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Alteration of posture-related cortical potentials in mild traumatic brain injury

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Abstract

This paper presents additional evidence showing the persistent functional deficits in concussed athletes as revealed by altered movement-related cortical potentials (MRCP) preceding whole body postural movements at least 30 days post-injury. Eight student-athletes participated in this study (a) prior to injury; and (b) 3, 10 and 30 days after MTBI. EEG was recorded while subjects produced static balance tasks and dynamic postural movements. All subjects were cleared for sport participation within 10 days post-injury based upon neurological and neuropsychological assessments as well as upon clinical symptoms resolution. There was a persistent reduction of MRCP amplitude prior to initiation of postural movement up to 30 days post-injury, although abnormal postural responses basically recovered within 10 days post-injury. The frontal lobe MRCP effects were larger than posterior areas. This supports the notion that behavioral symptoms resolution may not be indicative of brain injury resolution. Overall, persistent alteration of movement-related cortical potentials after MTBI may indicate residual disturbance of neuronal networks involved in preparation and execution of postural movements and a lower threshold for brain re/injury.

Keywords: Movement-related cortical potentials (MRCP); Mild traumatic brain injury (MTBI); Posture; Balance

There is a growing body of knowledge indicating long-lasting residual abnormalities [2,21] including the brain dysfunctions that may persist up to 10 years after mild traumatic brain injury [24]. Athletes with a mild traumatic brain injury (MTBI) often complain of symptoms long after injury, even though their acute clinical abnormalities and measurable cognitive and behavioral deficits are cleared. Incomplete recovery after a mild traumatic brain injury may also increase the risk of *second impact syndrome* [1], which is a significant health problem faced by the medical community today. Therefore, the ability to identify any residual functional impairment after MTBI is critical to prevention of brain re-injury.

In our previous work, we examined the residual effect of MTBI on movement-related cortical potentials (MRCP) preceding and accompanying isometric force production tasks [21]. Significant behavioral and electro-cortical changes between MTBI subjects and controls were observed especially when the complexity of force production task was increased. Specifically, there was a reduction of amplitude of MRCP prior to initiation of a higher degree of complexity force production task. This finding has been confirmed in a more recent line of research demonstrating the temporal course of MRCP in patients with contusions to the prefrontal cortex up-to 52 weeks post-injury [26,27]. Overall, these findings indicate prolonged malfunctioning of neuronal networks due to the incomplete recovery of the prefrontal cortex after MTBI.

Recently, the prominent MRCP preceding and accompanying whole body postural movements have been documented [18]. The observed MRCP waveforms, amplitude and spatial distribution were similar to those reported for voluntary finger [16,11], wrist [22], elbow [5], and/or leg [15] movement. The largest amplitude of MRCP was found at fronto-central electrode sites with maximum at Cz, suggesting the possible source in supplementary motor area (SMA) and the foot area of the sensori-motor cortex responsible for initiation of postural movements. The results from this study confirm the important role of higher cortical structures in regulation of postural equilibrium.

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Persistent balance problems in acute traumatic brain injury have been reported in numerous studies [4,6]. Postural instability in MTBI subjects has been attributed to disruption of brain functions within the brainstem, cerebellum and frontal lobe [6,7], but there have been no direct examinations of this hypothesis. We hypothesize that observed postural instability in MTBI subjects may be related to impaired cortical functions for balance. As stated by Jahanshahi and Hallett [8], the movement-related cortical potentials are not static, but may be altered as a function of recovery from brain injury. Accordingly, the primary aim of this study was (a) to examine MRCP associated with initiation of postural movement prior to mild traumatic brain injury; and (b) to identify residual alterations of MRCP associated with production of postural movement in individuals suffering from MTBI. To our knowledge this is the first study examining MRCP in the temporal course prior to and right after mild traumatic brain injury.

A total of 48 subjects were initially recruited for this experiment. All subjects were Pennsylvania State University athletes at risk for traumatic brain injury (collegiate football, ice hockey and rugby players), male, aged between 18 and 25 years (mean = 20.95 years). None of these subjects had a concussion history at the time of baseline testing. 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 based upon clinical symptoms resolution. A detailed description of inclusion criteria for MTBI can be found [21]. Consent forms approved by the Institutional Review Board of the Pennsylvania State University were obtained from each subject prior to each testing.

EEG 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 30 s (EEG data during static tasks will be fully discussed in our future reports). 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 respective stability boundary at comfortable speed without moving their feet. Subjects were instructed to produce postural sways with opened eyes at a self-paced rate of approximately once every 10 s. Subjects performed 60 postural sways in each session. There were two 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. A detailed description of the experimental procedure and setup can be found [21,22]. The continuous EEG activity from the scalp was recorded at 19 sites: FP1, FP2, FZ, F3, F4, F7, F8, CZ, C3, C4, T3, T4, T7, T8, PZ, P3, P4, O1, O2 according to the International 10–20 system [9a]. The ground electrode was located 10% anterior to FZ, linked earlobes served as references and electrode impedances were below 10 kΩ. The Ag/AgCl electrodes mounted in a Quickcap Electrode Helmet (NeuroScan, Inc., El Paso, TX, USA) were used in this study. EEG signals were recorded using a programmable DC coupled broadband SynAmps amplifier (NeuroScan, Inc.). The EEG signals were amplified (gain 1000, range set for ±55 mV) and bandpass filtered in the dc to 70 Hz frequency range. The EEG data were sampled at 250 Hz, using a separate 16-bit analog-to-digital converter for each channel. Data were collected using NeuroScan's Scan 4.2 software package and written to and stored on a Dell Precision 530 computer running an Intel XEON processor. A detailed description of EEG setup can be found [21,25]. Postural data was collected using an AMTI (Advanced Mechanical Technologies, Inc., Watertown, MA, OR6-7-1000) force plate. Data were collected at a 90 Hz sampling rate, sent to an AMTI Model ADI-32 interface box, processed through the ANTI MiniAmp MSA-6 amplifier, and synchronized with EEG records.

The EEG signals were first corrected for eye movements (ocular artifact reduction option of NeuroScan's Scan 4.1 software). The *Fy* signal from the force plate indicating the initiation of forward 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 1500 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 amplitude of MRCP was measured as: (i) the mean negativity measured between 600 and 500 ms prior to force onset referred to as the *Bereitschaftspotential* ($BP_{-600\,\text{to}\,-500}$) reflecting the cortical activation associated with the early stages for postural movement preparation; (ii) the mean negativity measured between 100 ms prior to force onset and force onset referred to as the *motor potential* ($MP_{-100\,\text{to}\,0}$) reflecting the cortical activation associated with later stages for postural movement preparation [10,11], (iii) the mean negativity measured from force-onset to 500 ms of movement production referred to as the *movement monitoring potential* (MMP) [5]. The MRCPs were measured at all of the above mentioned electrode sites representing the frontal, central, and parietal cortical areas.

The analysis for the force plate data was performed using AMTI's Biodaq analysis program, version 1.0 software package. Center of pressure (COP) measures were assessed in both eyes open and eyes closed conditions in both static and dynamic postural tasks. 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. The changes of the MRCP subcomponents amplitude in the temporal course prior to and after brain injury (main effect for the factor