct scores for two tasks; a recognition memory task that requires the discrimination of a series of abstract line drawings, and a memory task that requires the identification of a series of illuminated X's or O's after an intervening task (mouse clicking a number sequence from 25 to one). The first test taps immediate and delayed memory for visual designs and the second test measures short-term spatial memory (with an interference task).

(iii) The Reaction Time composite score represents the average response time (in milliseconds) on a choice reaction time, a go/no-go task, and the previously mentioned symbol match task (which is similar to a traditional digit symbol task).

(iv) The Processing Speed composite represents the weighted average of three tasks that are done as interference tasks for the memory paradigms.

In addition to the cognitive measures, ImPACT also contains a Postconcussion Symptom Scale that consists of 21 commonly reported symptoms (e.g., headache, dizziness, "fogginess"). The dependent measure is the total score derived from this 21-item scale.

The reliability [49–52] and validity [26, 53, 54] of the cognitive composite scores and the Postconcussion Symptom Scale [55–57], and the sensitivity of the battery to the acute effects of concussion [14, 15, 50, 58–62] have been examined in a number of studies. In three studies, the test-retest reliability of ImPACT over brief [49, 50] and long [52] retest intervals was considered adequate, and in one study stability was poor [51]. In regards to concurrent validity, the ImPACT composites measuring speed and reaction time showed medium correlations with the Symbol Digit Modalities Test, Trails B, and Digit Symbol [26, 53, 54], and the memory composites showed medium to large correlations with the Brief Visuospatial Memory Test [26, 53, 54].

3. Results

The athletes with previous concussions and the control subjects were compared on the four neuropsychological composite scores from ImPACT using multivariate analysis of variance (MANOVA) followed by univariate ANOVAs. Box's M Test was nonsignificant ($P = .65$), indicating that the covariance matrices were similar across the dependent variables. Levene's test was nonsignificant for all four composite scores, indicating that the assumption of homogeneity of variance was not violated. Across the two groups, however, there were some departures from normality on individual composite scores as assessed by the Kolmogorov-Smirnoff procedure. MANOVA and ANOVA tend to be quite robust to these violations of underlying general linear model assumptions. MANOVA, with the four cognitive composite scores as dependent variables and group membership as an independent variable, revealed no overall significant effect ($F(4, 47) = 1.6$, $P = .19$, observed power = .46). The power for this analysis was low, increasing the risk for a type II statistical error. Therefore, exploratory one-way analyses of variance were conducted using Verbal Memory, Visual Memory, Reaction Time, Processing Speed, and Postconcussion Symptom composite scores as dependent variables and group membership as the independent variable. There was a significant effect for only the Verbal Memory composite ($P = .028$, Cohen's $d = .63$, medium effect size). The Visual Memory and Postconcussion Symptom composite scores were both not significant in the one-way analyses ($P = .16$ and $P = .13$, resp.), although there were small-medium effect sizes for both ($d = .39$ and $d = .45$, resp.). Performances on the Processing Speed and Reaction Time composites were very similar between groups. A summary of the athletes' performance on ImPACT, including age-adjusted percentile rank normative scores, is presented in Table 1.

4. Discussion

The results of this study were provocative but not persuasive. There was modest evidence that athletes with multiple concussions could have a lingering deficit in memory. Athletes with three or more previous concussions performed more poorly on the Verbal Memory composite score (Cohen's $d = .63$, medium effect) than athletes with fewer concussions. In a previous study, Iverson and colleagues reported a nonsignificant trend for athletes with three or more concussions to have worse preseason memory performance on ImPACT than those with no previous concussions (Cohen's $d = .59$, medium effect size), and recently concussed athletes with a history of multiple concussions had significantly greater decrements on the memory composite compared to concussed athletes with no injury history [34]. The present study, like several previous studies, is underpowered, which might explain why the medium effect sizes for Visual Memory and the total score from the Postconcussion Scale were not significant.

The literature to date regarding cumulative effects is not definitive. Some researchers have reported that groups of athletes with a history of multiple concussions perform more poorly on neuropsychological testing compared to those with fewer or no concussions [2, 39], whereas others have not [27, 31, 32]. Some researchers have reported that athletes with a history of multiple concussions report more subjective symptoms during preseason testing [2, 28, 34, 45], whereas others have not [39]. In the previously discussed large-scale study with jockeys, Wall and colleagues [39] emphasized that those individuals with two or more concussions versus a single past concussion performed more poorly on the Stroop Color-Word test, suggesting that "multiple concussions may

Table 1: Performance on ImPACT in athletes with 3 or more previous concussions and matched controls.

ImPACT scores	3 or more previous concussions		No previous concussion		t-test	Effect size
	Mean	SD	Mean	SD	(P value)	(Cohen's d)
Raw scores						
Verbal memory	86.4	8.1	92.1	10.0	5.14 (.028)	.63
Visual memory	77.1	13.3	82.3	13.1	1.99 (.16)	.39
Processing speed	41.7	7.5	41.3	8.2	0.21 (.88)	.04
Reaction time	0.55	0.08	0.54	0.08	0.56 (.46)	.21
Total symptoms	4.2	7.8	1.6	3.5	1.54 (.13)	.45
Percentile ranks						
Verbal memory	44.4	31.8	71.3	27.2	10.75 (.002)	.91
Visual memory	50.5	33.0	63.1	31.1	2.00 (.16)	.39
Processing speed	66.5	29.2	66.8	28.9	0.002 (.97)	.01
Reaction time	54.3	33.1	58.6	29.9	0.24 (.63)	.14

Note: SD: standard deviation. "Percentile ranks" refer to the age adjusted percentile ranks for each raw score from the ImPACT normative data. By convention, Cohen's effects sizes are interpreted as follows: small: 0.2, medium: 0.5, and large: 0.8.

interfere with executive skills, including response initiation/inhibition, divided attention, and concentration" (page 519). However, there was no significant difference between jockeys who were multiply concussed and those who were never concussed on the Stroop test ($d = .22$). Moreover, the jockeys with one or more past concussions actually performed significantly better on a test of attention and processing speed (digit symbol-coding) than jockeys with no previous concussions ($d = .23$, small effect size). Therefore, although the authors emphasized the possibility of an adverse lingering neurocognitive effect of multiple concussions, the results from that study could be interpreted as equivocal.

In the previous large-scale NCAA football study, Collins and colleagues [2] reported that "a history of concussion is significantly and independently associated with long-term deficits in the domains of executive functioning and speed of information processing" (page 968). Indeed, those players with a history of two or more concussions performed more poorly on Trails B ($d = .41$) and the Symbol Digit Modalities Test ($d = .46$). These findings appear to support their conclusion regarding long-term deficits. However, it is interesting to note that players with one previous concussion performed significantly better than players with no previous concussions on Digit Span ($P < .03$; $d = .25$, small effect), Symbol Digit Modalities Test ($P < .03$, $d = .26$), Trails A ($P < .05$, $d = .23$), and Trails B ($P < .03$, $d = 26$). The effect sizes, however, were very small. Nonetheless, a trend toward better neuropsychological performance in those with a history of concussion is difficult to understand and reconcile.

Belanger and colleagues [63] conducted a meta-analysis of the literature on lingering effects of multiple concussions. Eight studies met inclusion criteria, contributing 614 cases of multiple concussions and 926 control cases of a single concussion. There was no significant overall effect of multiple concussions on symptom reporting or neuropsychological test performance. However, using specific cognitive domains as a moderator, they reported that worse performance in both executive functions ($d = .24$; small effect) and delayed memory ($d = .16$; small effect) was associated with multiple concussions. As noted in the Introduction, there are numerous and important methodological differences and limitations in this literature. There is considerable heterogeneity in sample sizes, outcome measures, research design, and data analyses. These methodological differences and limitations make the literature challenging to interpret and to compare. Nonetheless, the lack of convergent evidence across studies and the meta-analysis suggests that lingering neurocognitive difficulties (a) are difficult to detect with standard neuropsychological testing and/or (b) might be present in only a subset of athletes and are thus obscured in group analyses.

The present study, unfortunately, is not immune to some of those same limitations. First, the relatively small sample sizes decreased the statistical power of the study. Small sample sizes of multiply concussed athletes have been a common methodological limitation in previous studies (e.g., [27–31, 34]). This limitation is difficult to overcome because of the difficulty identifying a large cohort of multiply concussed athletes. From a database of 786 subjects, we were able to extract only 26 with history of three or more injuries. Second, we were unable to define the severity of prior concussions. This limitation also is common in the literature (e.g., [9, 27, 32–39]). Third, we were unable to determine when the prior concussions occurred. This limitation, too, is common in the literature (e.g., [9, 22, 27, 28, 30, 32, 34–38]). Lastly, similar to past studies [2, 22, 27–29, 32, 34, 35, 38, 39, 41], we utilized a cross-sectional methodology (not longitudinal). Thus, clear causal inferences cannot be made.

Despite these aforementioned limitations, this study used a careful matching methodology. The rigor of this matching process minimized the likelihood that any differences found in neurocognitive performance are attributable to extraneous factors, such as age, education, self-reported ADHD, academic, or learning problems; sport; position played, and school or organization. Of course, more research is needed to better understand the extent to which there are lingering

effects of multiple concussions, how the field best measures those effects, and how we use this information when making decisions regarding athletes' return to play.

Acknowledgments

Drs. G. Iverson and R. Echemendia have been reimbursed by the government, professional scientific bodies, and commercial organizations for discussing or presenting research relating to MTBI and sport-related concussion at meetings, scientific conferences, and symposiums. Dr. G. Iverson has received past research funding from ImPACT Applications, Inc. The developers or publishers of ImPACT did not provide funding for, nor influence the conduct of, this study.

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Hindawi Publishing Corporation
Rehabilitation Research and Practice
Volume 2012, Article ID 837301, 9 pages
doi:10.1155/2012/837301

Research Article

Driving Difficulties and Adaptive Strategies: The Perception of Individuals Having Sustained a Mild Traumatic Brain Injury

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Received 4 November 2011; Revised 22 December 2011; Accepted 16 January 2012

Academic Editor: Gina P. Armstrong

Introduction. After a mild traumatic brain injury (mTBI), individuals quickly resume driving. However, relatively little is known about the impact of mTBI on driving ability and, notably, on the perceived influence of postconcussive symptoms on driving. Hence, the objective of this study was to document the perception of driving abilities in individuals with mTBI. *Method*. Twenty-seven drivers with mTBI were interviewed to document their perception regarding their driving abilities. Both driving-related difficulties and compensatory strategies used to increase driving safety were documented. A mixed quantitative and qualitative analysis of the data was completed. *Results*. 93% of participants reported at least one difficulty perceived as having an impact on everyday activities. Most frequently named problems affecting driving were fatigue and reduced concentration. In addition, 74% of participants had adapted their driving or developed strategies to compensate for driving difficulties. *Discussion/Conclusion*. Postconcussive symptoms have repercussions on driving ability. However, people with mTBI tend to be aware of their difficulties and develop, over time, adaptive strategies. Preventive measures are thus warranted to increase health care professionals' awareness of the potential consequences of mTBI on driving ability and to promote guidelines for the safe resumption of driving after injury.

1. Introduction

Mild traumatic brain injury (mTBI) is a major public health problem disproportionately affecting young adults [1]. Incidence in the United States is about 600 per 100,000 population, though many do not seek medical care, and only 25% are admitted to hospital [1]. It is estimated that up to 25% of individuals presenting to the emergency department with mTBI have persisting postconcussive symptoms at 6 months following injury [2], such as headaches, fatigue, concentration deficits, delayed information processing, and vision and memory problems. Despite these problems, individuals generally resume driving shortly after their mTBI. However, very few studies have examined the potential consequences of mTBI on driving ability to promote positive strategies and guidelines for the safe resumption of driving after injury. In the present study, this issue will be investigated by obtaining the perspective of individuals who have sustained a mTBI in order to explore and describe the problems they experience in relation to driving, and the strategies they utilize to ensure safety when driving.

Driving is a complex, cognitively demanding activity [3]. It requires planning, concentration, inhibition of non-pertinent stimuli, foresight, anticipation, problem solving, a capacity to interpret complex situations with multiple stimuli quickly and efficiently, and the capacity to react

calmly, but rapidly and effectively [3]. For many people, driving is synonymous with independence [3], and, in most cases, persons who have experienced an injury wish to resume driving as quickly as possible [4]. However, the evaluation of driving is challenging for health care professionals, especially with the TBI clientele, since there are few standardized protocols of evaluation adapted to their needs [5, 6] and the efficiency of the existing measures is not well known [7].

Symptoms such as fatigue and difficulty concentrating are known contributors to "quasi accidents" (or near misses). Near misses are events that do not cause injury and have limited immediate impact [8]. However, near misses have been shown to be good indicators of drivers at higher risk of being implicated in accidents. If a person experiences drowsiness, near-misses are 14 times more common than actual accidents [8]. In terms of concentration, inattention of drivers plays a major role in road accidents for the general population. The National Highway Traffic Safety Administration (NHTSA) estimates that about 25% of accidents reported by the police involve a certain form of inattention of the driver—the driver is distracted, asleep or fatigued, or "lost in his/her thoughts" [9].

It has recently been shown that persons with mTBI are at a higher risk of collisions when driving [10]. Using a survey of on-road-related activities with a group of 80 college undergraduate students, Schneider and Gouvier found that persons who self-reported a history of mTBI also self-reported a significantly higher number of accidents than control subjects and were classified in a higher risk category for vehicular crashes [10]. Average age of participants was 22 years and average time following mTBI was 7.1 years. Preece et al. (2010) studied the risk incurred on driving for persons with mTBI in the first 24 hours after the trauma. Here, participants were recruited in the emergency department within 24 hours of their injury, had a Glasgow Coma Scale score between 13 and 15, and were between 18 and 65 years of age. Fifty-seven percent had sustained a mTBI secondary to an assault and 26% secondary to a fall. Using the Hazard Perception Test, a video simulation test of road scenes requiring participants to rapidly identify problematic situations (i.e., probable collisions), mTBI participants were significantly slower to react than participants having sustained an orthopaedic injury (450 ms slower) [4]. This slower reaction time would translate into an increased breaking distance of 7.50 m when travelling at a speed of 60 km/h. These results suggest an increased risk of accidents secondary to a mTBI in the first 24 hours; therefore abstaining from driving during this time is crucial to promote driving safety for this population [4].

Despite the potential consequences of mTBI on driving, few guidelines for return to driving have been published. This is surprising when one considers that driving is such a complex and high risk activity regarding potential injury to both self and others, seemingly as important as return to playing sports, where several clinical practice guidelines [11] ensure that athletes return to their sport at the safest time [12]. Considering the lack of clinical guidelines and the problems raised for clinical practice concerning the return to driving following a mTBI, the objectives of the study were to (1) document the perception of driving abilities in individuals having sustained a mTBI who remain symptomatic, (2) analyze the difficulties reported by individuals with mTBI in relation to their impact on driving, and (3) explore whether individuals with mTBI use compensatory strategies to facilitate safe driving.

2. Methodology

2.1. Study Design. A qualitative research method was used to attain the objectives of this study.

2.2. Participants. Twenty-seven drivers with postconcussive symptoms secondary to a mTBI participated in this study. Participants were recruited from the McGill University Health Centre and Hôpital du Sacré-Coeur de Montréal, two major tertiary trauma centers in the Montreal area, Canada, as part of a larger study on mTBI conducted by our research team. Potential participants with postconcussive symptoms were identified by their physicians and informed of our study. Those interested in the study were then contacted by the researchers. The inclusion criteria were the following: (a) diagnosis of mTBI confirmed by an expert physician, (b) presence of symptoms (regardless of the severity), and (c) between 18 and 65 years of age. Exclusion criteria included (a) litigation issues related to the TBI, (b) presence or history of neurological or psychiatric problems, and (c) substance abuse. The study was approved, as part of a broader study on the measure of complex everyday activities in individuals with mTBI, by the ethics committees of the McGill University Health Centre and the Centre for Interdisciplinary Research in Rehabilitation of Greater Montreal. All the participants signed a consent form.

2.3. Data Collection. The information was collected from participants using semistructured interviews based on the ADL Profile [13] and open-ended questions specific to driving. The ADL Profile is an evaluation tool used to document independence in everyday activities [13] that has well-documented psychometric properties for use with individuals with TBI [13–15]. The ADL Profile consists of 20 activities (six activities linked to personal care, five linked to domestic activities, and nine linked to community activities) [13]. Two methods of evaluation are included in the test: a performance-based measure of everyday activities and a questionnaire administered as a semi-structured interview. In the present study, only the portion of the semi-structured interview regarding independence in community activities was used, that is, in our case, independence in driving and self-perceived difficulties and their impact on everyday activities. Open-ended questions specific to driving covered the strategies used to overcome their driving difficulties and whether they felt safe when driving. With the consent of participants, 17 of the 27 interviews were recorded. These interviews were fully transcribed and the verbatim analyzed. Written notes taken during the interviews were used to analyze the results of those participants who refused to

be video taped. Sociodemographic and clinical information were also recorded.

Participants also completed the following tests: the Post-Concussion Scale Revised (PCSR) [16, 17], the Beck Anxiety Inventory (BAI) [18], the Posttraumatic Stress Disorder Checklist (PCL-S)[19], the Fatigue Severity Scale (FSS) [20], and the Beck Depression Inventory, Second Edition (BDI-II) [21].

2.4. Data Analysis. Both quantitative and qualitative analyses were performed on the data. First, a qualitative analysis of the transcripts was completed using a content analysis [22]. All transcripts were read several times, highlighting the text that appeared most informative of the repercussions of mTBI on driving ability and writing keywords or phrases (*preliminary codes*) using the participants' words. After open coding of three or four transcripts, the preliminary codes were assigned to the pertinent excerpts related to the research questions for all remaining transcripts, and new concepts were added when data that did not fit into existing codes was encountered. Once all transcripts were coded, the data within a particular code was either regrouped or eliminated when codes were deemed nonpertinent. During the last stage, the theoretical model of car driving proposed by Michon [23] was used to complete the final analysis of the data and to frame the presentation of the results regarding the perception individuals with mTBI have of their driving difficulties and of the strategies they use to overcome them. Data analysis was completed by two researchers (C. Bottari and M-P. Lamothe) to ensure reliability of the coding and of the interpretation of the data. Second, a quantitative analysis was completed to obtain frequencies of observed themes over all participants and scores on the ADL Profile interview.

2.5. The Hierarchical Model of Task Performance in Car Driving. The Hierarchical Model of Task Performance in Car Driving [23, 24] is a theoretical model that is largely cited in the scientific literature related to driving [5, 25–28]. This model looks at decision making in three levels: *strategic* (planning), *tactical* (manoeuvring), and *operational* (control) level. The *strategic* level refers to decision making that occurs over long periods of time, prior to taking the wheel. It involves general planning of trip goals, route, modal choice and the evaluation of costs and risks associated with the trip. The *tactical* level of decision making occurs on the road and is influenced by environmental conditions. Here the driver exercises manoeuvre control which allows him or her to interact with the circumstances faced on the road. This level calls upon complex cognitive functions, flexibility, and awareness of the actual demands of the road. Manoeuvres can be adapted to the situation, for example, choice of speed, overtaking a vehicle, distance between vehicles, and so forth, and are derived from the original goals set in the *strategic* level. This level of decision making takes place much more rapidly than strategic decisions as they occur over the space of seconds. Finally, it is within the *operational* (basic skill) level that immediate reactions are required. At this level, the individuals use the baseline skills of steering, braking, and so forth to cope with threats or perceived danger. These decisions occur over the space of milliseconds and depend essentially on the speed of perception of stimuli and driving skills.

Table 1: Demographic and clinical profile of participants.

Characteristics	Frequency (%)
Total	27 (100)
Women	13 (48)
	Mean (SD)
Demographics	
Age	32,15 (10,35)
Years of education	14,39 (3,16)
Time after TBI (months)	14,59 (19,08)
Test results	
Postconcussion Symptom Scale Revised	40,89 (20,58)
Beck Anxiety Inventory	12,37 (7,79)
Posttraumatic Stress Disorder Checklist	39,04 (13,17)
Fatigue Severity Scale	38,93 (15,58)
Beck Depression Inventory, Second Edition	15,67 (10,34)

3. Results

Of the 27 participants with postconcussive symptoms who participated in the study, all were currently active drivers except for one individual who was not currently driving as he had not yet received medical permission to drive. The sociodemographic characteristics of the participants, as well as the time after mTBI, are summarized in Table 1. Time after injury varied between two weeks and six years. Sixty-seven percent had sustained a sport-related accident and 19% a motor vehicle accident. Fifty-one percent had returned to work or school full time and 26% remained on medical leave of absence. Fifty-six percent of participants reported moderate or severe postconcussive symptoms (PCSR score > 21), 56% fatigue problems (FSS score > 36), 30% moderate or severe depression (BDI score > 20), 15% moderate or severe anxiety (BAI score > 22), and 33% reported posttraumatic stress disorder (PCLS score > 44). The average time between the accident and the return to driving for all participants was 19.8 days, with a standard deviation of 20.8 days. For the participants who waited the longest, the delay (one or two months) was secondary to the medical advice they had received. Alternately, four participants clearly mentioned not having waited at all to return to driving; they therefore drove in the first 24 hours following their mTBI.

3.1. Difficulties and Consequences in Everyday Functioning. At the time of the interview with the ADL Profile, 25 of the 27 participants (93%) mentioned at least one difficulty that limited them in the realization of their everyday activities. The difficulties reported by the participants, as well as the number of participants who identified them as having an impact on their participation in their overall activities of daily living (ADL), are indicated in Table 2. It is important

Table 2: Difficulties mentioned by the participants that have an impact on overall everyday activities.

Difficulties	*n*	%
Fatigue	21	77.78
Concentration	20	74.07
Memory	17	62.96
Anger easily	15	55.56
Fear, anxiety	14	51.85
Difficulty doing more than one thing at a time	13	48.15
Vision	10	37.04
Organisation	7	25.93
Headache	6	22.22
Hearing	5	18.52
Dizziness	3	11.11
Pain	2	7.41
Spatial orientation	2	7.41

to note that only those likely to interfere specifically with driving, according to the prerequisite skills for driving identified by Rizzo and Kellison [3], are reported in this study. Among the most prevalent problems, "fatigue" was present in 78% of participants; "concentration problems" and "memory problems" present, respectively, in 74% and 63% of our sample. In a slightly smaller proportion, we found "anger easily" (56%), "anxiety" (52%), and "difficulty doing more than one thing at a time" (48%).

3.2. Analysis of Driving Difficulties according to Michon's Model (1979, 1985). Table 3 illustrates the difficulties reported by the participants concerning their driving, while Table 4 presents an analysis of perceived driving difficulties according to the affected level of decision making using Michon's model of car driving [23, 24]. Difficulties affecting the *operational* level included vision problems, physical pain, and loss of automatic driving reflexes. Other issues such as fatigue, delayed response, dizziness, and concentration problems influenced both the *operational* and *tactical* levels of decision making. In the current study, participants reported that headaches, anxiety, decreased anticipation, memory problems, spatial orientation problems, and irritability influenced their *tactical* level of decision making during driving. Finally, one participant reported difficulties at the *strategic* level. This individual reported never questioning whether his physical and mental states were optimal for safe driving prior to getting behind the wheel of his car.

3.3. Strategies to Facilitate Safe Driving. Twenty of the 27 drivers (74%) reported having developed certain strategies or changed certain behaviors to compensate for their difficulties when driving. For the most part, strategies were developed only after having attempted a return to previous driving habits and having then becoming aware of unexpected driving difficulties and subsequent unsafe driving behaviors. These strategies were classified according to Michon's model of car driving [23, 24], depending on the decision-making level to which they belonged (see Table 5).

At the *strategic* level, the most common strategy used ($n = 8$) was reducing the travel distance or time spent driving. Four participants reported self-assessing their current aptness to drive prior to driving and reducing or abstaining from driving in the evening. These strategies were aimed at overcoming significant fatigue, concentration problems or significant headaches that interfered with their ability to drive a car. To explain the use of avoidance as a strategy to adapt to severe fatigue problems, one participant said:

> *And then, well, if one day when I'm tired because the night before, I admit, I did things that made me very tired, well you know, I'll maybe just not take the car because I'll decide that I do not have to take it that day.*

The third strategic-level strategy (not driving at night) compensated for fatigue problems and also addressed visual difficulties as some participants reported an increased difficulty with their visual acuity, especially in the evening. One participant reported:

> *But I do not drive at night (…) because at night I have a… since my accident, I have a problem with my vision (…) it's like I see stars.*

At the *tactical* level, not conversing with other passengers while driving, changing drivers along the way, and taking breaks or naps along the way were identified as strategies by three participants. These strategies helped overcome problems related to fatigue and concentration. For example, in talking about avoiding having a conversation with passengers, one participant said:

> *When I speak, I am less… a little like everyone; I am not very efficient at driving.*

Additional tactical-level strategies mentioned by the participants included reducing one's speed, decreasing risk taking behaviours, increasing the space between vehicles, avoiding traffic, or limiting the frequency of lane changes. Participants who reported using these strategies tended to also report high levels of anxiety and slower reaction times.

> *When I drive, I am maybe 90%. The 10% I miss, it's about my reaction speed. But I drive slower than before (…) well, 25% more slowly. I compensate.*

Strategies specific to the *operational* level were not explicitly mentioned by the participants. However, it is important to understand that Michon's three-level model is hierarchical and that decisions made at the tactical and strategic levels compensate for problems at the operational level.

Table 3: Examples of difficulties reported by the participants and their perceived repercussions on driving.

Difficulties	Verbatim examples that characterize the difficulties reported by the participants when driving
Fatigue	*I become . . .more tired a lot faster than before. . .*(Participant no. 300)
Vision problems	*Because at night I have a. . .since my accident, I have a problem with vision. (. . .) it's like I see stars.* (Participant no. 325)
Slowed reactions	*When I drive, I am maybe at 90%. (I'm missing 10%). It's due to my reaction speed.* (Participant no. 327)
Headaches	Evaluator*: You did not drive today because of your headache?* Participant*: Yes, I do not trust myself.* (Participant no. 314)
Anxiety	*I'm more stressed now about driving than before.* (Participant no. 323)
Loss of driving reflexes	*I loss the reflex of turning my head. (. . .) To check my blind spot, make the. . . good, you know: ≪OK, it's necessary that I check my blind spot. . . here. . . here. . . go!≫* (Participant no. 329)
Decreased anticipation	*When driving, taking an exit, often I take it at the last minute. People must find me stupid.* (Participant no. 308)
Dizziness	*It was more difficult, even at the beginning. I did not drive at all because I was really dizzy during the first days.* (Participant no. 334)
Memory problems	*Sometimes I just simply forget where I'm heading.* (Participant no. 311)
Concentration problems	*It's like I forgot some of what happened on the road. Like I was elsewhere, but it was as if I was not really there and I do not remember what happened.* (Participant no. 318)
Spatial orientation problems	*You know, remember where I turned, I am not able to. . . like my visual memory of places, I do not have it anymore. In effect, it's my sense of orientation.* (Participant no. 323)
Angers easily	*The traffic, I always had a hard time with that and it did not get better with time but what I realized, is that I am more irritable since I had this accident.* (Participant no. 324)
Pain	*I have a lot of fatigue when driving long distances, with my leg. At a given moment, I have to remove my leg and change to drive with my left leg because I have fatigue and pain in my leg.* (Participant no. 336)
Difficulty remaining in driving lane	*(. . .) I keep looking behind me because I have a feeling that someone is going to hit me from behind, just like in my accident. . .I avoided driving on highways. . . I had a lot of difficulty staying in my lane. But after, it did not really get better like it was before.* (Participant no. 328)
Questions his/her ability to drive	*I do not really ask myself whether I'm able to drive or not.* (Participant no. 311)

Table 4: Analysis of perceived driving difficulties according to the affected level of decision making based on Michon's model of car driving (1979, 1985).

Level of decision according to Michon's Model (1979)	Strategic	Tactical	Operational
Difficulties			
Fatigue		X	X
Vision problems			X
Slowed reactions		X	X
Headaches		X	
Anxiety		X	
Loss of driving reflexes			X
Decreased anticipation		X	
Dizziness		X	X
Memory problems		X	
Concentration problems		X	X
Spatial orientation problems		X	
Angers easily		X	
Pain			X
Difficulty staying in his own driving lane			X
Questions his/her ability to drive	X		

Table 5: Compensation strategies identified by participants.

Level of decision according to Michon's Model (1979)	Examples of compensatory strategies	*n*	%
Strategic-level strategies (before taking the wheel)	Avoiding driving due to significant fatigue or headaches	4	14.81
	Reducing the distance travelled or time of continuous driving	8	29.63
	Reduction/abstaining from driving at night	4	14.81
	Avoiding rush hour	2	7.41
	Using a GPS	2	7.41
Tactical-level strategies (on the road)	Avoiding conversations while driving	2	7.41
	Taking breaks while driving or naps before driving	2	7.41
	Changing drivers along the way	3	11.11
	Reducing one's speed	5	18.52
	Being more careful, taking fewer risks	4	14.81
	Minimizing lane changes	1	3.70
	Allowing more distance between vehicles	1	3.70

Table 6: Perceived level of independence of participants for driving and the use of compensation strategies.

ADL profile scoring	*n*	%	Compensation strategies
3: independent without difficulty	10	37.04	Without: 4 (40%) With: 6 (60%)
2: independent with difficulty	15	55.56	Without: 2 (13, 33%) With: 13 (86,67%)
1v/p: verbal and/or physical assistance	1	3.70	With: 1 (100%)
0: Dependant	1	3.70	N/A (does not have medical authorization to drive)

3.4. Level of Independence in Driving and Strategies That May Optimize Driving a Car Safely. During the interview, participants were asked to give their perception of their current level of independence in relation to driving, based on the four-point ordinal scale of independence of the ADL Profile (dependence, requires verbal and/or physical assistance, independence with difficulty, independence) (see Table 6). Ten of the 27 participants said they were independent, 15 said they were independent with difficulty, one needed physical or verbal assistance, and one reported being dependent.

Even though 10 participants reported being able to drive independently, five of these mentioned using strategies to overcome various difficulties when driving. Of these five, two reported being "more anxious while driving since their accident"; one was now overall "more cautious" when driving and the other "avoided driving in traffic all together." Still within the group of five who used strategies, two had reduced their time of continuous driving due to fatigue and two used somewhat risky strategies that included driving with his/her left leg when pain in the right leg was too severe and stopping on the side of the road to have a nap when overcome by fatigue.

For the 15 participants who perceived themselves as being independent with difficulty for driving, all but two reported using at least one strategy to compensate for difficulties that influenced their driving. One participant failed to put necessary strategies into place despite the presence of significant difficulties affecting his driving, that is, dizziness and important fatigue problems. This participant mentioned not questioning his physical or mental state prior to taking the wheel.

One participant reported needing assistance to drive; he stated that he only drives in more suburban or rural areas and asks his partner to drive for him in the city. One participant reported being dependent for driving, as he had not yet received the medical authorization to return to driving due to his persistent symptoms which were at risk of interfering with his driving.

4. Discussion

This study was one of the first to document the impact of mTBI on driving based on the perception of individuals with postconcussive symptoms at an average of 15 months after injury. Numerous postconcussive symptoms were clearly perceived by participants as having a negative impact on their driving ability (e.g., fatigue, difficulty concentrating or problems with memory, vision problems or hearing problems, dizziness, headaches, fear, and/or anxiety, etc.). However, participants tended to be aware of their difficulties and had developed strategies over time, and likely by trial and error (though this is not the safest approach), to compensate, and this helped them feel safer while driving. Indeed, awareness of one's own ability is a crucial skill for driving and is regarded as an important indicator of safe or unsafe driving [29].

4.1. Strategies Utilized to Facilitate Safer Driving. Individuals may fail to anticipate the repercussions of mTBI on their driving ability, resume their activities as prior to their accident, and only realize the need to modify their habits once they are confronted with driving difficulties [30]. Though this manner of resuming everyday activities may have little risk for certain everyday activities secondary to a mTBI, this adaptation period may carry greater risks for driving.

Individuals having sustained a mTBI tend to be more aware of their deficits than individuals with moderate or severe TBI [30]. According to Lundqvist and Alinder [5], individuals who are aware of their cognitive capacity are able to cope with cognitive impairments at the tactical level of decision making, as was seen in the present study. Awareness of deficits helps with developing compensatory strategies. In the present study, a majority of participants reported using at least one compensatory strategy to overcome their driving difficulties. Moreover, a person who is aware of having difficulties in the *operational* and *tactical* levels of driving will take appropriate decisions at the *strategic* level [5]. For example, participants in this study made *strategic*-level decisions to avoid night-time driving or reduce the distance they travel. Examples of *tactical*-level decisions were to slow down, minimize lane changes, and allow for a larger distance between vehicles. Such use of adaptive strategies at the *tactical* and *strategic* levels positively influences *operational* level decisions and can reduce accident risk [25].

At present, persons with mTBI's risk of minor accidents are likely greatest during the "exploration period" that occurs when first resuming driving after the accident. At this time, the person may not yet be aware of the impact of the mTBI on his driving ability and hence not have identified the need to modify his driving habits in consideration of his new condition. Therefore, the use of preventive interventions such as education programs on the potential impact of mTBI on driving and on potential strategies to reduce risk of accidents provided early on after the injury would merit further exploration.

4.2. Return to Driving following mTBI. Information concerning the delay between the occurrence of mTBI and the return to driving is not always available (both in general and in our participants), but the disparity in delays in the participants from this study deserves attention. In our study, at least four participants resumed driving within the first 24 hours of their mTBI. According to a study by Preece et al. [4], persons who have had a mTBI and are within the first 24 hours after mTBI were significantly slower to react in dangerous situations than a control group. The participants with mTBI in the present study were therefore potentially at risk when driving a car during the first 24 hours following their injury. Even though there are strict guidelines for return to play subsequent to mTBI, owing to the risks of re-injury and the aggravation of the symptomotology in athletes [31, 32], similar guidelines have not been well established for return to driving. Our results would suggest that persons having sustained, or suspected of having sustained, a mTBI should be informed that cognitive overload, which may occur secondary to a very rapid return to driving, can lead to an aggravation or a relapse of postconcussive symptoms even in individuals with mTBI who reported few symptoms [33]. Considering the safety concerns for the person with mTBI and society at large, strict guidelines to ensure the safe return to driving would need to be established with this population.

4.3. Limits to the Study. The aim of our study was to document the perception of driving abilities in individuals having sustained a mTBI who remain symptomatic. Our recruitment strategy specifically targeted symptomatic individuals which limits the generalization of these findings to non symptomatic individuals. The vast majority of individuals with mTBI are however likely to experience postconcussive symptoms within the first few days or weeks following their injury and equally likely to resume driving. Hence, results of the present study may generalize to acute mTBI and provide helpful insights to begin to improve clinical interventions in relation to mTBI and driving.

5. Conclusion

The principle objective of this study was to investigate the impact of mTBI on the resumption of driving from the perception of symptomatic and currently active mTBI drivers. It aimed to better understand the driving difficulties encountered by this population and the strategies used to compensate for the presence of these difficulties, the latter being an element of crucial importance to driving safety. It therefore provides a unique perspective to research related to the impact of mTBI on driving. Our results have demonstrated that persons with postconcussive symptoms report difficulties that slightly reduce their perceived level of independence in driving, as well as potentially increase the risk of minor driving accidents in individuals who fail to modify their habits to adapt to the presence of postconcussive symptoms. Nevertheless, they do report, for the most part, having developed strategies, over time and by trial and error (which is not the safest approach) for overcoming these difficulties. The risk of accidents is likely to be more elevated during the period where they try to resume driving while their strategies are not yet in place. We think that effective support of this clientele, by taking preventive actions and promoting positive driving strategies and guidelines for the safe resumption of driving after injury, could reduce the temporary safety risk. Additionally, further investigation into the consequences of mTBI on driving would be warranted to determine more effective methods of detecting overall risk when driving and readiness to resume driving. Knowledge translation strategies would also need to be identified to increase health care professionals' awareness of the potential consequences of mTBI on driving ability and to promote positive strategies and guidelines for the safe resumption of driving after injury.

Acknowledgments

This study was supported by the Canadian Institutes of Health Research (postdoctoral fellowships to C. Bottari and N. Gosselin). The authors are grateful to Dr. Diane Diorio, Mitra Feyz, Dr. Simon Tinawi, Elaine De Guise, Isabel Roy, Dr. Gilles Lavigne, and Kristina Martinu for their help to recruit subjects.

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Hindawi Publishing Corporation
Rehabilitation Research and Practice
Volume 2012, Article ID 625621, 6 pages
doi:10.1155/2012/625621

Research Article

Working with Mild Traumatic Brain Injury: Voices from the Field

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Received 4 November 2011; Accepted 16 January 2012

Academic Editor: Gina P. Armstrong

Mild traumatic brain injury (mTBI), also known as concussion, is an emerging public health issue in the United States. The estimated annual 1.2 million individuals who sustain this injury face a range of cognitive, psychological, and physical consequences for which rehabilitation protocols are being developed and implemented. On the frontlines of this developing area of rehabilitation work are professionals in a range of therapeutic settings whose practice wisdom has yet to be shared in the professional literature. This qualitative study aimed to fill this gap by exploring the experiences and insights of rehabilitation professionals serving mTBI patients in outpatient, civilian settings. An analysis of the qualitative data revealed five themes common in mTBI work, providing an in-depth look at this often challenging field of rehabilitation.

1. Introduction

In recent years, mild traumatic brain injury (mTBI) has emerged as a leading public health concern. Eighty-five percent of the 1.5 million traumatic brain injuries sustained by Americans every year are considered "mild" [1]. Mild traumatic brain injury, commonly known as concussion, is a serious neurologic condition that can have long-term cognitive, physical, emotional, and social consequences. Once believed to be a virtual rite of passage in childhood and sports, concussions are now largely understood by medical professionals to be a traumatic form of brain injury that requires careful diagnosis, management, and followup [2].

A traumatic brain injury (TBI) occurs when the brain experiences neurological or neuropsychological impairment as a result of an external trauma. This typically occurs when the head comes into direct contact with an object in situations commonly understood as "hitting your head" or "being hit in the head," such as car accidents, contact sports, or domestic violence assaults. A TBI can also occur during the rapid acceleration or deceleration associated with whiplash in which movement of the brain against the inside of the skull causes trauma [3]. Perhaps the least understood mechanism by which a mild TBI can occur is a blast injury, typical in modern military combat and other conflicts. Even without blunt force or acceleration/deceleration, trauma to the brain can arise from the sheer energy of the supersonic waves created from blasts. Blast injuries are so prevalent among service members and veterans that mTBI has been named the "signature injury" of the conflicts in Iraq and Afghanistan [4].

A diagnosis of traumatic brain injury requires a classification of severe, moderate, or mild based on an evaluation of consciousness, amnesia and a Glasgow Coma score. Diagnostic technology, including CT scans and MRIs, can be effective in the diagnosis of severe and moderate injuries. These technologies, however, are not able to reliably detect mild injuries, making diagnosis of mTBI far more difficult [5]. In large part, diagnoses of mTBI are based on patient self-report. Kennedy and colleagues [6] found that although emergency room and outpatient medical professionals understood mTBI and its complications, they typically failed

to assess for sequelae or make appropriate referrals. There have been significant advances, however, in diagnosis with the use of baseline testing. Many high school and college sports programs are requiring certain student athletes to get baseline neurological testing as a way to help diagnose and assess concussion [7]. The symptoms of mTBI can be cognitive, psychological, and physical. Cognitive symptoms include decreased processing speed, confusion or impaired cognition, problems with attention, impaired judgment, and amnesia, or other problems with memory, especially short-term memory. Psychological symptoms include irritability, anxiety, depression, and a change in personality. Physical symptoms include headaches, dizziness, diplopia, sensitivity to light or noise, tinnitus, and insomnia. These impairments often undermine academic and work performance, impacting employment, education, and relationships [8]. Generally symptoms resolve within a few weeks or months, yet for some, symptoms persist for long periods of time and can result in permanent disability [9].

Despite increases in awareness and concomitant increases in diagnosis, the development of evidence-based treatment and rehabilitation protocols for mTBI has lagged behind. A review of current practices [10] suggests that mTBI is largely treated using models developed for moderate and severe injuries, "despite strong evidence that they are distinct clinically and epidemiologically" and despite the fact that "rehabilitation designed for moderate and severe TBI has not been effective for mild TBI" [10, pp. 1590-1]. In 2007, the US Office of the Surgeon General and the Army Medical Department took an important step in addressing these treatment deficits with the establishment of a task force dedicated to developing best practices specific to treating mTBI in injured service members and veterans. Recommendations for best practices in both occupational and physical therapy have since been published for this population [11, 12].

Rehabilitation professionals who have direct experience with individuals diagnosed with mTBI have a wealth of knowledge of this injury from the hours they have spent with their patients. Despite having the most robust understanding of the current state of mTBI rehabilitation work, the experiences of mTBI professionals have yet to be included in published research. This qualitative study aims to fill this gap by giving voice to the practice experience of rehabilitation professionals working in mTBI.

2. Methods

This study was designed to gather information from rehabilitation professionals who had direct experience working specifically with patients diagnosed with mTBI. A participant recruitment letter, informed consent document, and interview protocol were developed and approved by West Chester University's Institutional Review Board prior to beginning data collection. Participants for the study were selected using nonrandom, snowball sampling techniques based on contacts with brain injury professionals. Participants were included in the study if they reported that they spent at least one quarter of their time with individuals diagnosed with mTBI.

In the spring of 2010, we conducted in-depth, semistructured interviews; either in person or via telephone, which lasted between 45 and 90 minutes each. Following a systematic process of informed and voluntary consent, we asked participants to describe their work with mTBI patients, beginning with the inquiry: "I'd like to hear about your experiences working with people with mild traumatic brain injuries." To facilitate the data analysis process, all interviews were digitally recorded using either a handheld digital recorder or a secure, web-based conferencing program. Recordings were transcribed by a professional organization using encryption software.

Fifteen licensed professionals employed in civilian (nonmilitary), outpatient brain injury rehabilitation positions, including physical, vestibular, cognitive, occupational, speech, and neuropsychology, were interviewed. Participants included 4 men and 11 women who worked in rehabilitation settings in the Mid-Atlantic region of the United States, with an average of 14 years in the field, ranging from 2 to 23 years of experience. Many worked in settings that require them to see a range of patients, although none spent less than one quarter of their time with individuals diagnosed with mTBI.

The qualitative data obtained in the interviews were analyzed using a systematic process of analysis, coding, and discussion. This study was based in grounded theory techniques [13] as a way to explore the lived experiences of mTBI rehabilitation professionals. Distinct from theoretical approaches such as ethnography or phenomenology, a grounded theory approach seeks "to build theory rather than test it" [13, p. 13]. As such, our coding scheme had not been developed prior to conducting the interviews. We were not seeking to test or confirm certain theories about mTBI work, but rather to develop them. Although we had impressions about the data given our experiences in the interviews, we resisted labeling and categorizing until interviews were transcribed and ready for analysis. We developed an initial coding scheme through an inductive process of analysis using three interviews. Subsequent interviews were coded using the scheme by at least two people through a process of constant comparison. When differences arose in the coding, we engaged in discussion until a consensus could be reached. The final analysis resulted in five major themes, pulled from eight that initially emerged.

The design of this study creates specific limitations. A snowball sampling technique was used to identify individuals specifically working with mTBI patients. This necessarily means the sample is not representative of rehabilitation professionals in general. Further, the research participants in this study worked with patients who had been referred for rehabilitation services; thus, their experience is limited to a group of patients whose characteristics and experiences likely differ from those individuals who are also injured but who do not receive rehabilitation services. While we might hypothesize that individuals with mTBI who are not receiving rehabilitation services are generally less symptomatic, it is possible that the severity of their impairments has interfered with the process of obtaining treatment.

3. Results and Discussion

Rehabilitation specialists reflected on their experiences working with the mTBI population from various perspectives. They spoke about their own first-hand experiences with this type of rehabilitation work, giving examples of the course of treatment with specific patients. Often rehabilitation professionals included their patients' words as a way of conveying the more subtle, complex elements of the work. Sometimes they quoted specific patients, but more frequently they quoted what seemed to be an "aggregate voice" of many patients. This plurivocal, narrative style [14] added to the richness of our findings, allowing for multiple voices to come through.

Five primary themes emerged from our analysis of the qualitative interview data: prevalence of misdiagnosis and misinformation, unpredictability of prognosis, complexity of symptoms, impaired self-awareness, and invisible nature of injury. The themes are described below in the order that they typically emerged in the interviews and often how they emerged in the practitioner-patient relationship.

3.1. Prevalence of Misdiagnosis and Misinformation. One prominent theme to emerge from this study was the prevalence of misdiagnosis and misinformation that occurs with mTBI. Research participants reported that their patients often had earlier experience with misdiagnosis prior to their rehabilitation. Largely, this was a result of the fact that negative results on medical imaging such as CT scans and MRIs are typically interpreted as sufficient reason to rule out a TBI of any kind despite the fact that diagnostic technology is not yet sensitive enough to pick up these mild injuries [5].

> *With mild traumatic brain injury, we really do not have a medical test that's accurate enough or detailed enough to really tell you which axons and dendrites might be sheared or which cell nuclei might be affected by the impact.*
>
> *My patients say, "But, my MRI looks normal. They keep telling me my MRI is normal."*
>
> *They hear, "Based on testing, you do not have a bleed. The CT scan and the MRI is negative and so you should be able to return to work or school but just take it easy."*

The lack of clear diagnostic tests that corroborate patients' symptomology contributes to feelings of frustration and misunderstanding. Rehabilitation professionals reported consistently hearing mTBI patients ask "Why am I having so much trouble if all my tests are normal?" In the face of symptoms that interfere with daily functioning, negative test results create a remarkably incongruous scenario, not only for patients but also for rehabilitation professionals.

Participants reported that their patients were also misinformed sometimes even by primary care physicians who made reassurances that mild brain injury is an uncomplicated event that resolves easily and quickly.

> *Family practitioners typically will tell them, "Oh, it's four to six weeks. You'll be better. It'll be fine. It's all good. Just go home, rest, do not push yourself."*
>
> *They go to a doctor, and they do not get as much support. "You'll be fine. You look great. Your CAT scans are normal. You know, give it a month. It'll be fine."*

Although important, sleep and rest do not necessarily produce the expected results in the type of atypical fatigue common in mTBI [15]. When efforts to "rest and take it easy" fail to produce expected recovery, even those who pursued further medical help were at times rebuffed.

> *He was sent to a psychologist who was basically saying, "You need to just push yourself through the situation. You cannot give in to the anxiety. If you push yourself through and get to the other side, you'll see that you can survive it."*
>
> *So many of our patients have been transferred from one physician to another and they haven't really gotten a solid answer.*

Research participants were acutely aware that mTBI patients who had been referred for rehabilitation services generally need more than just rest or a motivational shove. Too often, they found that their patients were left to manage without proactive medical care to provide accurate information about the complexity or anticipated duration of symptoms related to mTBI.

3.2. Unpredictability of Prognosis. Participants reported being routinely asked to give their patients a prognosis. Once patients find mTBI professionals who understand their injuries and are sympathetic to their often-complicated experiences of seeking support, they are eager to get a reliable and predictable prognosis. In part, patient demand for a clear prognosis stems from the sociocultural expectation that doctors not only diagnose but also predict outcomes. Increasingly, medical professionals are under pressure to cure ailments in a society that resists acceptance of death, pain, and suffering [16]. Participants expressed frustration with the inability to satisfy their patients' understandable desire for a clear and simple prognosis.

> *The hardest thing—a lot of people want an end date. They want an answer. They want—"When am I going to be back? When am I going to be healed? When am I going to be better?"*
>
> *"You'll be better in three months." You know, you cannot give that promise.*
>
> *I think the most challenging thing is that nobody really has a crystal ball, your ability to heal is individual and it's based on so many factors.*

Although patients want to know when they will feel better or when their situation will significantly improve, the ability to predict the duration and even severity of symptomology with a diagnosis of mTBI can be difficult [9].

> *You can have someone with a concussion rebound very quickly and get back to school or work without too much difficulty or you can have lingering symptoms that are very debilitating but they still have the same diagnosis.*

Understandably, many mTBI patients expect the prognosis to be consistent with their conceptualization of a mild diagnosis. As rehabilitation professionals know well, however, a diagnosis of mTBI does not always provide a clear treatment strategy in which quick and significant improvement is definite. Rehabilitation professionals reported needing to manage patients' disappointments given their expectations of recovery from a "mild" injury.

> *We have to help people manage expectations. We want to be encouraging at the same time not be over-encouraging, so you do not want them to look through rose-colored glasses but you do not want to dash their hopes, either.*

> *Across the board, patients do not realize that the side effects can be something that will be long lasting, and even permanent.*

The diagnostic use of the term mild can bear little relationship to the duration of symptoms or the time it takes for patients to actually feel symptom-free. The challenge for rehabilitation professionals is to keep patients motivated in the face of a complex and unpredictable injury.

3.3. Complexity of Symptoms. The complexity of symptoms associated with mTBI is another prominent theme that emerged from the analysis of the interviews with rehabilitation professionals. Respondents reported that this complexity adds to the challenge of working with mTBI survivors.

> *It's challenging, and it takes a lot of time to make sure that you're managing all of their symptoms, because they have so many, and trying to make sure that you're addressing everything.*

> *Even though the primary diagnosis is maybe a C5 spinal cord injury, the real problem is that they cannot remember anything and their attention is terrible, and they have all these behavioral problems.*

> *You can see personality changes, memory changes, social changes, different cognitive things—like initiation, planning, organization.*

> *All these realms are affected—sleeping, and sometimes vision, and they're dizzy, and they do not have balance.*

Rehabilitation professionals are not able to tell patients exactly what symptoms to expect because no two cases of mTBI present exactly the same. There are many different functional areas that can be affected by mTBI, including cognition, balance, vision, and emotional regulation [17].

The rehabilitation process can be further complicated by functional impairment, such as lack of attention, organizational deficits, or behavior changes. These types of symptoms can make it difficult for patients to be fully engaged in their own treatment. As one participant explained, "*Behavior becomes an issue, which interferes with their therapy*." Persistent dizziness, double-vision, or migraines due to vestibular and neurological symptoms can also make it difficult for patients to complete, or even participate in, the therapeutic activities of any given appointment [12]. Explains one rehabilitation therapist, "*The exercises we're making them do make them dizzy.*" The nature of symptoms and the subsequent treatment complications are mutually exacerbating which can contribute to the complexity of rehabilitation with this population.

3.4. Impaired Self-Awareness. It is not uncommon for survivors of mTBI to be unaware of their own symptoms or the extent of their own deficits [18, 19]. Participants in this study reported that their patients often do not recognize the exact nature of their condition.

> *I do not think some of them realize that they have changed—either the personality or their anger or their memory. They do not realize that it is all normal—it is all tied together.*

> *Maybe some of them thought, "Maybe my balance is not that bad," but then when we go through all the testing, it is bad …. And then as we talk with them, we might find more cognitive problems that they're having.*

> *I think some of them do not understand the limits that they have. They look fine so they try to push, push, push.*

> *They know they're not feeling good, but it's almost like until you start doing the evaluation and start talking to them about their symptoms, they do not fully really understand what they're experiencing.*

Participants also reported that some of their patients do not recognize their limitations or inabilities as symptoms. Other patients know of their symptoms but do not recognize the pervasiveness of the functional impairment.

> *Some say, "I do not understand. I was able to do all of this before. I have all these other abilities. But it's still so hard for me to initiate getting out of bed in the morning or putting a plan together for my day."*

> *They did not understand that they'd be facing so many challenges across a multitude of fronts.*

Because the preexisting misunderstandings that patients have about concussion are often coupled with impaired appreciation of deficits, working with mTBI patients can be both challenging and frustrating.

This lack of self-awareness is likely also a function of denial and minimization responses that are common and even vital in traumatic situations. Participants reported patients' resistance to acknowledging their deficits.

> *They ask, "Why is this so hard? That should not be hard. I should be able to do that."*

> *People kind of get back to the workplace and they do not want to come out and say, "I cannot handle all this right now. I'm not the same person as I was."*

> *They realize how bad they feel when they try to get back to their regular routine; when they try to go back to work.*

Though initially useful as a temporary coping mechanism in the face of a traumatic event, downplaying functional limitations that result from mTBI can make it difficult for practitioners to fully address their patients' rehabilitation needs.

3.5. Invisible Nature of Injury. A lack of reliable diagnostic technology, coupled with the invisible nature of mTBI, places patients at risk of experiencing suspicion of their condition, much like any "unseen" injury or disease. The TBI professionals in this study reported that their patients often experience suspicion from family, friends, employers, and even medical professionals about the extent of their injury.

> *I think because mild traumatic brain injury individuals look completely normal, even their family and their friends say. "What's wrong with them? Why cannot they do the things they did before?"*

> *People will be told by their doctors, their spouses, their family, "It's all in your head."*

> *They do not want to say anything because they might think that they're kind of making it up.*

The invisible nature of this injury is specifically difficult in employment settings. Participants shared multiple stories of patients who had experiences with employers and coworkers who were suspicious of their injuries.

> *One patient told me that people thought he was just trying to make a buck by being on a worker's comp.*

> *A lot of bosses, they brush it off, like, "You're walking, you're talking, you're good."*

> *They think that the person is being lazy, that the person's not motivated, that they purposefully are not being productive.*

> *A lot of times, I have patients who just muddle through because they're afraid that they'll lose their job, or their boss won't understand.*

Suspicion of invisible conditions, while not uncommon, is particularly challenging with an injury like mTBI that is so variable and unpredictable, even for medical professionals. The recent and multifaceted surge in advocacy and awareness raising in sports, media, and the military, however, promises to shift this trend, if slowly.

4. Conclusions

Rehabilitation with mTBI is complex and challenging work that requires professionals to address a range of psychosocial issues faced by their patients. Individuals with mTBI often had prior experience with misdiagnosis and misinformation and turned to their rehabilitation specialists for far more than standard rehabilitation services. Many sought clarification about their complex symptomology with the hope of also getting insight on a prognosis. Although unaware, at times, of their own limitations resulting from the trauma that was sustained, many patients were keenly aware of how the invisible nature of mTBI caused others to question the legitimacy of their injury.

This study suggests a need for further research in this area. It seems evident that mTBI rehabilitations is unexpectedly challenging for both workers and patients given the expectations associated with a "mild" injury. More information is needed about the emotional energy required to manage a caseload of mTBI patients, with specific attention to the type of training that rehabilitation professionals need to do this work both effectively and ethically. Lastly, although the trauma of mTBI refers to the external nature of the source of injury, this study suggests there may also be a traumatic element in the rehabilitation work as frontline professionals vicariously experience their patients' challenging and complex recoveries.

Acknowledgments

This research project was funded through the *Faculty Grants Development* program at West Chester University of Pennsylvania and would not have been possible without the excellent research assistance of Cathy Allen Plaisted. The authors would also like to thank S. Walker, J. Martin, L. Alexander, and S. Nath for their support.

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