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Predictors of performance monitoring abilities following traumatic brain injury: The influence of negative affect and cognitive sequelae

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ABSTRACT

Performance monitoring is a cognitive control process modulated by both cognitive and affective variables. This study examined the relative contributions of negative affect (NA) and cognitive sequelae to performance monitoring dysfunction following severe traumatic brain injury (TBI). We used the error-related negativity (ERN) and post-error positivity (Pe) components of the event-related potential (ERP) to test the hypothesis that NA and cognitive sequelae would predict performance monitoring dysfunction beyond time since injury, and injury severity. Nineteen survivors of severe TBI completed neuropsychological tests, measures of NA, and a computerized Stroop task. Scores on NA and neuropsychological measures were standardized to form magnitude of cognitive sequelae and negative affect composite scores. Separate hierarchical regression analyses with ERN and Pe amplitudes as dependent variables and injury severity, time since injury, magnitude of cognitive sequelae, and NA as independent variables indicated that NA and cognitive sequelae significantly predicted ERN amplitude, with a larger relative contribution of NA than cognitive sequelae. Increased levels of NA were associated with decreased amplitude ERN. Cognitive sequelae, but not NA, predicted Pe amplitude. Injury severity and time since injury were not significant predictors. Results suggest that both NA and cognitive sequelae play critical roles in performance monitoring decrements following TBI and indicate a possible dissociation between the ERN and Pe, with the ERN more related to affective processes and the Pe to cognitive processes.

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1. Introduction

Severe traumatic brain injury (TBI) typically results in significant long-term neuropsychological deficits (Zaloshnja et al., 2005). The neuropsychological domains of memory, attention, and speed of processing tend to be the areas associated with the greatest declines, even five years post injury (Millis et al., 2001). Other cognitive deficits associated with severe TBI frequently include increased agitation, confusion, disorientation, and alteration in psychomotor activity, mental inflexibility, and problems with vigilance and arousal (Rao and Lyketsos, 2000).

Difficulties with emotion regulation are also common following severe TBI (see Robinson and Jorge, 2005; Shenal et al., 2003). Depression and anxiety disorders are the most frequent psychiatric complaints following TBI (Silver et al., 2001), and depression and anxiety symptoms frequently co-occur in psychiatric and TBI populations (Jorge et al., 2004). There is also considerable overlap between the symptoms of depression and the sequelae of TBI. For example, survivors of TBI can present with diminished volition and motivation (Kant et al., 1998), attenuated executive functioning and impaired cognition (Rao and Lyketsos, 2000), disrupted sleep (Makley et al., 2008), and increased rates of anxiety and suicide (Simpson and Tate, 2007).

Independent of brain injury, neuropsychological impairment is often seen in individuals with depression and other mood or anxiety disorders. Depression can diminish a person's performance on tests of memory, problem solving, attention and concentration (e.g., Liotti and Mayberg, 2001; Marx et al., 1992; Mohanty and Heller, 2002). The hallmark of anxiety and depression is the presence of negative affect (NA), a construct describing the symptoms of affective distress with both depression and anxiety symptoms (Clark and Watson, 1991). Negative affect is related to deficits in memory and attention, decreased motor functions, and impaired executive functioning and cognitive control (Emerson et al., 2001, 2005; Mialet et al., 1996; Shenal et al., 2003; Sweeney et al., 2000; Trichard et al., 1995).

Cognitive control refers to the ability to coordinate thought and action with internal goals (Levine et al., 2002; Miller and Cohen,

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2001). One critical aspect of cognitive control is monitoring and evaluating performance for errors and adjusting performance when errors are made (Botvinick et al., 2001; Kerns et al., 2004). These performance monitoring functions can effectively be evaluated using the error-related negativity (ERN) and post-error positivity (Pe) components of the scalp-recorded event-related potential (ERP; Falkenstein et al., 1991; Gehring et al., 1993). The ERN is a frontally maximal response-locked deflection in ERP that peaks within 100 ms after the commission of an error (Falkenstein et al., 1991). The ERN is thought to reflect the detection of response conflict (i.e., the simultaneous activity of competing response options), the motivational or affective significance of errors, or a reinforcement-learning response to unexpected performance (see Olvet and Hajcak, 2008, for review).

The Pe is a positive deflection in the ERP that occurs between 100 and 400 ms after the ERN. The Pe is thought to reflect the cognitive evaluation of errors (see Overbeek et al., 2005, for review). For example, Pe amplitude is reliably associated with the recognition and awareness of errors, as it is reduced in amplitude when subjects are unaware of errors in performance (i.e., unable to identify errors) or have decreased awareness of their deficits following TBI (Endrass et al., 2007; Larson and Perlstein, 2009; Nieuwenhuis et al., 2001; O'Connell et al., 2009; Shalgi et al., 2009). Source localization studies of the ERN and Pe suggest neural generators within areas of the anterior cingulate cortex (ACC; Herrmann et al., 2004).

Studies using the ERN and Pe demonstrate that cognitive control and, more specifically, performance monitoring is altered following TBI and in individuals with affective disorders. For example, Larson et al. (2007a) studied error processing in a severe TBI sample and found attenuated ERN amplitude in TBI survivors relative to healthy controls, but no difference between groups in Pe amplitude. Error-related negativity amplitude is significantly elevated in individuals with OCD and increases in a linear fashion as symptom severity increases (e.g., Gehring et al., 2000). Error-related negativity amplitude is also greater in individuals who are moderately depressed (Luu et al., 2003; Tucker et al., 2003), and in individuals diagnosed with major depressive disorder (Chiu and Deldin, 2007).

Luu et al. (2000) report that the general presence of NA is associated with elevated ERN amplitudes during error monitoring tasks. Similar findings are reported by Hajcak et al. (2004) who measured ERN amplitude in high and low affect groups where the high NA group demonstrated overall elevated ERN and reduced Pe amplitudes. The studies looking at NA and ERN amplitudes have mostly included subjects with pre-existing NA characteristics. Wiswede, et al. (2009a) induced short-term NA by presenting negative images to healthy participants prior to flanker stimuli. Error-related negativity amplitude was elevated on the trials that were preceded by a negative image. Other studies demonstrate increased ERN amplitudes even when NA is induced by derogatory verbal feedback (Wiswede et al., 2009b). In other words, participants who are given negative feedback have larger ERN amplitudes relative to participants given positive feedback (Wiswede et al., 2009b). These studies demonstrate the lability of ERN amplitude in response to immediate situations, and the short-term induction of NA.

It is clear from these studies that both TBI and conditions involving NA can influence individuals' performance monitoring abilities. Considering the overlap of symptoms between TBI and affective disorders, Larson et al. (2009) examined the additive effect of severe TBI and NA on performance monitoring abilities and showed an interaction between NA and severe TBI. More specifically, ERN amplitude was disproportionately decreased in individuals with severe TBI and high levels of NA relative to those with severe TBI and low levels of NA or healthy controls with high levels of NA. In other words, neural reflections of performance monitoring such as the ERN are decreased in the combined conditions of TBI with NA than in either condition alone. Amplitude of the Pe did not differ as a function of group or NA. Overall, these findings raise the question as to the relative contributions of NA and cognitive sequelae on performance monitoring alterations following TBI.

Few studies to date have evaluated general cognitive functioning in relation to performance monitoring abilities. Kim et al. (2006) examined the relationship between ERN and Pe amplitudes and measures of neuropsychological functioning in individuals with schizophrenia and healthy controls. They found positive correlations between ERN amplitude and measures of executive functioning, including the Trail Making Test Part B and the number of categories achieved on the Wisconsin Card Sorting Test in individuals with schizophrenia, but not healthy controls. Larson and Clayson (2011) compared neuropsychological test performance and ERN and Pe amplitudes in a large sample of healthy individuals and found tests associated with attention and executive skills were related to ERN amplitude, even when other domains of cognitive functioning and NA were controlled. No other published studies have examined the relationship between cognitive sequelae and electrophysiological indices of performance monitoring, particularly in a group like those with TBI who have frequent and debilitating cognitive sequelae.

The purpose of the present study, therefore, was to examine the relative contributions of cognitive sequelae and NA to electrophysiological reflections of performance monitoring, particularly ERN amplitude, in individuals with severe TBI. We predicted that NA, cognitive sequelae, and indices of injury severity would all three contribute to TBI-related difficulties in performance monitoring reflected by the ERN, but that NA would make the largest relative contribution. We predicted no specific relationships with Pe amplitude. This research is important because, despite findings that NA is a common sequelae of severe TBI and is associated with worse injury outcomes, little research has explored the interactions and effects of NA on cognitive and behavioral performance post injury. Furthermore, the relative contributions of NA and cognitive sequelae to performance monitoring functions have not been thoroughly examined. Thus, this study can also provide insight into the potential influences of such abilities on the neural underpinnings of performance monitoring and the specific roles of ERP components such as the ERN and Pe.

2. Materials and methods

2.1. Participants

We have previously shown ERN and Pe amplitude differences between survivors of severe TBI and healthy controls (Larson et al., 2007a, 2009). Thus, we chose to focus our investigation into the relative contributions of cognitive and affective processes to these decrements in only individuals with severe TBI. Nineteen participants (four females) with severe TBI were included in the study. This study represents a re-analysis of the data from the same participants reported previously in the Larson et al. (2007a), Larson et al., 2009) studies. Severity of TBI was determined from medical record review of lowest post-resuscitation Glasgow Coma Scale (GCS) score (Teasdale and Jennett, 1974), with severe TBI defined as a GCS score<9. Duration of loss of consciousness (LOC) and duration of post-traumatic amnesia (PTA) were also acquired from medical record review or, when LOC and PTA information were not available in medical records, from structured participant and significant other interview (King et al., 1997; McMillan et al., 1996). We upgraded the TBI severity rating of one participant with a GCS of 11 from moderate to severe due to extended LOC (~6 h) and long-duration PTA (>28 days). All study procedures were approved by the Institutional Review Board at the University of Florida.

Demographic information, injury severity variables, and scores from measures of NA and neuropsychological functioning are presented in Table 1. Participants with TBI were, on average, 11.3

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