CHAPTER 3

EEG CHANGES AND BALANCE DEFICITS FOLLOWING CONCUSSION: ONE PIECE OF THE PUZZLE

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Abstract: This chapter explores the contribution that electroencephalographic (EEG) recordings and balance testing can make in the areas of concussion assessment and return to play decisions. Current literature in these areas and empirical research that combines these assessment tools is reviewed. Research findings support the view that it is not symptom resolution, rather resolution of the concussion pathology that should determine whether an athlete is ready to return to competition. The current gold standard measures used for evaluation of both concussion severity and return to play rely heavily on measures of loss of consciousness and post-traumatic amnesia. The limitations of these current standards are discussed and a proposal is made concerning how concussion assessment might be improved through the addition of functional motor tests and objective EEG measures that reflect cortical functioning. The combination of electroencephalographic recordings and motor testing allows practitioners to assess two crucial elements of athletic functioning. First, basic physical capabilities of an athlete are assessed via balance testing. Second, neuronal functioning of the cortex is assessed in both resting and task conditions to determine whether the athlete has returned to pre-injury levels of cortical functioning. New research in the field is revealing that by challenging concussed people to perform postural tasks while simultaneous electroencephalographic recordings are taken, insights into long lasting cognitive functional deficits can be revealed. Research applied to frequency analysis, event-related potentials, movement related cortical potentials and injury source localization associated with the electroencephalograph recordings will be presented and commented on. The goal of this chapter is to provide the reader with an overview of the current state of research in concussion assessment and diagnosis and to lead the reader through the groundbreaking work being performed using functional testing paradigms during electroencephalographic recordings. The reader will be left with an understanding of why the combination of motor and cerebral testing is essential in performing a sensitive and reliable measure of concussion severity and return to play readiness.

Keywords: Balance; Concussion; Diffuse Axonal Injury (DAI); Electroencephalogram (EEG); Low Resolution Brain Electromagnetic Tomography (LORETA); Traumatic Brain Injury (MTBI); Movement-Related Cortical Potential (MRCP.

1. INTRODUCTION

Despite the relatively simple mechanism of injury involved with concussion—blunt trauma, acceleration/deceleration and/or axial rotation of the head—the potpourri of symptoms that present in the hours to weeks post injury hint at the true complexity of the injury (Hugenholtz et al., 1988; Thatcher et al., 1989; Macciocchi et al., 1996; Wojtys et al., 1999; Barr, 2001; Guskiewicz et al., 2001; Oliaro et al., 2001; Powell, 2001). Any attempt to classify concussion as a traumatic event with predictable findings upon examination is erroneous. As mentioned in Chapter 1 of this book, most grading scales currently used to diagnose the severity of a head injury are based on loss of consciousness (LOC) and/or posttraumatic amnesia, both of which occur infrequently in mild traumatic brain injury (Guskiewicz et al., 2001), or the subjective reporting of physical symptoms which usually resolve themselves within a one-week period (Echemendia et al., 2001; Guskiewicz et al., 1997; Maddocks & Saling, 1996; Macciocchi et al., 1996; Macciocchi et al., 2001). The use of these measures as the basis for concussion diagnosis or return-to-play (RTP) decisions can have catastrophic results such as second impact syndrome (Thompson, 2005). The amazing plasticity of the brain may allow it to reallocate resources such that undamaged pathways and neurons are used to perform the cognitive or motor tasks being tested. This functional reserve gives the appearance that the person has returned to pre-injury health while in actuality the injury is still present (Randolph, 2001).

With this in mind we dedicate this chapter to two assessment tools that, when used together, greatly increase the sensitivity of concussion assessment and go beyond the methods currently used by the majority of sports medicine practitioners. This proposed evaluation technique integrates electroencephalograph (EEG) recordings and balance testing to determine injury severity and RTP readiness. The combination of these tests allows practitioners to assess two crucial elements of athlete functioning; first, the physical functioning of an athlete is assessed via balance testing, second, the neuronal function of the cortex is assessed while resting and during motor task conditions to determine whether it is functioning normally. Normalized functioning under both conditions is crucial for athletes prior to their return to the competitive sporting environment. If athletes return to competition too early they risk further injury due to slow or poor decision making, slowed reaction time or poor motor co-ordination.

2. EEG & THE DIAGNOSIS OF CONCUSSION

Electroencephalographic recordings measure the spontaneous rhythmic bioelectric potentials arising from the cortex (Shaw, 2002). They reflect the

temporal and spatial summated activity of both excitatory (EPSP) and inhibitory (IPSP) postsynaptic potentials generated by the pyramidal cells of the upper layers of the cortex (Shaw, 2002). EEG records the current flow in extracellular space, and therefore detects the synchronized activity of a large number of cells. Pyramidal cells receive inputs in the more superficial layers (layers II and III) from cortico-cortical inputs and in the deeper layers (layers IV and V) from thalamo-cortical inputs. EPSPs in the superficial layers and IPSPs in the deeper layers will both result in an upward (negative) waveform in the EEG. Conversely, EPSPs received by pyramidal cells in the deeper layers and IPSPs in the more superficial layers will result in a downward (positive) deflection in the EEG (Kandel et al., 2000). Since the cortico-cortical neurons are greater in number and synapse in the more superficial layers, they contribute more to the surface EEG potential.

EEG patterns are characterized by the frequency and amplitude of the electrical activity in the cortex. As the level of activation in the cortex increases, the EEG becomes increasingly desynchronized. EEG patterns are topographically localized in relation to nervous system organization. The interaction between specific and nonspecific sensory and cortical influences determines their frequency and cortical expression (Sterman, 1996). EEG is highly reliable and reproducible within the same individual upon repeat testing (Thatcher, 1999). This stability and reliability has been demonstrated with even small amounts of recorded EEG. Salinsky et al. (1991) reported that repeated 20 second samples were 82% reliable, 40 second samples were 90% accurate and 60 second samples were 92% reliable. The EEG is considered to be a more direct measure of cerebral function than either intracranial pressure (ICP) or cerebral blood flow (CBF) (Ommaya and Gennarelli, 1976), and provides a measure of the subject's level of arousal. Arousal control is considered to be an essential component of peak performance and this relationship has been documented and studied at length in sport psychology (Landers & Arent, 2001). We will return to this topic later in the chapter in the section on EEG event-related potentials.

Shear strain injury, otherwise known as diffuse axonal injury (DAI), is considered to be the primary pathologic feature of brain injury in all severity levels of concussion (Kushner, 2001). Diffuse axonal injury is frequently not detectable in MTBI using gross neuroimaging techniques such as magnetic resonance imaging (MRI) or computerized tomography (CT) scans (Barth et al., 2001). Therefore, it is necessary to study these effects using a diagnostic tool that is able to detect the effects of DAI, namely EEG. The first allusion to DAI dates to the 19th century. It was a mystery to neurologists how such a severe paralysis of neuronal function could occur in the absence of obvious anatomical damage (Shaw, 2002). In 1835, J. Gama proposed that "fibers as delicate as those of which the organ of mind is composed are liable to break as a result of violence to the head" (Shaw, 2002). DAI occurs from mechanically induced stretching, shearing or

tearing of nerve fibers. These forces are produced by acceleration or deceleration with angular rotation. This may result from trauma directly to the head or from trauma to the torso or axial skeleton with the force of the incident being transmitted indirectly to the brain matter (Amann, 2000). Holbourn (1943) and Strich (1961) described the primary microscopic feature observed in neural tissue post concussion as diffuse degeneration of white matter without obvious damage to the cortex (Gaetz, 2003). It was concluded that nerve fibers were torn or stretched at the time of injury. This conclusion was based on cadaver studies showing large numbers of nerve fibers with retraction balls (the appearance of severed axons with axoplasm extruded from the proximal and distal segments) (Gaetz, 2003). Recent findings by Smith et al. (1999) have hinted at neuronal axons ability to withstand large stretch forces and their high threshold for primary axotomy. They showed that no primary axotomy occurred in human neuronal cultures at tensile strains causing deformation of up to 65% of the neurons original length. However, a major consequence of this pathology is an increase in neuronal permeability, especially to Ca^{2+}, a possible loss of consciousness and/or post-traumatic amnesia (retrograde and/or anterograde) (Giza & Hovda, 2001). In addition, Smith et al (1999) showed that post injury axons showed a gradual recovery to their original shape, although there were multiple swellings along the length of many axons.

It is well known that brainstem reticular cells play a vital role in consciousness, and it has been suggested that it is damage to this structure that leads to decreased arousal or lack of consciousness following MTBI (Shaw, 2002). It may be the case, however, that it is not damage to the reticular cells themselves that causes this depression of arousal. Rather, it may be that mild DAI cause's damage to cortical structures that, under normal functioning, provide stimulation to the brainstem. As a result of damage to these cortical structures that provide excitatory inputs to the brainstem, reticular cells are suppressed due to lack of input (Gaetz, 2003). This could result in lowered arousal in subjects and decreased athletic performance. The importance of a neuroimaging tool that can detect DAI and not just neuronal tearing is paramount in concussion assessment. As a result of the method by which EEG records and represents human cortical activity, an electroencephaolograph can reflect the pathology of mild traumatic brain injury. It is evident from the above discussion concerning the relationship between concussion pathology and the symptoms following brain injury that a neuroimaging tool that can detect DAI and not just cortical lesions (which is all that MRI and CT detect) is essential in concussion assessment. EEG can provide important information regarding the pathology of a brain injury and additionally, the procedure is non-invasive and relatively inexpensive. Based on the above discussion and the information regarding concussion pathology given throughout this textbook, the value of EEG recordings in MTBI assessment is evident.

To aid with the discussion that follows I will note a few key terms used in the field of EEG. Frequency analysis divides the EEG into its frequency components measured in cycles per second (cps), or hertz (Hz). These can be in any size but are usually in 1 Hz or pre-set standard divisions (eg. 0.5-3.5, 3.5-8, 8-12, 12-20, 20-30). It is a measure of the amount of energy in each division and is referred to either as power or amplitude (the square root of power). Quantitative EEG (QEEG) refers to measures performed on the raw EEG recording. It encompasses any statistical or mathematical analysis made on the raw EEG recording. The QEEG can also provide graphical representations of the recorded electrical activity showing areas of the head in which specific EEG features are present.

A recent development that has greatly advanced the utility of EEG in localizing cortical damage is low-resolution brain electromagnetic tomography (LORETA). The Key Institute for Brain-Mind Research in Switzerland developed the LORETA inverse solution. This procedure computes, from the recorded EEG, the three-dimensional distribution of the electrically active neuronal generators in the brain as a current density value (amplitude/m^2) at each voxel (a voxel is the basic unit of computed tomography reconstruction, represented as a pixel in a computed tomography image display) (Korn et al., 2005). Three-dimensional LORETA images consist of 2,394 voxels in total and give a graphic representation of cortical gray matter similar to that of CT and MRI. Computations for source localization of EEG frequencies are limited to cortical gray matter and the hippocampus according to a digitized Probability Atlas (Brain Imaging Centre, Montreal Neurologic Institute). LORETA has been shown to be accurate in its generation of instantaneous scalp potential distribution maps to within 7mm (Korn et al., 2005). For a complete explanation of the LORETA inverse solution and its validation, please refer to Pascual-Marqui et al. (1994), Pascual-Marqui (1999), and Pascual-Marqui et al. (2002). By integrating LORETA into concussion assessment the clinician is given a tool that can detect the location of gray matter damage post MTBI even in the absence of cortical lesions, a process that until now has not been possible.

3. EEG RESEARCH ON CONCUSSION

3.1. EEG Frequency Analysis

Recent research (Thatcher et al., 1989, 1998a, 1998b, 2001; Shaw, 2002; Korn, 2005) has shown the validity and sensitivity of EEG frequency analysis in detecting structural damage post concussion and in evaluating the severity and extent of the injury. MRI and CT scans, on the other hand, are unable to detect cortical damage without the presence of lesions (Thatcher et

al., 1989, 1998a, 1998b, 2001; Barth et al., 2001; Guskiewicz, 2001; Kushner, 2001; Shaw, 2002). To date, studies using EEG frequency recordings to measure the negative effects of concussion on cognitive functioning have documented the following findings. Generally speaking, MTBI causes increased theta amplitude, reduced mean alpha frequency, reduced mean alpha amplitude, decreased beta amplitude, decreased amplitude differences between anterior and posterior regions, and decreased gamma frequency activity (Tebano et al., 1988; Thatcher et al., 1989; Montgomery et al., 1991; Hoffman et al., 1995; Watson et al., 1995; Thatcher et al., 1998a, 2001). In a much earlier study where they were able to record EEG's immediately following a boxing match, Larsson et al. (1954) showed overall reduced EEG amplitude and increased irregular theta activity in boxers within 15-30 minutes of a fight. These effects were more pronounced after being knocked out. By showing increased irregularity following a more serious injury, Larsson et al. demonstrated a correlation between injury severity and EEG abnormalities.

EEG recordings taken weeks or months after an injury have shown a gradual increase in the mean alpha frequency, for example from 9 Hz to 10 Hz. This is presumed to be a return to the subject's pre-injury dominant alpha frequency (Jung, 1953). A major shortcoming of this method of investigation is that the conclusion, made retrospectively, cannot be verified since within subject pre and post MTBI measures can not been compared. It is possible that they improve but never return to their own baseline standard. Also, this process requires repeat EEG recordings and is time consuming for both the subject and practitioner. Therefore, this method for testing a "return to pre injury baseline alpha frequency" is inefficient and remains an untested hypothesis. Currently, pre-season EEG baseline measures and post MTBI EEG measures are being taken on athletes by Thompson & Slobounov in an effort overcome this shortcoming and gain insight regarding within subject EEG changes pre and post injury.

The physiologic alterations following concussion are numerous and greatly affect the ionic channels of neuronal membranes (e.g. Na^{+}, K^{+}, Ca^{++}). These changes cause a reduction in EEG amplitude due to the reduced average current flux (Thatcher et al., 2001). One hypothesis is that following MTBI, the attenuation of EEG frequencies occurs because there are fewer functional ionic channels per unit volume (Thatcher et al., 2001). It should be noted that all of the above studies, except Thompson et al. (2005), recorded EEG in eyes-closed seated conditions. The cumulative effects of these neuronal changes after MTBI are (1) localized dysfunction specific to areas of maximal injury, and (2) overall diminished information processing capability and cognitive functioning (Thatcher et al., 1989 & 1998).

This range of findings in EEG and quantitative electroencephalogram (QEEG) studies should come as no surprise. EEG abnormalities are a result

of the pathology of MTBI. As all brain injuries differ in their mechanism, location, severity and symptomology, it is to be expected that EEG abnormalities post injury will differ between subjects. There is no special pathognomonic location and there is no special biological process that would produce pathognomonic changes in the EEG (Nuwer, 2005). Korn et al. illustrate this statement in a study published in 2005. EEG recordings and LORETA analysis demonstrated the sensitivity of LORETA to detect the varying sources of cortical damage post concussion. The EEG recordings taken in the MTBI population showed typical post injury findings of increased delta and decreased alpha power. The revolutionary findings for the study of MTBI came when LORETA was applied to the recorded EEG. Whereas controls displayed generators of delta rhythm in consistent voxels representing distinct cortical regions, MTBI subjects displayed increased intervoxel variability for delta generators, where the generators of maximal activity varied from patient to patient (Korn et al., 2005). These locations of abnormality were also compared to single photon emission computed tomography (SPECT), which is an invasive procedure compared to the non-invasive EEG. The location of abnormality given by the invasive SPECT procedure localized the source of the abnormal activity at or closely related (at the same cortical lobe) to the pathologic region observed in LORETA (Korn et al., 2005). Although all MTBI subject's abnormal rhythms were located in differing anatomical locations, as is to be expected based on the above discussion, the dysfunctional areas were generally found to originate in peripheral cortical regions compared to the mid-line restricted symmetric source that characterized the normal group (Korn et al., 2005). This is probably due to the proximity of the brain to the skull and the resulting impact at injury. These findings suggest that in concussed individuals the focal cortical abnormal generators that differ in location between patients, are related to the location of cerebral injury rather than to a single common pathologic generator or a general diffused cortical slowing (as one would expect in the case of a general stress-dependent mechanism, white matter lesion or lesion of a deep structure such as the thalamus or brain stem) (Korn et al., 2005). Based on their findings Korn et al. suggest several lines of evidence that support the view that cortical dysfunction, as reflected by EEG findings, may underlie PCS. First, three patients presenting with transient attacks of paraesthesia, suggestive of partial epilepsy, had a focal abnormal EEG localized to contralateral parietal cortex. Second, the correlation between EEG aberrations and reduced regional cerebral blood flow (rCBF) perfusion and blood-brain barrier (BBB) permeability suggests an association between the functional and anatomic lesions. Third, in eight patients with persistent PCS, EEG abnormalities were related anatomically to the SPECT findings and in one patient in whom the clinical syndrome was resolved, recovery was associated with parallel resolution of both EEG aberrations and SPECT findings (Korn et al., 2005).

Other recent work using EEG frequency analysis combined EEG recordings with alterations in posture (Thompson, et al., 2005). This testing procedure improved upon past studies by introducing testing conditions more appropriate to athletes who compete in dynamic competitive environments (that is, not just using seated eyes closed conditions which is the standard for most EEG databases). This research will be described with some detail to provide an example of current work being performed in the field.

In the Thompson et al. (2005) study a total of 24 subjects were included. They were divided into two groups based on concussion history. Subjects were included in the injury group if they had sustained a MTBI within 5 months of testing. All subjects in both groups were cleared for play based on current concussion return to play guidelines. EEG recordings were taken under four experimental conditions: eyes open (EO) seated, eyes closed (EC) seated, EO static standing, EC static standing. Seated trials consisted of 3 minutes of continuous data collection under each condition. Standing trials were performed using a bipedal stance on an AMTI force plate. For the static standing trials, subjects were instructed to remain as still as possible for 30 seconds. There were three trials in each standing/visual condition. A detailed description of experimental procedure and setup can be found in Thompson et al., 2005. The continuous EEG activity from the scalp was recorded at 19-sites: FP1, FP2, FZ, F3, F4, F7, F8, CZ, C3, C4, T3, T4, T5, T6, PZ, P3, P4, O1, O2 according to the International 10-20 system (Jasper, 1958). The ground electrode was located 10% anterior to FZ, linked earlobes served as references. Frequency bandwidths were divided according to the following six divisions: delta (0.5-3.5 Hz), theta (3.5-7.5 Hz), alpha (7.5-10 Hz), alpha2 (10-12.5 Hz), beta (12.5-17 Hz) and beta2 (17-19 Hz). Electrode-front-back groupings were used to find possible differences between the anterior (F3, Fz, F4), central (C3, Cz, C4), and posterior (P3, Pz ,P4) sites. Electrode-left-right groupings were used to analyze differences between electrodes on the left (F7, F3, T3, C3, T5, P3), central (Fz, Cz, Pz), and right (F8, F4, T4, C4, T6, P4) areas of the cerebrum. The confidence interval for all ANOVAs that were conducted was set at 95%.

To analyze the effects of injury, posture, and vision condition a 3 factor (EO vs. EC; injury vs. control; sitting vs. standing postures) ANOVA was performed with the dependent variable set to six EEG bandwidths (delta (0.5-3.5 Hz), theta (3.5-7.5 Hz), alpha (7.5-10 Hz), alpha2 (10-12.5 Hz), beta (12.5-17 Hz) and beta2 (17-19 Hz). In the Delta band, posture was not a significant factor at the 0.05 level ($p=0.615$). However, the interaction effect of injury x posture was significant ($p=0.035$). ANOVA performed exclusively on the standing condition showed a significant difference between groups ($p=0.01$) with the healthy group having much higher delta amplitude than the injured group. A second ANOVA holding posture

constant revealed that in the seated condition there was not a significant difference between groups (p= 0.536) (Figure.1).

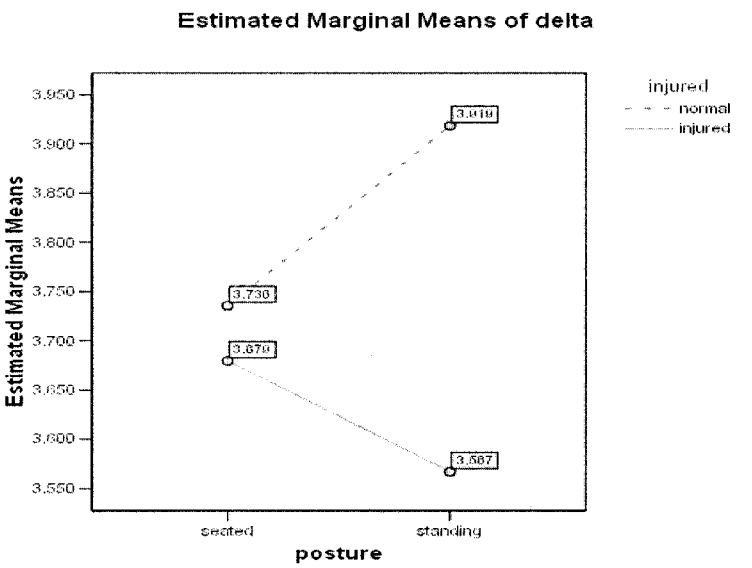

Fig.1. Delta Amplitude x Posture and Injury condition. (Re-printed, by permission, from Thompson, J., Sebastianelli, W., Slobounov, S. (2005). EEG and Postural Correlates of Mild Traumatic Brain Injury in College Athletes. *Neuroscience Letters, 377,* 158-163.)

The authors explained these findings as follows. In a subject who has sustained a MTBI the given task (trying to stand as still as possible) would place increased demands on the cognitive resources used to focus attention, since balance has been shown to be impaired in these individuals (Ingersoll & Armstrong, 1992; Haaland et al., 1994; Guskiewicz et al., 1997; Guskiewicz, 2001; Guskiewicz et al., 2001; Oliaro et al., 2001). Tasks that require increases in attentional focus have been shown to cause attenuation in the lower EEG frequencies, such as delta (Cripe, 2003). Individuals who have sustained a MTBI and are instructed to stand "as still as possible" will experience an increased cognitive load and that may be the underlying factor that causes a significant decrease in delta compared with the higher (more normal) delta measures when at rest in a seated posture.

Analysis performed with Theta as the dependent variable showed some other interesting findings. When ANOVA was performed with anatomical locations divided into anterior (F3, Fz, F4), central (C3, Cz, C4), and posterior (P3, Pz, P4), there was not a significant difference found between sites at the 95% confidence interval (p = 0.125). This was an unexpected finding based on the literature (Thatcher, 1989) and a further analysis was performed to distinguish if the averaging of normal and injured groups

caused there to be no effect found. Further analysis revealed that indeed a significant effect was present between electrode sites in the normal group ($p = 0.013$). Tukey post-hoc analysis revealed that differences lay between frontal and posterior sites ($p = 0.039$), and between central and posterior sites ($p = 0.021$). When the same analyses were performed on the data for the injured group there was no difference found between site locations for the injured group ($p = 0.797$). This lack of a difference held for the relationship between all areas as revealed by Tukey post-hoc analysis. The effect of concussive injury on theta amplitude in frontal, central and posterior areas is represented in figure2.

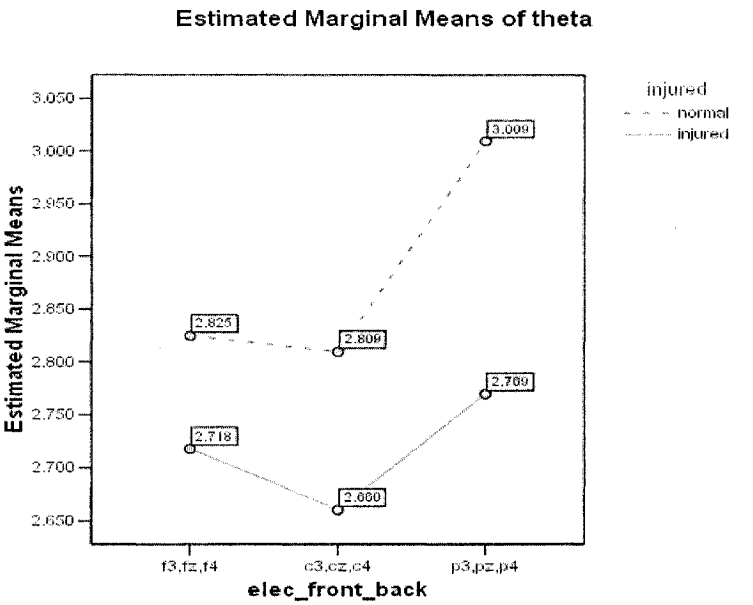

Fig. 2. Theta Amplitude x Injury and Electrode Site (front, central, posterior).

In the theta band, the normal group showed a significant increase in theta amplitude in the posterior region compared with their frontal and central regions. The injured group, however, showed no significant differences in theta amplitude between anterior, central, and posterior sites. This meant that in addition to the overall decrease in amplitude in the frontal and central sites, there was a dramatically larger decrease in theta amplitude in the posterior region of injured subjects compared to normals. This difference was not attributed to lower respiration rates in concussed individuals based on the Badawi et al. (1984) study that found no decrease in theta amplitudes with suppression of breath. The finding of decreased amplitude differences in the theta band between frontal and posterior sites

replicate the findings of Thatcher et al. (1989), which had the largest MTBI sample size to date (n = 608). In their study a decrease in power differences between anterior and posterior cortical regions was found. This was attributed to diminished functional differentiation.

Beatty et al. (1974) noted an interesting pattern related to posterior theta. In monotonous tasks requiring attentional focus, such as our posture task requiring subjects to remain as still as possible, theta suppression in the occipital-parietal areas benefited subjects who were otherwise most likely to show a degraded performance (Beatty et al., 1974). Those so-called "subjects who were otherwise most likely to show a degraded performance" are analogous to our injured population who post-injury have been shown to increase postural sway in static conditions. By learning (unconsciously) to suppress theta activity in the posterior region, injured subjects may improve their performance in monotonous tasks (such as bi-pedal static stance or elements of the neuropsychological testing battery) to levels close to, or equal to, normal subjects. Theta activity in normal subjects has been associated with performance of over-learned behavioral tasks (e.g. bipedal stance) and inactive processing conditions (Crews & Landers, 1993). In concussed individuals, the ability to remain still in a bi-pedal stance may no longer be handled like an over-learned behavioral task because it may require more conscious effort. This may help explain the suppression of theta activity under seemingly simple tasks in our concussed population compared with our healthy subjects. Further credence for our findings is found in the work of Montgomery et al. (1991). A reduction in theta amplitude occurred in their MTBI population between recordings taken immediate post-injury EEG and recordings taken at six weeks post injury. This is an interesting finding that suggests the effect of injury on theta amplitude is not immediately apparent and does not result from the acute alterations in cortical physiology. Also, athletes may be consciously unaware of these changes in cortical function. A lack of correlation has been shown between arousal levels—noted by individuals as cloudiness or drowsiness—and EEG changes (Watson, et al., 1995). The noted reduction in theta amplitude is not immediately apparent in concussed individuals post-injury but rather develops over a somewhat extended time period as shown by Montgomery et al (1991). During this time when pathological changes are obviously still occurring, many athletes may be allowed to return to play based on resolution of physical symptoms in spite of the fact that notable negative effects of the concussion are still present. This lack of sensitivity in detecting pathological deficits is a major shortcoming of current assessment tools.

Analysis of the alpha2 bandwidth was performed to determine what effect concussion had on this waveform. A 3 (electrode groupings left, center, right) x 2 (injury condition) ANOVA was run. A significant ($p = 0.048$) finding occurred for injury condition as well as for electrode pairings

by left, center, right (p = 0.000). Tukey post-hoc analysis revealed an abnormality in the MTBI group. The expectation was that there would be a significant decrease in alpha2 in the injured group in all electrode pairings when compared to the control group. It was also expected that the central electrode grouping would have higher overall power that the left and right groupings. This was not the case. There was no difference in alpha2 amplitude between the central and right side groupings (p = 0.481) in injured group, whereas the normal group showed significantly less alpha2 in the right side compared with the central region (p = 0.000). Between the normal and injured groups, the right side pairings of electrodes showed no difference in alpha2 amplitude (p = 0.921). This was unexpected since all other areas of the head have shown significant decreases in amplitude in all frequencies following concussion. The above is graphically represented in Fig. 3.

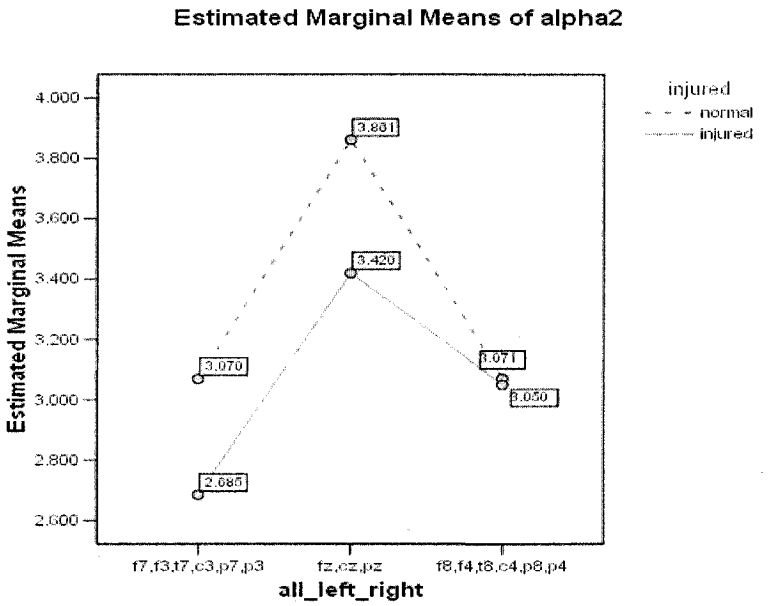

Fig. 3. Alpha 2 x Injury and Electrode Site (left, center, right). (Re-printed with permission from Elsevier: Thompson, J., Sebastianelli, W., Slobounov, S. (2005). EEG and Postural Correlates of Mild Traumatic Brain Injury in College Athletes. *Neuroscience Letters, 377,* 158-163.)

Crews and Landers (1993) showed that in movements involving simultaneous action of both sides of the body bilateral changes in alpha amplitude occurred in the motor cortex. It would therefore be expected that in a bipedal stance the left and right hemispheres would show similar levels

of activity. In the normal population this was the case ($p = 0.000$). However, as mentioned, the injured population had significantly higher alpha2 amplitude on the right side of the head compared with the left side of the head. It should be noted though that in the same study Crews & Landers demonstrated a positive correlation between decreases in right hemisphere activity and less error in a motor task. Error in our study can be viewed as movement by subjects, however subtle, when instructed to remain as still as possible during the testing conditions. The increased activity in the right side electrodes may correspond with subtle deficiencies in the ability of concussed subjects to remain motionless. This would be consistent with the premise that the right side of the head is associated with gestalt and holistic processing, and spatial tasks (Bennett & Trinder, 1977; Ray & Cole, 1985). Other possibilities also arise from the literature. It is possible that other frequencies have bilateral decreases in amplitude as a result of coup-contra coup injury. Due to its functional role in spatial awareness and not analytical processing, the alpha2 frequency band may not be as susceptible to or affected by the changes that occur in neuronal pathways and functioning as a result of concussion. EEG recordings of normal adult individuals show bilateral alpha activity.

It is uncommon to have exceptions to this bilateral symmetry in the alpha bands (Hoovey et al., 1972). In our recordings the normal population did have bilateral symmetry in the alpha band and coincides with the report by Hoovey et al. (1971). The fact that our injured population shows bilateral differences in alpha power can therefore be taken as a sign of their cognitive functional abnormality. Ray and Cole (1985) suggest that reduced alpha in the analytical left side of the brain would affect efficient cognitive processing and impair the ability of athletes to make quick decisions in the sport setting. Additionally, increased alpha in the left hemisphere is associated with reducing unneeded external stimulation (e.g. crowd) that can be equated to reducing the amount of distracting stimuli thus aiding in the efficiency of processing (Ray and Cole, 1985). Cumulatively, these reports suggest that the alpha asymmetry (i.e. the decrease in left side alpha amplitude) post concussion would cause detriments to athlete's performance and put them at potential risk for re-injury.

An analysis was also performed on the beta band in an attempt to replicate previous findings of decreased beta amplitude following concussion. The results of the 2 (injury) x 2 (eye condition) x 2 (posture) ANOVA with beta2 set as the dependent variable show a similar trend as the previous data shown in the theta, alpha, alpha2 and beta frequency bands. There were significant differences for injury ($p = 0.000$), eyes ($p = 0.000$), and posture ($p = 0.000$). There is a decrease in amplitude in the injured group compared with the normal subjects, there is an increase in amplitude in the eyes closed condition, and there is an increase in amplitude in the standing condition. ANOVA also revealed that significant differences

occurred between injury groups (p = 0.000) and between electrode sites (frontal, central, posterior) (p = 0.000) (Fig. 4).

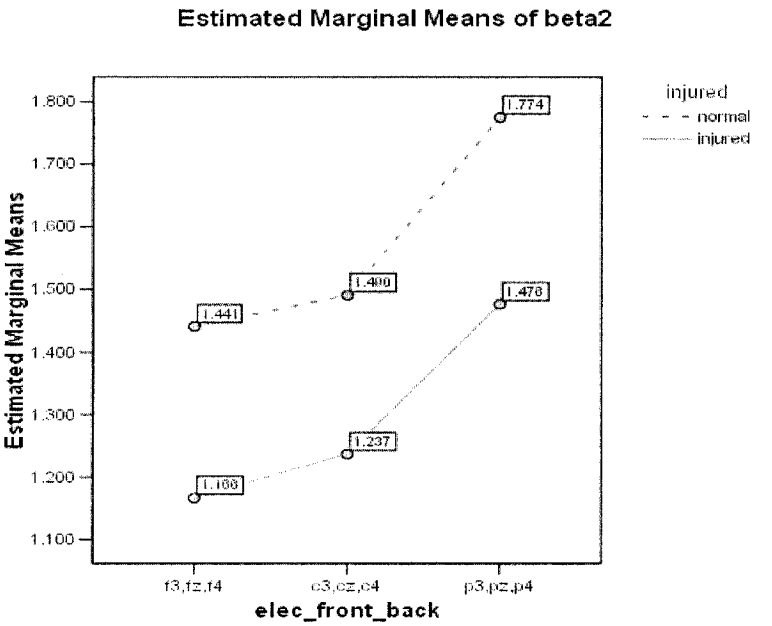

Fig. 4. Beta 2 x Injury and Electrode Site (front, center, posterior). (Re-printed with permission from Elsevier: Thompson, J., Sebastianelli, W., Slobounov, S. (2005). EEG and Postural Correlates of Mild Traumatic Brain Injury in College Athletes. *Neuroscience Letters, 377,* 158-163.)

The finding that dominates the literature on the cognitive effects of MTBI is an overall decrease in cerebral functioning. This is reflected in the EEG as decreased amplitude, especially in the higher frequencies (alpha2, beta, and beta2) (Thatcher et al., 1989; Thatcher et al., 1998; Claus, 2000). These changes can be present independent of the direction in which the brain impact the skull or even in the absence of impact with the skull, as is often the case in diffuse axonal injury (Amann, 2000; Kushner, 2001; Shaw, 2002). The EEG changes are a direct result of alterations in the cortical gray matter and/or white matter (Thatcher et al., 1998b). Thatcher et al. (1998a, 2001) also found significant decreases in EEG amplitude in the alpha, alpha2, beta, and beta2 frequency ranges. These had a significantly high negative correlation with MRI-derived T2 relaxation times and most strongly related to cortical gray matter injury. Damage within the gray matter would reduce the short-distance excitatory inputs to neurons and other pyramidal cells as well as reducing synchronization of active generators. The combination of these reductions is lower surface-recorded

EEG amplitudes (Thatcher et al, 1998a). It was concluded that these changes in the EEG were not related to the acute effects of concussive injury, rather they were related to the chronic consequences of MTBI (Thatcher, 1998a). The finding of reduced alpha and alpha2 amplitudes and reduced cortical excitability in general is consistent with the mechanisms of shear-strain and rotational forces leading to brain injury (Thatcher et al., 1989). These forces can damage both long and short axonal connections. The physiologic alterations following concussion are numerous and, as explained, greatly affect the ionic channels of neuronal membranes (e.g. Na^{+}, K^{+}, Ca^{++}). These changes cause a reduction in EEG amplitude due to the reduced average current flux. One hypothesis is that following MTBI the attenuation of EEG frequencies occurs because there are fewer functional ionic channels (Thatcher et al., 2001). The functional impacts of these changes are numerous. First, it is well established that the level of EEG recorded from the scalp is correlated with cortical activation (Sterman, 1996). Specifically, lower frequencies (theta) have been linked to drowsiness and under-aroused states, while higher frequencies indicate general arousal, cerebral excitation, attentional focus and cognitive processing (alpha, beta, beta2) (Beatty et al., 1974; Ray & Cole, 1985; Landers et al., 1991; Sterman et al., 1992; Beh, et al., 1996). The decrease in scalp-recorded EEG amplitudes is a reflection of the inability of concussed individuals to focus attention and perform cognitive tasks at their pre-injury level. Evidence for this is reflected in lower neurological test scores post-injury as well as subjective complaints of memory and concentration problems (Collins et al., 1990; Echemendia et al., 2001; Randolph, 2001).

The groundbreaking work by Roth, Sterman, and Clemente (1967) shed light on a cortical rhythm that previously (and in many cases since) was assumed to be alpha2 or low beta. The rhythm fell in the range of 12 – 20 Hz activity but was focused between 12 and 14 Hz. The production of this rhythm is associated with conscious inhibition of voluntary movement and can act as an index of motor attentiveness (Collins et al., 1990). Due to its direct association with the motor cortex and movement suppression, this specific rhythm in the alpha2/low beta range was termed the sensorimotor rhythm (SMR) (Roth, et al., 1967). The rhythmic activity of the SMR wave originates in the somatosensory relay nuclei of the thalamus, collectively known as the Ventrobasal (VB) nuclei (Sterman, 1996). The burst activity of the VB (which is relayed to the cortex via related pools of cortical cells and is seen as SMR in the EEG) is dependent upon hyperpolarization of the VB cells and their atypical response of a gradual decay of this hyperpolarized state due to a slow Ca^{++} influx. VB hyperpolarization is induced during inactive behavior (such as when we instructed our subjects to remain "as still as possible") by the attenuation of somatosensory inputs (Sterman, 1996).

In the concussed population, the ability to produce the SMR rhythm may be diminished for two reasons. First, the influx of extracellular Ca^{++} that results from MTBI may alter the rate at which the VB depolarizes following hyperpolarizing inputs or may extinguish the hyperpolarization all together. This would lower the amplitude of the EEG in this bandwidth on the cortical surface as we have seen in our concussed subjects. Second, concussion may affect the thalamus in such a way as to reduce its ability to gate afferent discharges from receptor pathways. This inability to attenuate the signal would cause increased stimulation of the sensorimotor cortex and thus increase movement. These subtle increases in movement would be noted by sensory receptors and relayed to the sensorymotor cortex, thus again increasing stimulation at this location. The individual, in an attempt to remain still, would then produce compensatory simple voluntary movements in order to correct any unwanted movements. This inability to relax, remain attentive, and reduce muscle tone (another requirement for the production of SMR) would decrease the amplitude of the cortical EEG measure in the SMR (12- 14 Hz) bandwidth consistent with our findings. This is also consistent with concussed individuals' self-reports of feeling antsy, inattentive and fidgety post-injury. These principles are consistent with findings of suppression in the 7 – 13 Hz alpha rhythm range with increased sensory stimulation, attentional demand, and motor performance (Sterman et al., 1994). Generally speaking, the results of this study showed significantly lower EEG amplitudes in MTBI subjects in spite of there being no significant differences in the physical measures of index of stability or symptom reports in the pre-testing interview. Although only a first step, functional tests that more closely resemble the sporting environment provide evidence that current assessment protocols (e.g., symptom and balance tests used alone) lack the sensitivity to detect the residual cortical damage that may result from sport concussions. With the addition of accurate yet inexpensive imaging techniques like LORETA, EEG is set to become the new gold standard in concussion management.

3.2. Event Related Potentials (ERP) and Movement Related Cortical Potentials (MRCP)

Though not as abundant in the literature, other works using EEG recordings following MTBI have looked at event-related potentials (ERP) and/or movement-related cortical potentials (MRCP). Both these features of EEG recordings have shown merit in distinguishing between healthy and concussed persons. ERPs are bioelectric responses that are deliberately elicited by feeding a stimulus into the nervous system. These stimuli are most often auditory, visual or somatosensory (Shaw, 2002). Unlike spontaneously produced, randomly distributed EEG activity, ERPs are

always time-locked to the onset of the sensory stimulus. The two most important features of an ERP are their amplitude and latency (time of the appearance of features of the waveform in relation to the stimulus). MRCPs work via the same principle except instead of being time-locked to an externally induced stimulus they are time-locked to a self-initiated movement. Since ERPs and MRCPs are generally much smaller in amplitude than higher amplitude EEG waveforms, they are next to impossible to detect on a conventional EEG. Therefore, the extraction of ERPs and MRCPs is done via signal averaging whereby multiple responses to the stimulus (or movement) are summed. This causes the random background EEG activity to be cancelled out while at the same time progressively increasing the definition of the averaged ERP or MRCP.

As with EEG amplitude studies, work with ERP recordings has been used to assess cerebral abnormalities following concussion. One of the most frequently occurring findings are amplitude changes in ERPs post concussion. In a study conducted by Dupuis et al. (2000) it was found that significant decreases in the amplitude of the P3 segment of the ERP (a positive shift occurring approximately 300ms following the stimulus hence the name P3) occurred in concussed individuals. This amplitude decrease is thought to reflect alterations in attentional-cognitive processes. Also notable in this study was the strong inverse relationship between the amplitude of the P3 and the severity of post-concussion symptoms (i.e. a larger P3 amplitude was associated with lower symptom severity). Lavoie et al. (2004) measured the P3 wave post injury and found that symptomatic athletes displayed longer reaction times, attenuated P3 waves and that the amplitude of the P3 wave varied inversely to the severity of post concussion symptoms. Potter and Barrett (1999) reported similar findings in a study using a demanding working memory task, the paced auditory serial addition task (PASAT). They showed reduced amplitude frontal negative potentials even in MTBI subjects who were asymptomatic. It has also been shown that post injury there is an enhancement of the negative deflection following the P3 wave in oddball task ERPs (Potter et al., 2001). The negativity is thought to be a reorienting negativity. If this is the case then it is suggested by Potter et al. that the MTBI group may be "more likely to shift resources from the task at hand and allocate them temporarily to the distracting novel stimuli". This could potentially spell disaster for athletes returning to competition before these cognitive deficits have resolved themselves. Generally speaking, these alterations in ERPs show a reduction in the allocation of attention resources at early stages of processing (Potter et al., 2001). Following a study in which they showed that MTBI patients had abnormal (reduced amplitude) ERPs, Solbakk et al. (1999) posit that MTBI patients allocate less processing resources to the task at hand than control subjects.

As mentioned, ERPs are time-locked to the onset of the stimulus. Not only has the amplitude of ERPs been shown to be affected by MTBI but so

have the latencies of the features of the waveform. In a study by Onofrj et al. (1991), P3 latencies in all subjects were above normal limits (+2SDs) (ie. delayed onset post stimulus). Over time these latencies progressively decreased (a shift toward normal latencies) during the course of recovery. Stelmack et al. (1993) conducted research in which they measured reaction time (RT), movement time (MT) and the amplitude and latency of the P3 wave. They concluded that the reduced P3 amplitude was associated with decreased attentional effort. Also, P3 latency was regarded as an index of stimulus evaluation time and there was a positive linear relationship between the two. Subjects who had increased stimulus evaluation times had increases in their P3 latency. In anther study that looked at both P3 amplitude and latency, Pratap-Chand et al. (1988) showed that following MTBI subjects had significant abnormalities of the P3 amplitude and latency. The study showed that P3 latency abnormalities occurred more often post concussion than did amplitude attenuation and P3 latencies also showed a larger abnormality than amplitude changes. As was the case in the study by Onofrj et al. (1991), the abnormalities found in the P3 wave resolved themselves over the course of time. Intracranial depth electrode and extracranial magnetic recordings have given evidence toward the cortical areas that produce the P3 wave, namely the amygdala and hippocampus. The P3 arises with the process of perception and cognition, and abnormalities in the P3 are indicative of damage to the above-mentioned structures that has occurred as a result of cerebral concussion (Pratap-Chand, 1988).

Slobounov et al. (2002), examined the residual effect of MTBI on movement-related cortical potentials (MRCP) preceding and accompanying isometric force production tasks. It was shown that in concussed subjects there was a concomitant reduction in the amplitude of MRCPs prior to the initiation of movements in force production tasks requiring increasing levels of complexity compared with normal subjects. Although not a study specifically related to changes in ERPs post concussion, an interesting study by Dirnbeger et al. (2004) should be noted. MRCPs were measured on fatigued subjects while performing a simple motor task (button press). Subjective fatigue (the measure used by Dirnberger et al.) is one of most common symptoms following concussion and has a direct effect on post injured subject's ability to perform physical tasks at pre-injury levels. Dirnberger et al. (2004) found that subjects who reported higher levels of fatigue had smaller amplitude MRCPs. You may also recall the previously mentioned link between athlete arousal levels and performance. Overall decreased arousal following a concussive incident is very relevant to sport performance. The inverted-U relationship, commonly referred to in sport psychology, is based upon the athlete attaining optimal levels of arousal in order to perform at their optimal level (Landers & Arent, 2001). And, as mentioned, a specific physiological measure of this state of arousal is the EEG. For optimal performance to occur, a change in brainwave patterns to a

beta or more aroused state must occur (Landers & Arent, 2001). A second skill related to level of arousal that is paramount to successful performance is the ability of an athlete to detect only relevant stimuli and filter out or ignore irrelevant stimuli in the environment (Easterbrook, 1959). The under-aroused performer has a broad perceptual range and therefore, accepts irrelevant cues uncritically (Easterbrook, 1959). Please also refer back to the earlier discussion on the work of Ray & Cole (1985) regarding increased alpha in the left hemisphere and its association with reducing unneeded external stimulation (e.g. crowd) and distracting stimuli. The optimal performance models described above emphasize the important roles of cognition and arousal in the proper execution of sport skills.

Slobounov et al. (2005a), researched the prominent MRCPs preceding and accompanying whole body postural movements and the role of the supplementary motor area (SMA) and sensory motor cortex in the maintenance of postural equilibrium. In this study 48 subjects were baseline tested during the pre-season. None of the subjects had suffered a prior MTBI. During the season 8 of the baselined athletes suffered a concussion. Three components of the MRCP, namely the *Bereitschafts* potential ($BP_{-600\ to\ -500}$), Motor Potential ($MP_{-100\ to\ 0}$), and Movement Monitoring Potential (mean negativity measured from force-onset to 500 ms of movement production) were measured during a self-initiated anterior sway. The *Fy* signal from the force plate, indicating the initiation of forward postural sway, was used as the trigger, and epochs were established 2500 ms before and 5000 ms after its onset. The baseline was derived from the average of the segment from 1500ms to 1200 ms before the trigger point for each channel. Each epoch was visually inspected and those containing artifacts were removed. At least 50 trials were averaged for each condition. The changes of the MRCP subcomponents amplitude in the temporal course prior to and after brain injury (main effect for the factor "testing day") at certain brain areas (main effect for the factor "electrode site") within subjects were subjected to repeated-measures ANOVA. The variable groupings were also used in a sectioned analysis that involved grouping the electrode sites based on anatomical location to detect differences between general functional areas within the brain. The anterior areas of the brain, but not the posterior areas of the brain, are highly vulnerable to damage after MTBI, resulting in long-term cognitive and behavioral impairment (Slobounov et al., 2005a). Therefore, the electrode-front-back groupings are used to find possible differences between the anterior (F3, Fz, F4), central (C3, Cz, C4), and posterior (P3, Pz, P4) sites. The confidence interval for all ANOVAs that were conducted was set at 95%. When using repeated-measures ANOVA, all *F*-ratios were assessed using degrees of freedom corrected with the Greenhouse-Geisser procedures for controlling Type1 error (Jennings et al., 1987).

Prior to brain injury, a slowly rising DC negativity ($BP_{-600\ to\ -500}$) was observed in all subjects under study, starting –1500 ms prior to initiation of postural sway predominantly at anterior and central electrode sites. From about 200 ms prior to the onset of postural sway, the amplitude ($MP_{-100\ to\ 0}$) increased more rapidly, and was maintained throughout the duration of postural sway (MMP). The maximal negativities of $MP_{-100\ to\ 0}$ were observed at Cz electrode site. Three days after injury, the $BP_{-600\ to\ -500}$ component was absent and negativity departed from baseline –250 ms prior to initiation of postural sway, although the $MP_{-100\ to\ 0}$ component was detected. MMP component did not show as pronounced. On day 10 and day 30 post-injury, the $BP_{-600\ to\ -500}$ component still was not pronounced and the amplitude of $MP_{-100\ to\ 0}$ gradually increased, but not reach baseline. The negativity of the MMP was not as pronounced and also did not return to baseline levels. For all three MRCP components under study ($BP_{-600\ to\ -500}$; $MP_{-100\ to\ 0}$; MMP), repeated measures ANOVA revealed that the main effect factor of "testing day" was significant ($p < 0.01$) and none of the MRCP components reached baseline level within 30 days post-injury. Also, the ANOVA revealed a significant main effect for the factor "electrode site" grouping ($p < 0.01$), suggesting the alteration of MRCP components predominantly at the anterior and central areas. For all MRCP components under study, repeated-measures ANOVA revealed a significant alteration of MRCP at anterior (F3, Fz, F4) and central (C3, Cz, C4) electrode sites ($p<0.01$) but not at posterior sites (P3, Pz, P4).

There are several specific findings of interest regarding the temporal course of MRCP alterations resulting from MTBI. First, the $BP_{-600\ to\ -500}$ component, which is traditionally reported as an index of preparation for self-initiated movement (Slobounov & Ray, 1998), was absent on day 3 post-injury and did not return to baseline level within 30 days post-injury. These results may indicate insufficient brain resource allocation (Gevins et al., 1979) and/or resource mobilization (McCallum et al., 1993) to initiate whole body postural sway within a stability region. In balance symptomatic individuals, the inability to focus attention on the task of postural recovery and efficiently recruit the cognitive resources needed for this task may be reflected in the reduced amplitude of $BP_{-600\ to\ -500}$ especially in the acute stage of MTBI. A steady increase of the BP component after one year post-TBI was reported by Wiese et al. (2004b), suggesting the use of enhanced cognitive resources during the preparation of self-initiated finger movements, partly due to recovery of frontal cortical systems. The long-term temporal course of whole body posture-related cortical potentials and underlying behavioral symptoms in subjects suffering from MTBI is awaiting future experimentations.

Numerous studies support the hypothesis that the early $BP_{-600\ to\ -500}$ reflects general aspects of voluntary movement preparation and is less sensitive to specific movement parameters (Jennings et al., 1987; Kristiva, et

al, 1990). In contrast, motor-task specific late components of the MRCP ($MP_{-100\ to\ 0}$) reflect "the central neural drive or scaling of the control signal excitation pulse" from the cortex to the involved muscle groups (Kristiva et al., 1990; Slobounov & Ray, 1998). The late $MP_{-100\ to\ 0}$ component of the MRCP, although present, was significantly reduced on day 3 post-injury and never returned to baseline level. Collectively the data from this study suggests that the residual abnormality of cortical control of postural movements in MTBI subjects is preserved up to at least 30 days following concussion. Previous studies in humans have reported that the pre-movement cortical negativity has a tendency to remain or even increase throughout the movement duration (Grunewald et al., 1983; Niemann et al., 1994). It was also reported that the longer the movement trial, the greater the MRCP duration (Niemann et al., 1994). Accordingly, this later component of the MRCP was called *the movement monitoring potential* (MMP). Thus, the magnitude and duration of the MMP may directly reflect the efficiency of regulatory mechanisms involved in motor task production (Slobounov et al., 2005b). The amplitude of the MMP significantly dropped on day 3 post-injury and remained lower than the baseline level during the entire testing period. This may indicate residual functional deficits of higher cortical structures that regulate posture and equilibrium. Finally, the alteration of the MRCP was predominantly observed at the frontal and central regions of the brain with a maximum at the Cz electrode site. It is well documented that frontal areas of the brain are highly vulnerable to damage after mild traumatic brain injury, resulting in long-term behavioral impairment (Struss, 2002). The reduced amplitude and delayed onset of MRCPs in concussed subjects may result from deranged neuronal input from the prefrontal cortex into the SMA. On the functional level, this may correspond to a loss of motor preplanning caused by frontal lobe damage (Weiss, 2004a). Posture recovery, an essential skill in any sport, has been shown to be a demanding task requiring cognitive work (Guskiewicz, 2003). As with the Thompson et al. (2005) study, by taking EEG measures during cognitive loading, residual damage from MTBI that would have otherwise gone unnoticed was detected.

4. BALANCE DEFICITS POST CONCUSSION

Balance and the maintenance of a stable posture are important features of athletic performance, and as such the level of postural degradation resulting from MTBI should be of paramount importance when assessing the effects of any such injury. Balance can be defined as the process of maintaining the center of gravity (COG) within the body's base of support (Guskiewicz, 2001). The system responsible for the maintenance of balance is a complex one and involves the integration of many cortical and

peripheral feedback mechanisms. The maintenance of balance is controlled through a hierarchy involving three separable levels (Guskiewicz, 2001). The highest level involves areas of the brain responsible for attention, concentration and memory, as well as the association cortex responsible for receiving and integrating input from other brain structures. The middle level involves the sensorimotor cortex, cerebellum, parts of the basal ganglia and some brainstem nuclei. Postural reflexes (afferent pathways from the eyes, vestibular apparatus, and proprioceptors) occur at this level, as do the efferent pathways (alpha motor neurons) controlling skeletal muscle, and the neurons of the integrating centers in the brainstem and spinal cord. The lowest level consists of the brainstem and spinal cord from which motor neurons exit (Guskiewicz, 2001).

In addition to the cognitive deficits reported following MTBI, it has been reported that areas of the brain associated with the maintenance of equilibrium (visual, somatosensory, and vestibular systems) are also negatively affected by concussion (Ingersoll & Armstrong, 1992; Haaland et al., 1994; Guskiewicz et al., 1997; Guskiewicz, 2001; Guskiewicz et al., 2001; Oliaro et al., 2001). In concussed individuals performing a bipedal stance, Geurts et al. (1999) found increases in the velocity of center of pressure and overall weight shifting speed when compared with normals. This was considered indicative of instability in both static and dynamic postures. In a study conducted on college athletes it was concluded by Slobounov et al., 2002, that there are transient functional changes in the brain that are associated with motor control and coordination in MTBI subjects. Slobounov et al., 2000, also found a decrease in EEG power in concussed individuals during a task requiring the recognition of unstable postures and thus inferred that the ability of people who have sustained a MTBI to recognize the limits of their functional boundaries may be impaired. These findings may result from damage to the brain that causes reduced local excitation as well as a reduced synchronization of the active generators of the higher frequency bands as measured by EEG (Thatcher et al., 1998).

Ratio scores were calculated in the Guskiewicz et al. (2001) experiment to reveal relative differences between the equilibrium scores of each of the sensory modalities involved with maintaining balance. Lower scores indicated an inability to compensate for disruptions in selected sensory modalities. The recovery of stability in the injured subjects coincided with reported ratio scores of the visual and vestibular systems suggesting that postural stability deficits in injured subjects could be linked to sensory integration problems that result from concussion (Guskiewicz et al., 2001). It is also suggested by Guskiewicz et al. (2001) that postural instability following a concussive incident could result from; (1) slowed subcortical activity and spatiotemporal disruption of postural responses (2) minor axonal disruption or (3) the abnormal metabolic cascade that may affect cortical

neurons responsible for sending information to centers responsible for the maintenance of posture. Timelines for the recovery to baseline levels of postural stability seem to run in the course of 1 – 3 days (Guskiewicz, et al., 2001). Other studies confirm the findings and recovery curves suggested by Guskiewicz et al. (2001). In a previous study, (Guskiewicz et al., 1997) and follow-up study (Guskiewicz, 2001), it was shown that injured subjects were significantly less stable than age matched normals on day 1 of testing and significantly less stable than their own pre-injury scores on day 3. Evidence of the recovery of postural stability in individuals suffering a mild injury was also shown in a study conducted by Ingersoll & Armstrong (1992), in which a difference between subject groups (all subjects injuries occurred greater than one year prior to testing) was not present for MTBI compared to normals but was present in the severely injured group.

Testing for balance impairments provides information concerning the functional abilities of the patient. The Romberg test has proven effective as a physical test of vestibular impairments (Ingersoll & Armstrong, 1992). Modifications to the Romberg test allow for the additional assessment of impairments in patients visual and proprioceptive systems. This adaptated test has been validated by Ingersoll & Armstrong (1992), Guskiewicz, (Guskiewicz, 2001; Guskiewicz et al., 2001) and Oliaro et al. (2001). The calculation of the functional area within which a person will move as a function of their base of support has been termed the index of stability (Slobounov et al., 1998). Testing the ability or willingness of subjects to move toward these limits of their base of support has been shown to be effective in distinguishing between concussed and non-injured individuals. As noted above, it is generally found that a return to baseline postural stability usually occurs within a 1 - 3 day period (Guskiewicz, 2001; Guskiewicz et al., 2001; Oliaro et al., 2001). These results are stable across different tests of stability, including such alterations as bi-pedal static posture, single legged stance, removal of visual inputs, manipulation of visual inputs, or manipulation of the testing platform or surface (Ingersoll & Armstrong, 1992; Haaland et al., 1994; Guskiewicz et al., 1997; Guerts et al., 1999; Guskiewicz, 2001; Guskiewicz et al., 2001; Oliaro et al., 2001). Our results are consistent with these findings. Thompson et al. (2005), utilized three stability tests; eyes open (EO) static posture, eyes closed (EC) static posture, and index of stability to discern between injured and normal subjects. Subjects were assigned to either a healthy (normal controls, n = 12) group or an injured (MTBI, n=12) group based on their complete medical and concussion history at the time of testing. Injured subjects (collegiate football, ice hockey and rugby players) were classified as those that had incurred a grade 1-2 MTBI as assessed by a team physician. Time between injury and testing date in the injured group ranged from 70 to 131 days (mean = 89.4 days). All subjects were asymptomatic at the day of testing and were cleared for sport participation based upon

neuropsychological assessment and clinical symptoms resolution. In the EO and EC conditions center of pressure (COP) was used as the stability measure. COP was measured as 95% ellipse area in inches. Independent-samples t-tests were run to determine if significant differences existed between injury groups in the EO condition and separately in the EC condition. In the EO condition there were not significant differences found between groups (see Fig.5a).

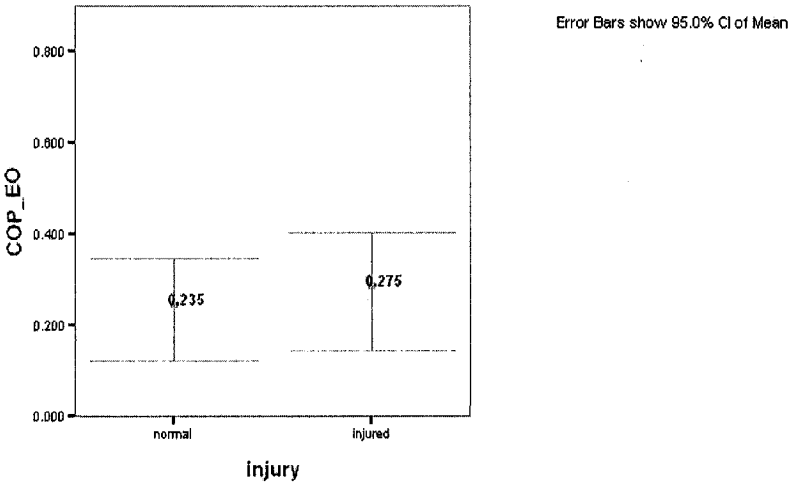

Fig. 5a. Averaged across subjects' the center of pressure (COP) area obtained from normal controls and MTBI subjects during bipedal stance in eyes opened (EO) testing condition. (Re-printed with permission from Elsevier: Thompson, J., Sebastianelli, W., Slobounov, S. (2005). EEG and Postural Correlates of Mild Traumatic Brain Injury in College Athletes. *Neuroscience Letters, 377,* 158-163.)

However, in the eyes closed (EC) condition the MTBI group showed an increased COP area ($p < 0.05$) (Fig.5b).

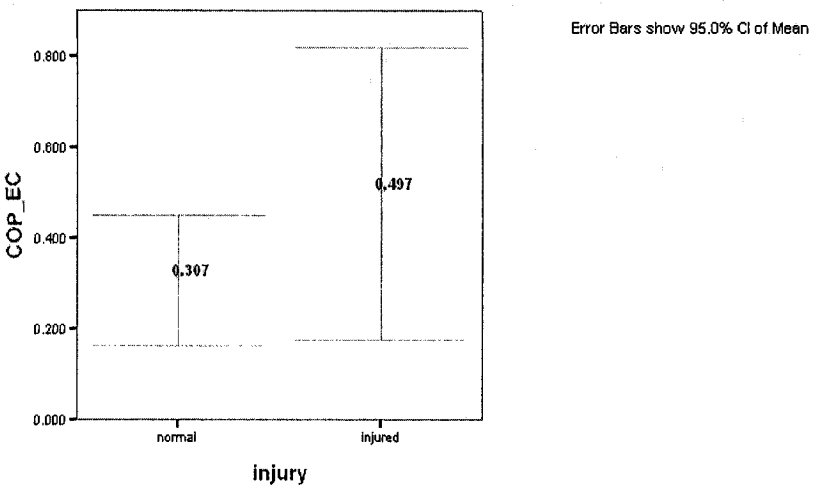

Fig. 5b. Averaged across subjects' the center of pressure (COP) area obtained from normal controls and MTBI subjects during bipedal stance in and eyes closed (EC) testing condition. (Re-printed, with permission, from Elsevier: Thompson, J., Sebastianelli, W., Slobounov, S. (2005). EEG and Postural Correlates of Mild Traumatic Brain Injury in College Athletes. *Neuroscience Letters, 377,* 158-163.)

Behaviorally, a systematic increase in the center of pressure (COP) area, an indication of postural instability (Slobounov et al., 2005a), was observed in concussed subjects performing postural task when vision was not available. This finding is consistent with our previous study that demonstrated motor deficiencies in MTBI subjects when the complexity of a force production task was increased (Slobounov et al. 2002). This is also in agreement with previous neuropsychological research suggesting that the differences in error and reaction time (RT) tests between concussed and control subjects became more obvious when behavioral task demands increased (Hugenholtz et al., 1988). The observed postural instability in concussed individuals under the no vision condition may occur because of an increased reliance on visual input during postural tasks. The negative effect of MTBI on postural stability, which was attributed to a sensory interaction problem between visual, vestibular and somatosensory systems (Guskiewicz, 2003), may be detected far beyond 7 days post-injury if properly assessed. Overall, behavioral data support the working hypothesis that long-term residual balance problems may be observed in concussed individuals provided that appropriate research methodology is implemented.

In a follow-up study, Slobounov et al. (2005a), tested 48 subjects prior to the start of their playing seasons. None of these subjects had a concussion

at the time of baseline testing based on current return to play standards. Eight of these subjects suffered a grade 1-2 MTBI within six months after baseline testing, as assessed by a team physician. These subjects were tested again on day 3, day 10 and day 30 post-injury. All subjects were asymptomatic at day 10 of testing and were cleared for sport participation based upon neurological and neuropsychological assessments as well as clinical symptoms resolution. Postural recordings were taken under three experimental conditions: static postural tasks - eyes open (EO) standing, eyes closed (EC) standing; and dynamic tasks - the whole body anterior-posterior (AP) postural movements. All standing trials were performed using a bipedal stance on an AMTI force plate. For the static standing trials, subjects were instructed to remain as still as possible for 30s. For the dynamic AP task, subjects were requested to produce self-initiated discrete whole body postural movement in the forward direction. Subjects were instructed to sway forward as far as they could to the limits of their stability boundary, at a comfortable speed without moving their feet. Subjects were instructed to produce eyes open postural sways at a self-paced rate of approximately one every 10s. Subjects performed 60 postural sways in each session. There were 2 sessions for this task condition. For the whole body AP postural movement subjects were instructed to lean forward and backward with maximal range of motion predominantly at the ankle joints. As with the Thompson et al. (2005) study, COP was measured as 95% ellipse area in inches. A repeated-measures ANOVA was conducted to test for the effects of testing date (injury condition) and vision conditions on COP measures. For the static balance tasks, the main effect of testing date was significant ($p < 0.05$). There was also an interaction of vision and testing day ($p< 0.05$), with significantly larger area of COP on day 3 post-injury (Fig.6). Independent-samples *t*-tests revealed significant differences between baseline testing and day 3 post-injury for both vision conditions ($p < 0.05$). Also, significant differences were observed between baseline testing and day 10 post-injury only for closed eyes conditions ($p < 0.05$). No differences were observed between baseline testing and day 30 post-injury regardless of vision conditions ($p >0.05$). Overall, a) the effect of injury on balance during static tasks was most obvious on day 3 post-injury; and b) vision availability was a significant factor influencing postural stability at least within 10 days post-injury; and c) balance problems during static tasks are fully cleared within 30 days post-injury.

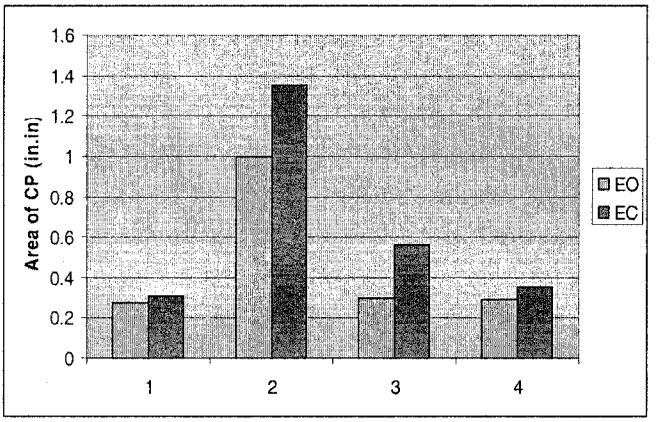

Fig.6. Temporal course of the area of Center of Pressure (CP) during static postural task performance with eyes open and eyes closed conditions. X-axis, 1- baseline testing; 2- day 3 post-injury; 3 – day 10 post-injury; 4 – day 30 post-injury. (Re-printed, by permission, from Elsevier: Slobounov, S., Sebastianelli, W., Moss, R. (2005). Alteration of Posture-Related Cortical Potentials in Mild Traumatic Brain Injury. *Neuroscience Letters, 383,* 251-255.)

For the dynamic postural task, the main effect of testing day was significant ($p< 0.05$), indicating the subjects' reduced mobility at least within 10 days post-injury (Fig.7). Independent-sample *t*-tests revealed significant differences between baseline testing and day 3 and day 10 post-injury ($p < 0.05$). No differences were observed between baseline testing and day 30 post-injury ($p >0.05$).

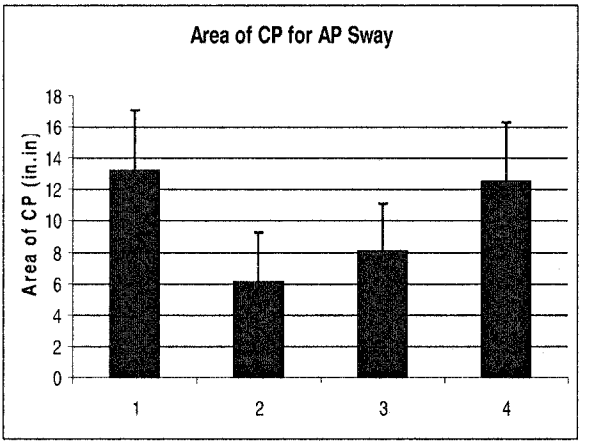

Fig. 7. Areas of the center of pressure (CP) during dynamic AP postural task performance. X-axis same as Fig.6. (Re-printed, by permission, from Elsevier: Slobounov, S., Sebastianelli, W., Moss, R. (2005). Alteration of Posture-Related Cortical Potentials in Mild Traumatic Brain Injury. *Neuroscience Letters, 383,* 251-255.)

Based on the results 2 main conclusions were drawn: (1) Indices of postural instability during static tasks were most pronounced at day 3 post-injury and basically resolved within 10 days post-injury, although, vision availability may influence postural stability in concussed subjects beyond 10 days post-injury. (2) Symptoms of reduced dynamic stability may be observed within 10 days post-injury if more challenging postural tasks are introduced (Slobounov et al., 2005a). A shortcoming of postural assessments for use in return to play measures lies in the transient nature of measurable balance impairments. Comparing the timeframes for recovery of neurologic pathology to postural stability, it seems as though the recovery times, as measured by the above studies, do not coincide. This may suggest that the normal pathways associated with control of balance and stability have not actually recovered since it has been shown that cortical damage from the injury has not fully recovered. Two plausible explanations may account for this mismatch in recovery times. First, the human nervous system has the ability to reorganize itself and adapt to changes. This remarkable trait is termed neural plasticity (Boroojerdi, et al., 2001; Staudt et al., 2002). This plasticity may allow alternate, undamaged neuronal pathways to perform the duties responsible for basic posture. Second, the currently used balance assessments may not adequately tax the systems involved in maintaining balance under the high demands of athletic competition. Alternatively, it may be a combination of the two above-mentioned shortcomings that may explain the mismatch between functional and physiological recovery.

CONCLUSION

Throughout this chapter the recurring message is that, following a MTBI, detectible effects on cortical functioning can be measured. Following a concussive incident the new state of equilibrium of the cerebral cortex is characterized by reduced amplitude in EEG frequencies. This EEG pattern is seen consistently in concussed individuals and persists over a prolonged time period, potentially years (Thatcher et al., 1989). The new state of functional organization may be helpful in the recovery of physical function in that it allows the individual to manipulate, operate on, and cope with sensory inputs, abstractions, and motor output demands. The importance of this reorganized EEG pattern may be the degree to which it allows injured subjects to utilize available cerebral resources (Thatcher et al., 1989). Although this may not cause day-to-day problems for many individuals, people who have been operating at or close to their capacity, in terms of cortical functioning and the allocation of resources to meet high cognitive and motor demands, may suffer significant consequences (Potter et al., 2001). High levels of competition require athletes to utilize numerous

cognitive resources simultaneously. If simple one-dimensional test conditions are used as the basis for return to play decisions (e.g., neuropsychological or balance testing used alone), residual impairments may go undetected. During ensuing competitions, when numerous cognitive resources need to be utilized simultaneously there may be detrimental effects on performance or, worse yet, risk to the athletes themselves, such as another concussion.

It has been shown that changes in surface EEG recordings represent the cortical activity involved in the performance of physical tasks (Aoki et al., 1999; Mima et al, 1999; Slobounov et al., 1999; Brown, 2000; Slobounov et al., 2001; Alegre, 2003), and that visual recognition of non-stable postures causes changes in the EEG (Slobounov et al., 2000). This information and results from numerous studies (Solbakk et al., Potter et al., Thompson et al., Thatcher et al. and numerous others mentioned in this chapter) lend support to the use of EEG combined with functional testing for athletes prior to their return to competition. Using this joint testing method, practitioners can improve upon the current return to play measures and increase the likelihood that the athletes they are sending back to the competitive sporting environment are cognitively ready to perform at their pre-concussion levels. Based on the above information, a concussion assessment that uses the combination of EEG, QEEG with LORETA analysis, and motor function tests is the best option currently available to assess concussion severity and upon which to base return to play measures. By integrating such multi-faceted tests into concussion protocol measurements, researchers are quickly moving toward the development of baseline and return to play evaluations that will give clinicians more valid and reliable measures upon which they can base mild traumatic brain injury diagnoses and return to play decisions.

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