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Error Awareness and Apathy in Moderate-to-Severe Traumatic Brain Injury

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ABSTRACT

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Moderate-to-severe traumatic brain injury (M/S TBI) is a growing public health concern with significant impact on the cognitive functioning of survivors. Cognitive control and deficits in awareness have been linked to poor recovery and rehabilitation outcomes. One way to research cognitive control is through awareness of errors using electroencephalogram and event-related potentials (ERPs). Both the error-related negativity and the post-error positivity components of the ERP are linked to error awareness and cognitive control processes. Attentional capacity and levels of apathy influence error awareness in those with M/S TBI. There are strong links between awareness, attention, and apathy. However, limited research has examined the role of attention, awareness, and apathy using electrophysiological indices of error awareness to further understand cognitive control in a M/S TBI sample.

The current study sought to elucidate the role of apathy in error awareness in those with M/S TBI. Participants included 75 neurologically-healthy controls (divided randomly into two control groups) and 24 individuals with M/S TBI. All participants completed self-report measures of mood, apathy, and executive functioning, as well as a brief neuropsychological battery to measure attention and cognitive ability. To measure awareness, participants completed the error awareness task (EAT), a modified Stroop go/no-go task. Participants signaled awareness of errors committed on the previous trial.

The M/S TBI group decreased accuracy while improving or maintaining error awareness compared to controls over time. There were no significant between-group differences for ERN and Pe amplitudes. Levels of apathy in the M/S TBI group were included in three multiple regression analyses predicting proportion of unaware errors, ERN amplitude, and Pe amplitude. Apathy was predictive of error awareness, although not in the predicted direction. Major analyses were replicated using two distinct control groups to determine potential sample effects. Results showed consistent results comparing both control groups to a M/S TBI group.

Findings show variable levels of awareness and accuracy over time for those with M/S TBI when compared to controls. Conclusions include varying levels of attention and awareness from the M/S TBI group over time, evidenced by improving awareness of errors when they are happening, but an inability to regulate performance sufficiently to improve accuracy. Levels of apathy are playing a role in error awareness, however, not in predicted directions. The study provides support for the role of attentional impairments in error awareness and encourages future studies to look for varying levels of performance within a given task when using populations linked to elevated levels of apathy and attentional deficits.

Keywords: traumatic brain injury (TBI), apathy, cognitive control, event-related potential (ERP), error-related negativity (ERN), post-error positivity (Pe), error awareness task (EAT)

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TABLE OF CONTENTS

Introduction 1

TBI and Awareness 2

Apathy 7

Electroencephalogram and Event-Related Potentials 12

Method 25

Participants 25

Error Awareness Task (EAT) 32

Electrophysiological Data Recording, Reduction, and Measurement 34

Neuropsychological Functioning, Mood, and Apathy Measures 36

Statistical Analyses 38

Results 43

Mood and Apathy Analyses 43

Neuropsychological Performance 44

Behavioral Analyses for the EAT 45

Early-to-Late Behavioral Performance 48

ERP Component Analyses 53

Severe TBI Analyses 57

ERP Analyses by Gender 60

Replication Analyses 60

The Role of Apathy in Error Awareness 62

Discussion 69

Limitations 79

Future Directions 83

Summary and Conclusions 84

References 87

LIST OF TABLES

1. Demographic Data including Means, Standard Deviation (SD), and Range by Group.....28

2. Description of TBI Participant Injury Severity and Verification.....31

3. Descriptive Data for Mood and Apathy Measures by Group.....44

4. Descriptive Data from the Frontal Systems Behavioral Scale (FrSBe).....45

5. Descriptive Data of Neuropsychological Measures by Group.....46

6. Behavioral Data for M/S TBI and Control Groups on the Whole Error Awareness Task.....47

7. Descriptive Data and Early to Late Behavioral Performance Change During the EAT as a Function of Group.....52

8. Descriptive Data for Number of Trials by Condition as a Function of Group.....53

9. Error Trial Means and Standard Deviation (SD) of Pe and ERN Amplitudes by Group.....54

10. Descriptive Data of and Early to Late Task Behavioral Performance Change During for the Severe TBI Group.....59

11. Error Trial ERP Means and Standard Deviation (SD) of Pe and ERN Amplitudes by Group and Gender.....61

12. Zero-order Correlations Between Unaware Accuracy, Apathy, Mood, and Neuropsychological Variables for the M/S TBI Group.....63

13. Hierarchical Regression Model with Arcsine-Transformed Unaware Error Proportion as the Dependent Variable.....65

14. Hierarchical Regression Model with ERN Amplitude as the Dependent Variable.....67

15. Hierarchical Regression Model with Pe Amplitude as the Dependent Variable.....67

LIST OF FIGURES

1. A pyramidal representation of awareness....3
2. Graphic representation of the EAT task....32
3. ERP component electrode locations....36
4. Line graph showing means and standard error for repeat and color no-go accuracy split by early and late sections of the EAT....50
5. Line graph showing means and standard error for repeat and color error awareness split by early and late sections of the EAT....51
6. Grand average waveforms for the ERN and Pe components by group....55
7. Topographical representation of Pe component mean voltages from 200-400 ms post response for correct, unaware, and aware trials by group....56
8. Topographical representation of ERN component mean voltages from 0-100ms post response for correct, unaware, and aware trials by group....57
9. Scatter plot of apathy scores and unaware error proportions from the M/S TBI group....66
10. Scatter plot of apathy scores and ERN amplitudes from the M/S TBI group....68
11. Scatter plot of apathy scores and Pe amplitudes from the M/S TBI group....68

Error Awareness and Apathy in Moderate-to-Severe Traumatic Brain Injury

Traumatic brain injury (TBI) and its effects are now a leading cause of death and disability in many countries (Koskinen & Alaranta, 2008; Kumar et al., 2009; Myburgh et al., 2008). In the United States alone, there are between 1.5 and 2.5 million TBIs each year (Beauchamp, Mutlak, Smith, Shohami, & Stahel, 2008; Hall, DeFrances, Williams, Golosinskiy, & Schwartzman, 2010), with a total cost of over $60 billion annually (K. L. Davis, Joshi, Tortella, & Candrilli, 2007; Gamboa, Holland, & Tierney, 2006; Woolhandler & Himmelstein, 2007; Wrona, 2006). Reports indicate that 19% of United States (U.S.) military personnel who have returned from Iraq and Afghanistan meet criteria for a TBI (Campbell et al., 2009). Indeed, between the years 2001 and 2007, 320,000 U.S. service men and women showed some effects of a TBI, ranging from mild symptoms to severe injury (Maruta, Lee, & Jacobs, 2010). Approximately 1.1% of the U.S. civilian population (3.17 million people) also live with some degree of TBI-related disability (Zaloshnja, Miller, Langlois, & Selassie, 2008).

The severity and types of disability following TBI vary depending on location of injury, extent of injury, and premorbid functioning (Lezak, Howieson, Bigler, & Tranel, 2012). In most TBIs, it is common to see diffuse axonal injury (DAI), or shearing of the white matter tracts, that can globally affect functioning (Dockree & Robertson, 2011; Maruta et al., 2010). Specific focal lesions in combination with global DAI may result in deficits in many areas of behavioral, emotional, cognitive, and executive domains (Bigler & Maxwell, 2012; Smith, Hicks, & Povlishock, 2013). Self-awareness and apathy are two commonly affected areas of functioning in those with moderate-to-severe (M/S) TBI (Lane-Brown & Tate, 2011). Although both awareness and apathy have been studied individually in this population there is very limited

information about the potential interaction of awareness and apathy in individuals with M/S TBI. Further, the neural mechanisms underlying both error awareness deficits and apathy following TBI are poorly understood.

TBI and Awareness

Awareness of behaviors, emotions, and cognitions is often affected following M/S TBI (Hart, Giovannetti, Montgomery, & Schwartz, 1998; Hart, Seignourel, & Sherer, 2009; Lanham, Weissenburger, Schwab, & Rosner, 2000; Port, Willmott, & Charlton, 2002; Sherer & Hart, 2003; Sherer et al., 1998). Individuals with M/S TBI often have difficulty recognizing their behavior and how functional deficits in behavior are connected to potential environmental problems (Dockree & Robertson, 2011). For example, repeating mistakes, committing social *faux pas*, and forgetting everyday tasks such as locking doors or taking medications are more commonly reported in individuals who have experienced a M/S TBI than in non-injured individuals (Dockree & Robertson, 2011). When awareness is present, those with M/S TBI are consistently more aware of their physical deficits than deficits associated with emotions, behaviors, and cognitions (Prigatano & Schacter, 1991). Poor behavioral, emotional, or cognitive awareness decreases chances for a successful outcome in the areas of rehabilitation (Lam, McMahon, Priddy, & Gehred-Schultz, 1988), vocational functioning (Sherer et al., 2002; Wise, Ownsworth, & Fleming, 2005), independence (Trudel, Tryon, & Purdum, 1998), and considerably increases the level of caregiver distress (Arnould, Rochat, Azouvi, & Linden, 2013; Hanks, Rapport, & Vangel, 2007; Nonterah et al., 2013)

Hart et al. (1989) suggested that error-related awareness research is a possible way to further understand the impact that TBI-related awareness deficits have relative to broader behavioral, emotional, and cognitive functioning. Crosson et al. (1989) put forth a pyramidal

model of levels of awareness that explains how awareness of specific deficits may be related to more general and broad self-awareness (see Figure 1). The first and most basic level of awareness is *intellectual awareness*: a person's ability to recognize his or her own impaired functioning. This may include a basic understanding that an individual is having difficulty in specific areas or a higher understanding of common errors in day-to-day functioning. For example, those with M/S TBI might be aware they have trouble remembering important things or they struggle to understand things people say. Higher-level intellectual awareness is necessary to understand the implications of the deficits. Examples of higher-level awareness may include people who can no longer remember directions to places must understand that they can no longer drive or travel unassisted or that someone with visual-spatial deficits may not be able to continue work in a graphics art field.

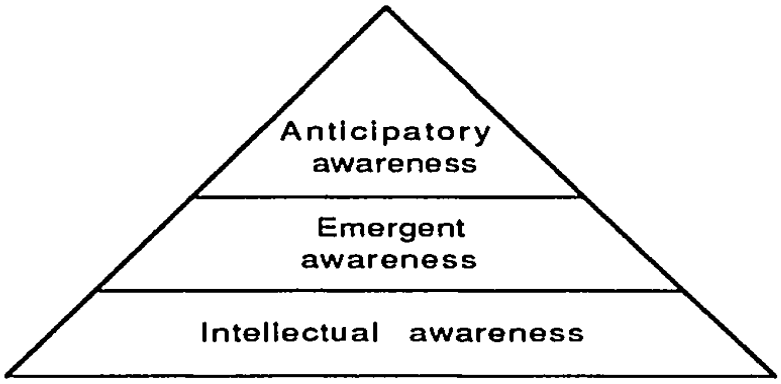

Figure 1. A pyramidal representation of awareness. Intellectual awareness is at the base and emergent awareness and anticipatory awareness build upon it. Anticipatory awareness requires some degree of intellectual Awareness. Adapted from "Awareness and Compensation in Postacute Head Injury Rehabilitation," by B. Crosson, 1989, *The Journal of Head Trauma Rehabilitation,* 4, p. 47. Copyright 1989 by Aspen Publishers.

Crosson et al. (1989) described intellectual awareness as based on concrete knowledge about the injury with two potential sources for its deficiency. A lack of knowledge is often seen

in situations where families and caregivers of those with M/S TBI do not have enough information about the symptoms and deficits they are seeing in the person with the injury so they attribute those deficits to laziness or other character traits. The other potential source is the brain pathology. Deficits in abstract reasoning from frontal lesions or DAI may make it difficult for the person to generalize understanding of deficits to other situations or environments. For example, Crosson et al. (1989) explain that a person with impaired abstract reasoning may only understand their deficits at a concrete level and in the context of a single situation. As such, they may be unable to generalize awareness of a situational impairment, leading to difficulty in spontaneously compensating in other environments or situations. Additionally, deficits in memory from basal forebrain or temporal lesions can decrease intellectual awareness that is based on learned behaviors from past experiences (Crosson et al., 1989).

The second level of the pyramid described by Crosson et al. (1989) is the person's ability to detect deficits as they are happening. This type of awareness is termed *emergent awareness* and relies on the person's ability to know they have deficits (intellectual awareness). Individuals with emergent awareness deficits are unable to detect problems as they occur due to difficulty in monitoring relationships between the environment and their actions. On a clinical level those with emergent awareness deficits are difficult to treat because they may have an understanding of their deficits and the consequences of them, but will continue to make the same mistakes and not apply compensatory strategies in the moment due to unawareness of the emerging issue. Individuals with emergent awareness problems typically require frequent external reminders from others telling them that a problem or deficit is occurring at that time.

The third level, *anticipatory awareness*, is a person's ability to predict when there will be difficulties as a result of their deficits (Crosson et al., 1989). Being able to anticipate difficulties

requires knowledge of the deficit (intellectual awareness) and awareness that the problem is occurring (emergent awareness.). Thus, anticipatory awareness builds on the other two levels. Deficits in anticipatory awareness impact the person's ability to initiate compensatory strategies once they are aware a known deficit is occurring. The individual with poor anticipatory awareness likely does not realize beforehand that application of a proven strategy can reduce the chances of problems related to their deficit(s).

The three levels of awareness become important to processing errors and error awareness in that those with M/S TBI can be affected at all three levels of awareness (Crosson et al., 1989). Error awareness takes all three levels into account through the process of being able to detect a conflict (intellectual awareness), recognize an error was made (emergent awareness), and take steps to ameliorate potential consequences by adjusting performance (anticipatory awareness). Individuals with M/S TBI seem to have the most difficulty with the later two levels of awareness in that they struggle to consciously detect having made an error and, thus, their anticipatory or compensatory responses are affected (Dockree & Robertson, 2011).

A secondary aspect related to error awareness in those with M/S TBI that plays a role in error detection is that of sustained attention (McAvinue, O'Keeffe, McMackin, & Robertson, 2005). Sustained attention is the ability to maintain mindful, conscious processing of repetitive, non-arousing stimuli whose qualities would otherwise lead to habituation and distraction over time (Robertson, Manly, Andrade, & Baddeley, 1997). Sustained attention tasks include those that require detection of targets that occur infrequently over a long period of time. Individuals with M/S TBI typically have impaired performance on such tasks (Whyte, Polansky, Fleming, Coslett, & Cavallucci, 1995; Wilkins, Shallice, & McCarthy, 1987) most likely related to difficulty in allocating attentional resources to task requirements prior to the application of

awareness levels. Additionally, individuals with M/S TBI have difficulty with tasks requiring continuous performance of sustained attention that require maintenance of response sets (e.g., sustained attention to frequent approach targets) and inhibition to infrequent avoidance targets (Chan, 2001; Robertson et al., 1997).

Error awareness and sustained attention processes have been connected through both physiological and theoretical means. First, fMRI data shows that both error awareness and sustained attention are executive processes that come from two distinct systems believed to have frontally-mediated connections (Westerhausen et al., 2010). Error awareness, as part of cognitive control has strong connections to the anterior cingulate cortex (ACC) and dorsolateral prefrontal cortices (Holroyd et al., 2004b; Hughes & Yeung, 2011; Yeung & Cohen, 2010; Yeung, Botvinick, & Cohen, 2004), while sustained attention is related to a frontal-parietal system that also involves the ACC (Westerhausen et al., 2010). Second, visual attention and awareness were described as separate but interconnected processes (Lamme, 2003). Lamme (2003) indicated that there are two types of consciousness or awareness: phenomenal consciousness and access awareness. He noted that most sensory stimuli are part of our phenomenal consciousness, which is short-lived and vulnerable to degradation. A small number of stimuli reach a stable level of access awareness where we are consciously aware of and able to report about the stimuli. Lamme (2003) theorized that the process that determines whether a stimulus moves from a phenomenal level to one of awareness is attention. Sustained attention, as an aspect of cognitive control, involves performance monitoring and evaluation of processing. Error awareness is the process of monitoring one's current performance in relation to a goal and detecting discrepancy between current and desired performance. McAvinue et al. (2005) demonstrated significant correlations between sustained attention and error awareness showing

that sustained attention is an executive process critical for the maintenance of error awareness.

Across situations where the levels of awareness are applied, individuals with TBI have reduced error awareness compared to controls (Hart et al., 1998; O'Keeffe, Dockree, & Robertson, 2004). Similarly, when error awareness is tested, there is a correlated reduction in sustained attention when comparing those with TBI to controls (McAvinue et al., 2005). Decreased sustained attention has been found in those with severe TBI (Slovarp, Azuma, & LaPointe, 2012). The reduction in error awareness and sustained attention seen in those with M/S TBI relates back to their difficulty in treatment and rehabilitation efforts to recognize deficits as they occur and to apply learned strategies to prevent increased functional problems. A further understanding of error awareness and the factors related to it may lead to more effective treatment and rehabilitation techniques, specifically in the areas of improving awareness of deficits, which in turn may be associated with improved post-injury outcomes. One factor related to error awareness and sustained attention that plays a significant role in error recognition and monitoring is negative affect and specifically apathy and anhedonia (Bressan & Crippa, 2005; M. J. Larson & Perlstein, 2009; M. J. Larson, Fair, Farrer, & Perlstein, 2011; Olvet, Klein, & Hajcak, 2010).

Apathy

Apathy is one aspect often associated with M/S TBI that may have an influence on levels of self-awareness. Apathy is conventionally defined as the absence of emotion, feeling, concern, and motivation (van der Wurff et al., 2003). Marin (1990) criticized this traditional definition as being too broad, in that it is possible to experience high levels of negative emotion, as can be the case in those with M/S TBI, but still lack motivation and initiative in most functional areas. Similarly, those with depression are often seen as apathetic, but commonly experience much

emotional pain and concern about their welfare and that of others. Marin gave a more consistent definition of apathy as a primary loss of motivation that is not affected by other factors related to consciousness, intellect, or emotional distress (Marin, 1990; 1996). A more recent paper proposed separate diagnostic criteria for apathy that includes a loss or diminished motivation from previous levels in two of three areas: behavior, cognition, and/or emotion that results in significant functional impairment and can be observed by the person themselves or by others (Arnould et al., 2013; Mulin et al., 2011).

Those with apathy, regardless of etiology, demonstrate symptoms of diminished motivation that are separate from and can not be attributed to emotional distress, cognitive impairment, or a decreased level of consciousness (Marin, 1990). Individuals with high levels of apathy are less able to formulate plans and goals, initiate and sustain behaviors, and to react to positive or negative stimuli in an emotional manner (Rao, Spiro, Schretlen, & Cascella, 2007). Such deficits make rehabilitation and improvements in functioning very difficult, particularly among those with M/S TBI (Lane-Brown & Tate, 2011). Those with M/S TBI who experience apathy are typically less engaged in rehabilitation activities and tend to have less appreciation for their functional gains. They are less interested in day-to-day activities and show a lack of ability to plan and develop future goals (Rao et al., 2007). This lack of functional behavior in turn leads to reduced vocational achievement (Prigatano & Fordyce, 1986) and overall decline in coping (Finset & Andersson, 2000).

Apathy following M/S TBI is rather prevalent (Starkstein & Pahissa, 2014). van Reekum, Stuss & Ostrander (2005) found that prevalence of apathy following M/S TBI was between 46.4% and 71.1%. Arnould, et al. (2013), in a recent review, found that an average of 48% (n = 554) of TBI participants were described as apathetic. In another study, those who had

focal frontal lesions apathy prevalence was as high as 89.3% (van Reekum, Stuss, & Ostrander, 2005). One concern with trying to study apathy is that apathy is often comorbid with depression. For example, one study reported apathy being comorbid with depression in 83% of M/S TBI cases with only 17% having apathy without depression (Kant, Duffy, & Pivovarnik, 1998).

Apathy has often been included as a symptom of depression in depression-related measures (Lane-Brown & Tate, 2009a; Marin, 1990; 1996; Marin, Firinciogullari, & Biedrzycki, 1993). Several researchers propose that apathy be considered a distinct syndrome and give diagnostic criteria to support their assertion (Marin, 1990; Mulin et al., 2011; Rao et al., 2007; Starkstein, 2008; Starkstein & Pahissa, 2014). Apathy-focused research in M/S TBI has demonstrated specific effects on cognitive functioning beyond the mood-related features of depression including the decreased ability to formulate plans for goal attainment, initiate the plan, and then cease activity when the goal is achieved (Lane-Brown & Tate, 2011). Apathy was distinguished from depression in a brain-injured population in that depression was related to affective and somatic symptoms while apathy was related to cognitive symptoms (Finset & Andersson, 2000). Finset and Andersson (2000) found that anhedonia and reduced initiative were common between both depression and apathy. Kant et al. (1998) found that in those with closed head injuries, apathy is pathophysiologically different than depression. Apathy is more likely a result of left inferior, medial, and superior frontal regions, insula, anterior cingulate cortex (ACC), and anterior temporal paralimbic areas including the amygdala and its related subcortical structures as well as white matter tracts including the corona radiate and the corpus callosum (Cummings, 1993; Duffy, 1997; Knutson et al., 2013; Marin, 1996).

The perceived overlap between apathy and depression can be linked to the use of apathy-related items in many, if not all, of the most frequently used depression scales (Levy &

Cummings, 1998). Apathy symptoms that commonly overlap with depression on such scales include psychomotor slowing, social inactivity, and decreased interest levels. In addition, overlap may be due to similar neural circuits (i.e., ACC, prefrontal cortex, amygdala) being involved in cognitive control and affective regulation (Cummings, 1993; Duffy, 1997; Marin, 1996).

In addition to a difference in physiological lesion location being found in TBI patients, apathy is a distinct syndrome and more common than depression in those with Huntington's, Parkinson's, and Alzheimer's diseases (Kirsch-Darrow, Fernandez, Marsiske, Okun, & Bowers, 2006; Landes, Sperry, & Strauss, 2005; Naarding, Janzing, Eling, van der Werf, & Kremer, 2009). In Huntington's disease patients, studies indicate that apathy was related to cognitive deterioration and functional declines, but depression was not (Naarding et al., 2009). All of these disorders, including TBI, commonly affect the frontal areas of the brain as well as the subcortical limbic areas with the cingulate cortex being the linking neural connection between the two areas (Cummings, 1993; Lezak et al., 2012; Stuss, van Reekum, & Murphy, 2000).

As the ACC and the prefrontal cortex are highly involved in the processes of evaluation and regulation of cognitive control (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Kerns, Cohen, MacDonald, & Cho, 2004), deficits related to apathy may alter a person's ability to detect errors and monitor performance. Similarly, apathy has been related to other neurobehavioral deficits in impulse control connected to cognitive control and performance monitoring (Ciurli, Formisano, Bivona, Cantagallo, & Angelelli, 2011). The neural connection between apathy and performance monitoring becomes important due to the high rates of apathy in those with conditions that affect the anterior subcortical nuclei and frontal cortical areas such as TBI, Parkinson's, Huntington's, and Alzheimer's (Andersson, Gundersen, & Finset, 1999a).

Apathy has been linked to deficits in sustained attention with studies in older adults suggesting that greater variability in reaction times on sustained attention tasks is a significant predictor of a lack of perseverance, or the ability to maintain task focus when a task is boring or difficult (Arnould et al., 2013; Rochat et al., 2013). The Rochat et al. (2013) study used the Sustained Attention to Response Task (SART) where participants must withhold responses to infrequent and unpredictable stimuli while maintaining rapid responses to frequent approach stimuli (Robertson et al., 1997). The SART has been validated as sensitive to deficits in sustained attention following TBI (Dockree et al., 2004; McAvinue et al., 2005). Arnould et al. (2013) concluded that attentional difficulties could be a significant contributor to apathy due to loss of interest as part of a progressive attentional disengagement resulting in a compromise of task and goal achievement. Arnould et al. (2013) found group difference between a severe TBI group and controls on measures of performance maintenance (i.e., response times) for both initial task performance and rate of performance deterioration over time. They characterized the group differences as waning responsiveness, increasing response times, and response time variability, concluding that the changes in performance were consistent with attentional lapses and decreased arousal level (Arnould et al., 2013). They implicated apathy as the source of decreased arousal leading to the attentional lapses (Arnould et al., 2013). As previously shown, there is a significant relationship between attention and error awareness.

If, as suggested, apathy does influence attentional ability, then apathy may also influence overall error awareness as all three components (apathy, attention, and error awareness) are connected physiologically through medial frontal structures and neural connections and have significant evidence supporting their associations. Through a more thorough understanding of the neurophysiologic manifestations of apathy I hoped to be able to inform future research aimed

at improving treatment and outcomes in general error awareness and performance monitoring for those with these debilitating conditions. Especially in those with M/S TBI, it is important to understand how apathy affects cognitive processes so as to assist in improved awareness of deficits and regulation of functioning in this growing population.

Electroencephalogram and Event-Related Potentials

Neurophysiologic markers of error awareness and performance monitoring can be measured using electroencephalogram (EEG) and scalp-recorded event-related potentials (ERPs). Electroencephalography was first used in the late 1800s on studies with animals and was later employed for use with humans in the early 20th century (Luck, 2005; Niedermeyer & da Silva, 2012; Swartz & Goldensohn, 1998). Electroencephalography has developed into the premier technology for studying automatic processes in the human brain including error awareness and performance monitoring due to the capability of measuring brain activity at a millisecond level and employment of both stimulus and response-locked recording (Luck, 2005; Niedermeyer & da Silva, 2012; Swartz & Goldensohn, 1998). The brain has an electrical charge that is maintained by billions of neurons. Neurons transport charged ions of either potassium or sodium across their membranes. When the ions are moved they can push or pull like-charged ions with them creating waves of electric currents in a process called volume conduction. When the waves come into contact with the scalp, the ions interact with the metal in the EEG electrodes and this change in voltage can then be measured. Looking at the differences between electrode sites over time produces the EEG recording.

The main neurological source for acquiring EEG data is the pyramidal neurons that comprise about 85% of the cortex. Pyramidal neurons are ideal for electrophysiological recording as their axons are oriented perpendicular to the cortical surface (Davidson & Jackson,

2000). Even so, the electric potential of a single neuron is too small to be detected by EEG. The simultaneous excitation of thousands or even millions of neurons in a similar spatial orientation is required for the electrical potential to be recorded (Nunez & Srinivasan, 2006). Even with the simultaneous firing of multiple pyramidal neurons, the electrical activity must be amplified to provide a visual signal of the waveforms.

Event-related potentials are used to better understand the neural functioning of patients or research participants. Data from EEG is very complex as it consists of electrical signals from hundreds of neural sources collected through sensors placed on the scalp. Embedded within the EEG data are electrical neural responses associated with specific time-locked cognitive, sensory, and motor events. Specific events can be isolated and extracted from the EEG data through a process of averaging (Luck, 2005). The extracted data are seen as specific waveforms of varying amplitude, latency, and polarity and are generally called ERPs as they are electrical potentials associated with specific events (Luck, 2005).

Event-related potentials are patterns of neural activity that are time-locked to either the presentation of a stimulus or to a response. In order to isolate the ERP signals it is necessary to filter out as much "noise" as possible from the recorded signal (Talsma, 2005). Noise can come from many different sources in the environment or from the person themselves in the form of eye blinks, movement, or forming circuits with objects that conduct electricity (Fabiani, Gratton, & Coles, 2000). As such, techniques have been developed that average data across multiple trials and filter out "noisy data." These methods provide for a higher signal-to-noise ratio and allow for a cleaner picture of the ERPs that are being examined. Each type of ERP of a particular amplitude and latency corresponds to specific neural reactions to environmental stimuli, either external or internal (Luck, 2005). This study focused on two particular ERP components that

have been linked to error-awareness and performance monitoring in cognitive control: the post-error positivity (Pe) and the error-related negativity (ERN).

Post-error positivity (Pe). An important ERP component related to error awareness and performance monitoring is the Pe. The Pe is a positive deflection in the ERP occurring 300-500 milliseconds following an error (Falkenstein, Hoormann, Christ, & Hohnsbein, 2000; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005b). Using low resolution electromagnetic tomography (LORETA) techniques on EEG data the Pe component was localized to the rostral portion of the ACC (Herrmann, Römmler, Ehlis, & Heidrich, 2004). However, there seems to be heterogeneous opinion about the actual source of the Pe, with most sources supporting a medial-parietal/ACC origination (Carter & Van Veen, 2007; Hester, Foxe, Molholm, & Shpaner, 2005; O'Connell et al., 2007; Ullsperger, Harsay, Wessel, & Ridderinkhof, 2010; Wessel, Danielmeier, & Ullsperger, 2011). Error awareness, which has been consistently associated with the Pe component, was specifically related to a principal component analysis-derived centro-parietal subcomponent of the Pe (Endrass, Klawohn, Gruetzmann, Ischebeck, & Kathmann, 2012a). In a substantial review of the literature, Overbeek et al. (2005) gave several hypotheses providing insight into the functional significance of the Pe. Hypotheses include the affective-processing, behavior-adaptation, and the error-awareness hypotheses.

The affective processing hypothesis states that error awareness has emotional correlates and the neuroaffective processes involved in the emotional appraisal of the error is reflected on the scalp as the Pe. The basis for this hypothesis is a study demonstrating that those who made more errors had smaller Pe amplitudes than those who made fewer mistakes (Falkenstein et al., 2000). Falkenstein et al. (2000) somewhat reluctantly concluded that those with more errors had smaller Pe amplitudes than those with fewer errors because the emotional significance of errors

was reduced when making more errors resulting in decreased component amplitudes. However, findings related to the affective processing hypothesis have not been replicated in other studies showing that subjects with high negative affect had smaller amplitude Pe components (Hajcak & McDonald, 2003; Hajcak, McDonald, & Simons, 2004). There is limited support for the affective processing hypothesis based on failure to replicate the initial findings, as well as considerable debate about the neural source of the Pe and its connection to other known emotion-related structures following more recent fMRI studies (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005b).

The behavioral adaptation hypothesis defines the Pe as being a representation of conscious changes in behavior to apply more resources to conflict detection and adaption to avoid erroneous responses on future trials (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005b). Behavior adaptation is most commonly thought of through the exposition of post-error slowing. Post-error slowing is a phenomenon where after an error has been committed, the response times for succeeding trials will slow, presumably as part of a system to reduce the number of errors by applying more resources and attention to the task at hand. Studies have demonstrated that this post-error slowing only occurs following an aware-error signaled by an amplified Pe (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001), and others have demonstrated that post-error slowing occurs following an amplified ERN (Debener, 2005; Gehring, Goss, & Coles, 1993). Overbeek, Nieuwenhuis, & Ridderinkhof (2005) suggested that, as a solution to this discrepancy, there are potentially two parallel systems that can signal a need for increased attention and resources depending on the type of error-response needed. The first system would be a rapid preconscious signal generated in the ACC that is seen on scalp-generated EEG as the ERN. The second, a slower system that responds to error salience and evaluates the significance

of the error, can be seen on the scalp as the Pe. The amplitude of the Pe has been attributed to the motivational salience of the error (Ridderinkhof, Ramautar, & Wijnen, 2009), meaning that the more salient the error is the greater the amplitude of the Pe. Behavioral adaptation theory is a plausible explanation as to why those with ACC, and particularly rostral-cingulate zone, lesions still have intact post-error slowing especially when the errors have more salience as seen in Stroop and go/no-go tasks (Ridderinkhof et al., 2009).

The most accepted hypothesis of the functional significance of the Pe is that of error awareness. The error awareness hypothesis reflects that the Pe is a representation of conscious error awareness in that the amplitude of the waveform covaries with the degree of awareness of the error (Dockree & Robertson, 2011; Nieuwenhuis et al., 2001; O'Connell et al., 2007). Nieuwenhuis et al. (2001) demonstrated greater Pe amplitudes for errors participants detected when compared to undetected errors on an antisaccade (eye movement) task involving participants being told to move their eyes towards a target stimulus, but were cued with a distractor on 50% of trials. The participants were asked to signal if they made an eye movement towards the distractor cue with Pe components compared for errors they signaled and errors they were unaware of, but were recorded as saccades. Further evidence in support of this theory comes from findings showing that the salience of error-inducing information is positively correlated with Pe amplitude (Leuthold & Sommer, 1999; Ridderinkhof et al., 2009). Variation found with error awareness indicated that the component was sensitive to the salience of an error and that salience secondarily may trigger error awareness (Endrass, Klawohn, Preuss, & Kathmann, 2012b; O'Connell et al., 2007; Ullsperger et al., 2010).

Questions still remain as to whether the Pe component is the actual expression of error awareness or if it represents processes that lead to conscious error awareness (Overbeek,

Nieuwenhuis, & Ridderinkhof, 2005b). More complexity underlying the significance of the Pe comes from data showing that the Pe is found not only for aware errors, but also for responses where the respondent was unsure of the accuracy of their response (Hewig, Coles, Trippe, Hecht, & Miltner, 2011).

The Pe, as far as it can be attributed to conscious error-awareness, can be generalized to represent conscious performance awareness beyond just error awareness on task completion (M. J. Larson & Perlstein, 2009; M. J. Larson, Kaufman, Kellison, Schmalfuss, & Perlstein, 2009; Wessel et al., 2011). A similar Pe to controls was found in those with M/S TBI and is more elevated when a person is aware that they have made an error compared to when they are unaware that the error was committed (Dockree & Robertson, 2011; Endrass, Reuter, & Kathmann, 2007; M. J. Larson & Perlstein, 2009). Pe amplitude is decreased in those with severe depression, which has been attributed to apathy (Schrijvers et al., 2009). The connection of apathy with Pe amplitude combined with the findings of Pe amplitude being correlated with motivational salience of errors (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005b), and the high prevalence of apathy in M/S TBI provides impetus for the use of the Pe as a strong measure of potential effects that apathy has on the ability of those with M/S TBI to detect errors in performance. Given that survivors of M/S TBI have higher levels of apathy, apathy is linked to decreased Pe amplitude, and that Pe amplitude is correlated with error monitoring, I used the Pe component as an electrophysiological measure of error awareness.

Error-related negativity (ERN). The ERN is a response-locked, negative deflection in the ERP that occurs 50-100 milliseconds following an error (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991). Based on studies involving source localization (Hughes & Yeung, 2011; Yeung & Cohen, 2010) including dipole modeling techniques (Dehaene & Posner, 1994) and

functional imaging (Taylor, Stern, & Gehring, 2007) the ERN is generated in the anterior cingulate cortex (ACC) with connections to the anterior insular cortex (Ullsperger et al., 2010).

There are two main theories of the functional significance of the ERN: the reinforcement learning theory and the conflict monitoring theory. The reinforcement learning theory is based on previous research supporting a link between the basal ganglia and prefrontal cortex. The basal ganglia is involved in prediction of whether ongoing events will turn out to be better or worse than expected (Barto, 1995; Houk, Adams, & Barto, 1995; Montague, Dayan, & Sejnowski, 1996). The basal ganglia send phasic signals in the form of positive or negative error messages based on whether the outcome is predicted to be better or worse to the frontal cortex and then back to the basal ganglia through the dopamine system (Schultz, 1998; 2002). An error, in which a person responds in a way contrary to goals is described as an unexpected event associated with a lack of expected reinforcement following a response. The reinforcement learning theory of the ERN uses this same theoretical framework to suggest that the ACC interacts with the dopamine system to enhance task performance (Holroyd & Coles, 2002). The ACC is thought to act as a filter between the different neural sources from which it receives motor commands (e.g. prefrontal cortex, orbitofrontal, amygdala). Through this process the ACC uses the dopamine system to optimize and select which of the sources will be best suited to control the motor system and reinforces that selection for future utilization of the most appropriate resource given a particular situation (Schultz, 1998; 2002). The amplitude of the ERN is then modulated by phasic shifts of either more dopamine (things are better than expected) producing a smaller ERN, or less dopamine (things are worse than expected) producing a larger ERN (Holroyd, Nieuwenhuis, & Mars, 2004a). Thus, when a person makes an error, negative reinforcement learning signals are sent to the ACC in the form of the ERN

component, resulting in improved motor control through more efficient resource allocation. As such, unpredicted errors and error feedback produce the ERN in the ACC.

A second theory of the role of the ERN is the conflict monitoring theory. The conflict monitoring theory predicts that the ERN signals response conflict and represents a precursor to conscious error detection (Steinhauser & Yeung, 2010). The conflict monitoring theory suggests that the ACC is involved in the detection of concurrent but incompatible representations in the environment (Carter & Van Veen, 2007). The conflict monitoring theory holds that the ERN is the product of ACC activation as it detects conflict in the environment and calls for greater resources from the dorsolateral prefrontal cortex. This call for resources then provides increased attentional capacity to handle the conflict and adjust performance as needed (Carter & Van Veen, 2007; Yeung & Cohen, 2010). The conflict monitoring theory posits that the ERN is a reflection of the dynamics of response conflict and selection and not just that of error detection or prediction (Hughes & Yeung, 2011).

Multiple studies manipulating response conflict by altering attention to competing stimuli support the conflict monitoring theory and suggest that greater attention to the target stimulus (i.e., correct response option), and not error detection alone, is related to ERN amplitude. For example, Hughes and Yeung (2011) altered the degree of response conflict by comparing trials during a conflict condition (on a traditional flanker task) to trials during a masked condition (using masked congruent flanker trials to manipulate the level of attention to the target stimulus). A flanker is a task where participants see a stimulus arrow with arrows on either side pointing in a congruent direction as the center arrow (<<<<<) or an incongruent direction (<<><<). Participants are to signal which direction the center stimulus arrow is facing. There were no behavioral (measured through response times and accuracy) differences between conditions, but

ERN amplitudes were significantly greater for errors committed in the conflict condition compared to the masked condition, suggesting that the ERN is sensitive to changes in the amount of attention to the conflict stimulus during the response selection process.

Several studies have demonstrated that changes in ERN amplitude reflect manipulations in levels of response conflict. For example, Stahl and Gibbons (2007) measured ERN amplitude differences based on variations in the stimulus onset asynchrony on a stop-signal task, hypothesizing that conflict would increase with increased delays in the presentation of the stop signal due to increased attention to the correct response option that then conflicts with the incorrect response. The authors observed a larger ERN for correct response trials (long-delay stopped trails) relative to error trials (short-delay non-stopped trials), not only adding further evidence that the ERN is not an index of error detection, but also providing evidence that the ERN is sensitive to dynamic changes in conflicting responses due to increased attention to the conflicting correct response option. Danielmeier, Wessel, Steinhauser, and Ullsperger (2009) and Maier, Pellegrino, and Steinhauser (2012) also observed a larger ERN for errors in which flanker stimuli were spaced farther apart and when flankers were larger in size, respectively, indicating that ERN amplitude is related to the degree of attention to the target stimulus. When flankers were located far from the target stimulus or were smaller, less attention was directed to the flankers and more attention was directed to the correct-response target. Thus, when an error occurred there was greater conflict between the correct and incorrect response options leading to larger ERN amplitudes. Similarly, following enhancements of cognitive control on high-conflict trials, subsequent errors are associated with larger ERN amplitude, reflective of greater attention to target stimulus following recruitment of attentional processes to improve task performance (M. J. Larson, Clayson, & Baldwin, 2012b). That is, ERN amplitudes are more negative on

errors committed on incongruent trials preceded by incongruent trials compared to errors on incongruent trials that were preceded by congruent trials, suggesting enhanced detection of conflict between the correct target stimulus and committed erroneous response. Together, ERN amplitudes are sensitive to changes in the amount of conflict between the correct and incorrect response options, not solely the detection of an error.

Using current research explaining the Pe and ERN components, it is possible to gain a better understanding of performance monitoring and error awareness. Although thought to reflect only unconscious error detection or conflict and to be unconnected to the Pe (Hughes & Yeung, 2011), the ERN was shown to be affected by conscious error awareness in an antisaccade task (Wessel et al., 2011). The Wessel (2011) and Hughes and Young (2011) studies also demonstrated that when an error was committed and a larger ERN was produced the response time for the person to signal that they recognized they made an error was significantly shorter. A change in response time demonstrates, perhaps, that a larger ERN represents more certainty in the assessment of the conflict or error made, that is then shown by a quicker conscious response to signal the error commission and is also represented in a larger Pe (Wessel et al., 2011). Results are contradictory regarding whether error awareness is necessary for the generation of an ERN. Several studies indicate no differences in ERN amplitude based on conscious error awareness (Dhar, Wiersema, & Pourtois, 2011; Endrass et al., 2007; Endrass, Franke, & Kathmann, 2005; Nieuwenhuis et al., 2001; O'Connell et al., 2007), suggesting that the ERN is independent of error awareness and may represent preconscious conflict processing. In contrast, Dhar et al. (2011) and Hewig et al. (2011) observed increased ERN amplitudes for aware relative to unaware errors, possibly suggesting that ERN amplitudes may index response conflict that is dependent, at least in part, on conscious awareness.

Error awareness methods. Recent research demonstrates methodological means for investigating how ERP components are affected by and indicate conscious error awareness. One specific task, the error awareness task (EAT), was designed and tested for use as a measure of conscious error awareness and provides an effective platform for eliciting a significant number of both conscious and unconscious errors (Ullsperger et al., 2010). The EAT is a modified motor go/no-go task that involves participants making commission errors of which they are either aware or unaware. The task requires that participants respond to a series of Stroop-type color words in incongruent fonts (e.g., the "RED" written in blue ink) with two different types of no-go lures where participants are to withhold their response. The lure types include a word repeated twice consecutively and a congruent color word trial where the word matches the ink color (e.g., the word "RED" written in red ink). By having competing types of response inhibition rules, the aim is to vary the strength of stimulus-response relationships, resulting in competitive suppression of rule representations such that the more prepotent rule would suppress the weaker rule (Hester et al., 2005). The suppression of the weaker rule then produces a significant number of errors, a small proportion of which may go unnoticed due to a primary focus on the prepotent rule. In the case of the EAT the goal is to use the human overlearned word reading behavior as a prepotent response and suppress awareness of word color. The rule competition predisposes the participants to attend to the repeat trials and not the Stroop congruent color trials. Participants are then trained to press an error awareness button on the trial following an error to signal conscious awareness.

Hester et al. (2005) initially used the EAT as a functional magnetic resonance imaging (fMRI) task seeking to confirm previous findings related to the ERN and Pe components as distinct aspects of error processing and awareness. They demonstrated ACC activation

following errors, but that presence of the ACC activation alone was insufficient for conscious error awareness and that a later activation, hypothesized to be the Pe component, was indicative of error awareness. They found significant activity in bilateral prefrontal cortices and parietal regions when participants signaled error awareness leading them to the conclusion that ACC activation, while necessary for error awareness is insufficient. Hester et al. (2005) provided support for the ERN's involvement in conflict detection and the Pe's involvement in conscious error-awareness through localization of error-awareness processes. They later used the EAT to demonstrate that there was impaired error awareness on behavioral measures in clinical populations of cocaine (Hester, Simões-Franklin, & Garavan, 2007) and chronic cannabis users (Hester, Nestor, & Garavan, 2009) when compared to controls. O'Connell et al. (2007) strengthened the methodological rigor of error awareness studies in EEG/ERP research by using the EAT to increase the number of errors, show reliable ERP components, and improve the process for subjectively reporting and recording error awareness in a control population (Wessel, 2012). A next step is to apply the EAT task to other clinical populations, such as M/S TBI, to better understand the role of conscious error awareness demonstrated through electrophysiological components.

An important question related specifically to the EAT and task performance when using a M/S TBI group relates to the pattern of performance across the task. Given the high rate of apathy, attentional dysregulation, and decreased awareness in those with M/S TBI there is reason to believe that performance may change throughout tasks that are perceived as, or become more difficult, as a result of decreased engagement or decreased cognitive reserve and attentional ability. The question of variable performance over task duration comes from a recent hypothesis proposing a relationship between effort and apathy in those with brain damage (Arnould et al.,

2013). The hypothesis indicated that effort and apathy are related through the influence of cognitive deficits such as attentional and executive impairments, and that those with a TBI are likely to recruit more effort early in a task that is easy in order to compensate for deficits, but disengage more quickly when the task becomes difficult (Arnould et al., 2013).

Several studies have looked at how the ERN and Pe components of the ERP are affected by TBI (M. J. Larson & Perlstein, 2009; M. J. Larson, Kaufman, Schmalfuss, & Perlstein, 2007; Wessel et al., 2011). The ERN is attenuated in those with M/S TBI, but the Pe and post-error slowing are generally similar to those of healthy controls (M. J. Larson et al., 2007; M. J. Larson & Perlstein, 2009). Negative affect, as seen through anxiety and depression levels, inversely correlates with ERN amplitude and level of cognitive deficits predicts Pe amplitudes in those with severe TBI (M. J. Larson et al., 2009; 2011). As noted above, depression and co-occurring apathy are very common in those with a TBI where 46%-71% have apathy (van Reekum et al., 2005), and 83% have comorbid apathy and depression (Kant et al., 1998).

No previous studies have examined how apathy influences performance monitoring and conscious error awareness in those with M/S TBI as seen through the use of ERPs as well as other neuropsychological measures. A greater understanding of the relationship between negative affect, specifically apathy, and error awareness will guide future research aimed at the development of more efficient and cost effective rehabilitation and treatment techniques. An understanding of how apathy relates to a person's ability to detect deficits in their behaviors, specifically errors, may provide clinicians with more accurate expectations of what their patients with M/S TBI are capable of and where to start working with them on developing coping and compensatory abilities. Thus, the current dissertation had the following specific aims:

Aim 1: To determine the presence or absence of group differences between those with M/S TBI and non-TBI controls on behavioral and electrophysiological indices of conscious error awareness.

- Hypothesis 1: Individuals with M/S TBI will have fewer aware errors than demographically-similar controls.
- Hypothesis 2: There will be group differences between M/S TBI participants and non-TBI controls for Pe amplitudes on aware error trials and ERN amplitudes on unaware error trials.

Aim 2: To determine the relationship between apathy and behavioral and electrophysiological indices of error awareness when controlling for injury severity and cognitive functioning in only those with M/S TBI.

- Hypothesis 1: Increased levels of apathy will be associated with a higher proportion of unaware errors relative to aware errors in those with M/S TBI.
- Hypothesis 2: Increased levels of apathy will be associated with decreased Pe and ERN amplitudes in those with M/S TBI.

Method

Participants

Participants consisted of two separate groups: individuals with M/S TBI and healthy controls. Due to the nature of the EEG recording there is a risk for excessive artifact and the need to exclude participants due to "noisy" data or equipment malfunction. Noise in the EEG comes from multiple sources including external electrical interference and internal participant sources such as movement artifacts and blinks (Fabiani et al., 2000). In order to account for participants that would be excluded due to excessive noise I recruited 75 control participants and

randomly matched 30 control participants with the M/S TBI group. I then created a second set of 30 control participants from the remaining control participants to use as a secondary control group to confirm principal behavioral and ERP analyses (a sensitivity analysis).

Inclusion and exclusion criteria. All participants were between the ages of 18 and 56 years old. They were all native-English speakers and all but two of the M/S TBI participants were right-handed (one of which was not included in the ERP analyses). It was important to use participants who are right-handed because there are potential hemispheric differences in those who are left-handed compared with right-handed individuals (Zhavoronkova, 2000). However, I determined that inclusion of two left-handed individuals was of minor risk due to the small percentage (approximately 25-30%) of left-handed individuals who demonstrate some level of hemispheric language and memory differences (Lezak et al., 2012). Only one of the left-handed individuals was included in ERP analyses. I used native-English speakers due to different patterns of neural activity found when a person is reading materials that are not in their native tongue (Sakai, 2005) and all materials used in this study were written and standardized in English.

Exclusion criteria included: history of learning disability, ADHD, psychotic or bipolar disorder, severe depression, uncorrected vision, language comprehension deficits, recent substance dependence or history of neurological impairment other than TBI (i.e., stroke, epilepsy). Healthy controls were excluded if they had any history of mental health diagnosis in addition to the previous exclusionary criteria. All participants were screened for and excluded if they had color blindness using the Ishihara pseudo-isochromatic color plates (Clark, 1924).

Participants included. For the principal analyses, 26 participants with a M/S TBI were recruited, of which five participants were excluded from ERP analysis due to insufficient

numbers of either aware or unaware error trials or excessively noisy data, one was excluded because they were unable to adequately learn the EAT task, and one other due to excessive visual acuity and comprehension problems when completing the Ishihara Color Blindness Test. Due to that participant's inability to complete a basic color blindness test he was excluded. Fifteen control participants were excluded due to noise and insufficient numbers of trials. Trials were considered bad and removed if more than 15% of channels were marked bad. Channels were marked bad if the fast average amplitude exceeded 100μV or if the differential average amplitude exceeded 50μV. I established that any person with fewer than six errors in any trial category would be excluded due to a lack of stability and reliability in the average component waveform with fewer trials in adult participants (Olvet & Hajcak, 2009b). This left a total sample size of 49 for EEG analysis and evaluation (control n=30, TBI n=19). The sample size used for behavioral analysis included all participants from the TBI group (n= 24) that completed the EAT and the 30 randomly selected controls.

There was a significant difference in years of age between the control group and the M/S TBI groups, with an independent samples t-test showing that the control group was younger, $t(1,52) = -2.98$, $p = .004$ (see Table 1). There was no difference in years of education between the TBI and control groups, $t(1,52) = -1.03$, $p = .31$. A *chi*-squared test indicated that there was also a significant difference between the groups on gender distribution, $\chi^2(1) = 3.80$, $p = .05$, with the M/S TBI group having a higher proportion of males to females than the control group. The M/S TBI group had 16 males and 8 females (66.7% male) and the control group consisted of 12 males and 18 females (40.0% males).

Table 1

Demographic Data including Means, Standard Deviation (SD), and Range by Group

	M/S TBI (n = 24)			Control (n = 30)		
	Mean	*SD*	Range	Mean	*SD*	Range
Age (years)	30.29	11.69	18 – 56	22.87	6.32	18 – 49
Education (years)	14.79	2.45	11 – 22	14.25	1.34	12 – 16

Participants were recruited via flyers placed throughout the Brigham Young University and Utah Valley University campuses and local community. Specific recruiting for the M/S TBI group occurred at the Utah Valley Regional Medical Center, TBI support groups, Intermountain Medical Center's neurorehabilitation facility in Murray, UT, the Brigham Young University Comprehensive Clinic, the Utah Brain Injury Alliance, local medical providers, and through compiled lists of previous research participants who expressed interest in further participation in research. Control participants were recruited from undergraduate psychology classes that offer extra credit for research participation and flyers posted throughout the local community. Participants received course credit or $35 for participation.

Assessing injury severity. The M/S TBI group consisted of participants who sustained a TBI between approximately six months prior to participation and less than ten years from study participation. Waiting six months following an injury was important due to spontaneous recovery following TBI where the person will recover some levels of functioning as the brain undergoes healing (Myburgh et al., 2008; Novack, Alderson, Bush, Meythaler, & Canupp, 2000). TBI severity was determined using three possible indices obtained from medical records and structured interviews (see below) with participants. The three indices included duration of loss

of consciousness (LOC), duration of post-traumatic amnesia (PTA), and Glasgow Comma Scale (GCS) score (Teasdale & Jennett, 1974). Following current classification standards, moderate TBI was defined as the lowest post-resuscitation GCS score in a range of 9-12, PTA between 1 and 7 days, and LOC of more than 30 minutes, but less than 6 hours (Bigler, 1990; Bond, 1986; Lezak et al., 2012). Severe TBI was defined as a GCS score of less than 9, LOC of greater than 6 hours, or PTA of more than 7 days (Bigler, 1990; Bond, 1986; Lezak et al., 2012). The main criterion for determining injury severity was the lowest post-resuscitation GCS, when available through documentation, with PTA and LOC acting as alternate criteria for assessment of injury severity when records were unavailable or insufficient to document GCS.

Participants were asked to bring with them or provide copies of medical records and neuroimaging for review to determine level of severity. If participants did not have access to their medical records, a signed release was requested in order to obtain copies of the records from their health care provider(s). In addition, if there were discrepancies between the participant report and medical records, or medical records were unavailable, comprehensive interviews were conducted with the participant and/or significant other/caregiver to further determine level of severity. While determination of LOC and PTA based on retrospective interview has been criticized due to confusion between disorientation and PTA (Shores, Marosszeky, Sandanam, & Batchelor, 1986) and the presence of isolated recall of events detached from continuous memory, termed "islands of memory," (Gronwall & Wrightson, 1980), I employed retrospective interviewing methods to minimize such confusion. Retrospective techniques have been shown to be reliable and valid for determining injury severity based on PTA (King et al., 1997; McMillan, Jongen, & Greenwood, 1996). Specifically, I asked very specific questions related to what the participants themselves could

remember and not what others had told them happened (e.g. "What is the last thing you remember before the accident?). Table 2 contains a summary of the TBI group severity classification information and whether or not the information was obtained through structured clinical interview or through medical records.

Briefly, of the 24 M/S TBI participants there were 13 classified as severe and 11 as moderate. Classifications were determined for nine of the participants from medical records and the remaining 15 from participant and/or family member accounts gathered through the previously mentioned structured clinical interview. Three participants refused to sign medical releases of information. There were GCS scores reported in three (GCS = 3, 7, and 14) of the nine participants with medical records. The participant with a GCS score of 14 was not seen until the second day following the injury, after which he/she was hospitalized for 10 days and remained in PTA for four days, according to medical records. One severe TBI participant had no documented LOC, a reported Ranchos Los Amigos Cognitive Scale score of three, and a documented diagnosis of severe TBI. For the remaining participants I based injury severity classifications on retrospective clinical interview (n = 15) or other medical record information (n = 6) related to duration of PTA and LOC. Average LOC for the moderate TBI participants was 0.51 (SD = 0.34) hours with PTA being 45.03 (SD = 40.23) hours. The severe TBI participants had an average LOC duration of 366.93 (SD = 377.06) hours and PTA duration of 994.62 (SD = 1556.17) hours.

Table 2

Description of TBI Participant Injury Severity and Verification

Age	Sex	Etiology	LOC Hours	PTA Hours	Months Post	Medical Record	Patient Account
18	M	Fall	>0.50	0.75	8		X
27	F	BFT	504	504	99		X
41	F	Bike	.50	24	82	X	
22	M	MVA	336	336	99	X	
24	M	MVA	384	1440	54	X	
30	M	Fall	1	96	150	X	
21	M	Fall	0.10	72	6		X
26	F	MVA	96	240	72		X
31	M	MVA	1080	336	69	X	
35	M	MVA	0.50	1	16		X
28	F	Fall	0.33	36	98		X
24	M	MVA	144	144	27		X
23	F	MVA	*	40	66		X
19	M	Fall	1	1.50	19		X
23	F	MVA	0.03	2016	31		X
24	M	Bike	>0.05	336	26	X	
56	M	MVA	0.50	72	35		X
45	F	Fall	336	336	120		X
52	M	Bike	0.92	120	8	X	
26	M	Bike	0.25	32	60		X
45	M	BFT	672	1344	39	X	
51	M	Bike	18	18	105	X	
18	M	Fall	120	120	6		X
18	F	MVA	1080	5760	25		X
		Mean:	198.98	559.39	54.92		
		SD:	330.09	1223.84	41.09		

Note. BFT = blunt force trauma, Bike = cycling accident, MVA = motor vehicle accident

Error Awareness Task (EAT)

The computerized EAT was completed during EEG recording. The EAT was originally developed by Robert Hester, PhD. and was adapted and used in this research with his permission (Hester et al., 2005; 2009).

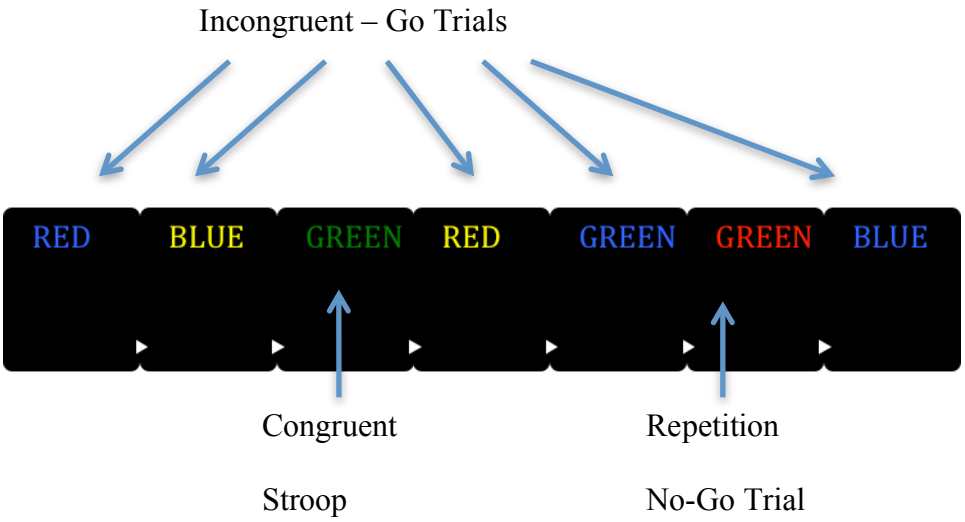

Figure 2. Graphic representation of the EAT task. The EAT presents a serial stream of single color words in incongruent fonts, with the word presented for 900 milliseconds followed by a random inter-trial interval between 1000 and 1500 milliseconds. Participants were trained to respond to each of the words with a single 'Go trial' button press, and withhold this response when either of two different circumstances arose. The first was if the same word was presented on two consecutive trials (Repeat No-go), and the second was if the word and color font of the word match (Congruent Stroop No-go). To indicate 'error awareness' participants were trained to press the error button on the trial following any commission errors. Adapted from "Neural Mechanisms Involved in Error Processing: A Comparison of Errors Made With and Without Awareness," by R. Hester, J. J. Foxe, S. Molholm, M. Shpaner, & H. Garavana, 2005, NeuroImage, 27, p. 603. Copyright 2005 by Elsevier, Inc.

The EAT consists of a practice condition and the main task. The main task is summarized in Figure 2. The purpose of the EAT is to create a scenario where the participant is

faced with a task that is sufficiently difficult to illicit a significant number of errors based on two different rules they must pay attention to. Of the two rules one is more prepotent than the other and as such the person will be more inclined to focus on this rule and not be as aware they are making errors related to the other.

In the EAT practice there are four steps. During the first step, the participants saw the color words "RED," "GREEN," "BLUE," etc. written in a colored font that does not correspond with the word presented. For example, the word "RED" written in blue ink or the word "GREEN" written in red ink. Participants were instructed to press "1" for each stimulus. In the second step participants were instructed to continue with the previous instructions, but also told that if a word was repeated twice in a row they were not to press any key when the repeated word was displayed (the word "Red" followed by "Red") a second time (consecutively repeated word equals no-go stimulus). Participants were then instructed that if they made a mistake and pressed the "1" button when they should have withheld their response on a repeated word they needed to press "2" on the next trial in order to indicate awareness of the error. In the third step the second rule was introduced. They were instructed to continue to press "1" for each incongruent stimulus (color word presented in a different color of font, e.g., "RED" written in blue ink), however, if the word was written in the same color of font as the written word they do not press any key (congruent stimulus equals a no-go trial). The last step reminded participants that if they did press a key on either a congruent trial or a repetition trial they were to signal they made an error by pressing "2" on the subsequent trial regardless of the type of stimulus shown during the "awareness" trial. The first phase (steps one and two) of the practice consisted of 50 trials and the second phase (steps three and four) consisted of 100 trials to ensure adequate learning of each rule. If a participant did not meet a 75% criterion indicating mastery of each step in the practice

they were allowed to repeat that portion of the practice up to two more times in order to meet rule mastery criteria. Only one of the TBI participants was unable to meet the necessary mastery level and was excluded from the study. No other participants from either group required more than one extra practice on either phase to meet the 75% mastery level.

Following the practice trials, participants began the EAT task. The task employed all of the rules the person was taught during the practice. They were to press "1" if presented with an incongruent stimulus (color word and font do not match). If they saw a congruent stimulus (matching color word and font) or they saw a consecutively repeated word, they were not to press any key. If they did press a key when not indicated they were to signal that they made an error by pressing "2" on the next trial. Each word was presented for 900 milliseconds with a random inter-trial interval (ITI) of between 1000 and 1500 milliseconds. The task consisted of four blocks of 225 trials including 46 no-go trials (23 incongruent and 23 repetitions) and 179 go trials for a total of 900 trials (717 go and 183 no-go).

Electrophysiological Data Recording, Reduction, and Measurement

Electroencephalogram data was recorded from a geodesic sensor net with 128 scalp sites and Electrical Geodesics, Inc., (EGI; Eugene, Oregon) amplifier system (20K gain, nominal bandpass = .10-100Hz). Electrode placements enabled recording vertical and horizontal eye movements reflecting electro-oculographic (EOG) activity. Data from the EEG was referenced to the vertex electrode and digitized continuously at 250Hz with a 24-bit analog-to-digital converter. A right posterior electrode approximately two inches behind the right mastoid served as common ground. Electrode impedance was maintained at or below 50kΩ. Electroencephalographam data was segmented off-line and single trial epochs rejected if voltages exceeded 100μV, transitional (sample-to-sample) thresholds were greater than 100μV,

or eye-channel amplitudes were above 70μV. Data was digitally re-referenced to an average reference then digitally low-pass filtered at 30Hz. Eye movement artifacts including blinks, saccades, and movements were corrected using independent component analysis as part of the open source ERP Toolkit in Matlab (Dien, 2010). Independent component analysis (ICA) allows for automatic artifact correction through identification of prototypical blinks and saccades that are applied to a template. Waveform data was then analyzed using the template to remove artifacts.

In order to understand potential differences between early and late performance on the EAT task I further segmented the ERP data into early and late halves with the intention of evaluating electrophysiological differences in error awareness due to potential differences in early and late performance across the task. However, there were insufficient trials in multiple categories for 13 out of the 19 M/S TBI participants leaving insufficient sample size to complete a full analysis. Early and late analyses of the ERPs were, therefore, not conducted.

Analysis of the Pe and ERN was achieved through similar processes as used by Larson et al. (2007) in a study of the Pe and ERN in individuals with M/S TBI and healthy control participants. Event-related epochs were response-locked and extracted with a duration starting 400 ms prior to stimulus response, and ending 800 ms after response, with -400 milliseconds (ms) to -200 ms serving as the baseline. Due to the absence of a distinct peak, the Pe was identified as the averaged activity from 200 ms to 400 ms post-response from six centro-parietal sites (54, 55, 61, 62, 78, 79, see Figure 3). The ERN was identified as the peak negative amplitude deflection from 0 ms to 100 ms from five fronto-central sites (Ref [Cz], 7, 106, and 6 [FCz], see Figure 3) averaged across 15 ms pre- to 15 ms post-peak amplitude in order to control for group-wise latency differences. The use of an adaptive mean procedure improves robustness

to noise in both ERN and Pe analyses when compared to peak amplitudes (Clayson, Baldwin, & Larson, 2013). Correct-response data for both components was collected using the same time window and electrodes to include in data analysis for use as an error-trial comparison (Clayson et al., 2013).

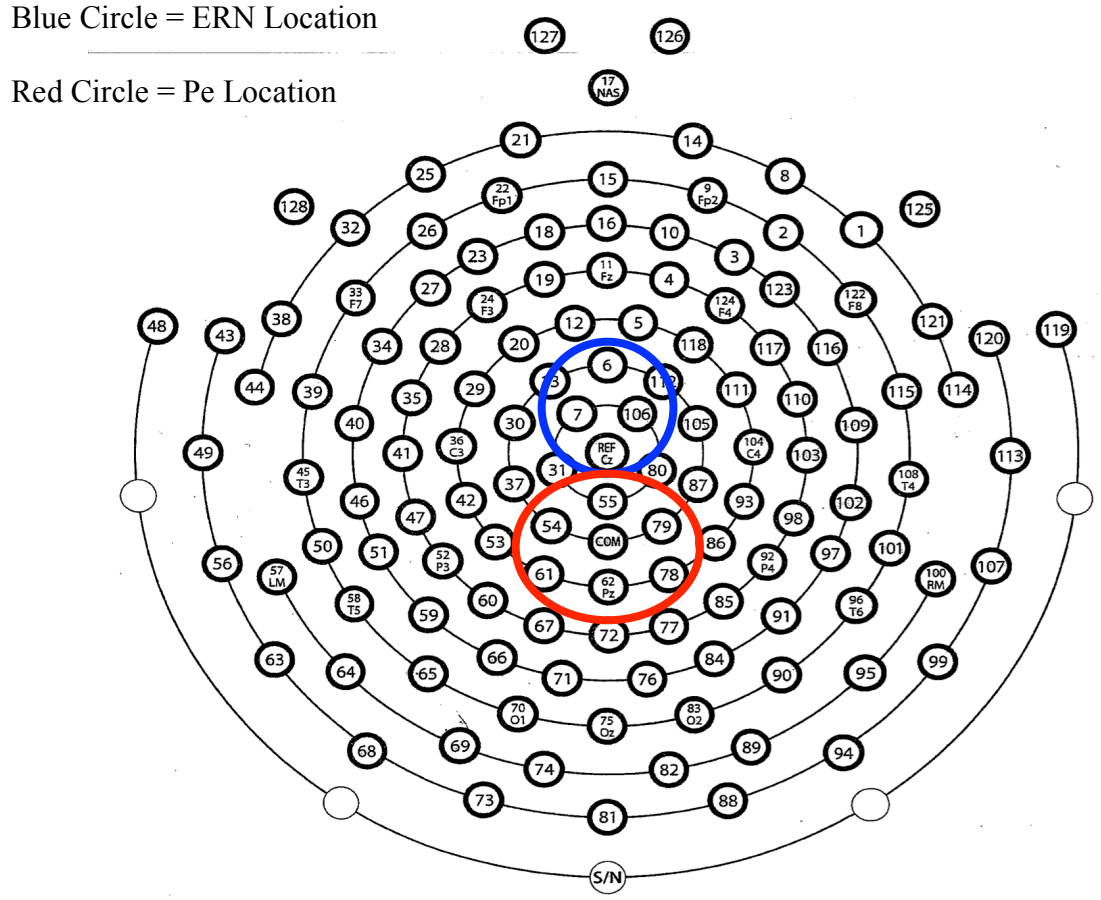

Figure 3. ERP component electrode locations.

Neuropsychological Functioning, Mood, and Apathy Measures

Participants completed a short battery of measures aimed to characterize their current neuropsychological functioning, current mood, and levels of apathy. Measures were

administered through the use of both paper-and-pencil tasks as well as electronically through use of an online survey tool (http://www.byu.qualtrics.com).

Apathy Evaluation Scale – Self-Rating Form (AES). The AES is an 18-item self-report apathy measure that demonstrates good reliability (internal consistency = 0.86-0.94) and validity (Glenn et al., 2002; Marin, 1991). Scores on the AES range from 18-72 with a mean of 28 and standard deviation of +/-6 on the original validation sample (Marin, 1991). The AES has been used with TBI populations and found to be a sensitive measure at determining presence of apathy (Clarke et al., 2011) and differentiating apathy from anxiety and depression (Kant et al., 1998). (Lane-Brown & Tate, 2009b) recently validated the AES in a M/S TBI population and established a score of 37 as a reliable cutoff for presence of apathy in this population.

Beck Depression Inventory – Second edition (BDI-II). The BDI-II is a depression measure used to screen for and identify levels of depression in clinical and normal populations. It has a internal consistency reliability of a=0.92 (Beck, Steer, Ball, & Ranieri, 1996). The BDI-II provides a depression rating score and cutoff scores have been defined for use with M/S TBI populations (Homaifar, Brenner, & Gutierrez, 2009) with a score of 35 being needed to meet criteria for clinical depression in a M/S TBI population.

Wechsler Test of Adult Reading (WTAR). The WTAR is a 50-item measure of premorbid intellectual functioning. This measure was demonstrated as valid in estimating pre-injury intelligence in a severe TBI population (Green, Melo, Christensen, Ngo, Monette, & Bradbury, 2008). This measure was used to describe the nature of our M/S TBI group.
Frontal Systems Behavior Scale – Self-Rating Form (FrSBe). The FrSBe is a 46-item behavior rating scale originally designed to measure behavioral change associated with frontal lobe injury. Psychometrically the FrSBE has demonstrated good reliability (internal consistency 0.96; split

half 0.93) and validity (Clarke et al., 2011; Zgaljardic, Borod, Foldi, & Mattis, 2003). The FrSBe gathers information regarding behavioral changes related to functioning in natural settings from the patient (self-report) and significant others. Also included are self- and other ratings of premorbid behavior to use as comparison data. The FrSBE includes an overall composite score and three subscales that include questions assessing apathy, disinhibition, and executive function.

Repeatable Battery for the Assessment of Neuropsychological Status (RBANS). The RBANS, although initially designed as a screening tool for the assessment of dementia, has gained considerable use as a neuropsychological screening instrument for neurologic and psychiatric disorders based on its short administration time, co-normed index scores, inclusion of a summary score, and equivalent A and B forms for use across multiple test administrations (Randolph, Tierney, Mohr, & Chase, 1998). The RBANS consists of twelve subtests measuring five primary domains: immediate memory, delayed memory, visuospatial/constructional abilities, language, and attention/processing speed (Randolph et al., 1998). Index scores are provided for each domain along with a Total scale score. Studies demonstrate the reliability and validity of the RBANS in detecting cognitive impairment in individuals with TBI (McKay, Casey, Wertheimer, & Fichtenberg, 2007; McKay, Wertheimer, Fichtenberg, & Casey, 2008). The Total scale score was used to assess general cognitive functioning and the five domain indices to further delineate group neuropsychological status.

Statistical Analyses

Statistical analyses were conducted using the statistical software package SPSS 21 (SPSS IBM, New York, NY) and the ERP PCA Toolkit (Dien, 2010). I initially ensured normality of data distribution and variance for all observed variables through the visual observation of scatterplots and box plots to ensure normality and Levene's test to ensure equality of variances

(Levene, 1960). Further data cleaning was done through identification of potential outliers observed on scatterplots and box plots that had scores beyond two interquartile ranges from the median, as the median is unaffected by outliers. Using this method no outliers were identified. Means, standard deviations, and ranges were calculated for ERP component amplitude, behavioral data (RT, accuracy, and error awareness), number of trials as a function of trial type (go, correct, error, awareness level, and lure type), neuropsychological measures (total and subscales where available), self-report mood and apathy data, and demographic variables. Zero-order correlations and independent-samples *t*-tests were used to evaluate the relationship between and compare groups on ERP component amplitudes, behavioral, trial, neuropsychological, and self-report data. Significance for all analyses was set at the $p = .05$ level.

I subsequently examined demographic variables as a function of group to ensure groups were similar on age, education, number of trials for ERP analysis, and gender ratio using independent-samples *t*-tests and *chi*-square analysis, respectively. All accuracy and error awareness percentages were transformed using an arcsine transformation. I used the arcsine transformation because accuracy and error awareness percentages were derived from count data resulting in increased risk for binomial distributions and a significant negative skew. Due to significant skew the accuracy and error awareness percentages required the arcsine transformation to normalize the distribution. Additionally, in order to account for differences between moderate and severe TBI groups I repeated all non-significant M/S TBI group analyses with only the severe TBI group ($n = 13$ for behavioral and mood analyses and $n = 10$ for ERP component analyses).

To address Aim 1, Hypothesis 1 (the M/S TBI group will have fewer aware errors compared to controls) robust ANOVAs were calculated using the ERP PCA Toolkit to evaluate arcsine-transformed error awareness rates between M/S TBI and control groups. Robust ANOVAs were used in order to overcome the biasing effects of nonnormality, (co)variance heterogeneity between groups, non-orthogonal groups, and to reduce Type I error (Dien, 2010; Keselman, Wilcox, & Lix, 2003). Robust ANOVA statistics are interpreted similarly to traditional ANOVAs, but avoid susceptibility to assumption violations in the same way as traditional ANOVAs. I decomposed significant interactions using Fisher's least significant difference approach, controlling for family-wise Type I error. The seed for the number generation was set at 1,000, and the number of iterations used for bootstrapping was 50,000 for all robust ANOVA analyses (Clayson & Larson, 2012; Dien, Franklin, & May, 2006; Dien, Michelson, & Franklin, 2010; M. J. Larson, Clawson, Clayson, & South, 2012a). I expected that the TBI group would have a significantly lower proportion of aware errors than controls. I also completed additional robust ANOVAs for arcsine transformed no-go accuracy, arcsine transformed accuracy rates for color and repeat trials, and response times for go, error, aware errors, unaware errors, and awareness response trials between groups. I then used a 2-Group (M/S TBI, control) x 2-Trial Type (go, error) robust ANOVA to detect differences in RTs.

To address potential differences in task performance over time related to impairments in attention or fatigue, I split the participant task into an early half from trials 1-450 and a late half from trials 451-900. I also completed separate 2-Group x 2-Time (early, late) robust ANOVAs to compare groups on first and second half behavioral performance for each RT and accuracy condition.

For Aim 1, Hypothesis 2, (there will be group differences in the Pe and ERN amplitudes on error-aware trials and error-unaware trials, respectively) I conducted 2-Group (M/S TBI, control) x 2-Error Type (aware, unaware) robust ANOVAs on Pe and ERN amplitudes using the ERP PCA Toolkit (Dien, 2010). I expected that the M/S TBI group would have significantly lower Pe amplitudes on aware error trials and lower ERN amplitudes on unaware error trials relative to the control group. I did not complete robust ANOVAs comparing early to late performance for ERN and Pe component amplitudes due to too few error trials across participants for adequate sample size (see Electrophysiological Data Recording, Reduction, and Measurement section above). Groups were then compared by gender for ERN and Pe amplitudes using a 2-Group x 2-Gender x 2-Error Type robust ANOVA.

To address Aim 2, I used a previously completed confirmatory factor analysis (CFA) of a shortened version of the AES and BDI-II that showed distinct apathy and depression factors with the purpose of determining appropriate items that loaded onto an apathy composite for inclusion in multiple regression analyses (Kirsch-Darrow, Marsiske, Okun, Bauer, & Bowers, 2011). It is generally recommended that there should be ten times the number of subjects in a factor analysis as the number of variables with a minimum of 100 subjects (Warner, 2008). As the current sample size is below this recommendation and insufficient for a reliable factor analysis I used the Kirsch-Darrow et al. (2011) factor analysis due to their sample size of 146 Parkinson's disease patients. While their CFA was not with a M/S TBI population, apathy has been shown to be a consistent construct and differentiated from depression across multiple neurological disorders (Kirsch-Darrow et al., 2006; Landes et al., 2005; Naarding et al., 2009). The Kirsch-Darrow et al. (2011) CFA found a four-factor model of the AES and BDI-II with the following factors: (1) apathy, (2) dysphoric mood, (3) loss of interest/pleasure, and (4) somatic concerns. The apathy

factor consisted of nine items from the AES (4, 5, 6, 7, 9, 10, 11, 12, and 13). Factor loadings for item parcels were between .646 and .870. The other three factors included the BDI-II items and the remaining AES items. The overall fit of the 4-factor model was considered good (see Kirsch-Darrow et al., 2011). I attempted to replicate the Kirsch-Darrow et al. (2011) factor analysis, but was unable to support an adequately fitting model, most likely due to a significantly smaller sample size in the current study. As such, I used the factor structure from the above model and included results from the specific questions listed above in an apathy variable for the Aim 2 regression analyses.

I determined that the regression analyses would be completed with only the M/S TBI group, as the control group did not have an injury to which the injury severity measures can be applied. Furthermore, the intent of Aim 1 was to demonstrate that there was a difference between the M/S TBI group and the non-TBI group relative to accuracy of error awareness leading to distinct analysis of the correlation of apathy and accuracy in the M/S TBI group.

I next conducted three separate multiple regression analyses for the M/S TBI group with the newly derived apathy variable, duration of LOC and PTA, and RBANS Total score as independent (i.e., predictor) variables. For Hypothesis 1 (increased levels of apathy will be associated with a higher proportion of unaware errors relative to aware errors in those with M/S TBI), I used multiple regression to predict the arcsine transformed unaware errors percentage in the M/S TBI group, when controlling for apathy, duration of LOC and PTA, and RBANS scores. For Hypothesis 2 (increased apathy will be related to decreased Pe and ERN amplitudes), I predicted both Pe error amplitude and ERN amplitude in separate analyses from level of apathy while controlling for injury severity (LOC and PTA duration), and RBANS scores. I used the

variance inflation factor (VIF) to test for multicollinearity (Kleinbaum, Kupper, Muller, & Nizam, 2007).

In order to account for differences between controls that were randomly selected and the remaining controls from the original sample, the remaining control participants were combined (n = 30 for all analyses except ERP analyses where n = 16) and used in a separate set of replication analyses to determine any potential differences due specifically to the subset of controls included in the principal analyses. Any discrepancies between the original control group and the replication control group were subjected to a combined control group analysis to determine results.

Results

Mood and Apathy Analyses

Data for measures of mood and apathy as a function of group are presented in Tables 3 and 4. There were significant between-group differences on levels of depression and apathy reported in the Beck Depression Inventory–II, $t(1,30.14) = -4.27$, $p < .001$, the Apathy Evaluation Scale, $t(1,33.98) = -3.46$, $p = .001$, and the composed apathy score, $t(1,49) = -2.21$, $p = .03$, with the M/S TBI group reporting higher levels of both broad depression and specific apathy compared to non-injured controls on all measures. Notably, neither group's mean depression scores met the threshold for the mild depression lower-bound score of 14 on the BDI-II. Similarly, neither group met criteria for elevated levels of apathy using the cut-score for elevated levels of apathy above 34 on the AES (Andersson, Krogstad, & Finset, 1999b).

The before and after injury scores on the FrSBe subscales of Apathy, Disinhibition, and Executive Functioning and Total score for only the M/S TBI participants indicated significantly decreased post-injury functioning for all scales (see Table 4 for descriptive data and significant

pre-to-post score differences). That is, participants reported worse overall functioning and higher levels of apathy, disinhibition, and executive functioning after injury. The FrSBe was only administered to the M/S TBI group because it is specific to people who have suffered an injury and gives pre- and post-injury scores.

Table 3

Descriptive Data for Mood and Apathy Measures by Group

	M/S TBI (n = 24)			Control (n = 30)			
	Mean	SD	Range	Mean	SD	Range	Significance
BDI - II	11.75	7.84	0 – 28	4.40	3.46	0 – 14	*
AES	30.33	7.16	20 – 50	24.70	3.95	18 – 33	*
Apathy composite	15.54	4.23	10 – 27	13.30	2.98	9 – 20	*

Note. BDI-II = Beck Depression Inventory – II, AES = Apathy Evaluation Scale – Self-report, Apathy composite is the composite apathy scale formed from the significant apathy factor composed of questions from the BDI-II and AES. Scores on the AES range from 18-72. * = $p < .05$, ** = $p < .01$.

Neuropsychological Performance

Analysis of neuropsychological data indicated that there were no significant differences between the M/S TBI and control groups on the RBANS Total score, $t(1,52) = 1.27$, $p = .21$, nor on the WTAR, $t(1,52) = .38$, $p = .71$, indicating no between-group differences on predicted pre-injury cognitive functioning or measured post-injury overall cognitive scores. No group differences for the RBANS domains of immediate and delayed memory, attention, and visuospatial processing (ts < 1.33, ps > .19; see Table 5) were found when comparing M/S TBI

participants to healthy controls. There was a significant difference between the M/S TBI and control groups on the Language subscale, $t(1,52) = 2.02$, $p = .05$, with the M/S TBI group performing below controls indicating decreased performance on verbal fluency and naming tasks in those with a M/S TBI.

Table 4

Descriptive Data from the Frontal Systems Behavioral Scale (FrSBe)

	Before Injury			After Injury			
	Mean	SD	Range	Mean	SD	Range	Significance
FrSBe Total	91.57	12.46	65 – 120	106.75	18.02	64 – 142	*
Apathy	25.54	4.91	17 – 37	31.17	8.03	17 – 45	*
Disinhibition	33.21	4.48	27 – 46	36.50	5.05	28 – 48	*
Executive Function	33.00	6.35	19 – 48	39.08	8.64	19 – 60	*

Note. * = $p < .05$, ** = $p < .01$.

Behavioral Analyses for the EAT

Accuracy and error awareness. Accuracy and response time data for the M/S TBI participants and controls are included in Table 6 below. For tests of Aim 1, Hypothesis 1, there were no significant differences between M/S TBI and control participants on percentage of aware errors, $T_{WJt}/c(1.0,28.6) = 2.20$, $p = .15$. Similarly, there were no significant between-group differences for other measures of EAT accuracy including, no-go accuracy, $T_{WJt}/c(1.0,41.0) =$

0.01, $p = .93$, and no-go accuracy broken down by lure type (i.e., repeat and color) T_{WJt}/cs < 0.06, and ps > .81. Error awareness and error awareness separated by lure type (i.e., repeat and color) showed no significant group differences between M/S TBI and control participants (T_{WJt}/cs < 2.63, ps > .12).

Table 5

Descriptive Data of Neuropsychological Measures by Group

	M/S TBI (n = 24)			Control (n = 30)		
	Mean	SD	Range	Mean	SD	Range
WTAR (WAIS-III Predicted)	109.29	9.53	82 – 121	110.50	7.38	89 - 120
RBANS Total	95.83	22.32	54 – 145	102.10	13.84	77 – 127
Immediate Memory	98.00	19.31	57 – 136	100.53	14.85	78 – 130
Visuospatial	104.29	13.60	75 – 126	107.47	16.17	62 – 126
Language	93.50	19.62	51 – 130	102.53	13.08	78 – 124
Attention	96.29	20.52	55 – 128	99.13	13.43	64 – 120
Delayed Memory	92.63	21.34	48 – 131	98.33	8.95	83 - 112

Note. RBANS = Repeatable Battery for the Assessment of Neuropsychological Status, WTAR = Predicted Wechsler Adult Intelligence Scale -III Full Scale Intelligence Quotient score of the Wechsler Test of Adult Reading.

Response times (RTs). There were no significant differences when comparing the M/S TBI and controls groups on RTs for overall performance on the EAT, T_{WJt}/cs < 45.9, and ps > .08. A separate 2-Trial Type (Go, Error) by 2-Group robust ANOVA for RTs indicated a

significant main effect of accuracy, $T_{WJt}/c(1,43.9) = 15.30$, $p < .001$, with slower error- than go-trial RTs. There was no significant Trial Type x Group interaction, $T_{WJt}/c(1,43.9) = 2.02$, $p = .16$, or main effect of group, $T_{WJt}/c(1,44.2) = 1.73$, $p = .19$ (see Table 6).

Table 6

Behavioral Data for M/S TBI and Control Groups on the Whole Error Awareness Task

	M/S TBI (n = 24)		Control (n =30)	
	Mean	SD	Mean	SD
No-Go Accuracy (% correct)	.49	.23	.49	.19
Repeat No-Go accuracy	.57	.23	.56	.20
Color No-Go accuracy	.40	.25	.42	.22
Error Awareness (% of Aware errors)	.65	.24	.74	.12
Repeat error awareness	.59	.27	.70	.17
Color error awareness	.70	.25	.82	.28
Unaware Error Proportion	.35	.24	.25	.11
Go RT (ms)	533.17	91.16	491.74	77.34
Error RT (ms)	543.88	96.52	519.25	99.10
Aware error RT	550.07	104.51	510.96	96.94
Unaware error RT	537.04	101.44	527.53	107.17
Error Awareness RT (ms)	438.70	92.79	395.38	69.98

Note. Accuracy (percentage of correct responses) data presented in Table 6 are not arcsine transformed and represent the observed overall accuracy rate and percentages of errors a participant was aware of.

Early-to-Late Behavioral Performance

Table 7 contains data comparing EAT first half accuracy and RT performance with second half accuracy and RT performance as a function of group. Figures 4 and 5 contain line graphs showing significant Group x Time interactions for accuracy and error awareness by error trial type. Overall, patterns of performance over time differed between the M/S TBI group and controls with the M/S TBI showing decreasing accuracy and improving awareness over the course of the task. Controls improved accuracy and awareness. Different patterns between color and repeat no-go trials are reported below.

Color no-go error awareness. Robust 2-Group x 2-Time (e.g., early, late) ANOVA comparisons of accuracy for early and late EAT performance showed a significant main effect of time for awareness of color no-go errors, $T_{WJt}/c(1,33.6) = 108.70$, $p < .001$, indicating that both the M/S TBI and control groups improved their awareness of color no-go errors. There was no main effect of group, $T_{WJt}/c(1,36.4) = 0.49$, $p = .49$, or Group x Time interaction, $T_{WJt}/c(1,33.6) = 3.92$, $p = .06$, for color no-go awareness. Both groups improved awareness of color no-go errors over the course of the EAT.

Color no-go accuracy. When comparing the M/S TBI and control groups on early and late color no-go accuracy percentage there was no significant main effect of time, $T_{WJt}/c(1,44.2) = 0.87$, $p = .36$. However, there was a significant main effect of group, $T_{WJt}/c(1,26.5) = 14.07$, $p < .001$, and a significant Group x Time interaction, $T_{WJt}/c(1,44.2) = 24.24$, $p < .001$. Decomposition of the interaction shows that M/S TBI performance on color no-go trials decreased from early to late, $T_{WJt}/c(1,21.0) = 29.28$, $p < .001$, while controls performance increased, $T_{WJt}/c(1,27.0) = 5.63$, $p = .03$. Interestingly, the M/S TBI group had a significantly elevated first-half accuracy percentage, $T_{WJt}/c(1,35.0) = 36.68$, $p < .001$, compared to controls,

but no difference during the later half of the task on color no-go trials, T_{WJt}/c(1,44.1) = 0.00, p = .94.

Repeat no-go awareness. Comparisons of repeat no-go awareness for early and late EAT performance indicated a main effect of time for awareness of repeat no-go errors, T_{WJt}/c(1,37.4) = 5.19, p = .03, suggesting that both the M/S TBI and control groups improved their awareness of repeat no-go errors over time. There was no main effect of group, T_{WJt}/c(1,31.6) = 1.19, p = .29, but there was a Group x Time interaction, T_{WJt}/c(1,37.4) = 5.20, p = .03 for repeat no-go awareness. Decomposition of the interaction showed that M/S TBI awareness of repeat errors remained similar from early to late, T_{WJt}/c(1,21.0) = 0.00, p = 1.00, whereas controls awareness increased during the second half of the task, T_{WJt}/c(1,27.0) = 17.38, p < .001.

Repeat no-go accuracy. There was a significant main effect of time, T_{WJt}/c(1,45.1) = 7.36, p = .01, a significant main effect of group, T_{WJt}/c(1,30.1) = 8.63, p = .01, and a significant Group x Time interaction, T_{WJt}/c(1,45.1) = 17.86, p < .001. Decomposition of the interaction shows an initially worse repeat trial accuracy during the early half of the task for controls, T_{WJt}/c(1,33.8) = 23.88, p < .001, followed by an improvement in repeat no-go accuracy by controls during the later half, T_{WJt}/c(1,27.0) = 27.60, p < .001, that accounts for the interaction. The M/S TBI group showed no difference between repeat no-go accuracy early and late performance when compared to controls, (ps >.33).

Comparisons of early and late performance for RTs indicated a significant main effect of time for go trials, T_{WJt}/c(1,46.6) = 17.50, p < .001, error trials, T_{WJt}/c(1,45.0) = 13.96, p < .001, awareness response trials, T_{WJt}/c(1,46.4) = 23.23, p < .001, and aware and unaware errors, T_{WJt}/c(1,38.6) = 14.62, p < .001 and T_{WJt}/c(1,43.5) = 5.62, p = .02, respectively with both groups

showing a decrease in RTs over time. There were no significant main effects of group or Group x Time interactions for RTs (T_{WJt}/cs < 0.82, ps > .37).

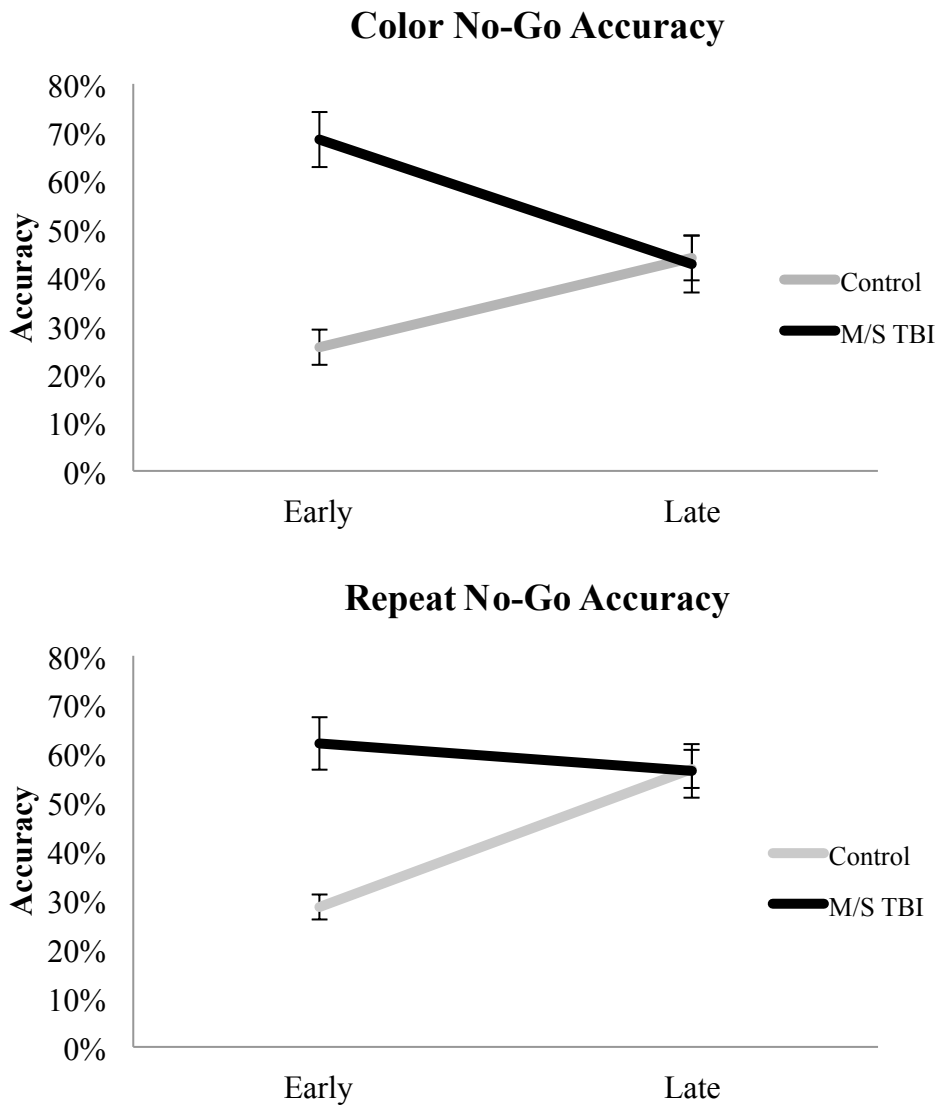

Figure 4. Line graphs showing means and standard error for arcsine-transformed repeat and color no-go accuracy split by early and late sections of the EAT. Error bars represent the standard error.

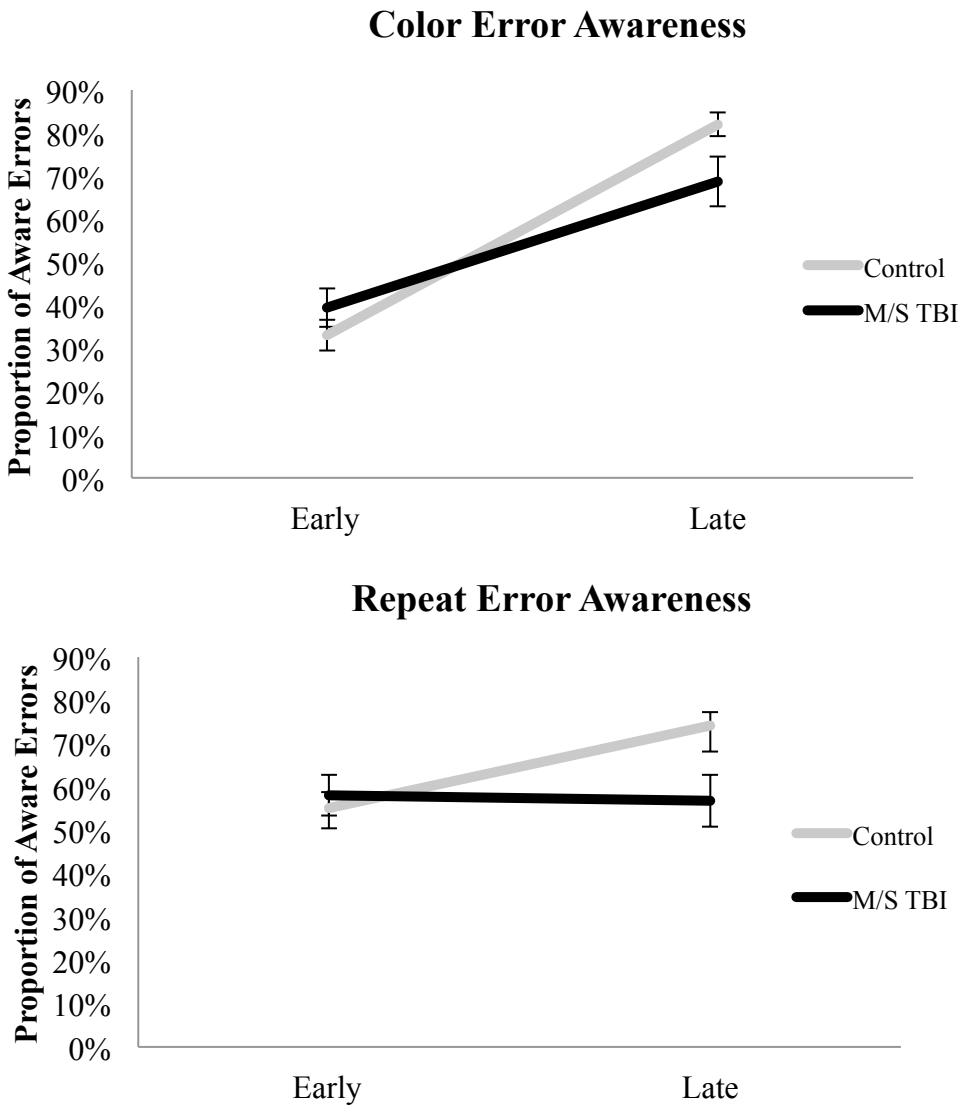

Figure 5. Line graph showing means and standard error for arcsine-transformed repeat and color error awareness split by early and late sections of the EAT. Error bars represent the standard error.

Table 7

Descriptive Data and Early to Late Behavioral Performance Change During the EAT as a Function of Group

	M/S TBI (n = 24)					Control (n =30)				
	Early		Late			Early		Late		
	Mean	SD	Mean	SD	p	Mean	SD	Mean	SD	p
No-Go Accuracy (% correct)	.48	.20	.50	.26	.40	.48	.18	.51	.21	.22
Repeat No-Go accuracy	.62	.26	.56	.27	.30	.29	.14	.57	.21	<.001
Color No-Go accuracy	.69	.28	.43	.29	<.001	.26	.20	.44	.25	.04
Error Awareness (% Aware errors)	.66	.25	.64	.26	.67	.72	.12	.78	.13	.01
Repeat error awareness	.58	.23	.57	.29	.97	.55	.20	.74	.17	<.001
Color error awareness	.39	.22	.69	.28	<.001	.33	.20	.82	.15	<.001
Go RT (ms)	545.07	87.18	521.52	101.12	.03	531.23	84.82	493.27	93.92	<.001
Error RT (ms)	558.64	96.17	524.89	116.29	.03	535.19	96.24	504.69	103.25	.01
Aware error RT	565.92	105.49	527.84	118.50	.05	534.82	95.03	493.73	107.33	.001
Unaware error RT	550.52	103.09	514.93	110.19	.09	534.54	105.43	518.08	110.56	.12
Error Awareness RT (ms)	453.60	91.65	424.45	100.37	.01	426.36	72.19	385.38	81.34	<.001

Note. Accuracy (percentage of correct responses) data presented in Table 7 are not arcsine transformed and represent the observed overall accuracy rate and percentages of errors a participant was aware of. *P*-values represent paired-samples *t*-tests comparing early and late arcsine transformed accuracy and error rates, as well as RTs within groups.

ERP Component Analyses

Waveforms, scalp maps, and descriptive data for the Pe and ERN are presented in Tables 8-9 and Figures 6-8. Groups did not significantly differ on numbers of trials used for ERP analyses for any condition (see Table 8). For amplitude of the Pe, there was a significant main effect of awareness, T_{WJt}/c (1,32.9) = 33.94, $p < .001$, showing that the awareness of errors corresponded with increased Pe amplitude for both groups. There was no significant main effect of group, T_{WJt}/c (1,43.0) = 0.03, $p = .86$, or Group x Awareness interaction, T_{WJt}/c (1,32.9) = 0.41, $p = .52$, when comparing M/S TBI with controls on Pe amplitudes.

For the amplitude of the ERN, there was no significant main effect of awareness, T_{WJt}/c (1,31.7) = 0.03, $p = .87$, or Group x Awareness interaction, T_{WJt}/c (1,31.7) = 3.05, $p = .10$, when comparing M/S TBI and control groups. There was a significant main effect of group for the ERN with the M/S TBI group having more negative ERN amplitude than controls, T_{WJt}/c (1,40.3) = 4.37, $p = .04$ (see Table 9 and Figures 6 and 8).

Table 8

Descriptive Data for Number of Trials by Condition as a Function of Group

	M/S TBI			Control			
	Mean	SD	Range	Mean	SD	Range	Significance
Aware	45.42	21.09	8 - 77	52.60	23.44	17 - 106	
Unaware	32.42	25.37	7 - 94	19.87	13.16	6 – 54	
Correct	413.52	85.26	264 - 512	427.67	71.08	240 - 516	

Note. Included in Table 8 are mean numbers of trials by condition included in ERP component analyses for the ERN and Pe components. No significant differences between M/S TBI and control groups on number of trials was found.

Table 9

Error Trial ERP Means and Standard Deviation (SD) of Pe and ERN Amplitudes by Group

	TBI (*n* = 19)						Control (*n* = 30)					
	Unaware			Aware			Unaware			Aware		
	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range
Pe	0.08	1.30	-2.02-3.67	1.92	2.20	-2.04-5.68	-0.04	1.61	-2.81-3.55	2.21	2.12	-2.05-6.16
Pe (severe only, *n = 10*)	0.54	1.33	-0.81-3.67	1.45	1.81	-1.25-4.00						
ERN	-0.17	1.64	-2.66-3.51	-0.59	1.64	-2.04-5.68	0.26	1.57	-2.61-5.21	0.73	1.69	-1.98-4.05
ERN (severe only, *n* =10)	-0.46	1.31	-2.66-1.90	-0.66	1.83	-4.61-2.14						

Note. Pe = Post-error positivity, ERN = Error related negativity.

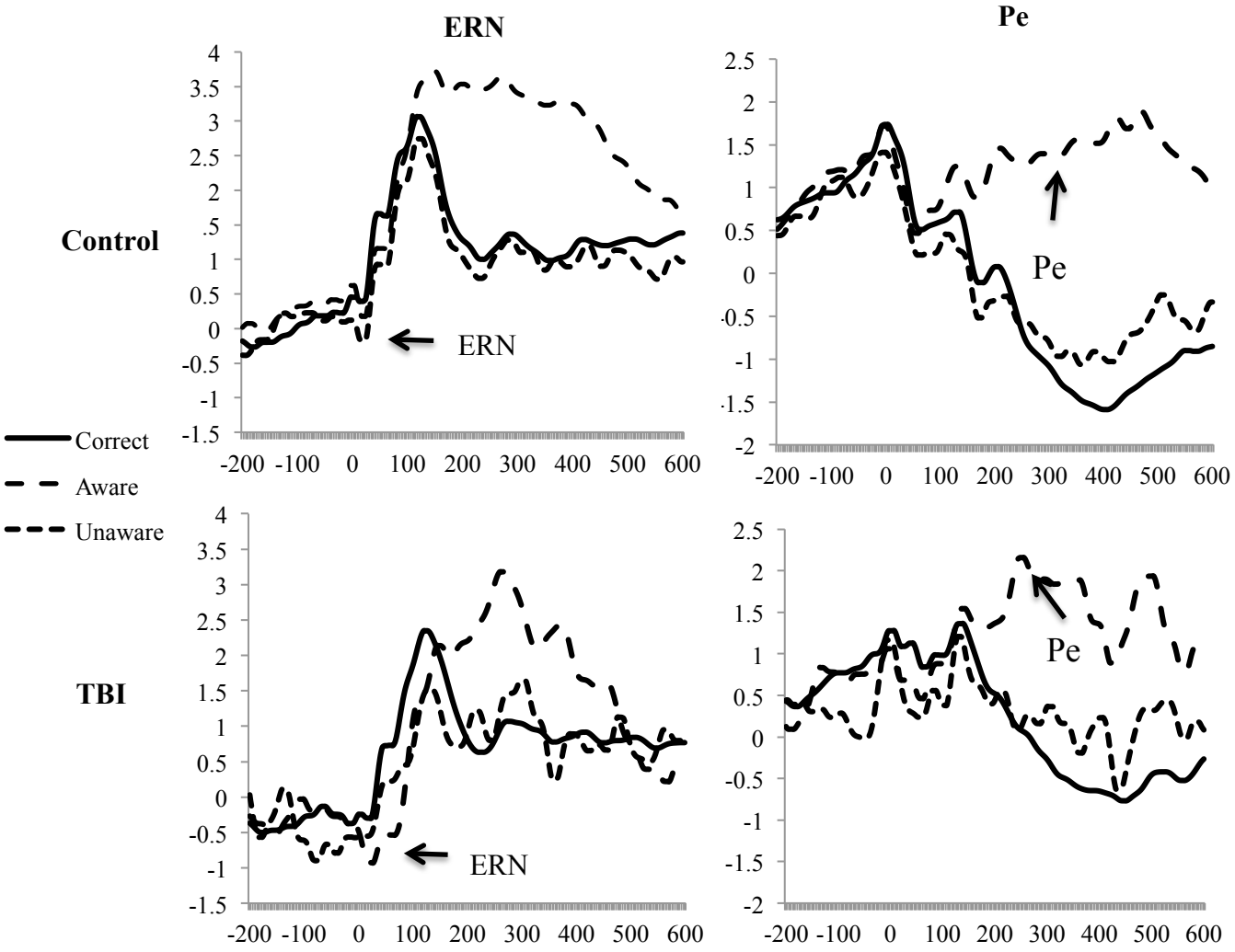

Figure 6. Grand average waveforms for the ERN and Pe components by group. Waveform figures were smoothed using 3-median smoothing techniques alternated with a 3-median skip. Original segmented epochs are response-locked at -400-800ms. The waveforms shown are epoch windows from -200-600ms.

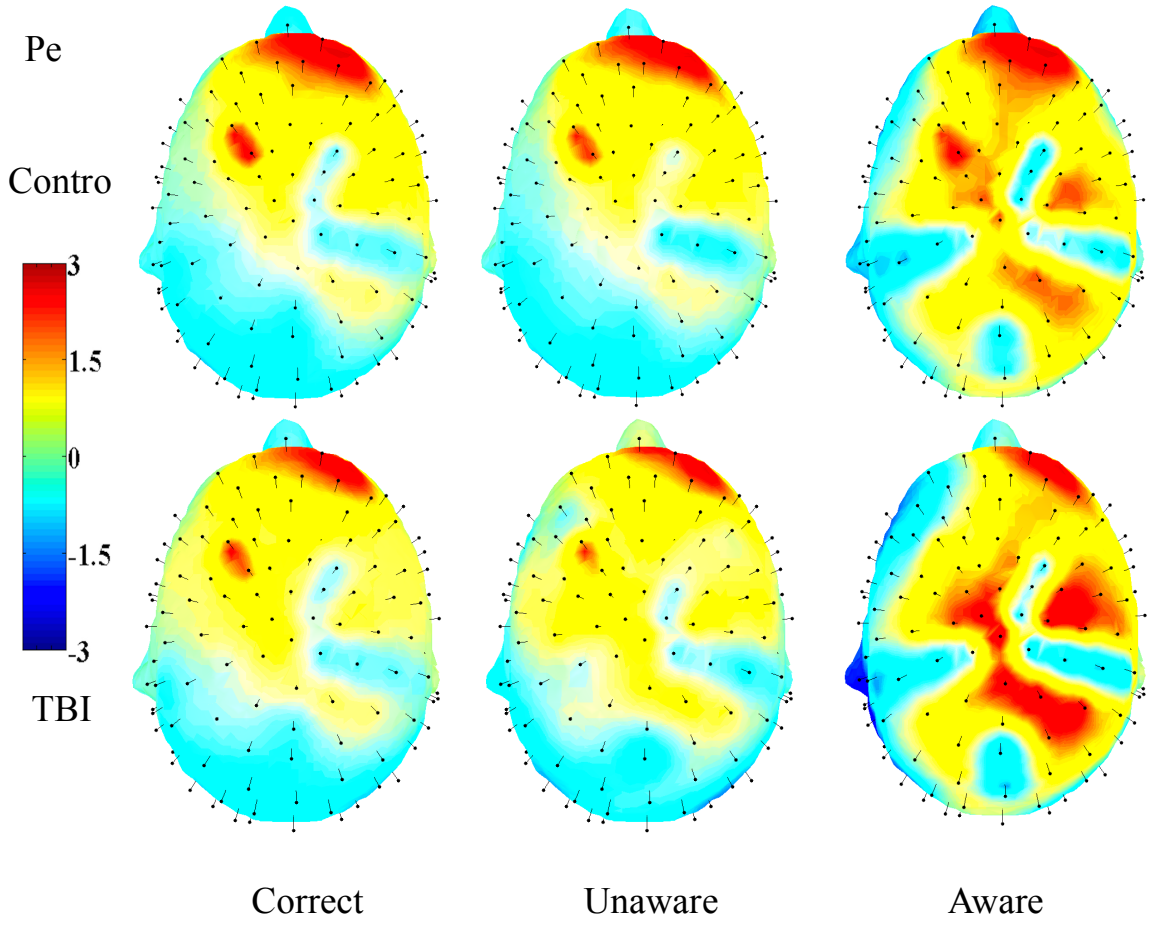

Figure 7. Topographical representation of Pe component mean voltages in microvolts (μV) from 200-400 ms post response for correct, unaware, and aware trials by group. Scalp maps created with ERP LAB (Lopez-Calderon & Luck, 2014).

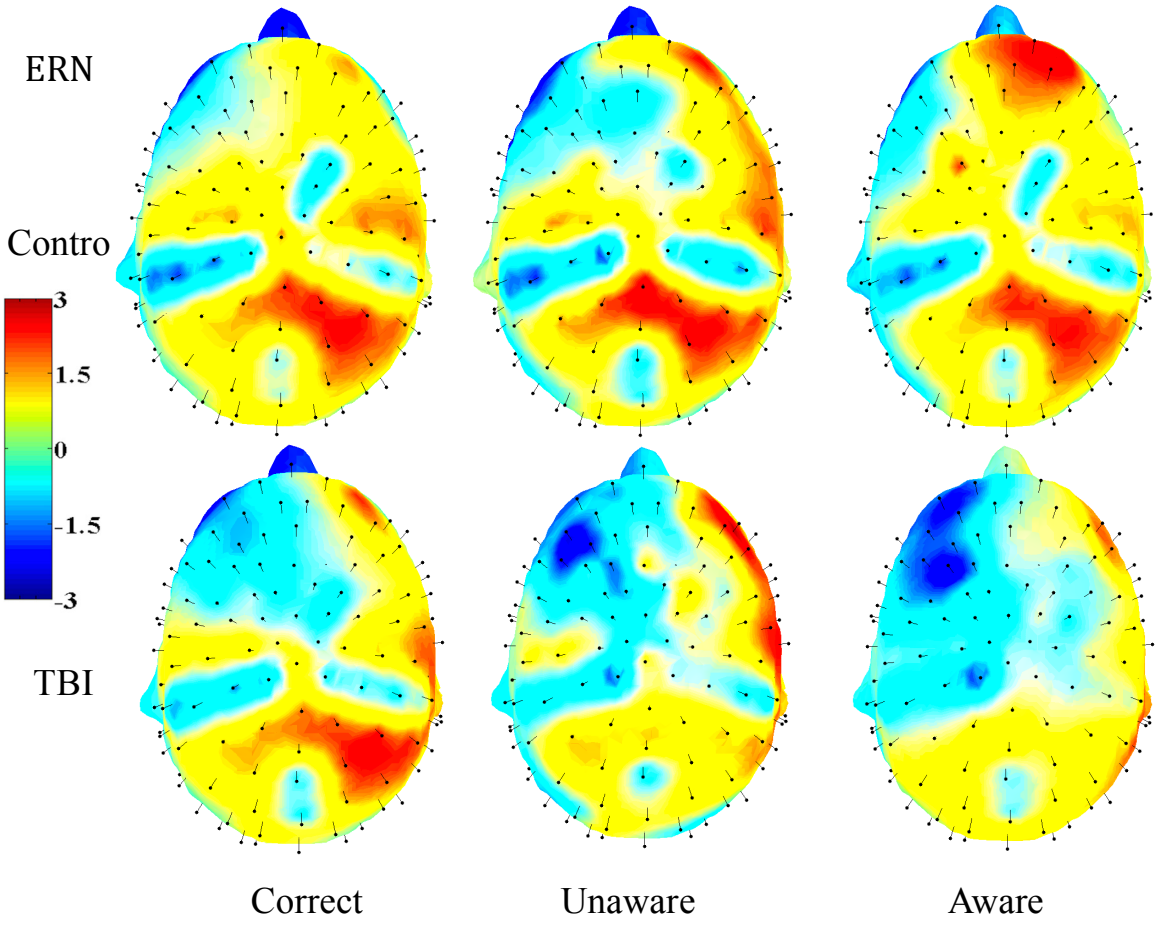

Figure 8. Topographical representation of ERN component mean in microvolts (μV) voltages from 0-100ms post response for correct, unaware, and aware trials by group. Scalp maps created with ERP LAB (Lopez-Calderon & Luck, 2014).

Severe TBI Analyses

Neuropsychological analyses. Further analysis comparing only the participants who experienced a severe TBI (excluding those with moderate TBI) to control participants indicated significant differences between the severe TBI and control groups on the RBANS Total scale, $t(1,41) = 2.21$, $p = .03$ ($M = 88.85$, SD = 25.53), Language subscale, $t(1,41) = 2.90$, $p = .01$ ($M = 88.23$, SD = 18.43), and Attention subscale, $t(1,41) = 2.75$, $p = .01$ ($M = 81.23$, SD = 29.62) showing that controls performed significantly better than severe TBI participants on overall

cognitive, language, and attention tasks. There were no group differences between severe TBI and control groups on Wechsler Adult Intelligence Scale-III (WAIS-III) Full Scale Intelligence Quotient (FSIQ)-predicted WTAR scores, $t(1,52) = 0.53$, $p = .60$, indicating no predicted pre-injury differences in intelligence.

Behavioral analyses of the EAT. There were nonsignificant group differences between severe TBI participants and controls for no-go accuracy, $T_{WJt}/c(1.0,19.9) = 0.53$, $p = .48$, and for error awareness, $T_{WJt}/c(1.0,25.5) = 3.72$, $p = .07$, when comparing groups on whole task performance. Similarly, with only the severe TBI participants there were no group differences for RTs ($T_{WJt}/cs < 2.31$, $ps > .16$) when compared to controls. Results of a 2-Trial Type (Go, Error) by 2-Group robust ANOVA for RTs using only the severe TBI group indicated a significant main effect of trial type, $T_{WJt}/c(1,18.8) = 7.00$, $p = .03$, again with error trials having slower RTs than go trials for both groups. There was no significant main effects of group, $T_{WJt}/c(1,22.0) = 0.44$, $p = .51$, or Group x Trial Type interactions, $T_{WJt}/c(1,18.8) = 1.46$, $p = .24$. In sum, the severe TBI group, when compared to controls, performed similarly to the M/S TBI group.

Early-to-late behavioral performance. When comparing early-to-late EAT performance for only the severe TBI group fewer differences were found for RTs, accuracy, and error awareness performance (see Table 10). Between-groups comparisons with controls and severe TBI groups showed no significant interactions ($T_{WJt}/cs < 3.72$, $ps > .07$). The lack of significant findings could be due to the limited sample size of severe TBI participants.

Table 10

Descriptive Data of Early to Late Behavioral Performance Change for the Severe TBI Group

	Early		Late		
	Mean	SD	Mean	SD	*p* -value
No-Go Accuracy (% correct)	.43	.20	.44	.27	.85
Repeat No-Go accuracy	.57	.31	.52	.30	.61
Color No-Go accuracy	.65	.29	.35	.27	.001
Error Awareness (% of Aware errors)	.61	.29	.63	.28	.56
Repeat error awareness	.53	.25	.51	.34	.88
Color error awareness	.33	.22	.70	.27	.01
Go RT (ms)	531.54	93.23	509.97	106.80	.20
Error RT (ms)	541.25	97.55	513.18	126.95	.25
Aware error RT	561.52	115.88	518.63	132.46	.27
Unaware error RT	516.99	95.54	493.16	95.90	.36
Error Awareness RT (ms)	446.86	109.42	413.79	113.50	.04

Note. Accuracy (percentage of correct responses) data presented in Table 10 are not arcsine transformed and represent the observed overall accuracy rate and percentages of errors a participant was aware of. *P*-values represent paired-samples *t*-tests comparing early and late arcsine transformed accuracy and error rates, as well as RTs within groups.

ERP component analyses. Descriptive data for ERN and Pe amplitudes for the severe TBI group were included in Table 9 above. There was a significant main effect of awareness, T_{WJt}/c (1,16.0) = 17.90, p = .001, but nonsignificant main effect of group, T_{WJt}/c (1,21.1) = 0.02,

p = .88, and Group x Awareness interaction, T_{WJt}/c (1,16.0) = 3.35, p = .09, for Pe amplitudes when comparing severe TBI participants with controls.

For ERN amplitudes, comparing only severe TBI participants to controls there was no significant main effect of awareness, T_{WJt}/c (1,14.0) = 0.25, p = .63, or Group x Awareness interaction, T_{WJt}/c (1,14.0) = 1.30, p = .28. There was also a nonsignificant main effect of group for ERN amplitudes, T_{WJt}/c (1,16.5) = 4.14, p = .06, indicating that the severe TBI group had similar amplitude ERN components when compared to controls.

ERP Analyses by Gender

Table 11 contains results of gender comparisons for electrophysiological indices of error awareness by group. Analyses indicated no significant gender differences for either ERP component, T_{WJt}/cs < 0.42, ps > .52, and no significant Group x Gender interactions, T_{WJt}/cs < 0.40, ps > .50. There are no detected gender differences for ERP waveforms.

Replication Analyses

I used the second control group, as previously described, to conduct a sensitivity analysis by replicating primary analyses from the main study to provide confirmation of results. Results of replication analyses using an alternate control group compared to the M/S TBI group indicated similar outcomes on demographics, measures of mood and apathy, and neuropsychological tests. Behavioral results including RTs and accuracy showed similar findings when using both control groups. Results from robust ANOVAS of early to late performance comparisons for accuracy and RTs for the alternate control group mirrored the previously reported results. However, there was one point of discrepancy between the two sets of results related to repeat-trial error awareness.

Table 11

Error Trial ERP Means and Standard Deviation (SD) of Pe and ERN Amplitudes by Group and Gender

	TBI (n = 19)							
	Male (n = 12)				Female (n = 7)			
	Unaware		Aware		Unaware		Aware	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Pe	-0.18	1.06	2.13	2.39	0.53	1.63	1.55	1.93
ERN	0.05	1.78	-0.84	2.01	-0.53	1.40	-0.17	0.55
	Control (n = 30)							
	Male (n = 12)				Female (n = 18)			
	Unaware		Aware		Unaware		Aware	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Pe	0.48	1.45	1.35	2.12	-0.39	1.65	2.77	1.98
ERN	0.41	1.89	0.46	1.69	0.16	1.36	0.90	1.71

Similar to the original control group, when comparing the alternate control group to M/S TBI there was a main effect of time, $T_{WJt}/c(1,47.8) = 9.23$, $p < .001$, indicating that both groups improved their awareness of repeat errors. There continued to be no main effect of group, $T_{WJt}/c(1,40.9) = 0.00$, $p = .99$, as well. The difference was in that there was not a Group x Time interaction, $T_{WJt}/c(1,47.8) = 0.20$, $p = .66$ for repeat no-go awareness, as was previously seen. Results from a combined control group analysis of repeat error awareness indicated a trend-level main effect of time, $T_{WJt}/c(1,31.6) = 3.68$, $p = .06$, and a trend-level Time x Group interaction, $T_{WJt}/c(1,31.6) = 3.64$, $p = .07$, with controls showing improvements in repeat-trial awareness from early-to-late in the task while the M/S TBI group maintained a similar performance. There was no significant main effect of group, $T_{WJt}/c(1,28.0) = 0.88$, $p = .35$. Analysis of ERP results

indicated accurate replication of findings from the original control group. Given these findings, it appears that there is fairly consistent replication of findings between the two control groups with the exception of the repeat-trial error awareness. Results related to repeat error awareness were interpreted cautiously given the inconsistency in findings.

The Role of Apathy in Error Awareness

Results of the correlation and regression analyses for Aim 2 were completed with only the M/S TBI sample because of the need to control for injury severity in the analyses and the lack of range in the control participant data. Zero-order correlations between arcsine transformed unaware accuracy and apathy, mood, and neuropsychological variables are shown in Table 12. As expected, the AES, BDI-II, and FrSBe post-injury Total and Apathy scales were all highly correlated with each other, but were not significantly correlated with error awareness. The RBANS total score was significantly correlated with percentage of unaware errors.

Table 13 shows regression coefficients and individual predictor values while Figure 9 contains a scatter plot of apathy scores and unaware error arcsine-transformed unaware error percentages for the M/S TBI group. Analysis of Aim 2, Hypothesis 1 revealed a significant model, $F(4,19) = 4.65$, $p = .01$ $R^2 = 49.5\%$. Apathy was a significant negative predictor of the proportion of arcsine-transformed unaware errors when controlling for injury severity (LOC and PTA durations) and neuropsychological functioning (RBANS). Duration of PTA also accounted for a significant proportion of the variance in the model. Contrary to predictions, the model indicated that as apathy increased the proportion of unaware errors actually decreased.

Table 12

Zero-order Correlations Between Unaware Accuracy, Apathy, Mood, and Neuropsychological Variables for the M/S TBI Group

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. AES	1												
2. BDI-II	0.69**	1											
3. FrSBe Total	0.48*	0.74**	1										
4. Apathy	0.62**	0.63**	0.86**	1									
5. Disinhibition	0.05	0.39	0.58**	0.18	1								
6. Executive Function	0.40	0.73**	0.95**	0.77**	0.45*	1							
7. RBANS Total	-0.01	-0.08	-0.15	-0.08	-0.24	-0.09	1						
8. AQ Total	-0.13	-0.74**	-0.50*	-0.27	-0.26	-0.64**	0.02	1					
9. Unaware Error Rate	-0.01	0.10	0.15	0.08	0.06	0.21	-0.34*	-0.22	1				
10. ERN	-0.10	-0.28*	-0.15	-0.06	-0.38	-0.00	0.36*	-0.13	-0.24	1			
11. Pe Unaware	0.31*	0.11	0.13	0.13	-0.17	0.26	0.02	-0.40	-0.02	0.22	1		
12. Pe Aware	-0.03	-0.05	-0.01	0.11	-0.35	0.13	0.26	0.0.10	-0.03	0.18	0.28	1	
13. Apathy composite	0.82**	0.87**	0.82**	0.88**	0.25	0.75**	-0.05	-0.46*	-0.08	-0.40	0.08	-0.24	1

Note. AES = Apathy Evaluation Scale - Self-report, BDI-II = Beck Depression Inventory; FrSBe = Frontal Systems Behavioral Scale; FrsBe subscales include: Apathy, Disinhibition, and Executive Functioning; RBANS = Repeatable Battery for the Assessment of Neuropsychological Status; AQ = Awareness Questionnaire – Self-report form; ERN = Adaptive mean amplitude (μV) for the error related negativity; Pe = Adaptive mean amplitude (μV) for unaware and aware error trials of the post-error positivity. Unaware Error Rate is the arcsine-transformed proportion of total errors that were unaware errors. All subscales and Total scale correlations from the FrSBe are from post-injury self-ratings. The Apathy composite consists of a subset of AES questions compiled following a factor analysis showing specific questions which loaded on an apathy-related factor from both the AES and the BDI-II completed by Kirsch-Darrow et al. (2011).

* - $p < .05$, ** - $p < .01$.

Table 13

Hierarchical Regression Model with Arcsine-Transformed Unaware Error Proportion as the Dependent Variable

	B	*B (Std. Err.)*	β	*t*	*p*-value	Partial *R*	VIF
Apathy	-.02	.01	-.34	-2.07	.05	-.43	1.01
RBANS Total Score	-.01	.01	-.41	-1.93	.07	-.40	1.75
LOC Duration	.00	.00	-.36	-1.46	.16	-.32	2.33
PTA Duration	.00	.00	.60	2.82	.01	.54	1.70

Note. Loss of consciousness (LOC) and post-traumatic amnesia (PTA) duration were calculated in hours. The symbol "*B"* represents unstandardized coefficients while "β" is the standardized coefficient. Partial *R* is the correlation between the predicted variable and the predictor variable when controlling for all other predictors.

Tables 14 and 15 contain regression-specific data related to ERN and Pe amplitude as dependent variables. Figures 10 and 11 are scatter plots of apathy scores and ERN and Pe amplitudes. Relative to Hypothesis 2 of Aim 2, the model predicting ERN amplitude was not significant, $F(4,14) = 2.86$, $p = .06$, $R^2 = 45.0\%$. The model predicting Pe amplitude was nonsignificant, $F(4,14) = 0.39$, $p = .81$, $R^2 = 10.1\%$.

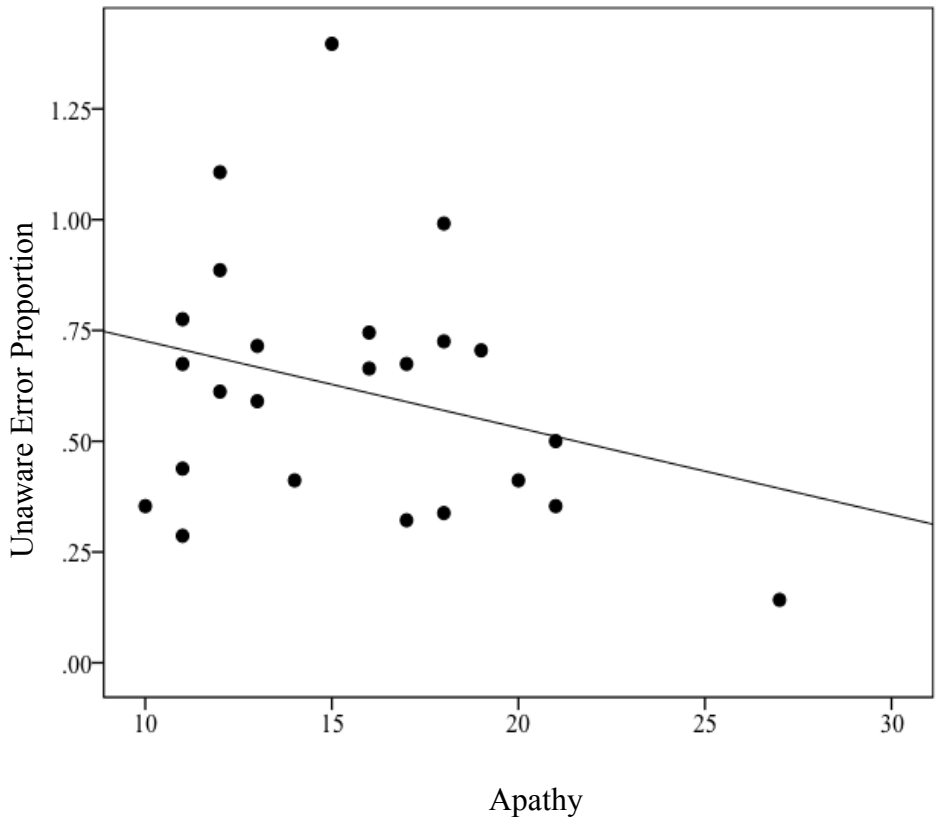

Figure 9. Scree plot of apathy scores and unaware error proportions from the M/S TBI group. Unaware error proportions are arcsine-transformed allowing for improved distribution normality and scores above 1.00.

Table 14

Hierarchical Regression Model with ERN Amplitude as the Dependent Variable

	B	*B (Std. Err.)*	β	*t*	*p*-value	Partial *R*	VIF
Apathy	-.07	1.51	-.22	-1.09	.29	-.28	1.05
RBANS Total Score	-.07	.01	.85	3.22	.01	.65	1.77
LOC Duration	.00	.00	.44	1.33	.21	.34	2.79
PTA Duration	.00	.00	.06	0.21	.84	.06	2.10

Note. Loss of consciousness (LOC) and post-traumatic amnesia (PTA) duration were calculated in hours. The symbol "*B"* represents unstandardized coefficients while "β" is the standardized coefficient. Partial *R* is the correlation between the predicted variable and the predictor variable when controlling for all other predictors.

Table 15

Hierarchical Regression Model with Pe Amplitude as the Dependent Variable

	B	*B (Std. Err.)*	β	*t*	*p*-value	Partial *R*	VIF
Apathy	.02	.11	.05	.18	.86	.05	1.05
RBANS Total Score	.02	.02	.29	.85	.41	.22	1.77
LOC Duration	.00	.00	.16	.38	.71	.10	2.79
PTA Duration	.00	.00	-.21	-.58	.57	-.15	2.07

Note. Loss of consciousness (LOC) and post-traumatic amnesia (PTA) duration were calculated in hours. The symbol "*B"* represents unstandardized coefficients while "β" is the standardized coefficient. Partial *R* is the correlation between the predicted variable and the predictor variable when controlling for all other predictors.

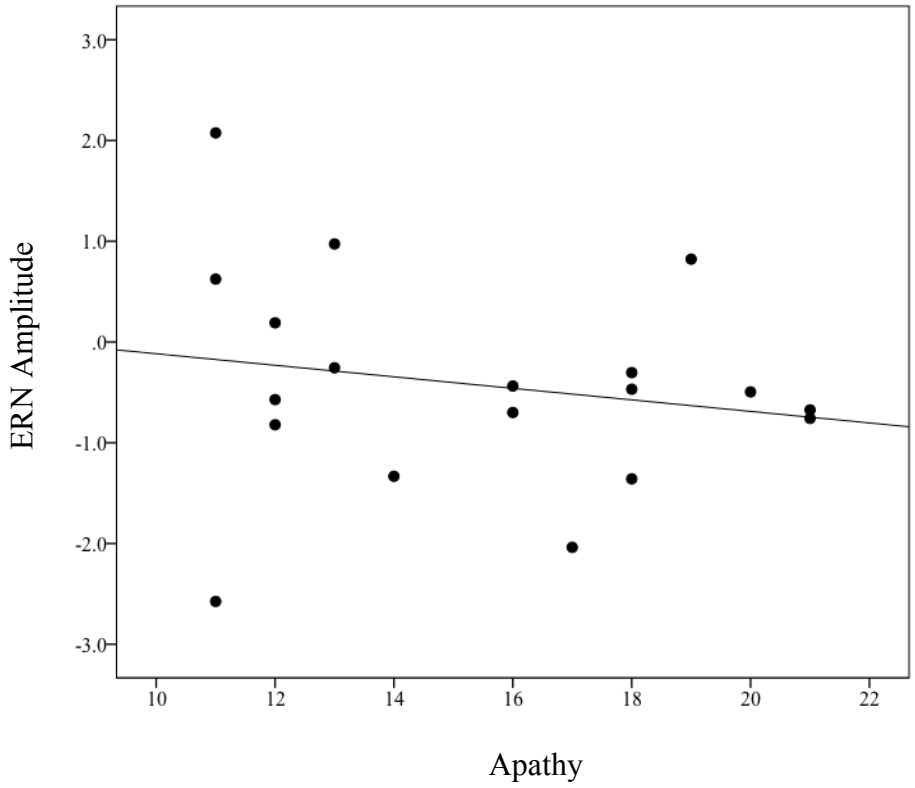

Figure 10. Scree plot of apathy scores and ERN amplitudes from the M/S TBI group.

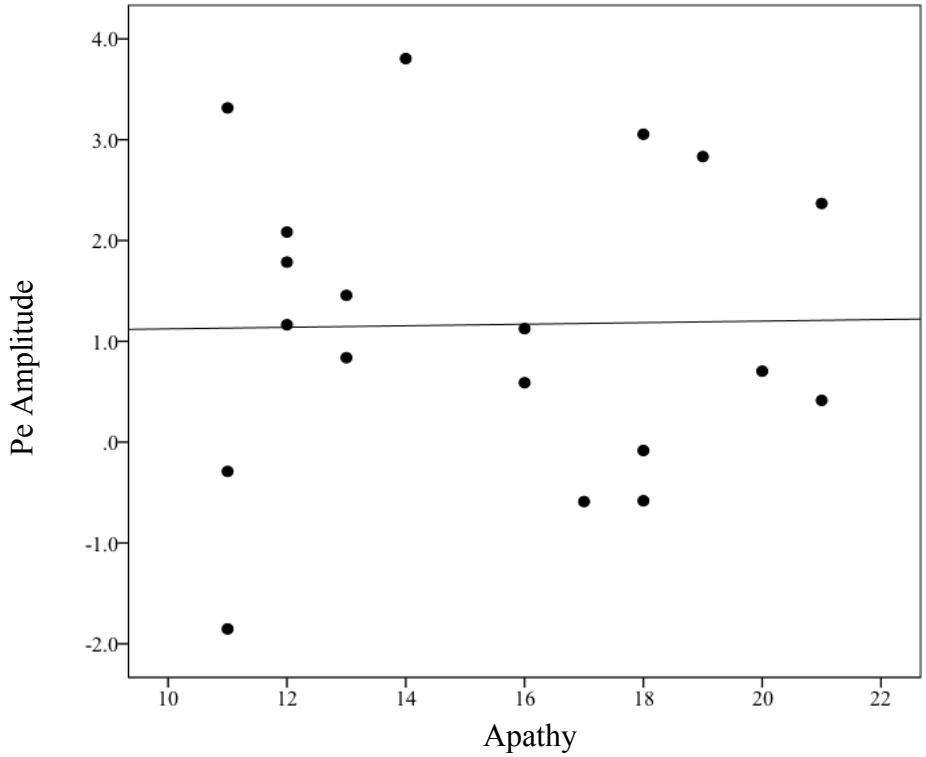

Figure 11. Scree plot of apathy scores and Pe amplitudes from the M/S TBI group.

Discussion

The purpose of this dissertation was to provider greater understanding of the role of apathy in cognitive control and specifically error awareness in those with M/S TBI. I intended to add to the growing body of error awareness in M/S TBI literature by looking at how levels of self-reported apathy influenced electrophysiological and behavioral measures of error awareness. This study aimed to determine: first, if there were group differences on electrophysiological and behavior indicators of conscious error awareness, and; second the relationship of apathy with the same indices of error awareness. The study hypotheses were: (1) that those with M/S TBI would have fewer aware errors (measured through the proportion of aware to unaware errors) than controls; (2) that there would be group differences between the M/S TBI group and controls for Pe component amplitudes on aware trials and ERN component amplitudes on unaware error trials; (3) that increased levels of apathy would be associated with a higher proportion of unaware to aware errors for the M/S TBI group; and (4) that higher apathy levels would be associated with decreased Pe and ERN component amplitudes in the M/S TBI group.

Findings from this study include an older M/S TBI than control group, as well as increased levels of apathy and depression, and decreased neuropsychological functioning for the M/S TBI group compared to control participants. Groups were similar on electrophysiological indicators of error awareness. However, the M/S TBI group had better early-task accuracy than controls, which decreased over time, whereas the control group improved performance over time completing the task. Awareness of color errors improved for both groups, but awareness of repeat errors only improved for control participants over time. Apathy predicted the proportion of unaware errors for the M/S TBI group, but in the opposite direction as expected.

In general, the results of the current study were mixed. Results indicated some significant differences between the groups on behavioral measures related to accuracy and error awareness. However, relative to the first hypothesis, there was not a difference between-groups on the proportion of aware errors when looking at performance over the entire task. Differences began to emerge only when looking at levels of error awareness and accuracy over time by comparing early task performance with later task performance. Results show that individuals in the M/S TBI group started the task with better accuracy for color and repeat trials and either decreased their level of performance, as was the case for color trials, or maintained similar performance for repeat trials. In comparison, controls started with relatively lower accuracy than the M/S TBI group for both types of trials and improved to similar levels as the M/S TBI group. The pattern of change in error awareness and accuracy indicated that the M/S TBI group started out more accurate and less aware of errors than controls, but performance decreased or stayed the same while control accuracy and awareness increased.

The results of differential awareness as a function of group and time on the task are mixed relative to previous work in the area of error awareness in a M/S TBI sample. Similar to previous studies, results from the current study show improvements in RTs across groups over time (M. J. Larson et al., 2007; 2009; M. J. Larson & Perlstein, 2009); however, current results showed significant accuracy differences between groups over time. Previous studies did not look at differential task performance in the early and late halves of their respective tasks. Current findings show no differences between groups when looking at the entire EAT, but analyses comparing early and late performance between groups show significant behavioral differences over time. The group differences over time provide evidence to support accuracy and awareness differences between M/S TBI and control groups that may not conflict with previous studies, and

may provide some additional support for their findings. An interesting question would be to reanalyze the previous awareness datasets (e.g., M.J. Larson & Perlstein, 2009) to determine if there were behavioral accuracy differences between groups at different time points in the task. Taken together, results form early-to-late analyses support Aim 1 Hypothesis 1, in that on behavioral measures there were group differences between the TBI and control groups, but only as a function of time completing the task.

The findings that the M/S TBI group had decreasing accuracy over time while controls improved their accuracy, connected with increased awareness of color errors in both groups indicates that the M/S TBI group was possibly attending less to errors. A conclusion of decreased attention comes from the finding that the M/S TBI group was making more errors at the end of the task, potentially a result of attentional slips leading to decreased performance. The M/S TBI group improved awareness of errors for color trials indicating that, despite initial errors of commission, they improved their evaluative performance monitoring ability at a similar rate as controls. However, the M/S TBI group did not correct and regulate performance in the same way as controls. Not only did the M/S TBI group not correct and improve their performance over time in the same way as controls, for color trials they actually got worse. The decrease in performance indicates that whereas the M/S TBI group did successfully evaluate performance through increasing error awareness, they did not regulate that performance, as accuracy decreased over time. Maintenance of evaluative monitoring but not regulative improvements relates back to the Crosson et al. (1989) model of awareness in that whereas the M/S TBI group had intellectual awareness of errors at a level allowing error recognition they did not develop awareness at the level of emergent or anticipatory awareness. By not developing emergent or anticipatory awareness they were unable improve performance in the moment of the color or

repeat no-go trial, and were unable to anticipate and make cognitive changes to plan how to improve future performance on similar no-go trials.

One potential explanation for why the TBI group became less accurate as the task progressed, but became more aware of errors may be related to deficits in attention. Whereas controls showed improved performance and error awareness over time due to better attentional abilities and a lack of attentional slips. In comparison to controls, the M/S TBI group was unable to adjust performance sufficiently to improve accuracy. This discrepancy is most likely related to deficits in emergent and anticipatory awareness (see the Crosson et al., 1989 model of awareness in the Introduction) where higher levels of awareness may not be accessible to the M/S TBI group due to associated deficits in sustained attention (Lamme, 2003; McAvinue et al., 2005). The M/S TBI group did not attend well enough to the task to stop them from making errors on similar types of trials as they were happening. However, increases in error awareness signify that they recognized the error, but only after it had occurred and not in a way that they could attend sufficiently to prevent a similar error in the future. Whereas RBANS-measured attention was similar between the M/S TBI and control groups, the severe TBI group demonstrated impaired attention when compared to controls. Despite the measured attentional discrepancies there is cause to question if the RBANS was measuring the same type of attention and vigilance as is required for constant monitoring of performance. Based on considerable evidence from research into the subscales of the RBANS, it is my contention that the RBANS does not address the issue of attending to and being conscious of performance deficits in those with TBI in real-world settings.

A contention about the RBANS Attention subscale is based on the types of tasks included in the Attention subscale and the wide variety of abilities that can fall under the construct of

attention such as mental control, working memory, hemi-neglect, focused attention, and divided attention (Zomeren & Brouwer, 1994). Convergent validity analysis of the Attention subscale indicates poor validity in that it is not significantly correlated with other measures of attention such as the Trailmaking Test A and B and Line Cancellation (E. Larson, Kirschner, Bode, Heinemann, & Goodman, 2005). However, the tests comprising the Attention subscale, Digit Span and Coding, do correlate highly with the Wechsler Adult Intelligence Scale-III Working Memory index (Gold, Queern, Iannone, & Buchanan, 1999). It can be safely concluded that while working memory is an important aspect of attention other aspects of attention such as vigilance, sustained attention, and mental control are more involved in task and error monitoring once initial requirements have been learned in the EAT, and the Attention subscale of the RBANS may not adequately represent those aspects of attention.

Two hypotheses from M/S TBI group results lead to further research questions: 1) deficits in complex attentional processes may lead to results showing differential patterns in accuracy, and 2) attentional deficits may be associated with decreased levels of error awareness over time for the M/S TBI group despite similar physiological responses as controls to errors. The question of whether or not complex attentional processes are leading to decreased error awareness is an interesting question that has received some support. No-go errors (false positive button presses) during the Sustained Attention and Response Task (a task similar to the EAT, but with a focus on sustained attention over time) were associated with impaired error awareness, suggesting that lapses in sustained attention or inhibition may result in greater numbers of unaware errors (McAvinue et al., 2005). Of note is that TBI survivors who have DAI, particularly to the white matter innervating the frontal lobes, exhibit similar patterns of executive and attentional deficits as those with various focal frontal lesions (Dockree & Robertson, 2011;

Stuss, 1998). Executive deficits can be seen through deficits in sustained attention, inhibition of prepotent responses, and monitoring of environmental changes. All of these are processes that may be employed during a specific task (Stuss, 2011), such as the EAT task. Sustained attention, inhibition of prepotent responses, and monitoring of environmental changes draw upon resources from the environment and/or require endogenous behavioral control to maintain a goal-directed focus, which can be compromised following a TBI and lead to increased attentional lapses and decreased awareness of errors (O'Keeffe, Dockree, Moloney, Carton, & Robertson, 2007). O'Keeffe et al. (2007) proposed that more cognitively simple tasks will increase the challenge of maintaining attention and alertness to combat the monotony of the task, but more cognitively challenging tasks will be more stimulating and increase alertness to task demands.

The EAT task does have increased initial cognitive demands compared to later in the task, in that the person must learn and remember two competing rules and various instructions related to the signaling of an aware error. Results show that participants were able to quickly master those rules and procedures without difficulty, as evidenced by the fact that all but one severe TBI participant were able to learn the task requirements on the first practice session. Due to the length of the task and the speed at which stimuli are presented cognitive demands, while not reduced, may wane once participants are engaged in the task due to monotony and fatigue. There is some automation of responses with the majority of trials being go-trials, potentially resulting in difficulty maintaining attention and vigilance to performance. The characterization of decreased awareness of errors due to attentional drift is consistent with other studies in M/S TBI survivors (Dockree & Robertson, 2011; McAvinue et al., 2005; O'Keeffe et al., 2007).

Results suggest a similarity between groups on electrophysiological measures of error awareness. For the ERPS, the Pe component showed significant differences for awareness

demonstrating increased amplitude Pe for aware compared to unaware errors—consistent with previous findings (Charles, Van Opstal, Marti, & Dehaene, 2013; O'Connell et al., 2007). Contrary to Hypotheses 2 of Aim 1, the findings from the Pe component, representing conscious error awareness, showed no significant differences between the M/S TBI and control groups. A lack of group differences on the Pe would seem to indicate that the Pe is intact in those with M/S TBI and signals conscious awareness of errors. In other words, similar Pe amplitudes between the M/S TBI group and controls indicates that both groups had similar electrophysiological representations of conscious error awareness. There is some debate about whether or not the Pe is a binary indicator of error awareness or if it corresponds to error awareness inputs from other sources such as the ERN (Overbeek, Nieuwenhuis, & Ridderinkhof, 2005a; Shalgi & Deouell, 2013; Steinhauser & Yeung, 2010). Elevated Pe amplitude is thought to represent awareness of errors with amplitudes of unaware errors being similar to correct responses (Hughes & Yeung, 2011; Riesel, Weinberg, Endrass, Meyer, & Hajcak, 2013), as is the case in this study. Analogous to current findings, the Pe component did not differentiate between TBI and controls in other studies not related to conscious error awareness (M. J. Larson et al., 2007; 2009). However, previous results also show that levels of deficit awareness drawn from differences in self-reported and significant other-reported deficit awareness are positively correlated with Pe amplitudes (M. J. Larson & Perlstein, 2009). Further work is needed to confirm if awareness of deficits correlates with conscious awareness of errors in real time evaluation of the Pe component of the ERP.

The ERN, in contrast, had several significant between-groups differences. Notably, however, a visual inspection of the grand average waveforms for the ERN component did not match with expected waveform characteristics and brings into question the validity of the ERN

component in this study. The ERN component waveforms, while present, do not have similar overall ERN component amplitude differences between error and correct trials that are typically seen with this component. For example, one recent study using healthy controls found average ERN amplitudes to be -0.3μV, with a difference between ERN and correct trials to be -1.6μV (M. J. Larson, Steffen, & Primosch, 2013). The current study found an ERN/correct trial average difference of -0.16μV across groups with a mean ERN of -0.43μV for the TBI group and 0.53μV for controls. The difference between the current ERN and comparable ERNs from other studies is that the current ERN component shows a 10 times reduction in difference between correct and error trials. A decreased differentiation between correct and error trials could be due to the introduction of two competing no-go conditions (repeat and color-congruent trials) in the EAT, resulting in a higher degree of uncertainty for correct trials and therefore increased activation of monitoring processes (O'Connell et al., 2007).

Previous research shows that the ERN is affected by task requirements, with many different types of tasks being employed across studies to elicit the ERN. The use of multiple different tasks resulted in multiple variations of the ERN component with varying conclusions about the role of the ERN in error awareness (Gründler, Cavanagh, Figueroa, Frank, & Allen, 2009; Olvet & Hajcak, 2009a; Riesel et al., 2013). For example, Grundler et al. (2009) found ERN amplitude differences between patients with obsessive-compulsive disorder when comparing across a more complex probabilistic learning task and a less complex response-conflict flanker task. Results indicated that the flanker task resulted in more elevated ERN amplitudes than with the probabilistic learning task. Riesel et al. (2013) compared ERN and Pe component characteristics from healthy controls on three different tasks commonly used to study the ERN and Pe components: a modified flanker, a go/no-go, and a Stroop task. They found

behavioral performance differences between the three tasks as well as component amplitude differences, with the stroop task showing the most attenuated ERN amplitudes when compared to the other two tasks. Riesel et al. (2013) related the decrease in ERN amplitude to task difficulty, with the Stroop task being the most difficult of the three compared tasks. The finding that task difficulty is negatively correlated with ERN amplitude is consistent with several other studies (Falkenstein, 2004; Hoffmann & Falkenstein, 2010; Pailing & Segalowitz, 2004). The current study employed the EAT as a task designed to elicit the ERN and Pe components. The EAT is a hybrid Stroop and go/no-go task with complex instructions and multiple rules and procedures for participants to remember. Such extensive task requirements increase the difficulty of the task and the cognitive resources needed to adequately complete the task. It is highly probable, given previously mentioned results from other studies, that there is a continuum where increasing task difficulty relates to decreasing ERN amplitude, and the EAT is higher on that continuum than other more common tasks.

It may also be the case that the EAT does not elicit strong ERN components due to the conscious monitoring nature of the task. Attenuated and lower amplitude ERN components have been reported when using the EAT and connected to continued error processing at a conscious level across trials, signaling and supporting continued performance adjustments (O'Connell et al., 2007). Furthermore, evidence supports the ERN as part of a broader error-awareness and performance-monitoring system. Hewig et al. (2011) indicated that the ERN is necessary, but not sufficient for there to be full error recognition at a conscious level, and Charles et al. (2013) supported that the ERN reflects only part of an error-awareness hierarchy.

Another possibility for why there was a diminished ERN could be due to elevated levels of depression in the TBI group, with depression shown to attenuate ERN component amplitudes

(M. J. Larson et al., 2009; M. J. Larson, Perlstein, Stigge-Kaufman, Kelly, & Dotson, 2006). This is an unlikely possibility due to the fact that ERN amplitudes were more attenuated in the control group and although the TBI group was the group with higher levels of reported depression, levels were below clinical cutoffs.

With respect to the role that apathy plays in error awareness for survivors of M/S TBI from Aim 2, Hypothesis 1, apathy did significantly predict the proportion of unaware errors, but not in the hypothesized direction. The model indicated that as apathy increased the proportion of unaware errors decreased. In other words, the more apathetic a person with M/S TBI was the more awareness they had of errors. Previous findings indicated that apathy results in reduced goal-directed behavior through impairments of multiple executive processes such as inhibition, set shifting, and rule finding (Arnould et al., 2013). Additionally, apathy negatively affects maintenance and sustaining of attention in those with TBI (Arnould et al., 2013). Given these previous findings connecting apathy to other cognitive functions it would seem likely that there would be a positive and not a negative relationship between apathy and error awareness.

Relative to Aim 2, Hypothesis 2, apathy was not a significant predictor of ERN nor Pe amplitudes. Findings showing that increases in apathy did not predict decreases in ERN amplitudes could have several possible interpretations. First, previously discussed concerns about the nature of the ERN and reliability of the current ERN waveforms put the validity of these results in question. Further study and replication is needed to confirm this result. I would expect that future studies find results showing that apathy does predict ERN amplitudes, with potential interpretations related to the role of the ERN. The most common of these interpretations is that the ERN component signals response conflict and represents a precursor to conscious error detection (Steinhauser & Yeung, 2010). Thus, reduced ERN amplitude would

likely be predicted by increased apathy meaning that as apathy levels increase, salience of competing responses signaled as errors would reliably decrease, resulting in attenuated ERN amplitudes. Essentially, errors are less significant to those with higher levels of apathy.

The finding that apathy did not significantly predict Pe amplitudes relates back to previously reported findings and characterization of the Pe as a binary indication of error awareness (Shalgi & Deouell, 2013). Elevated Pe amplitude is thought to represent awareness of errors with unaware errors being similar to correct responses (Hughes & Yeung, 2011; Riesel et al., 2013), as was found in this study. However, previous results show that levels of deficit awareness are positively correlated with Pe amplitudes with inferences drawn between self-reported deficit awareness on the FrSBe and error awareness (M. J. Larson & Perlstein, 2009), but negative affect measured by a composite of depression and anxiety measures did not significantly predict Pe amplitudes (M. J. Larson et al., 2011). As apathy has been shown to be a different construct than depression (Kirsch-Darrow et al., 2006; 2011), and can be classified as a substantial decrease or lack of affect and emotion there is insufficient evidence to believe that elevated apathy levels would influence Pe amplitudes in the same way as negative affect.

Limitations

One important limitation of this study is the nature of the sample itself. There is a high degree of heterogeneity in the TBI sample in the areas of injury severity, cognitive functioning, and awareness of deficits. The broad range of characteristics within the TBI sample is indicative of real-world characteristics of the TBI population and the variety of presentations that can be seen from survivors of M/S TBI. Such real-world representation provides generalizability to the population as a whole. However, a heterogeneous sample in terms of injury severity and associated awareness limits the ability to detect differences and provide specific conclusions.

There is a concern that the M/S TBI group was cognitively similar to controls as the only differences in cognitive functioning were related to worse language performance for the M/S TBI group. I was able to show that there were broader cognitive differences between controls and the severe TBI group in overall decreased cognitive functioning as well as specific language and attentional deficits for the severe TBI group. These differences lead to a potential greater degree of confidence in the results from the severe TBI group analyses and comparisons, but also lead to a question related to sample size and available power from the smaller severe TBI sample. The answer to the power question will require study replication with a larger severe TBI sample.

Another sample-related limitation is that participants were found through convenience methods and from a highly educated population. Sample recruitment may have played a role in the limited differences seen between M/S TBI and controls in that there were pre-injury factors such as education, health, and fitness levels that could be affecting results. Whereas there were no differences between groups on level of education, samples represent a population with higher education completion and/or aspirations as a majority of both samples were gathered from BYU and the surrounding area. Multiple studies have shown that education level is a significant predictor of functional and cognitive outcomes following M/S TBI (L. C. Davis et al., 2012; Jeon et al., 2008; Novack, Bush, Meythaler, & Canupp, 2001). However, the use of the WTAR in this study shows that education levels in both groups are estimated to be similar and not above average levels. Thus, it is not reasonable to assume that there were premorbid intellectual differences between groups leading to a high level of recovery in the M/S TBI group.

The current sample had a wide range of injury severity that may have impacted the reliability of findings. However, in order to account for this we completed analyses with only

the severe TBI group, where appropriate. In the case of apathy, where we were comparing the severe TBI group to a control group the sample size was notably smaller and likely played a role in the results found. Future studies with a more homogeneous severe TBI sample are needed to further elucidate the role of apathy in the complex process of error awareness. The use of measures that test both broad executive skills and specific facets of attention will provide more understanding to the relationship between attention, apathy, and error awareness, as I found no differences in electrophysiological indicators of error awareness.

Related to the sample characteristics is the fact that several of the TBI participants were unable or unwilling to provide access to medical records and/or requests for medical records to providers were not answered. Additionally, some records that were received did not contain sufficient information to adequately document injury severity and required clinical interpretation to extrapolate severity. For example, one record noted, "there was decreased GCS during transportation," with no further information related to whether the decrease was to a 14 or to a three on the Glasgow Coma Scale. In situations where medical records were not available or insufficient a comprehensive retrospective interview was completed with the participant and available family members.

A lack of pertinent medical records does have implications for generalizability and reliability of study findings in that it reduces confidence that some of the participants met study injury severity criteria. This deficit in confidence applies more specifically to the participants classified as having a moderate TBI as they are closer to the lower end of the severity spectrum and often did not have sufficient medical records documenting injury severity. To address this concern I attempted to complete as many analyses as possible with only the severe TBI

participants to ensure that severity was not playing a factor in results and ultimately the conclusions made from those results.

I note that while medical records do provide additional confirmation of severity there are potential confounds in using indices such as GCS and LOC for severity classification due to medical procedures such as intubation, induced coma, and surgery (Lezak et al., 2012). Lezak et al. (2012) reported that it is not uncommon to see misclassification for someone with an initially high GCS or little or no LOC who later has deterioration of mental status due to delayed hematoma, cerebral edema, or other trauma related problems. They further note that PTA is used in clinical settings as a reliable measure of injury severity as it correlates well with GCS ratings (Levin, Benton, & Grossman, 1982). Further, it was concluded that fine-tuned accuracy of PTA duration was not necessary and that larger estimates of time in hours, days, and weeks is sufficient for clinical documentation (Lezak et al., 2012). As the medical records obtained in this study are from clinical settings they follow this pattern of larger estimates of PTA and are similar to reports from the participants themselves in level of specificity for estimating both LOC and PTA (King et al., 1997; McMillan et al., 1996). There are additional classification systems proposed for research purposes with greater sensitivity to distinguish between moderate and severe TBI participants; however; those systems were not available for this study (Lezak et al., 2012).

Another potential limitation of the current study was the exploratory use of the EAT task with a M/S TBI sample. There has been no previous use of this type of task with this population and there will need to be replication in order to determine reliability of results. However, the EAT has been successfully used in fMRI studies with chronic substance abuse populations

(Hester et al., 2007; 2009), and in healthy controls for previous ERP studies with similar results across studies (O'Connell et al., 2007; Orr & Hester, 2012).

One question that has been addressed relative to the EAT task requirements relates to the motor response and increased activation due to having to press an alternate button when signaling error awareness following an error. The concern being that by having increased motor activation and response options there could be contamination of the error awareness activations. Hester, et al., (2005) addressed this concern and demonstrated that differences between aware and unaware responses were unrelated to the awareness signaling process through the use of an oddball task where they had participants respond in an identical fashion as they would to aware errors, but removed the error component from the task by having participants press the awareness button following a neutral stimuli. The EAT task was also tested using electrodermal skin conductance to show that the secondary awareness response did not contaminate the electrophysiological indicators of error awareness (O'Connell et al., 2007).

Future Directions

One potential direction would be to replicate current study findings with a larger sample of TBI participants, ideally with more severe TBI participants or only severe TBI participants. By including a larger sample with more severe TBI participants, results could be strengthened and confirmed, especially those results showing trend-level significance such as repeat error awareness. Similarly, by applying the EAT to other samples of TBI participants, there would be further data adding to the growing body of literature supporting task-related differences in electrophysiological and behavioral indices of error awareness. For example, there is growing evidence supporting that ERN amplitudes are task-dependent and related to task difficulty (Riesel et al., 2013).

Another area where further study is needed is in the area of apathy and its connection to error awareness evidenced through electrophysiological indices, specifically the Pe component. Previous evidence showing that increased awareness of deficits correlates with elevated Pe amplitudes and increased negative affect predicts reduced Pe amplitudes in those with a M/S TBI compared to controls (M. J. Larson et al., 2009) could be expanded to include indices of conscious error awareness and detection through use of the EAT task or similar tasks requiring error signaling. Similarly, more research is needed in order to delineate the role that apathy plays in error awareness and how a substantial decrease in emotion and motivation contributes differently than negative affect. Determining the role of apathy could be further explored through the use of a simpler task, such as a flanker task, to determine first if task requirements contributed to a lack of findings in the current study relative to a relationship between apathy and the Pe component. Additionally, by comparing participants with elevated levels of apathy, but not negative affect on indices of error awareness using an error awareness task would provide information about how these two constructs influence error awareness. For example, the use of various populations where apathy has been found separate from depression, such as in Parkinson's Disease and M/S TBI, would be good populations from which to gather participants.

Summary and Conclusions

In this dissertation I found that individuals with M/S TBI have a different process of error awareness using behavioral measures than healthy controls. Behavioral results indicated that the M/S TBI group showed decreases in accuracy over time while control participants improved their accuracy. Awareness of errors remained constant or improved overtime for both groups. Conclusions from these results support that those with M/S TBI demonstrate deficits in higher-level awareness and regulative attention restricting them from modifying performance.

Behavioral differences were not replicated in the electrophysiological responses of error-related ERP components, specifically the ERN and the Pe. The lack of differential findings related to the ERP components was interpreted to mean that the M/S TBI group was attending less to or less aware of their errors at a regulative level, but when they were able to attend they had similar physiological responses as healthy controls. Whereas the Pe was seen as consistent with previous research, the ERN component was not reliably produced in this study and results related to the ERN should be interpreted cautiously. However, there is evidence to support that task requirements may be responsible for reduced amplitude ERN components and error versus correct differences across groups. Replication is needed to determine if the ERN components produced in this study were a result of some set of task requirements.

I found that apathy was counter intuitively predictive of increases in error awareness behaviorally. More research in this area is needed to delineate potential implications of elevated apathy and decreased attentional processes in survivors of M/S TBI.

In conclusion, findings from the current dissertation show differences in error awareness processes between M/S TBI participants and neurologically-healthy controls. Differences are related to variations in no-go accuracy and error awareness occurring over time and not delineated by comparisons of an entire task. The awareness model presented by Crosson (1989) provides a viable framework for understanding the deficits in performance from the M/S TBI group indicating that awareness at an intellectual level is most likely intact and accounts for maintenance of error awareness. However, higher-level emergent and anticipatory awareness are impaired due to attentional deficits resulting in decreased accuracy in the M/S TBI group over time. The current dissertation provides support for continued exploration of performance across task duration and the effects of task requirements on behavioral and electrophysiological

indicators of error awareness. The current dissertation also sets the stage for future research into the role of apathy in attention and error awareness for those with a M/S TBI potentially leading to clinical research aimed at improving rehabilitation efforts.

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