INTRODUCTORY CHAPTER

CONCUSSION IN ATHLETICS: ONGOING CONTROVERSY

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Abstract: Multiple traumas to the brain are the most common type of catastrophic injury and a leading cause of death in athletes. Multiple brain injuries may occur as the long-term disabilities resulting from a single mild traumatic brain injury (MTBI, generally known as concussion) are often overlooked and the most obvious clinical symptoms appear to resolve rapidly. One of the reasons of controversy about concussion is that most previous research has: a) failed to provide the pre-injury status of MBTI subjects which may lead to misdiagnosis following a single brain injury of the persistent or new neurological and behavioral deficits; b) focused primarily on transient deficits after single MTBI, and failed to examine for long-term deficits and multiple MTBI; c) focused primarily on cognitive or behavioral sequelae of MTBI in isolation; and d) failed to predict athletes at risk for traumatic brain injury. It is necessary to examine for both transient and long-term behavioral, sensory-motor, cognitive, and underlying neural mechanisms that are interactively affected by MTBI. A multidisciplinary approach using advanced technologies and assessment tools may dramatically enhance our understanding of this most puzzling neurological disorder facing the sport medicine world today. This is a major objective of this chapter and the whole book at least in part to resolve existing controversies about concussion.

Keywords: Injury; Concussion; Collegiate coaches; EEG and Postural stability.

1. INTRODUCTION

Over the past decade, the scientific information on traumatic brain injury has increased considerably. A number of models, theories and hypotheses of traumatic brain injury have been elaborated (see Shaw, 2002 for review). For example, using the search engine *PubMed* (National Library of Medicine) for the term "brain injury" there were 1990 articles available between the years of 1994-2003, compared to 930 for the years 1966-1993. Despite dramatic advances in this field of medicine, traumatic brain injury, including the mild

traumatic brain injury (MTBI), commonly known as a concussion, is still one of the most puzzling neurological disorders and least understood injuries facing the sport medicine world today (Walker, 1994; Cantu, 2003). Definitions of concussion are almost always qualified by the statement that loss of consciousness can occur in the absence of any gross damage or injury visible by light microscopy to the brain (Shaw, 2002). According to a recent *NIH Consensus Statement*, mild traumatic brain injury is an evolving dynamic process that involves multiple interrelated components exerting primary and secondary effects at the level of individual nerve cells (neuron), the level of connected networks of such neurons (neural networks), and the level of human thoughts or cognition (NIH, 1998).

The need for multidisciplinary research on mild brain injury arises from recent evidence identifying long-lasting residual disabilities that are often overlooked using current research methods. The notion of transient and rapid symptoms resolution is misleading since symptoms resolution is not indicative of injury resolution. There are no two traumatic brain injuries alike in mechanism, symptomology, or symptoms resolution. Most grading scales are based on loss of consciousness (LOC), and post-traumatic amnesia, both of which occur infrequently in MTBI (Guskiewick et al. 2001, Guskiewick, 2001). There is still no agreement upon diagnosis (Christopher & Amann, 2000) and there is no known treatment for this injury besides the passage of time. LOC for instance, occurs in only 8% of concussion cases (Oliaro et al., 2001). Overall, recent research has shown the many shortcomings of current MTBI assessments rating scales (Maddocks & Saling, 1996; Wojtys et al., 1999; Guskiewicz et al., 2001), neuropsychological assessments (Hoffman et al., 1995; Randolph, 2001; Shaw, 2002; Warden et al., 2001) and brain imaging techniques (CT, conventional MRI and EEG, Thatcher et al., 1989, 1998, 2001; Barth et al., 2001; Guskiewicz, 2001; Kushner, 1998; Shaw, 2002).

The clinical significance for further research on mild traumatic brain injury stems from the fact that injuries to the brain are the most common cause of death in athletes (Mueller & Cantu, 1990). It has been estimated that in high school football alone, there are more than 250,000 incidents of mild traumatic brain injury each season, which translates into approximately 20% of all boys who participate in this sport (LeBlanc, 1994, 1999). It is conventional wisdom that athletes with uncomplicated and single mild traumatic brain injuries experience rapid resolution of symptoms within 1-6 weeks after the incident with minimal prolonged sequelae (Echemendia et al., 2001; Lowell et al., 2003; Macciocchi et al., 1996; Maddocks & Saling, 1996). However, there is a growing body of knowledge indicating long-term disabilities that may persist up to 10 years post injury. Recent brain imaging studies (MRS, magnetic resonance spectroscopy) have clearly demonstrated the signs of cellular damage and diffuse axonal injury in subjects suffering from MTBI, not previously recognized by conventional imaging (Garnett et

al., 2000). It is important to stress that progressive neuronal loss in these subjects, as evidenced by abnormal brain metabolites, may persist up to 35 days post-injury. Therefore, athletes who prematurely return to play are highly susceptible to future and often more severe brain injuries. In fact, concussed athletes often experience a second TBI within one year post injury. Every athlete with a history of a single MTBI who returns to competition upon symptoms resolution still has a risk of developing a post-concussive syndrome (Cantu & Roy, 1995; Cantu, 2003; Kushner, 1998; Randolph, 2001), a syndrome with potentially fatal consequences (Barth et al., 2001).

Post-concussive syndrome (PCS) is described as the emergence and variable persistence of a cluster of symptoms following an episode of concussion, including, but not limited to, impaired cognitive functions such as attention, concentration, memory and information processing, irritability, depression, headache, disturbance of sleep (Hugenholtz et al., 1988; Thatcher et al., 1989; Macciocchi et al., 1996; Wojtys et al., 1999; Barth et al., 2001; Powell, 2001), nausea and emotional problems (Wright, 1998). Other signs of PCS are disorientation in space, impaired balance and postural control (Guskiewicz, 2001), altered sensation, photophobia, lack of motor coordination (Slobounov et al., 2002d) and slowed motor responses (Goldberg, 1988). It is not known, however, how these symptoms relate to damage in specific brain structures or brain pathways (Macciocchi et al., 1996), thus making accurate diagnosis based on these criteria almost impossible. Symptoms may resolve due to the brain's amazing plasticity (Hallett, 2001).

Humans are able to compensate for mild neuronal loss because of redundancies in the brain structures that allow reallocation of resources such that undamaged pathways and neurons are used to perform cognitive and motor tasks. This functional reserve gives the appearance that the subject has returned to pre-injury health while in actuality the injury is still present (Randolph, 2001). In this context, Thatcher (1997, 2001) was able to detect EEG residual abnormalities in MTBI patients up to eight years post injury. This may also increase the risk of *second impact syndrome* and multiple concussions in athletes who return to play based solely on symptom resolution criteria (Barth et al., 2001; Kushner, 2001; Randolph, 2001).

2. NEURAL BASIS OF COGNITIVE DISABILITIES IN MTBI

There is a considerable debate in the literature regarding the extent to which mild traumatic brain injury results in permanent neurological damage (Levin et al., 1987; Johnston et al., 2001), psychological distress (Lishman, 1988) or a combination of both (McClelland et al., 1994; Bryant & Harvey,

1999). Lishman's (1988) review of the literature suggested that physiological factors contributed mainly to the onset of the MTBI while psychological factors contributed to the duration of its symptoms. As a result, causation of MTBI remains unclear because objective anatomic pathology is rare and the interaction among cognitive, behavioral and emotional factors can produce enormous subjective symptoms in an unspecified manner (Goldberg, 1988).

To-date, a growing body of neuroimaging studies in normal subjects has documented involvement of the fronto-parietal network in spatial attentional modulations during object recognition or discrimination of cognitive tasks (Buchel & Friston, 2001; Cabeza et al., 2003). This is consistent with previous fMRI research suggesting a supra-modal role of the prefrontal cortex in attention selection within both the sensori-motor and mnemonic domains (Friston et al., 1996, 1999). Taken together, these neuroimaging studies suggest the distributed interaction between modality-specific posterior visual and frontal-parietal areas service visual attention and object discrimination cognitive tasks (Rees & Lavie, 2001). Research on the cognitive aspects in MTBI patients indicates a classic pattern of abnormalities in information processing and executive functioning that correspond to the frontal lobe damage (Stuss & Knight, 2002).

The frontal areas of the brain, including prefrontal cortex, are highly vulnerable to damage after traumatic brain injury leading to commonly observed long-term cognitive impairments (Levin et al., 2002; Echemendia et al., 2001; Lowell et al., 2003). A significant percentage of the mild traumatic brain injuries will result in structural lesions (Johnston et al., 2001), mainly due to diffuse axonal injury (DAI), which are not always detected by MRI (Gentry et al., 1988; Liu et al., 1999). Recent dynamic imaging studies have finally revealed that persistent post-concussive brain dysfunction exists even in patients who sustained a relatively mild brain injury (Hofman et al., 2002; Umile et al., 2002).

Striking evidence for DAI most commonly involving the white matter of the frontal lobe (Gentry et al., 1998) and cellular damage and after mild TBI was revealed by magnetic resonance spectroscopy (MRS). Specifically, MRS studies have demonstrated impaired neuronal integrity and associated cognitive impairment in patients suffering from mild TBI. For example, a number of MRS studies showed reduced NAA/creatine ratio and increased choline/creatine ratio in the white matter, which can be observed from 3-39 days post-injury (Mittl et al., 1994; Garnett et al., 2000; Ross & Bluml, 2001). The ratios are highly correlated with head injury severity. More importantly, abnormal MR spectra were acquired from frontal white matter that appeared to be normal on conventional MRI. Predictive values of MRS in assessment of a second concussion are high, because of frequent occurrence of DAI with second impact syndrome (Ross & Bluml, 2001). The language, memory and perceptual tasks sensitive to frontal lobe

functions have been developed because a disruption in frontal-limbic-reticular activation system following closed head injury has been hypothesized (Johnston, 2001). Patients with MTBI performed poorly in these tasks. Long-term functional abnormalities, as evidenced by fMRI have been documented in concussed individuals with normal structural imaging results (Schubert & Szameitat, 2003; Chen et al., 2003). Overall, abnormal brain metabolism may present between 1.5 – 3 months post-injury indicating continuing neuronal dysfunction and long-term molecular pathology following diffuse axonal brain injury.

3. POSTURAL STABILITY AND MTBI

Human upright posture is a product of an extremely complex system with numerous degrees of freedom; posture, like other physical activities, undergoes dramatic changes in organization throughout life. The nature of postural dynamics is more complex than a combination of stretch reflexes (Shtein, 1903) or voluntary movements aimed at counterbalancing the gravitational torque in every joint of the human body (McCollum & Leen, 1989). Human posture includes not only the maintenance of certain relative positions of the body segments but also fine adjustments associated with various environmental and task demands. It follows from this perspective that neither accounts of the neural organization of motor contraction synergy (Diener, Horak & Nashner, 1988) and feedforward control processes (Riach & Hayes, 1990) nor solely somatosensory cues attenuating the body sway (Jeka & Lackner, 1994; Barela et al., 2003) can explain the nature of postural stability unless we consider the more global effects of the organism-environment interaction (Gibson, 1966, Riccio & Stoffregen, 1988).

Traditionally, postural stability has been measured indirectly by determining the degree of motion of the center of pressure at the surface of support through force platform technology (Nashner, 1977; Goldie et al., 1989; Nashner et al. 1985; Hu & Woollacott, 1992; Slobounov & Newell, 1994 a,b; 1995; Slobounov et al, 1998 a,b). The location of the center of pressure is generally assumed to be an accommodation to the location of the vertical projection of the center of gravity of the body in an upright bipedal stance (Winter, 1990). The positive relationship between a measure of increased sway and loss of balance was established by Lichtenstein et al. (1988). More recently, postural sway, reaction time and the Berg Scale have been used to determine reliable predictors of falls (Lajoie et al., 2002). It was shown that postural sway values in the lateral direction associated with increased reaction time could be used as a predictor of falls.

However, Patla et al. (1990) have suggested that increased body sway is not an indication of a lesser ability to control upright stance and is not predictive of falls, because the task of maintaining a static stance is quite

different from the requirements needed to recover from postural instability due to a trip or slip. This suggestion is consistent with notion that the center of pressure sway during quiet stance is a poor operational reflection of postural stability (Slobounov et al., 1998a). We have shown that the ratio of the area of the center of pressure to the area within the stability boundary, defined as *stability index*, is a strong estimate of postural stability both in young, elderly and concussed subjects (Slobounov et al., 1998b; Slobounov et al., 2005a).

Several previous studies have identified a negative effect of MTBI on postural stability (Lishman, 1988; Ingelsoll & Armstrong, 1992; Wober et al., 1993). Recently, Geurts et al. (1999) showed the increased velocity of the center of pressure and the overall weight-shifting speed indicating both static and dynamic instability in concussed subjects. Interestingly, this study also indicated the association between postural instability and abnormal mental functioning after mild traumatic brain injury. It is worth mentioning that research on the relationship between cognitive functions and control of posture is a new and expanding area in behavioral neuroscience (Woollacott & Shumway-Cook, 2002). The use of postural stability testing for the management of sport-related concussion is gradually becoming more common among sport medicine clinicians. A growing body of controlled studies has demonstrated postural stability deficits, as measured by Balance Error Scoring System (BESS) on post-injury day 1 (Guskiewicz et al., 1997; 2001; 2003; Rieman et al., 2002; Volovich et al., 2003; Peterson et al., 2003). The BESS is a clinical test that uses modified Romberg stances on different surfaces to assess postural stability. The recovery of balance occurred between day 1 and day 3 post-injury for the most of the brain injured subjects (Peterson et al., 2003). It appeared that the initial 2 days after MTBI are the most problematic for most subjects standing on the foam surfaces, which was attributed to a sensory interaction problem using visual, vestibular and somatosensory systems (Valovich et al., 2003; Guskiewicz, 2003). Despite the recognition of motor abnormalities (Kushner, 1998; Povlishock et al., 1992) and postural instability resulting from neurological dysfunction in the concussed brain, no systematic research exists identifying how dynamic balance and underlying neural mechanisms are interactively affected by single and multiple MTBI.

Additional evidence supporting the presence of long-term residual postural abnormalities was provided in a recent study showing a destabilizing effect of visual field motion in concussed athletes (Slobounov et al., 2005c). In this study, postural responses to visual field motion were recorded using a virtual reality (VR) environment in conjunction with balance and motion tracking technologies. When a visual field does not match self-motion feedback, young controls are able to adapt via shifting to a kinesthetic frame of reference, thus, ignoring the destabilizing visual effects (Keshner & Kenyon, 2000-2004). The conflicting visual field motion

in concussed athletes within 30 days post-injury produces postural instability. Concussed subjects were found to be significantly dependent on visual fields to stabilize posture. It was suggested that visual field motion produced postural destabilization in MTBI subjects due to trauma induced dysfunction between sensory modalities and the frontal cortex. Again, it should be noted, the frontal areas of the brain are highly vulnerable to damage in subjects after traumatic brain injury, resulting in behavioral impairments (Stuss & Knight, 2002).

4. EEG RESEARCH OF MTBI

Electroencephalography (EEG) reflecting the extracellular current flow associated with summated post-synaptic potentials at the apical dendrites in synchronously activated vertically oriented pyramidal neurons (Martin, 1991), with sources of either a cortico-cortical or thalamo-cortical origin (Barlow, 1993), was first developed by Hans Berger in 1925 in attempt to quantify the cortical energetics of the brain. Since then there has been a plethora of both basic and applied scientific study of the cognitive and motor functions using EEG and its related experimental paradigms (see Birbaumer et al., 1990; Pfurtscheller & de Silva, 1999; Nunez, 2000 for reviews).

EEG, due to its sensitivity to variations in motor and cognitive demands, is well suited to monitoring changes in the brain-state that occur when a performer comes to develop and adopt an appropriate strategy to efficiently perform a task (Gevins et al., 1987; Smith et al., 1999; Slobounov et al., 2000a,b). Sensitivity of the EEG in the *alpha* (8-12Hz), *theta* (4-7Hz) and *beta* (14-30Hz) frequency bands to variations in motor task demands has been well documented in a number of studies (Jasper & Penfield, 1949; Pfurtscheller, 1981). Moreover, the functional correlates of *gamma* (30-50 Hz) activity, initially defined as a sign of focused cortical arousal (Sheer, 1976), which accompany both motor and cognitive task, are also now being widely investigated (Basar et al., 1995; Tallon-Baudry et al., 1996, 1997; Slobounov et al., 1998c).

EEG work related to understanding human motor control has a long history. With the early work of Kornhuber and Deecke (1965) in Europe and Kutas and Donchin (1974) in the United States, there have been studies examining human cortical patterns associated with movement in both time – movement-related cortical potentials, MRCP (Kristeva et al., 1990; Cooper et al., 1989; Lang et al., 1989; Slobounov & Ray, 1998; Slobounov et al., 2002a,b,c; Jahanshahi & Hallett, 2003, for review) and frequency (Pfurtscheller & da Silva, 1999, for review) domains.

There are numerous EEG studies of MTBI. For instance, early EEG research in 300 patients clearly demonstrated slowing of major frequency bands and focal abnormalities within 48 hours post-injury (Geets & Louette, 1985). A more recent study by McClelland et al. (1994) has shown that

EEG recordings performed during the immediate post-concussion period demonstrated a large amount of "diffusely distributed slow-wave potentials," which were markedly reduced when recordings were performed six weeks later. A shift in the mean frequency in the alpha (8-10 Hz) band toward lower power and overall decrease of beta (14-18Hz) power in patients suffering from MTBI was observed by Tebano et al. (1988). In addition, the reduction of theta power (Montgomery et al., 1991) accompanying a transient increase of alpha-theta ratios (Pratar-Chand, et al., 1988; Watson et al., 1995) was identified as residual organic symptomology in MTBI patients.

The most comprehensive EEG study using a database of 608 MTBI subjects revealed (a) increased coherence and decreased phase in frontal and frontal-temporal regions; (b) decreased power differences between anterior and posterior cortical regions; and (c) reduced alpha power in the posterior cortical region, which was attributed to mechanical head injury (Thatcher et al., 1988). A more recent study by Thornton (1999) has shown a similar data trend in addition to demonstrating the attenuation of EEG within the high frequency gamma cluster (32-64 Hz) in MTBI patients. Focal changes in EEG records have also been reported by Pointinger et al. (2002) in early head trauma research. In our work, significant reduction of the cortical potentials amplitude and concomitant alteration of gamma activity (40 Hz) was observed in MTBI subjects performing force production tasks 3 years post-injury (Slobounov et al.,2002,d). More recently, we showed a significant reduction of EEG power within theta and delta frequency bands during standing postures in subjects with single and multiple concussions within 3 years post-injury (Thompson, et al., 2005).

Persistent functional deficits revealed by altered movement-related cortical potentials (MRCP) preceding whole body postural movements were observed in concussed athletes at least 30 days post-injury (Slobounov et al., 2005b). It should be noted that all subjects in this study were cleared for sport participation within 10 days post-injury based upon neurological and neuropsychological assessments as well as clinical symptoms resolution. Interestingly, the frontal lobe MRCP effects were larger than posterior areas. The fact that no behavioral signs of postural abnormality were observed on day 30 post-injury despite the persistent presence of cerebral alteration of postural control may be explained by the enormous plasticity at different levels of the CNS allowing compensation for deficient motor functions. Specific mechanisms responsible for this plasticity and compensatory postural responses are awaiting future examinations. The results from this report support the notion that behavioral symptoms resolution may not be indicative of brain injury pathway resolution. As a result, the athletes who return to play based solely on clinical symptom resolution criteria may be highly susceptible to future and possibly more severe brain injuries. There is no universal agreement on concussion grading and return-to-play criteria.

However, recent evidence in clinical practice indicates underestimation of the amount of time it takes to recover brain functions from concussion. Accordingly, the alteration of brain potentials associated with postural movement clearly observed within 30 days post-injury could potentially be considered within the scope of existing grading scales and return-to-play criteria.

CONCLUSION

There is still considerable debate in the literature whether mild traumatic brain injury (MTBI) results in permanent neurological damage or in transient behavioral and cognitive malfunctions. We believe that one of the reasons for this controversy is that there are several critical weaknesses in the existing research on the behavioral, neural and cognitive consequences of traumatic brain injury. First, most previous research has failed to provide the pre-injury status of MTBI subjects that may lead to misdiagnosis of the persistent or new neurological and behavioral deficits that occur after injury. Second, previous research has focused selectively on pathophysiology, cognitive or behavioral sequelae of MTBI in isolation. Third, previous research has focused primarily on single concussion cases and failed to examine the subjects who experienced a second concussion at a later time. Finally, previous research has failed to provide analyses of biomechanical events and the severity of a concussive blow at the moment of the accident. Biomechanical events set up by the concussive blow (i.e. amount of head movement about the axis of the neck at the time of impact, the site of impact etc.) ultimately result in concussion, and their analysis may contribute to a more accurate assessment of the degree of damage and potential for recovery. Overall, a multidisciplinary approach using advanced technologies and assessment tools may dramatically enhance our understanding of this puzzling neurological disorder facing the sports medicine world today.

We believe that the currently accepted clinical notion of transient and rapid symptoms resolution in athletes suffering from even mild traumatic brain injury is misleading. There are obvious short-term and long lasting structural and functional abnormalities as a result of mild TBI that may be revealed using advanced technologies. There is a need for the development of a conceptual framework for examining how behavioral (including postural balance), cognitive and underlying neural mechanisms (EEG and MRI) are interactively affected by single or multiple MTBI. A set of tools and advanced scales for the accurate assessment of mild traumatic brain injury must be elaborated including the computer graphics and virtual reality (VR) technologies incorporated with modern human movement analysis and brain imaging (EEG, fMRI and MRS) techniques. Semi-quantitative

estimates of biomechanical events set up by a concussive blow should be developed using videotape analysis of the accident, so they may be correlated with other assessment tools. Current research studying student-athletes prior to and after brain injury has provided strong evidence for the feasibility of the proposed approach utilizing technologies in examining both short-term and long-lasting neurological dysfunction in the brain, as well as balance and cognition deterioration as a result of MTBI.

OUTLINE OF THE BOOK

We will now provide a few more details on the organization of book's content There are five main parts, providing multidisciplinary perspectives of sport-related concussions. This book covers conceptual, theoretical and clinical issues regarding the mechanisms, neurophysiology, pathophysiology, and biomechanics/pathomechanics of traumatic brain injuries which constitutes **Part 1**.

Numerical scales, categories, and concussion classifications which are well-accepted in clinical practice are contained in **Part 2** of the book. It is important to note that existing limitations, controversy in aforementioned scales are discussed within the **Part 2** of this book.

Fundamentals of brain research methodology, in general, and the application of various brain imaging techniques such as EEG, MRI, fMRI, CT, and MRS, in specific, are developed in **Part 3** of the book.

Part 4 of the book constitutes a number of chapters on experimental research in humans along life-span suffering from single and multiple concussions. This research is presenting biomechanical, neurophysiological, and pathophysiological data obtained from brain injured subjects.

Finally, **Part 5** of the book concentrates on current information pertaining to care, clinical coverage and prevention of sport-related concussion as well as the medical issues, rehabilitation practitioners' responsibilities and psychological aspects of concussion in athletes. This part is focused on specialized treatment and rehabilitation of brain injured athletes. A special chapter is developed on the perception and concerns of coaches in terms of prevention of sport-related concussions. Also, a special emphasis within **Park 5** of this book is devoted to case studies, current practices dealing with concussed athletes and future challenges.

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