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Performance Monitoring and Cognitive Control in Individuals with Mild Traumatic Brain Injury

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Abstract

Literature suggests that individuals with mild traumatic brain injury (mTBI) show subtle abnormalities in the cognitive control process of performance monitoring. The neural bases of performance monitoring can be measured using the error-related negaitivity (ERN) and post-error positivity (Pe) components of the scalp-recorded event-related potential (ERP). Thirty-six individuals with mTBI and 46 demographically similar controls completed a modified color-naming Stroop task while ERPs were recorded. Separate repeated-measures analyses of variance were used to examine the behavioral (response times [RT] and error rates) and ERP (ERN and Pe amplitudes) indices of performance monitoring. Both groups showed slower RTs and increased error rates on incongruent trials relative to congruent trials. Likewise, both groups showed more negative ERN and more positive Pe amplitude to error trials relative to correct trials. Notably, there were no significant main effects or interactions of group for behavioral and ERP measures. Subgroup and correlational analyses with post-concussive symptoms and indices of injury severity were also not significant. Findings suggest comparable performance to non-injured individuals in some aspects of cognitive control in this sample. Neuropsychological implications and comparison with other cognitive control component processes in individuals with TBI are provided. (*JINS*, 2012, *18*, 323–333)

Keywords: Cognitive control, Performance monitoring, Traumatic brain injury, Concussion, Stroop, ERN

INTRODUCTION

Cognitive control refers to the ability to regulate cognitive processes, detect conflict, and signal for the allocation of cognitive resources for corrective action (Botvinick, Carter, Braver, Barch, & Cohen, 2001; Kerns et al., 2004). Understanding cognitive control impairment in individuals with neurologic disorders can clarify the neurologic abnormalities underlying aberrant behavior in everyday life. For example, individuals with moderate-to-severe traumatic brain injury (TBI) show impaired regulative and evaluative cognitive control processes relative to neurologically healthy controls (Larson, Kaufman, Kellison, Schmalfuss, & Perlstein, 2009; Larson, Kaufman, & Perlstein, 2009; Larson, Kaufman, Schmalfuss, & Perlstein, 2007; Perlstein, Larson, Dotson, & Kelly, 2006; Scheibel et al., 2007, 2009). These impairments may underlie the cognitive rigidity and perseverative errors frequently demonstrated in individuals following moderate-to-severe TBI. As a result of these and other such findings, treatment approaches have begun to emphasize cognitive-control deficits in rehabilitation to target the underlying problem (e.g., Ownsworth, Fleming, Desbois, Strong, & Kuipers, 2006; Ownsworth, Quinn, Fleming, Kendall, & Shum, 2010). Although a large amount of research has focused on elucidating cognitive control impairments in individuals with moderate-to-severe TBI, it remains unclear the extent to which individuals with mild TBI (mTBI) may experience alterations in cognitive control processes.

Studies of behavioral manifestations of cognitive control (e.g., response times [RTs] and error rates) in individuals with mTBI report inconsistent findings. For example, several studies find mTBI to be associated with longer RTs on speeded tasks (Bohnen, Jolles, & Twijnstra, 1992; Ellemberg, Leclerc, Couture, & Daigle, 2007; Hartikainen et al., 2010), whereas other studies find no such RT differences, although this could be related to level of task difficulty (Broglio, Pontifex, O'Connor, & Hillman, 2009; Larson, Farrer, & Clayson, 2011; Pontifex, O'Connor, Broglio, & Hillman, 2009). Additional studies of

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post-error slowing, a regulative control index of performance monitoring, show little difference between individuals with mTBI and controls (Larson, Perlstein, Demery, & Stigge-Kaufman, 2006; Pontifex et al., 2009).

Neuropsychological indices of cognitive control in individuals with mTBI are also inconsistent; some show potential long-term cognitive deficits following injury (Pertab, James, & Bigler, 2009; Vanderploeg, Curtiss, Luis, & Salazar, 2007), whereas others maintain that individuals with mTBI will quickly return to baseline functioning after the acute phase of injury (Binder, Rohling, & Larrabee, 1997; Frencham, Fox, & Maybery, 2005). Considering inconsistencies in behavioral and neuropsychological research of mTBI, physiological measures, such as cognitive event-related potentials (ERPs), may provide insight into potential cognitive control deficits not easily detected by traditional neuropsychological or behavioral measures.

Several studies of individuals with mTBI have examined ERP components thought to reflect different aspects of cognitive control. For example, the N2 and P3 ERP components putatively reflect the regulative process of attention allocation to a task. One study found that individuals with chronic mTBI showed significant attenuation of N2 and P3 amplitudes compared to control participants despite comparable cognitive functioning between groups (Broglio et al., 2009). These findings support earlier studies that show attenuated P3 amplitudes among concussed college athletes (Dupuis, Johnston, Lavoie, Lepore, & Lassonde, 2000; Lavoie, Dupuis, Johnston, Leclerc, & Lassonde, 2004). In contradiction to these studies, a recent study found that athletes tested 2 or more years after injury had P3 amplitudes comparable to control participants, whereas concussed athletes that sustained an injury within a year of testing had attenuated P3 amplitudes (Theriault, De Beaumont, Gosselin, Filipinni, & Lassonde, 2009).

Larson et al. (2011) examined both regulative and evaluative components of cognitive control in individuals with mTBI by assessing ERP components thought to reflect neural processes underlying conflict monitoring and conflict adaptation. According to the conflict monitoring theory, following high conflict, strategic adjustments in control bias attention on task-relevant information processing to maximize performance efficiency (Botvinick et al., 2001; Kerns et al., 2004). Conflict adaptation refers to this increase in cognitive control following conflict to more proficiently complete subsequent tasks that require more attention to task-relevant information (i.e., target stimulus) such as during incongruent trials (Kerns et al., 2004). Larson et al. (2011) reported comparable conflict-monitoring processes in individuals with mTBI relative to controls; however, individuals with mTBI showed deficits in the compensatory recruitment of cognitive control suggesting that, whereas evaluative control processes may remain intact, the regulative processes may be abnormal in individuals with mTBI.

Three primary ERP components are thought to reflect performance monitoring cognitive control functions. The error-related negativity (ERN) reflects evaluative aspects of cognitive control related to performance monitoring. The ERN is a negative deflection in a response-locked ERP waveform occurring within 100 ms after the commission of an error and is related to the response conflict detected when an error is committed (Falkenstein, Hohnsbein, Hoormann, & Banke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993). The correct-related negativity (CRN) is an ERP component with identical temporal characteristics and scalp topography as the ERN, but occurs following correct responses (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000). The post-error positivity (Pe) is a positive deflection in the ERP with centro-parietal scalp distribution that occurs within 200–400 ms after conscious erroneous responses (Falkenstein et al., 1991; Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001). Various theories implicate the Pe as a reflection of conscious error processing (Larson & Perlstein, 2009; Nieuwenhuis et al., 2001) or a reflection of an emotional response to conscious errors (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005).

Only one study that we are aware of has examined evaluative control processes, as reflected by the ERN and Pe, in individuals with mTBI. Pontifex et al. (2009) examined the ERN in concussed athletes in the chronic stage of injury (mean of 2.9 $\pm$ 2.9 years post-injury) along with an ImPACT neurocognitive assessment. These researchers found that, even in the presence of normal functioning on the ImPACT, individuals with mTBI had significantly attenuated ERN amplitudes compared to controls. Considering the varying results on previous ERP studies of cognitive control in mTBI, follow-up studies are needed to confirm the findings. In addition, how researchers measure mTBI becomes an important factor. For example, research points to post-traumatic amnesia (PTA) as among the strongest predictors of functioning one year post TBI (e.g., Hiekkanen, Kurki, Brandstack, Kairisto, & Tenovuo, 2009), but no research to date has looked at the association between injury severity variables such as PTA and loss of consciousness (LoC) and neural indices of cognitive control.

Some methodological limitations of the study by Pontifex et al. (2009) may account for the mild-TBI-related findings evidenced that the current investigation will address. The study by Pontifex et al. used a spatial filter and principle components analysis (PCA) to remove ocular artifact. However, spatial PCA inadequately separates ocular artifact from ERP data as a spatial filter distorts the spatial distribution of ERP activity (Jung et al., 1998; Lagerlund, Sharbrough, & Busacker, 1997). The present examination used independent component analysis (ICA) to preserve the spatial distribution of the data (Jung, Makeig, Humphries, et al., 2000; Jung, Makeig, Westerfield, et al., 2000). This approach may be better suited for clinical and neurologic populations where movement and ocular artifact may be more prevalent (Jung, Makeig, Westerfield, et al., 2000). Furthermore, Pontifex et al. (2009) extracted ERN amplitudes as the most negative-going peak amplitude. Peak measures are more contaminated by noise in the data than mean amplitude measures (Luck, 2005). As such, the present examination used a novel statistical approach, temporospatial PCA (see Dien, 2010a), to isolate

ERN and Pe activity and subsequently extract the mean amplitude of these components.

The purpose of the present study was to investigate performance-monitoring-related cognitive control processes in individuals with mTBI and to replicate the findings by Pontifex et al. We hypothesized that injury severity would predict ERN amplitude with those experiencing more LoC and PTA having attenuated ERN amplitudes relative to those with no LoC or PTA and controls.

METHOD

All study procedures were completed in compliance with Institutional Review Board at Brigham Young University. Descriptive information, including injury characteristics and neuropsychological test data are provided in Table 1. All participants were screened for the presence of psychiatric disorders using the Mental Health Screening Form-III (Carroll & McGinley, 2001). Other exclusion criteria for both groups were current or previous psychiatric disorder, learning disability, alcohol or substance abuse, other acquired brain disorders (e.g., epilepsy, stroke), anti-epileptic or psychotropic medication use, color-blindness, uncorrected visual impairment, or participation in current litigation.

Initial study enrollment initially included 43 individuals with mTBI and 52 healthy controls. One individual with mTBI was excluded due to psychotropic medication use. To maintain an adequate signal-to-noise ratio, six individuals with mTBI and six controls with fewer than six error trials after artifact correction were also excluded (Olvet & Hajcak, 2009). Thus, final enrollment included 36 individuals with mTBI and 46 neurologically healthy controls.

Participants in the mTBI group were recruited *via* flyer and advertisement at local hospital facilities, student-athlete facilities, and campus buildings. Control participants were recruited *via* flyer and advertisement on campus and in the local community. All individuals in the mTBI group sustained a mTBI as defined by the American Congress of Rehabilitation Medicine (Kay et al., 1993) and reiterated by the World Health Organization (WHO; Carroll, Cassidy, Holm, Kraus, & Coronado, 2004). Specifically, participants endorsed one or more of the following after a blow to the head: confusion/disorientation, LoC for 30 min or less, PTA less than 24 h or Glasgow Coma Scale (GCS) score between 13 and 15 after 30 min of the injury. Mild TBI was determined by comprehensive patient and significant other interview and, when available, reviews of medical records. To ensure accurate classification we followed the interview guidelines outlined by Ruff, Iverson, Barth, Bush, and Broshek (2009) for the assessment of mTBI, but we note the limitation that LOC and PTA data were primarily provided by participant and significant-other report.

Mechanism of injury for the mTBI participants was primarily from sports-related incidents, including football, rugby, and soccer ($n = 25$; 69%). Additional injuries were sustained from falls ($n = 7$; 19%), motor vehicle accidents ($n = 2$; 6%), a bicycle accident ($n = 1$; 3%), and a sledding accident ($n = 1$; 3%). Participants were tested at least 1 month following their injury, with an average time since injury of 7 ± 8 months (range: 1 month to 45 months). Twenty-four of 36 participants (67%) reported loss of consciousness (LoC); average length of LoC was approximately 1.65 min (±2.23; range: .1 to 10 min). Twenty-five of 36 participants (69%) experienced post-traumatic amnesia

Table 1. Descriptive Information for Mild TBI and Control Participants

	Mild TBI ($n = 36$)		Control ($n = 46$)		Analysis	
	Mean	*SD*	Mean	*SD*	*t*	*p*
Age (yrs)	21.6	2.4	20.7	2.2	−1.79	.08
Average educational level (yrs)	14.3	1.2	14.1	1.5	−.6	.52
BDI-II score	8.1	7.7	4.2	3.7	−3.0	.004
STAI-State	50.2	18.4	28.3	7.3	−7.4	<.001
STAI-Trait	34.7	9.0	32.1	6.3	−1.5	.13
Rivermead Post-Concussion score	20.0	14.0	0.5	3.1	−9.2	<.001
Rey-AVLT total recall (trials 1-5)	55.1	7.7	57.1	7.5	1.2	.23
Rey-AVLT short-delay recall	11.5	2.2	12.5	2.0	2.0	.05
Rey-AVLT long-delay recall	11.5	2.8	12.0	2.9	0.7	.48
WMS-R Logical Memory I total	27.2	6.8	26.2	8.7	−.6	.55
WMS-R Logical Memory II total	23.1	6.8	22.4	9.7	−.4	.72
Digit Span forward (max# digits)	10.9	2.0	10.7	2.0	−.5	.61
Digit Span backward (max# digits)	7.9	2.4	7.8	2.0	−.2	.85
Trail Making Test Part A (seconds)	17.8	5.7	16.8	3.8	−.94	.35
Trail Making Test Part B (seconds)	42.2	13.6	44.4	13.2	0.7	.46
COWAT total	45.5	9.0	43.5	9.4	−1.0	.34
Category fluency total	23.4	4.6	23.5	3.7	0.1	.89

Note. BDI-II = Beck Depression Inventory-2nd Edition; STAI = State Trait Anxiety Inventory; Rey-AVLT = Rey Auditory-Verbal learning Test; WMS-R = Wechsler Memory Scale-Revised Edition; COWAT = Controlled Oral Word Association Test.

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(PTA); average estimated PTA was 118.60 ± 346.75 min (range: .1 to 1440 min). Twenty-three of 36 participants (64%) reported previous mTBIs; median number of prior mTBIs was 2 (range: 1 to 6).

Male-to-female ratio did not significantly differ between groups, $\chi^2(1) = 0.04$, $p = .85$; there were 18 males and 18 females in the mTBI group and 22 males and 24 females in the control group. All participants completed the Beck Depression Inventory, 2nd Edition (BDI-II; Beck, 1996) and State-Trait Anxiety Inventory (STAI; Speilberger, Gorusch, & Lushene, 1970). Post-concussion symptoms were assessed using the Rivermead Post-Concussion Symptoms Questionnaire (King, Crawford, Wenden, Moss, & Wade, 1995). Individuals with mTBI showed increased levels of state anxiety, depression, and post-concussive symptoms compared to controls (see Table 1).

Assessment of Cognitive Functioning

To characterize the cognitive functioning of the sample, all participants completed a battery of neuropsychological tests. Measures administered included the Rey Auditory-Verbal Learning Test (Rey, 1964), the Wechsler Memory Scale-Revised (WMS-R) Logical Memory I and II subtests (Wechsler, 1987), the Digit Span forward and backward subtests from the Wechsler Adult Intelligence Test–Third Edition (Wechsler, 1997), the Trail Making Test Parts A and B (Reitan, 1958), the Controlled Oral Word Association Test [COWAT] and Category Fluency (Benton & Hamsher, 1976). Individuals with mTBI did not significantly differ from control participants on the majority of measures of neuropsychological functioning; the only exception being the Rey Auditory-Verbal Learning Test short delay recall (Table 1).

Computerized Experimental Task

Participants performed a modified color-naming version of the Stroop task wherein they were presented with a Stroop stimulus with the words red, green, or blue printed in red, green, or blue font. Congruent trials consisted of words presented in their same color of font (e.g., BLUE printed in blue font); incongruent trials consisted of color-words shown in a different color of font (e.g., BLUE printed in red font). Participants were instructed to respond as quickly and accurately as possible to the color of the word with a button press to one of three response keys using the index, middle, and ring fingers of their right hand. Color-to-key mapping was practiced before task performance using 40 presentations of each color-key combination. Stimuli were presented for 1.5 s followed by a 1.5-s duration fixation cross. Participants were presented five blocks of 100 trials (500 total trials); 50% of trials were congruent and 50% of trials were incongruent.

Electrophysiological Data Recording

Electroencephalogram (EEG) was recorded from 128 scalp sites using a geodesic sensor net and Electrical Geodesics, Inc. (EGI; Eugene, OR) amplifier system (20 K nominal gain, bandpass = .10–100 Hz). Electroencephalogram was referenced to the vertex electrode and digitized continuously at 250 Hz with a 24-bit analog-to-digital converter. Impedances were maintained below 50 kΩ consistent with recommendations of the manufacturer. Data were high-pass filtered at .1 Hz and low-pass filtered at 30 Hz.

Event-related Potential Measurement

Individual-subject response-locked averages were calculated using a window from 300 ms before participant response to 700 ms following participant response. Trials containing errors of omission were excluded from averages. Eye blinks were removed from the segmented waveforms using ICA in the ERP PCA Toolkit (Dien, 2010b). The ICA components that correlated at .9 with the scalp topography of two blink templates, one generated based on the current data and another provided by the ERP PCA Toolkit author, were removed from the data (see Dien, Michelson, & Franklin, 2010). Channels were marked bad if the fast average amplitude exceeded 100 μV or if the differential average amplitude exceeded 50 μV. Data were average re-referenced and waveforms were baseline corrected using a 200 ms window from −300 ms to −100 ms before stimulus presentation.

To extract the aforementioned ERP components, temporospatial PCA (Dien, 2010a) was conducted (see Foti, Weinberg, Dien, & Hajcak, 2011, for a similar approach) using the ERP PCA Toolkit (Dien, 2010b). We followed previously published guidelines to extract ERP components (see Dien, 2010a; Dien, Beal, & Berg, 2005; Dien, Khoe, & Mangun, 2007). All single subject averages were included in the PCA; factors were chosen based on scree plots using the parallel test (Horn, 1965). A temporal PCA with Promax rotation using all time points from single subject averages as variables with participants, correct and error trials, and electrodes as observations was first conducted and yielded 16 temporal factors (TFs). A spatial PCA with Infomax rotation using electrode sites as variables and participants, correct and error trials, and temporal factors as observations followed and yielded six spatial factors (SFs; Dien, 2010a; Dien et al., 2007).

Electrode sites for analysis were chosen based on the scalp distribution of the temporospatial factors reflecting the ERP components of interest and previous research (e.g., Falkenstein et al., 2000; Gehring et al., 1993; Overbeek et al., 2005). Temporal factor 3 spatial factor 1 (TF3SF1) matched the expected scalp topography and timing of the CRN and ERN (see Figure 1); thus, the ERN was averaged across electrodes that showed robust differences between correct and error trials (electrodes: 7, 31, 55, 80, 106, Ref [Cz]; see Clayson & Larson, 2011 for sensor layout). Correct-trial and error-trial ERN amplitudes were extracted as the mean amplitude from 0 to 200 ms following the response. For the Pe, TF2SF1 matched the temporal and topographic characteristics of the Pe (see Figure 1). The Pe was averaged across electrodes showing robust correct- and error-trial

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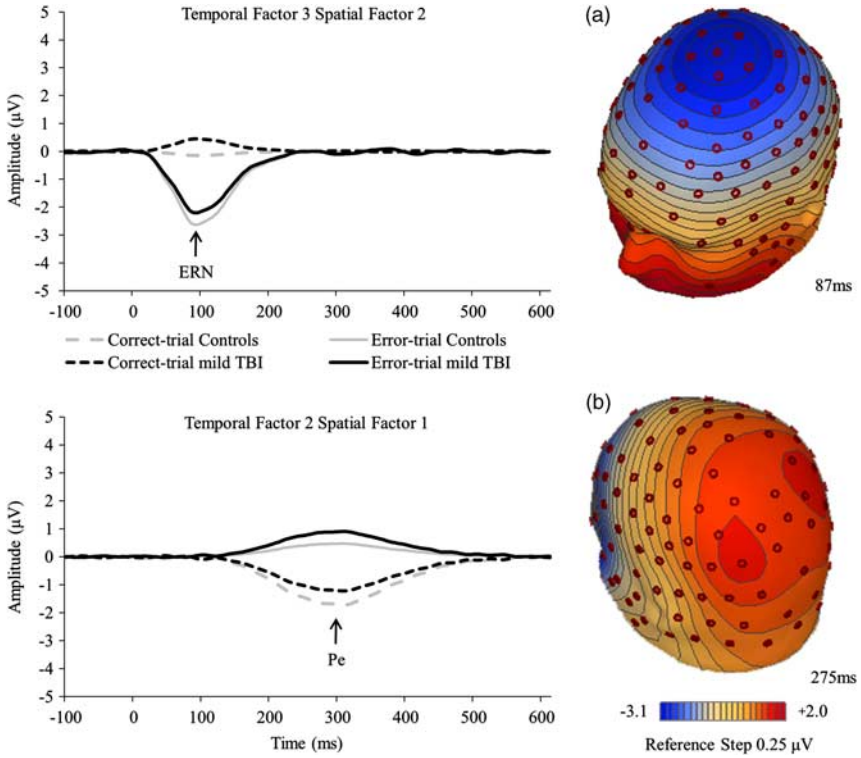

Fig. 1. Spline-interpolated voltage maps are displayed for the error-minus-correct difference activity for A) temporal factor 3 spatial factor 1 (ERN) and B) temporal factor 2 spatial factor 1 (Pe). The grand average waveforms as a function of group and accuracy representing temporal factor 3 spatial factor 1 for the error-related negativity (ERN) and temporal factor 2 spatial factor 1 for the post-error positivity (Pe).

differences (electrodes: 54, 55, 61, 62 [Pz], 78, and 79). Error-trial and correct-trial Pe amplitudes were extracted as the mean amplitude from 200 to 400 ms post-response.

Previous research indicates that it is inappropriate to analyze latency differences after conducting a temporal PCA (Dien, Spencer, & Donchin, 2004); thus, a centroid latency measurement on single subject averages before PCA was used. As a traditional peak measurement of latency represents the mode of the waveform rather than the central tendency, we used a centroid measurement that better characterizes the central tendency of the ERP component latency by using the area under the curve (see Dien et al., 2004 for formula; see also Luck, 2005). The centroid latency was derived using the abovementioned range of time points of interest for the ERN (0 to 200 ms) and Pe (200 to 400 ms).

Data Analysis

To overcome the potential biasing effects of non-normality and (co)variance heterogeneity between groups as well as to reduce Type I error (see Dien & Santuzzi, 2005), robust analyses of variance (ANOVAs) were conducted on trimmed means (5% symmetric trim) using the ERP PCA Toolkit. Although robust statistics are more conservative than conventional ANOVAs, *p*-values are interpreted in the same manner.

Separate robust 2-Group (mTBI, controls) × 2-Congruency (congruent, incongruent) ANOVAs were conducted for RTs and error rates. Robust 2-Group × 2-Accuracy (correct, incorrect) ANOVAs were used to analyze post-correct and post-error RTs and error rates as well as ERN amplitude and Pe amplitude. Pearson's correlations were used to investigate the relationship between indices of TBI severity and ERP indices. For correlation analyses in individuals with mTBI, time since injury, length of LoC, length of PTA, number of previous head injuries, and Rivermead Post Concussion Symptoms scores were correlated with ERP component amplitudes and error minus correct difference waves.

RESULTS

Behavioral

Mean RT and error rate data as a function of group are presented in Table 2. The Group × Congruency robust ANOVA on RTs indicated a significant main effect of congruency, with longer RTs shown for incongruent trials compared to congruent trials, $T_{WJt}/c(1.0,73.9) = 356.08$, $p < .001$. The main effect of group, $T_{WJt}/c(1.0,57.8) = 0.90$, $p = .35$, was not significant. The Group × Accuracy interaction was not significant, $T_{WJt}/c(1.0,73.9) = 1.59$, $p = .21$.

A Group × Congruency robust ANOVA on error rates indicated larger error rates for incongruent than congruent trials, $T_{WJt}/c(1.0,54.7) = 34.54$, $p < .001$. The main effect

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Table 2. Mean Behavioral and Electrophysiological Summary Data as a Function of Group

	Mild TBI ($n = 36$)		Control ($n = 46$)	
	Mean	*SD*	Mean	*SD*
Congruent-trial RT (ms)	623	88	603	75
Incongruent-trial RT (ms)	713	101	699	101
Congruent-trial error rates (%)	2	2	3	6
Incongruent-trial error rates (%)	5	3	8	15
Post-correct RT (ms)	666	94	648	85
Post-error RT (ms)	726	105	727	117
Post-correct error rates (%)	4	2	5	8
Post-error error rates (%)	7	7	9	16
CRN amplitude (μV)	0.3	0.7	0.2	0.6
ERN amplitude (μV)	−1.0	1.0	−1.0	1.2
ERN difference amplitude (μV)	−0.6	0.7	−0.6	0.6
Correct-trial Pe amplitude (μV)	−1.1	1.0	−1.5	1.3
Error-trial Pe amplitude (μV)	1.0	1.8	0.5	1.9
Pe difference amplitude (μV)	1.0	1.0	1.0	1.0

Note. RT = response time; CRN = correct-related negativity; ERN = error-related negativity; Pe = post-error positivity; difference = error minus correct.

of group, T_{WJt}/c (1.0,74.0) = 0.09, $p = .79$, and the Group × Accuracy interaction were not significant, T_{WJt}/c(1.0,54.7) = 0.88, $p = .37$.

The Group × Accuracy robust ANOVA on post-correct and post-error RTs indicate significantly slower RTs following error trials than following correct trials when collapsed across groups, T_{WJt}/c(1.0,72.3) = 76.06, $p < .001$. The main effect of group, T_{WJt}/c(1.0,59.1) = 0.27, $p = .60$, and the Group × Accuracy interaction were not significant, T_{WJt}/c(1.0,72.3) = 1.21, $p = .27$.

A Group × Accuracy robust ANOVA on post-correct and post-error error rates indicated that more errors were committed following an error trial than following a correct trial; this difference was supported by a significant main effect of accuracy, T_{WJt}/c(1.0,71.7) = 12.24, $p = .003$. As before, neither the main effect of group, T_{WJt}/c(1.0,73.5) = 0.07, $p = .79$, nor the Group × Accuracy interaction were significant, T_{WJt}/c(1.0,71.7) = 0.02, $p = .89$.

Event-Related Potentials

Grand averaged ERN, CRN, and Pe waveforms as a function of group are presented in Figure 2. Mean ERN and Pe TFSF component amplitude data as a function of group are presented in Table 2. Grand averaged ERN, CRN, and Pe TFSF waveforms as a function of group are presented in Figure 1. For controls, ERPs contained an average ± standard deviation of 469 ± 46 for correct trials and 22 ± 37 for error trials. For individuals with a mTBI, ERPs contained an average of 473 ± 45 for correct and 17 ± 11 for error trials. No between group differences were shown for the number of trials retained for averaging for either condition (|*t*s| <0.8, *p*s > .43).

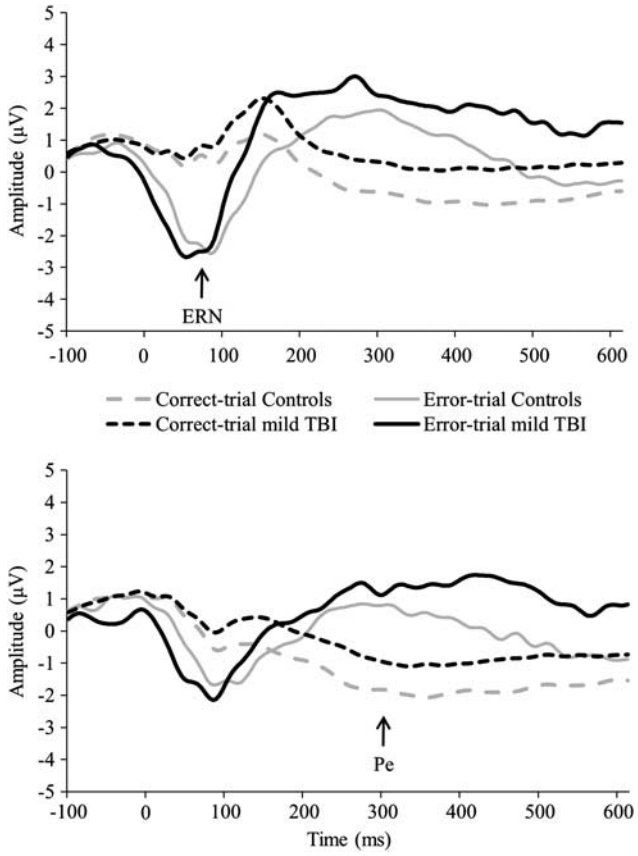

Fig. 2. Grand average waveforms as a function of group and accuracy representing the error-related negativity (ERN) averaged across central electrode locations and the post-error positivity (Pe) averaged across centro-parietal electrode locations.

The Group × Accuracy robust ANOVA on ERN amplitude[1] showed the expected main effect of accuracy with more negative ERN amplitudes shown for error trials compared to correct trials, T_{WJt}/c(1.0,73.8) = 73.14, $p < .001$. The main effect of group was not significant, T_{WJt}/c(1.0,73.4) = 0.11, $p = .75$. Importantly, the Group × Accuracy interaction was also not significant, T_{WJt}/c(1.0,73.8) = 0.22, $p = .64$. A Group × Accuracy robust ANOVA on ERN centroid latency showed a non-significant main effect of accuracy, T_{WJt}/c(1.0,73.4) = 1.01, $p = .32$. Individuals with a mTBI showed longer latencies than controls as supported by a significant main effect of group, T_{WJt}/c(1.0,71.2) = 5.16, $p = .03$. The Group × Accuracy interaction was also not significant, T_{WJt}/c(1.0,73.4) = 3.16, $p = .08$.

[1] Pontifex et al. (2009) analyzed non-factored single subject averages using a peak measure and found the largest effect for ERN amplitude at FCz and for Pe amplitude at Pz. When conducting analyses with identical time windows and component data extraction as the Pontifex study, Group × Accuracy robust ANOVAs on ERN amplitude and Pe amplitude showed non-significant main effects of group (T_{WJt}/cs < 0.4; *p*s > .53) and non-significant Group × Accuracy interactions (T_{WJt}/cs < 2.1; *p*s > .15). When using a peak amplitude measure for the ERN TFSF component (negative peak for ERN and positive peak for CRN), the Group × Accuracy robust ANOVA showed a non-significant main effect of group and a non-significant Group × Accuracy interaction (T_{WJt}/cs < 0.2; *p*s > .74).

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The Group × Accuracy robust ANOVA on Pe amplitude showed more positive Pe amplitudes to error trials than to correct trials as indicated by a main effect of accuracy, $T_{WJt}/c(1.0,73.9) = 90.12$, $p < .001$ (see footnote 1). The main effect of group was not significant, $T_{WJt}/c(1.0,71.5) = 0.01$, $p = .94$. The Group × Accuracy interaction was not significant, $T_{WJt}/c(1.0,73.9) = 0.94$, $p = .34$. A Group × Accuracy robust ANOVA on Pe centroid latency showed longer latencies to error trials than to correct trials as indicated by a main effect of accuracy, $T_{WJt}/c(1.0,73.4) = 58.24$, $p < .001$. The main effect of group was not significant, $T_{WJt}/c(1.0,71.1) = 1.57$, $p = .21$. The Group × Accuracy interaction was not significant, $T_{WJt}/c(1.0,73.4) = 1.30$, $p = .26$.

Subgroup Analyses

To ensure that findings are not primarily the result of too mild of injuries, we conducted two separate subgroup analyses. The first subgroup analysis examined 24 individuals from the mTBI that reported LoC and 46 controls. The Group × Accuracy robust ANOVAs on ERN and Pe amplitude showed non-significant main effects of group ($T_{WJt}/cs < 1.54$; $ps > .21$) and non-significant Group × Accuracy interactions ($T_{WJt}/cs < 0.8$; $ps > .36$).

A second subgroup analysis examining 25 individuals with PTA and 36 controls yielded similar findings to the overall analysis. The Group × Accuracy robust ANOVAs on ERN amplitude and Pe amplitude showed non-significant main effects of group ($T_{WJt}/cs < 0.4$; $ps > .55$) and non-significant Group × Accuracy interactions ($T_{WJt}/cs < 1.1$; $ps > .30$).

Considering the large range of time since injury in the current study, we conducted a subgroup analysis examining 33 individuals with mild TBI and a time since injury less than 12 months and the study control group. Group × Accuracy robust ANOVAs on ERN amplitude and Pe amplitude showed non-significant main effects of group ($T_{WJt}/cs < 0.3$; $ps > .59$) and non-significant Group × Accuracy interactions ($T_{WJt}/cs < 1.6$; $ps > .21$), consistent with the findings including all participants.

Correlational Analyses

After excluding three outliers[2] (two individuals with time since injury of over 25 months [over two *SD*s above the mean] and one individual with a length of PTA of 1440 min [over five *SD*s above the mean]), more positive error-trial Pe amplitude and Pe difference waveform (error minus correct) amplitude were associated with longer length of PTA, $r(21) = .47$, $p = .02$; $r(21) = .58$, $p = .004$, respectively. Less positive error-trial Pe amplitude[3] and Pe difference waveform amplitude were associated with longer time since injury, $r(31) = -.42$, $p = .02$; $r(31) = -.40$, $p = .02$, respectively. Longer CRN centroid latency was associated with shorter LoC, $r(20) = -.55$, $p = .008$. Longer ERN centroid latency was also related to fewer prior TBIs, $r(31) = -.48$, $p = .03$. None of the other correlations between time since injury, length of LoC, length of PTA, number of previous head injuries, or Rivermead Post Concussion Symptoms scores and electrophysiological measures were significant in individuals with mTBI ($|rs| < .38$; $ps > .07$).

DISCUSSION

We examined differences in cognitive control processes related to performance monitoring between individuals with mTBI and neurologically healthy controls. As expected, both groups showed longer RTs and higher error rates to incongruent trials than congruent trials. For post-error trials, both groups showed longer RTs and higher error rates compared to post-correct trials. No group differences were shown for any behavioral measures. These behavioral findings are consistent with current neuropsychological similarities between groups and suggest that cognitive control processes related to error monitoring may be intact in this sample of individuals with mTBI.

This conclusion that cognitive control processes in individuals with mTBI in the current sample may be intact is further supported by electrophysiological findings. Both groups showed more negative ERN amplitudes and more positive Pe amplitudes to error trials than to correct trials—but there were no significant group differences. In an attempt to replicate previous findings by Pontifex et al. (2009) of reduced-amplitude ERN in individuals with mild TBI, PTA and LoC subgroup analyses were conducted to ensure that current findings were not the result of differences in mTBI classification. Results were generally consistent with the overall analyses and showed no group-related differences in ERP amplitudes.

These findings stand in contrast to those of Pontifex et al. It is possible that variability in TBI severity between study samples, including potential differences in injury location and severity account for the between-study differences. We find this explanation unlikely, however, as a subset of individuals from this sample were used in a previous study of conflict adaptation that showed intact evaluative control processes (similar to the conflict-related ERN), but impaired regulative control processes (Larson et al., 2011). Indeed, the current findings, in conjunction with those of Larson et al. (2011), suggest that ACC-mediated conflict monitoring processes may be largely intact in individuals with mild TBI.

[2] Exclusion of these individuals from the overall analyses of behavioral and ERP data do not alter any findings. Group × Congruency robust ANOVAs on RTs and error rates showed non-significant main effects of group ($T_{WJt}/cs < 0.3$; $ps > .62$) and non-significant Group × Accuracy interactions ($T_{WJt}/cs < 1.3$; $ps > .28$). Group × Accuracy robust ANOVAs on ERN amplitude and latency and Pe amplitude and latency showed non-significant main effects of group ($T_{WJt}/cs < 2.0$; $ps > .15$) and non-significant Group × Accuracy interactions ($T_{WJt}/cs < 1.6$; $ps > .20$).

[3] For correlations, when examining only those individuals who received a head injury in the last 12 months, less positive error-trial Pe amplitude and Pe difference waveform amplitude were associated with longer time since injury, $r(30) = -.39$, $p = .03$; $r(30) = -.36$, $p = .04$, respectively. There were no changes to the group of individuals with PTA.

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According to the cognitive control theory, conflict is detected by the ACC, which in turn signals the dorsolateral prefrontal cortex (dlPFC) for compensatory attentional control to diminish conflict and improve subsequent performance (Botvinick, et al., 2001; Carter & van Veen, 2007; Cohen, Botvinick, & Carter, 2000; Egner & Hirsch, 2005a, 2005b; Hanslmayr et al., 2008; Kerns et al., 2004). The dlPFC minimizes conflict by providing top-down biasing of frontal and posterior systems to conflict and increase attentional focus (Corbetta & Shulman, 2002; Desimone & Duncan, 1995; Egner & Hirsch, 2005a; Rainer, Asaad, & Miller, 1998). The link between the ACC and dlPFC is known as the conflict-control loop (Carter & van Veen, 2007). There is little response conflict on a correct trial as the executed response matches the primed response of the target stimulus (Botvinick et al., 2001; Yeung, Botvinick, & Cohen, 2004); however, there is presence of stimulus-conflict processing on an incongruent trial that occurs before response execution. Previous research shows impaired conflict adaptation processing in mTBI while showing intact ACC-mediated stimulus-conflict processing (Larson et al., 2011). The current study further supports these findings by evidencing intact ACC-mediated response-conflict processing. Thus, ACC-mediated conflict processing appears to be intact in mTBI. With regard to the conflict-control loop, it appears that the dlPFC or possibly other frontal and parietal systems involved in attentional control are more affected following a mTBI.

Procedural and methodological differences may also account for the discrepancies between the current study and the study by Pontifex et al. (2009). Pontifex et al. used a modified 400-trial Eriksen flanker task in which participants responded to the direction of a target stimulus equiprobably flanked by either congruent or incongruent arrows. Moreover, a jittered inter-trial interval (1100, 1300, or 1500 ms) was used. The current study used a 500-trial Stroop task with equiprobable congruent and incongruent stimuli as well as a fixed 1500 ms inter-trial interval. Considering previous findings that show similar patterns of ERN amplitudes when using the Eriksen flanker and Stroop tasks in individuals with major depressive disorder (e.g., Chiu & Deldin, 2007; Holmes & Pizzagalli, 2008) and schizophrenia (e.g., Kim et al., 2006; Morris, Yee, & Nuechterlein, 2006), we believe that task differences do not likely account for non-significant group differences. However, to our knowledge, no research has directly compared ERN amplitudes across various tasks to find the optimal task structure for eliciting the ERN. Such research, including specific information about timing and stimulus onset asynchronies, would be beneficial particularly when examining group differences between controls and neurologic, psychiatric, or developmental populations.

Other explanations may account for the non-significant group differences in the current study. The mTBI group in the current study consisted of 25 individuals (69%) that received a sports-related mTBI, which is commonly less severe than a head injury received during, for example, a motor vehicle accident. However, when only examining those individuals with PTA, one of the strongest predictors of functioning following TBI (e.g., Hiekkanen et al., 2009), and LoC in hopes of sifting out individuals who received less severe mTBIs, group differences for ERN amplitude did not emerge. Furthermore, the sample in the study by Pontifex et al. (2009), used participants exclusively recruited from sports teams; thus, the high number of sports injuries does not likely account for the differences between studies. Another possible limitation may be that controls showed less state anxiety and higher depression levels than individuals with mTBI. Individuals with mild depression levels have shown similar ERN amplitudes to controls (Compton et al., 2008), and individuals with high state anxiety have shown attenuated ERN amplitudes relative to low state anxiety (Compton et al., 2007). However, higher state anxiety should have biased the current findings resulting in attenuated ERN amplitude in the mTBI group relative to controls, which was not the case. Finally, the current study used ICA to remove ocular artifacts and temporospatial PCA for data analysis as recommended by Jung et al. (2000) for clinical and neurologic populations where movement and ocular artifact may be more prevalent, rather than a spatial PCA as used by Pontifex et al. (2009) (see above for methodological reasons for these different approaches to artifact removal).

Although electrophysiological group differences were not shown in the omnibus analyses, correlations between ERP indices and TBI-related variables showed that more severe head injury was related to ERP indices of performance monitoring. Longer PTA was related to more positive error-trial Pe and Pe difference waveform amplitudes. In light of theories of Pe generation, individuals with mTBI with longer PTA may require greater neural activation for conscious error processing or may have a greater affective response to errors. Attenuated error-trial Pe amplitude associated with shorter time since injury may reflect that participants were not aware of committed errors resulting in a blunted error-trial Pe. Paradigms in which participants signal whether an error was committed on the trials following errors (e.g., Hester, Foxe, Molholm, Shpaner, & Garavan, 2005) would be beneficial to dissociate whether the above-mentioned differences are primarily the result of impairments in conscious error recognition or rather the amount of neural activation requisite to achieve conscious error processing. Time since injury may also play a role, as increased time since injury was associated with decreased Pe amplitudes. We note, however, that the time course of cognitive deficits following mTBI remains controversial and unclear. Lastly, contrary to previous research (Pontifex et al., 2009), number of head injuries was not associated with any ERP amplitudes or latencies.

Taken together, the current study found no significant differences in behavioral or electrophysiological cognitive control indices related to error monitoring between individuals with mTBI and controls. Both ACC-mediated stimulus-conflict (Larson et al., 2011) and response-conflict (current study) processes appear to be largely intact in mTBI; however, when examining processes related to the recruitment of cognitive control impairments emerge. Two future steps in

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this line of research include independent replication of findings to further understand performance-monitoring processes in individuals with mTBI and studies investigating brain regions involved in the conflict-control loop to clarify the extent to which regulative cognitive control processes are impaired in individuals with mTBI.

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