THESIS

INVESTIGATING PAIN MODULATION THROUGH VISUAL IMAGERY AND INJURY PERCEPTION POST-MILD TRAUMATIC BRAIN INJURY: AN ERP STUDY

Submitted by

Kailee A. Kriesel

Department of Psychology

In partial fulfillment of the requirements

For the Degree of Master of Science

Colorado State University

Fort Collins, Colorado

Summer 2026

Master's Committee:

Advisor: Deana Davalos

Michael Thomas
Marti Amberg
Jaclyn Stephens

ABSTRACT

INVESTIGATING PAIN MODULATION THROUGH VISUAL IMAGERY AND INJURY PERCEPTION POST-MILD TRAUMATIC BRAIN INJURY: AN ERP STUDY

Traumatic brain injury (TBI) is a significant health concern in the United States, with mild traumatic brain injuries (mTBI) occurring most frequently among adolescents and young adults. Although mTBI is often considered less severe, individuals may continue to experience persistent symptoms, including pain and psychological distress. Emerging research suggests that cognitive factors, like injury perceptions, may influence how these symptoms are experienced and processed. However, limited research has examined how these perceptions relate to the neural processing of pain using neurophysiological methods. The present study investigated whether perceived control over injury-related symptoms was associated with neural responses to pain-related imagery and whether injury status moderated this relationship. Participants ($N = 54$) completed self-report measures of depressive symptoms, pain catastrophizing, and injury perceptions, followed by a visual imagery task during which EEG was recorded. Event-related potentials (ERP), specifically P2 and P300, were analyzed in a subset of participants with usable EEG data ($N = 21$).

Results showed no significant group differences in P2 and P300 amplitudes. Perceived control was not associated with ERP responses or moderated by injury status. Exploratory analyses revealed significant group differences in self-report measures, with control participants reporting greater psychological distress and emotional representations, and the mTBI group reporting higher illness coherence. These findings suggest that self-reported cognitive-emotional

factors may not directly map onto neural responses to pain-related imagery, emphasizing the need for multimodal approaches in mTBI research.

TABLE OF CONTENTS

INTRODUCTION

Traumatic brain injury (TBI) is becoming an increasing concern in the United States with the most recent data reporting 586 TBI-related hospitalizations per day (CDC, 2024). However, hospitalization data primarily reflect more severe injuries and may not capture the full burden of mild traumatic brain injuries (mTBI). Mild traumatic brain injury is typically defined as a disruption in brain functioning caused by an external force that results in temporary neurological symptoms, such as confusion, headache, or brief loss of consciousness, without the prolonged unconsciousness or extensive structural brain damage commonly seen in moderate or severe TBI. Specifically, mTBI frequently result in visits to the emergency department and are most common amongst younger populations between the ages of 15-24 (Meske et al., 2019; CDC, 2024). Prior research has found that sports and motor vehicle accidents are the leading causes of mTBIs in young adults (Gaw & Zonfrillo, 2016). Symptoms typically arise immediately after injury with pain, specifically headaches, being the most common complaint (Uomoto & Esselman, 1993).

While pain after TBI is common and generally resolves once the tissues have recovered, a subset of individuals, particularly those with mTBI, may experience chronic pain or persistent sensory symptoms, such as heightened sensitivity to light, sound, or touch (Nampiaparampil, 2008; Karr et al., 2022). These symptoms can lead to increased anxiety, depression, and panic, which has been associated with pain catastrophizing tendencies (Granot & Ferber, 2005). High levels of pain catastrophizing have been associated with reports of higher pain intensity, increased psychological distress, and the inability to return to daily activities (e.g., work, school, social interactions) (Ferdek et al., 2019). While the behavioral outcomes can be incapacitating, there may be discrepancies between the amount of pain reported and the amount of pain being

experienced. Specifically in college students, who may be at increased risk for mTBI due to participation in contact sports, these injuries can cause deficits in academic performance leading to negative consequences such as poor grades, increasing stress, and lower employment outcomes (Meske et al., 2019). Eventually, this could develop into poor mental health and behavioral issues (Pavlovic et al., 2019).

Recognizing the impact of a brain injury is important for positive health outcomes, mental health, and quick reintegration into everyday routines (Robertson & Schmitter-Edgecombe, 2015). Individuals who struggle to understand the way their injury is impacting their lives tend to form harmful belief systems, creating feelings of helplessness and low confidence in their abilities, adopt passive coping strategies, and experience poorer psychological and functional outcomes (Medley et al., 2010). These negative beliefs about the consequences of the injury correlate with worse functional outcomes and with the experience of post-concussion symptoms that do not consistently align with traditional clinical measures of injury severity (Polinder et al., 2018). Metacognitive impairments are also common with mTBI, showing as impaired self-awareness and performance monitoring (Robertson & Schmitter-Edgecombe, 2015; Yoshida et al., 2023). These impairments may present as overestimating one's abilities or underestimating the difficulties of a task, which can compromise safety, motivation, and the ability to set realistic goals for oneself.

Although self-report methods provide important information about symptom experience, they are inherently subjective and may be influenced by cognitive and emotional factors such as pain catastrophizing (Medley et al., 2010; Ferdek et al., 2019). As a result, incorporating neuroimaging methods may provide complementary insight into neural processes underlying sensory processing, attention allocation, and visual perception. Specifically,

electroencephalography (EEG) provides an objective, physiological marker of how the brain responds to stimuli and can help identify neural deficits associated with mTBI (Sun et al., 2017). Event-related potentials (ERPs) derived from EEG can index pain processing (Sun et al., 2022). ERPs can also be very useful for assessing attention and perception, especially because the brain processes information rapidly (Woodman, 2010). Additionally, with EEG being portable, inexpensive, and non-invasive, it is the ideal neuroimaging method for researching neural activity related to mTBI, pain, and perception compared to fMRI, which is costly and involves a restricting environment.

Statement of Problem

The current literature does not adequately address the relationship between mTBI, pain post-injury, and visual perception or imagery while accounting for subjects' beliefs about their injury. Previous research has revealed deficits in vividness of visual imagery and perception following mTBI (Oostra et al., 2012; Bartolomeo et al., 2012), including evidence of double dissociations between imagery and perceptual abilities (Bartolomeo et al., 2012). Research has also identified high rates of chronic pain following mTBI (Harrison-Felix et al., 2024; Lonsdale, 2010), as well as deficits in self-awareness and perception of injury consequences (Medley et al., 2010; Yoshida et al., 2023). However, little research has examined how individuals with mTBI and varying levels of perceived control over the consequences of their injury cognitively process pain-related thought using neurophysiological methods. It has been reported that many people who struggle with the aftermath of their brain injury experience intrusive mental images of their pain that causes intense discomfort (Lonsdale, 2010; Gombay & Andrews, 2021). Whether the pain is imagined or real, it is important to address this issue as it could cause increased feelings of helplessness, depression, anxiety, and panic (Medley et al., 2010; Ferdek et al., 2019; Robertson & Schmitter-Edgecombe, 2015). Particularly in college students, these problems can

lead to academic, social, and emotional difficulties that would make it difficult for someone to remain successful in school and their everyday lives (Meske et al., 2019; Pavlovic et al., 2019).

LITERATURE REVIEW

Mild Traumatic Brain Injury

Mild traumatic brain injury (mTBI) represents approximately 75-80% of all brain injury cases in the United States making it the most common form of TBI (National Academies of Sciences, Engineering, and Medicine, 2022). mTBI, also commonly referred to as a concussion, is caused by a bump, blow, or jolt to the head or body that results in the brain shifting or rotating inside the skull (Brain Injury Association of America, 2025). Post-injury symptoms include dizziness, temporary loss of consciousness and amnesia, sensory overload, pain, and more (Oldenburg et al., 2015; Nampiaparampil, 2008). However, research has shown that deficits in cognition and emotion regulation can still be present even years after the initial injury (Konrad et al., 2010). While mTBI is generally considered less severe than moderate or severe TBI, the long-term consequences can be substantial and negatively impact daily living (Konrad et al., 2010; Carroll et al., 2014; Meske et al., 2019; Pavlovic et al., 2019).

TBI is a major cause of death and disability worldwide, with its true prevalence likely underestimated due to underreporting and limitations in data collection (National Academies of Sciences, Engineering, and Medicine, 2022). This underestimation is particularly relevant for mTBI, which often goes unrecognized because individuals may not seek medical care or may be treated in non-hospital settings. In the United States, millions of individuals are living with TBI-related disability (Karamian et al., 2024), although this number likely underrepresents the full burden of milder injuries. Young adults are at elevated risk for TBI, particularly through motor vehicle accidents and sports-related injuries, which are common mechanisms of mTBI in this

population (National Academies of Sciences, Engineering, and Medicine, 2022). The highest rates of nonfatal TBI-related hospitalizations due to motor vehicle accidents occur among individuals aged 15-34 years (Peterson & Thomas, 2021), highlighting a critical risk period of mTBI exposure.

Cognitive effects of mTBI can persist for years following injury (Konrad et al., 2010; McInnes et al., 2019). Even six years post-injury, individuals may experience considerable impairments across several domains like learning, attention, working memory, long-term memory, and executive functioning (Konrad et al., 2010). However, the severity and persistence of these deficits vary across studies, reflecting the heterogeneity of mTBI outcomes (McInnes et al., 2019). In addition to cognitive challenges, mTBI is associated with emotional and psychological disturbances. Many individuals experience increased rates of anxiety, depression, and emotional dysregulation, which can significantly impact daily functioning and quality of life (Pavlovic et al., 2019). The combination of cognitive and emotional symptoms emphasizes the complexity of mTBI recovery and highlights the need for additional research on its long-term effects. Given the persistent cognitive and emotional consequences of mTBI and the variability in symptom presentation, objective neurophysiological methods such as EEG and ERPs have been widely used to detect subtle neural disruptions that may not be captured through clinical or behavioral assessment (Gosselin et al., 2011; Haneef et al., 2013).

Mild Traumatic Brain Injury and EEG/ERPs

Electroencephalography (EEG) and event-related potentials (ERP) are valuable tools for brain injury research, as they can capture cognitive and neural abnormalities that may not appear in standard clinical assessments. mTBI causes electrophysiological abnormalities that are visible on EEG recordings, and EEG may be more sensitive to detect TBIs than other clinical or neuroimaging methods (Ianof & Anghinah, 2017). In one study, while 86% of patients with

abnormal clinical exams also had abnormal EEGs, the reverse was less common – only 23% of those with abnormal EEGs also had abnormal exams (Ianof & Anghinah, 2017). However, EEG changes do not present the same for all individuals, with some patients exhibiting normal EEGs as early as 15 minutes post-injury, likely due to variations in injury severity (Ianof & Anghinah, 2017).

ERPs, which measure neuronal activity in response to specific stimuli, are very valuable for assessing the integrity of neural processing in TBI patients (Sun et al., 2017). The P300 component, which is widely recognized as an index of cognitive function, provides insight into deficits in attention, working memory, and decision-making that may not be noticed through behavioral measures alone (Sun et al., 2017). Additionally, EEG's millisecond-range temporal resolution allows for studying functional properties and temporal dynamics of cognitive processes better than other neuroimaging methods. Recent studies have shown reduced amplitudes and delayed latencies in TBI patients compared to healthy controls, further emphasizing the role of ERPs in detecting subtle cognitive impairments (Liu et al., 2021).

The long-term impact of mTBI on cognition has been debated with some studies finding no lasting deficits, while other studies show executive dysfunction and working memory impairment 1-year post-injury, particularly in athletes and veterans (Arciniega et al., 2021). Notably, cognitive deficits related to mTBI have been detected in undergraduates with a history of mTBI up to 3.8 years post-injury, while similar deficits were observed in student athletes just 17 days post-injury (Arciniega et al., 2021). Given that post-concussion symptom presentation overlaps with conditions such as major depression, chronic pain, and somatization disorders, symptom-based assessments alone may lack specificity in identifying underlying neural mechanisms (Eierud et al., 2014). As a result, the use of EEG and ERPs in mTBI research is

important for providing objective neurophysiological measures that complement clinical symptom reporting and may help clarify persistent neural disruptions associated with mTBI. While EEG and ERP research has primarily focused on cognitive dysfunction following mTBI, these methods are also well-suited for examining altered sensory and affective processing, including the neural mechanisms underlying pain perception, which is a common and persistent symptom following brain injury.

Pain Perception and Mild Traumatic Brain Injury

Pain perception and its relationship with mTBI is complex and involves several brain areas including the primary and secondary somatosensory cortices (S1 and S2), the anterior cingulate cortex (ACC), the insular cortex (IC), the prefrontal cortex (PFC) and the thalamus. These regions form the "pain neuromatrix", a network responsible for processing the sensory and emotional components of pain (Melzack, 2001). Although originally described as a general model of pain processing, this framework is relevant for understanding altered pain experiences following mTBI. Disruptions in pain modulation and anticipation have been observed following mTBI, suggesting altered top-down control of pain processing networks (Strigo et al., 2014). Additionally, individuals with post-traumatic headache, a common sequela of mTBI, show altered longitudinal patterns of pain-evoked brain activation, further supporting changes in this network over time (Ku et al., 2025). Collectively, these findings suggest that mTBI may be associated with dysregulation of pain processing systems that contribute to heightened or prolonged pain experiences.

The primary and secondary somatosensory cortices are involved in locating the pain and determining its intensity. In chronic pain, including those following mTBI, these regions show increased and more widespread activation, which makes the pain harder to localize (Lithwick et al., 2013). This cortical reorganization may manifest as pain in response to non-painful stimuli

and an amplified perception of painful stimuli (allodynia and hyperalgesia, respectively). Emotional processing regions, like the anterior cingulate cortex and the insula, also display heightened activity with chronic pain, which can amplify emotional and sensory processing (Lithwick et al., 2013). Lastly, the thalamus, which relays pain signals, also shows increased activity both acutely and chronically following a TBI, adding to pain sensitivity.

Alterations in prefrontal regions, particularly the medial prefrontal cortex (mPFC), have been implicated in disrupted attentional control and maladaptive cognitive patterns such as pain catastrophizing (Granot & Ferber, 2005; Lonsdale, 2010). Consistent with this, research in mTBI populations indicates that catastrophizing is associated with greater symptom reporting and poorer recovery outcomes, highlighting the role of cognitive appraisal in post-injury pain experience (Chaput et al., 2016; Shi et al., 2024).

Psychological factors, including catastrophizing and anxiety, significantly impact the experience of pain. Pain catastrophizing is the tendency to exaggerate the threat posed by pain and feeling helpless in managing it. Individuals who catastrophize tend to experience heightened pain intensity and more severe disability (Granot & Ferber, 2005). Additionally, emotional disturbances, like depression and anxiety may either exacerbate pain perception or result as a consequence of chronic pain, making it a difficult cycle to break (Lonsdale, 2010). Although neuroimaging methods like fMRI are ideal for spatial localization of pain-related brain regions, EEG and ERP approaches offer high temporal resolution, making them useful for examining the rapid and dynamic neural processes involved in pain perception, modulation, and cognitive appraisal following mTBI.

Pain and EEG/ERPs

EEG and ERPs have been widely used to study how the brain processes pain, due to their ability to track rapid changes in brain activity down to the millisecond (Luo et al., 2022; Sun et

al., 2022). Unlike other neuroimaging methods, EEG records electrical activity from neurons to capture the timing of pain responses. Painful stimuli tend to cause consistent changes in brain wave patterns. Specifically, delta and beta power often increase in the frontal and temporal regions, respectively. Gamma activity, which has been linked to how strongly people feel pain, also increases in the prefrontal and frontocentral areas (Zis et al., 2022).

Pain triggers specific ERP responses, especially the N2/P2 complex, which shows up around 250-350 milliseconds after a painful stimulus. These signals are linked to areas like the ACC, which is involved in processing the pain itself as well as the emotional responses to it, such as during placebo effects (Lyby et al., 2011). The size of the ERP signal often reflects how intense the pain feels and can change when pain is reduced. ERPs also reveal the order in which pain is processed by different brain areas. Typically, the ACC and S1 respond first, followed by the insula and motor cortex (M1) (Sun et al., 2022).

EEG studies focused on brain connectivity have also discovered individual differences in pain processing, like pain catastrophizing. Those who catastrophize tend to exhibit stronger beta activity from the somatosensory to temporal areas but show weaker activity from temporal to frontal regions. This pattern could suggest linked stronger sensory and emotional processing towards pain while being unable to regulate the experience (Ferdek et al., 2019). These findings underscore the value of EEG and ERPs in discovering the neural mechanisms underlying the sensory and emotional components of the pain experience. In addition to sensory processing of pain, EEG and ERP research has also begun to clarify how internally generated cognitive representations, such as mental imagery, interact with pain perception and its neural correlates.

Visual Imagery in Mild Traumatic Brain Injury and Pain Processing

Previous research has explored the relationship between mental imagery and TBI, highlighting various deficits and rehabilitation approaches. Studies have shown that damage to

specific regions of the brain can impair mental imagery independently of perceptual deficits. For example, Bartolomeo et al. (2012) show how patients with damage to the right hemisphere exhibit “imaginal neglect”, which is failing to visualize the left side of an imagined scene, even with intact perception. Similarly, Moro (2008) discovered patients with brain damage could experience severe impairments in generating and manipulating mental images despite having preserved visual perception. This suggests that distinct neural mechanisms underlie mental imagery and perception.

Other studies have assessed the use of mental imagery as a form of rehabilitation. Gagnon et al. (2015) used guided mental imagery in an active rehabilitation program for adolescents recovering from sports-related concussions. It was reported that adolescents experienced significantly less post-concussion symptoms and even showed improvements in mood and cognitive functioning. Niemeier et al. (2001) also demonstrated the effectiveness of visual imagery techniques in improving visual attention deficits following a stroke and brain injury. Additionally, research suggests that self-referential imagery can enhance memory performance in individuals with neurological damage. Grilli and Glisky (2010) found that self-imagination, which involves integrating personal thoughts, emotions, and sensory details, improved memory recall in patients with episodic memory impairments and healthy subjects. Piolino (2007) extended this research to TBI patients, finding that deficits in autobiographical memory and self-perspective in visual imagery were associated with executive dysfunction.

Research on mental imagery and pain has shown how imagined experiences can influence pain perception and neural processing. Christian et al. (2015) demonstrated that painful scenarios from the first-person perspective led to higher pain ratings and greater activation in brain regions associated with interoceptive awareness and emotional processing, like the anterior

insula. Their findings suggest that perspective in mental imagery modulates both the subjective experience of pain and its neural correlates. Similarly, Fardo et al. (2015) found that mental imagery can both amplify and diminish pain perception. When participants imagined a lesion on their forearm, pain intensity and unpleasantness increased, whereas imagining a protective glove led to a reduction in perceived pain. EEG data revealed that these effects were mediated by differential activation in brain regions associated with cognitive control and emotion regulation. Research by Gosden et al. (2013) and Philips (2011, 2013) further underscores the role of pain-related mental imagery in chronic pain sufferers. These studies indicate that pain imagery is common, often distressing, and linked to heightened emotional responses and cognitive biases, such as overestimation of pain-related threats. These findings suggest that internally generated mental representations can significantly shape both emotional and sensory experiences, raising important questions about how individuals with mTBI perceive and interpret their own injury and associated symptoms.

Mild Traumatic Brain Injury and Perception of Injury

Individuals with mTBI often exhibit impairments in self-awareness and distorted perceptions of the injury, which can affect their ability to accurately recognize deficits and engage in rehabilitation effectively (Medley et al., 2010; Robertson & Schmitter-Edgecombe, 2015; Yoshida et al., 2023). Research suggests that following mTBI, individuals may underestimate the severity of their condition and the impact of their symptoms. Medley et al. (2010) found that those with mTBI reported significantly fewer difficulties than their clinicians or significant others, suggesting reduced awareness of deficits and a tendency towards underreporting. This reduced awareness may be influenced by coping styles, with individuals in high optimism and low control/ambivalent groups demonstrating lower reliance on emotion-focused coping and equally impaired self-awareness.

Similarly, Yoshida et al. (2023) highlighted that those with mTBI exhibit impaired metacognitive efficiency in predicting task difficulty, leading to overestimation of their abilities and underestimation of task demands. This metacognitive bias, despite poor decision-making performance, may contribute to behavioral inflexibility and difficulty adapting to new situations. Additionally, Robertson and Schmitter-Edgecombe (2015) found that metacognitive awareness is particularly impaired in the acute phase of recovery, making it difficult for individuals to accurately judge task performance, engage effectively in rehabilitation, and apply appropriate cognitive effort. Although some improvement in metacognitive awareness is possible over time, error monitoring remains a challenge, limiting the ability to recognize and correct mistakes.

Jones et al. (2017) further demonstrated that negative illness perceptions, such as longer expected recovery timelines and more severe symptomology, were associated with greater perceived brain damage, highlighting how subjective beliefs about injury may influence long-term outcomes. The underutilization of support services by students, as shown by Meske et al. (2019), further underscores the disconnect between perceived and actual impairment, as those with mTBI were less likely than their uninjured peers to seek academic or psychological assistance, despite reporting significant difficulties with coursework, relationships, and mental health. Differences in how individuals perceive and interpret their injury may also be influenced by the extent to which they feel able to control or manage their symptoms, highlighting perceived control as a key psychological factor in mTBI recovery (Medley et al., 2010; Robertson & Schmitter-Edgecombe, 2015; Granot & Ferber, 2005).

Mild Traumatic Brain Injury and Perceived Control

Perceived control is a key component of illness or injury perception and refers to the extent to which individuals believe they can influence or manage their symptoms and recovery (Leventhal et al., 1980, 2016). Within the context of mTBI, perceived control has been shown to

play an important role in shaping psychological and functional outcomes. Individuals with lower perceived control are more likely to report greater symptom severity, poorer psychological adjustment, and reduced engagement in adaptive coping strategies (Medley et al., 2010). In contrast, higher perceived control is associated with more effective coping, better emotional regulation, and improved functional recovery. This construct is especially relevant in mTBI populations where the symptoms are often subjective and variable, increasing reliance on individual interpretation and appraisal of the injury.

Perceived control is also closely linked to maladaptive cognitive patterns such as pain catastrophizing, as individuals who feel less control over their symptoms may be more likely to engage in rumination, magnification, and feelings of helplessness (Granot & Ferber, 2005; Chaput et al., 2016). These cognitive-emotional processes can amplify the experience of pain and contribute to more psychological distress. Importantly, emerging evidence suggests that these factors may influence not only subjective symptom reporting but also underlying neural processing, particularly in systems involved in attention allocation and sensory processing. Taken together, these findings suggest that perceived control may play a central role in shaping both psychological and neural responses to pain and injury-related cognition following mTBI, underscoring the need for research that integrates these constructs within a unified neurophysiological framework.

Statement of Purpose

This study investigated how individuals with mTBI process visualized pain scenarios and how their perception of injury influenced neural responses. Using EEG, we examined how differences in injury perception – such as perceived impact, control, and emotional response – affected brain activity during pain-related imagery. Specifically, we analyzed the P2 and P300 ERP components, which reflect sensory processing and attention allocation.

Current research primarily relies on self-report measures, leaving a gap in understanding the real-time neural mechanisms underlying injury perception and pain processing. This study provides crucial insights into the cognitive and affective dimensions of pain processing in mTBI populations. Additionally, exploratory analyses explored the influence of mental health and pain catastrophizing on neural responses. Findings from this research can help refine interventions aimed at improving perceived control, pain management, and recovery outcomes in those with mTBI.

Hypothesis 1

Individuals with a history of mild traumatic brain injury will differ from controls in P2 and P300 amplitudes during pain-related imagery, such that individuals with mTBI will exhibit reduced amplitudes relative to controls.

Hypothesis 2

Perceived control will significantly predict P2 and P300 amplitudes during pain-related imagery, such that lower perceived control will be associated with larger ERP amplitudes.

Hypothesis 3

Injury status will moderate the relationship between perceived control and ERP amplitudes, such that the association between perceived control and P2 and P300 amplitudes will differ between individuals with mTBI and controls.

METHODS

Participants

We recruited 54 participants between the ages of 18 and 34 through a university participant pool and campus advertisements. This age range captures young adults at a college-age level, a population at elevated risk for mTBI, while also ensuring a sufficient and diverse sample for recruitment. All participants contributed behavioral (survey-based) data, and all 54 completed the full experimental session including EEG recording. The final sample ($N = 54$) had a mean age of 20.61 years ($SD = 3.25$). The sample was predominantly female (72.22%) compared to male participants (27.78%). In terms of racial/ethnic identity, the majority of participants identified as White (75.93%).

Based on a priori power analysis conducted using G*Power, a total sample size of 102 participants was initially estimated to achieve 80% power at an alpha level of 0.05. However, a review of similar studies using comparable methodologies and populations indicates that sample sizes typically range around 33 participants (Proverbio et al., 2023; Oostra et al., 2012; Niemeier et al., 2015; Ferdek et al., 2019; Liu et al., 2021; Yoshida et al., 2023). For example, Nandrajog et al. (2017) investigated ERP and neuropsychological differences in individuals with mTBI using a total of 36 participants and conducted multiple t-tests and non-parametric comparisons between mTBI and control groups. They reported statistically significant group differences in P300 latency and amplitude, demonstrating that meaningful neural effects can be detected with smaller samples. Similarly, Gosselin et al. (2011) analyzed ERP and fMRI data from 14 mTBI participants and 23 matched controls, using independent t-tests and ANOVAs to examine group differences in ERP components. Despite their limited sample size, they were able to detect significant group x task interaction effects and brain-behavior correlations, supporting the

sensitivity of ERP measures even in modest samples. This study aimed for a sample size within this range while maintaining statistical rigor.

Participants were classified into mTBI and control groups based on self-reported injury history collected during screening. Individuals with a history of mTBI were required to report an injury within the past 18 months in order to qualify to participate. Injury classification was based on structured self-report of mechanism of injury (e.g., sports-related injury, falls, motor vehicle accidents, etc.) and typical concussion-related symptoms (e.g., headache, dizziness, confusion, etc.). No clinical diagnostic records or Glasgow Coma Scale scores were available. Control participants were defined as individuals with no lifetime history of TBI.

Prior to coming to the lab, participants completed a screening survey to assess brain injury characteristics, demographics, and mental imagery abilities through the Vividness of Visual Imagery Questionnaire (VVIQ; Marks, 1973). Exclusion criteria included currently taking prescribed medication for pain, inability to provide informed consent, individuals with non-traumatic brain injuries (e.g., stroke or tumor), individuals who have undergone neurological surgery within the last 18 months at the time of participation, history of neurological disorders, inability to generate mental images (e.g., aphantasia), and a brain injury that occurred outside of the last 18 months at the time of participation. Additionally, corrected hearing and vision was necessary to receive instructions and complete the task.

Although all 54 participants contributed behavioral data, only a subset ($N = 21$) provided usable ERP data following preprocessing. Participants were excluded due to excessive artifact contamination or insufficient artifact-free trials.

General Procedure

First, participants were required to provide their written consent before participating further. Then, the following surveys were completed: Patient Health Questionnaire (PHQ-9;

Kroenke et al., 2001), Pain Catastrophizing Scale (PCS; Sullivan, 1995), and the Illness Perception Questionnaire (IPQ-R; Moss-Morris et al., 2002). These measures have been used in mild traumatic brain injury populations to measure depression, pain catastrophizing, and injury perceptions, respectively (Fann et al., 2005; Naugle et al., 2021; Snell et al., 2010). For this study, the IPQ-R was revised further to include the word "injury" in replacement of "illness", consistent with prior research that has applied this measure to brain injuries (Medley et al., 2010). This adaptation aligns with recommendations in the literature that the terminology can be modified to suit the specific condition under investigation (Hill, 2010; Moss-Morris et al., 2002). The PHQ is widely used to assess symptoms of depression, which is common in individuals with mTBI and may influence cognitive and emotional outcomes. The PCS measures pain-related catastrophic thinking, which is an important factor in understanding the psychological response to injury and its potential impact on symptom persistence. Finally, the IPQ evaluates individuals' beliefs and perception about their condition, providing insight into how cognitive and emotional factors influence recovery trajectories.

After completing the surveys, the participants were fitted with an EEG cap and given instructions to complete the visual imagery task. Once the task was complete, participants were debriefed and given compensation in the form of course credit or $25.

Measures

Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001): Participants reported their depressive symptoms over the past two weeks on a four-point Likert scale. The scale consists of the statements, "Not at all", "Several days", "More than half the days", and "Nearly every day". The PHQ contains nine items that correspond to the diagnostic criterion for major depressive disorder (e.g., "Little interest or pleasure in doing things", "Feeling tired or having little energy"). Total scores range from 0 to 27, with higher scores indicating greater depressive

symptom severity. Scores are typically interpreted as follows: 0-4 (minimal), 5-9 (mild), 10-14 (moderate), 15-19 (moderately severe), and 20-27 (severe). A score of 10 or higher is commonly used as a cutoff indicating clinically significant depressive symptoms and the potential need for further assessment. This measure has high internal reliability (α range = .86 –.89; Kroenke et al., 2001). See Appendix A for a copy of the measure.

Pain Catastrophizing Scale (PCS; Sullivan, 1995): Participants indicated the degree to which they have specific thoughts and feelings that may be associated with pain on a five-point Likert scale. The scale consists of the statements, "Not at all", "To a slight degree", "To a moderate degree", "To a great degree", and "All the time". The PCS consists of 13 items that assess past experiences with pain associated with rumination (e.g., "I keep thinking about how much it hurts"), magnification (e.g., "I wonder whether something serious may happen"), and helplessness (e.g., "It's awful and I feel that it overwhelms me"). This measure has high internal reliability (α range = .91 –.93; Wheeler et al., 2019). See Appendix A for a copy of the measure.

Illness Perception Questionnaire (IPQ-R; Moss-Morris et al., 2002): Participants reported their beliefs and perceptions about their condition. The questionnaire consists of three main sections: the identity subscale assesses whether participants have experienced specific symptoms and attribute them to their illness (e.g., pain, headaches, dizziness); the cognitive and emotional subscales evaluate beliefs about the timeline, consequences, personal and treatment control, coherence, and emotional impact of their condition using a five-point Likert scale (e.g., "This illness will pass quickly", "My illness has major consequences on my life"; ranges from "Strongly disagree" to "Strongly agree"), and the causal subscale is used to identify and rank potential causes they believe contributed to their condition (e.g., "Chance or bad luck", "My own behavior"). This measure has good internal reliability (α range = .71 –.87; Rivera et al., 2024).

Control participants were instructed to complete this questionnaire by imagining how they would respond if they had experienced a mild traumatic brain injury. As such, control responses reflect hypothetical representations of injury rather than lived experience. For the purposes of this study, analyses focused on the perceived control subscale of this measure.

See Appendix A for a copy of the measure.

Visual Imagery Task: The task was created using STIM^2 stimulus presentation software (Version 4.3; Compumedics Neuroscan, 2003). Elements of the task were adapted from Proverbio et al. (2023). During the task, participants were shown a short paragraph describing a scenario of experiencing pain following a brain injury from a first-person point of view. Following the paragraph, a blank black screen was shown as a cue to begin visual imagery of the scenario just read. The task began with instructions, informing the participants they were about to begin 10 practice trials. Following the practice trials, another set of instructions appeared, instructing the participants to follow the same instructions as before for the next 50 real trials. Each set of instructions remained on screen for 20 seconds, each scenario paragraph remained on screen for 15 seconds, and each blank screen remained on screen for 10 seconds. The stimuli were presented in white text centered in the middle of the screen, Times New Roman font, and with a black background. The task did not require any response from the participant. The task continued to play for an estimated 25 minutes. For analysis purposes, ERPs were time-locked to the onset of the paragraph (trial onset), which marked the beginning of the continuous 25-second stimulus window. Epochs were extracted relative to this event to capture neural activity associated with the processing and subsequent imagery of each pain-related scenario. See Appendix B for a copy of the task instructions and stimuli.

EEG Procedure

EEG was recorded at a sampling rate of 1000Hz using a SynAmps2/RT 64 channel system (Curry 9.0.2, Compumedics Neuroscan, USA, Model: 8050). EEG signals were collected using a 64 electrode Quik-Cap Neo Net, equipped with sewn-in or adhesive silver/silver-chloride sintered electrodes (Diameter = 1.5cm, Compumedics Neuroscan, USA, Inc.). Electrode placement followed the international 10-20 system, as implemented in the Quik-Cap 64 configuration. A .01 Hz high-pass filter and a 100 Hz low-pass filter were applied during data acquisition.

The electrode series included all standard positions (Fp1, Fpz, Fp2, AF3, AF4, F11, F7, F5, F3, F1, Fz, F2, F4, F6, F8, F12, FT11, FC5, FC3, FC1, FCz, FC2, FC4, FC6, FT12, T7, C5, C3, C1, Cz, C2, C4, C6, T8, TP7, CP5, CP3, CP1, CPz, CP2, CP4, CP6, TP8, M1, M2, P7, P5, P3, P1, Pz, P2, P4, P6, P8, PO7, PO3, POz, PO4, PO8, O1, Oz, O2, Cb1, Cb2, VEOG, HEOG, EKG, EMG). Eye movements were monitored with electrode placement directly below the center of the left eye to record vertical ocular movements (VEOG). All channels were referenced to Cz during recording. The ground electrode was placed at AFz. Stimulus event markers were synchronized with EEG recordings using the StimTracker Duo (Cedrus Corporation, USA) to ensure precise timing accuracy between stimulus presentation and EEG acquisition.

Data Analysis

The recorded EEG signals were segmented into epochs of 100 ms before and 2000 ms after stimulus presentation. A baseline correction was applied using the 100 ms pre-stimulus interval. Data were analyzed in MATLAB. Trials exceeding a voltage of +/- 100 μV were removed. The mean number of usable trials retained per participant was 25.81 (SD = 13.03), with a range of 6-44 trials across participants. ERPs were averaged across remaining artifact-free trials for each participant. EEG data quality requirements resulted in substantial attrition for ERP

analyses. Specifically, ERP data from 33 participants were excluded due to excessive noise artifacts and failure to meet a minimum requirement of at least 6 artifact-free trials following preprocessing. This threshold was applied to ensure a minimum level of usable data for ERP averaging while retaining as many participants as possible. Importantly, exclusion criteria were applied uniformly across groups, reducing the likelihood of systematic bias in data retention. This resulted in a final ERP sample of 21 participants (n = 9 mTBI, n = 12 control).

For each participant, EEG data were first averaged across artifact-free trials to produce individual ERP waveforms. Mean amplitudes were then computed within each region of interest (ROI) by averaging across selected electrodes for each component, yielding a single mean amplitude per participant per component. The amplitude of P2 was measured as the mean voltage between 150 and 250 ms after stimulus onset at electrodes C1, C2, P3, and P4. The selection of these electrodes was based on prior research by Proverbio et al. (2023) and are consistent with literature indicating that the P2 component is commonly observed over central and posterior regions associated with early attentional processing. The amplitude of P300 was measured as the mean voltage between 300 and 500 ms after stimulus onset at electrodes AF3, AF4, FC1, FC2, F3, F4, and Fz. The selection of these electrodes was also based on prior research by Proverbio et al. (2023) as well as prior findings that P300 scalp distribution can vary depending on task demands, especially in paradigms involving attention and cognitive evaluation. Given the imagery-based and attentionally engaging nature of the present task, these regions were chosen to capture later stage cognitive processing associated with stimulus evaluation.

To further evaluate the spatial distribution of the P300 component, exploratory visualizations were also conducted at centroparietal electrode sites (Pz and CPz), which are

typically associated with maximal P300 amplitude. These analyses were not included in the a priori hypotheses and were conducted for descriptive purposes only.

To address Hypothesis 1, two independent t-tests were conducted to compare the P2 and P300 ERP amplitudes between participants with a history of mTBI and control participants with no history of a brain injury. These analyses were used to examine whether injury status was associated with differences in neural responses during pain-related imagery.

To address Hypothesis 2, simple linear regression analyses were conducted to examine whether perceived control, as measured by the Perceived Control subscale of the Illness Perception Questionnaire-Revised (IPQ-R), predicted P2 and P300 amplitudes. Perceived control was treated as a continuous predictor variable to preserve individual variability and maximize statistical power.

To address Hypothesis 3, moderation analyses were conducted using linear regression to test whether injury status (mTBI vs. control) influenced the relationship between perceived control and ERP amplitudes. Interaction terms between perceived control and injury status were included in the models to determine whether the strength or direction of the association differed between groups. Overall, these analyses aimed to examine both the independent and interactive effects of perceived control and injury status on neural responses to pain-related mental imagery. This approach allowed for a more nuanced understanding of how cognitive factors, such as perceived control, may influence pain processing following mTBI.

RESULTS

Descriptive Statistics

Descriptive statistics are provided in Table 1. The total sample consisted of 54 participants (mTBI = 25, Control = 29). On the PHQ-9, participants reported a mean score of 9.15 (SD = 6.27), indicating mild depressive symptoms on average. On the PCS, the mean score was 21.31 (SD = 13.96), meaning participants were experiencing moderate levels of catastrophizing bordering on a clinically significant level (scores > 30). Perceived injury representations were assessed using the IPQ-R. The perceived control subscale had a mean score of 15.94 (SD = 3.49). In addition to the primary subscale of interest, two additional subscales were included for exploratory analyses: illness coherence and emotional representations. Mean illness coherence was 13.13 (SD = 4.86) and mean emotional representations were 12.60 (SD = 6.08).

ERP analyses were conducted on a subset of participants with useable EEG data following artifact rejection (N = 21, mTBI = 9, Control = 12). Due to preprocessing constraints, the number of artifact-free trials per participants were variable and, in some cases, relatively low, which may increase variability in ERP amplitude estimates. For this subsample, the mean P2 amplitude was 0.48 μV (SD = 0.87), and the mean P300 amplitude was -1.32 μV (SD = 3.29).

Primary Analyses

Hypothesis 1 – Group Differences in ERP Components

To examine whether individuals with a history of mTBI differed from control participants in neural responses to pain-related imagery, two independent-samples t-tests were conducted for P2 and P300 amplitudes.

For the P2 component, there was no significant difference in amplitude between the mTBI group (M = 0.25 μV) and the control group (M = 0.65 μV), $t(17.48) = 1.15$, $p = .265$, 95% CI [-0.34, 1.14], $d = 0.47$.

For the P300 component, there was also no significant difference between the mTBI group (M = -0.71 μV) and the control group (M = -1.79 μV), $t(18.14) = -0.79$, $p = .439$, 95% CI [-3.94, 1.78], $d = -0.32$ (small).

Given the unexpected scalp distribution of the P300 in the primary analysis, additional descriptive waveforms were inspected at centroparietal sites (Pz and CPz), where this component is typically most pronounced. These plots showed a clearer positive-going deflection consistent with the expected P300 morphology (See Figure 3).

Hypothesis 2 – Main Effect of Perceived Control

To examine whether perceived control predicted neural responses to pain-related imagery, two simple linear regression analyses were conducted with perceived control (IPQ-R Perceived Control subscale) as a continuous predictor of P2 and P300 amplitudes.

For the P2 component, perceived control was not a significant predictor of amplitude, b = -0.002, SE = 0.057, $t(19) = -0.03$, $p = .978$, 95% CI [-0.121, 0.118]. The overall model was not significant, $F(1,19) = 0.001$, $p = .978$, with negligible variance explained ($R^2 < .001$). The standardized coefficient was near zero ($\beta = -0.006$), indicating no meaningful relationship between perceived control and P2 amplitude.

For the P300 component, perceived control was also not significant, b = 0.10, SE = 0.21, t(19) = 0.48, p = .639, 95% CI [-0.35, 0.55]. The overall model was not significant, F(1,19) = 0.***23***, p = .639, and explained a small portion of the variance ($R^2 = .012$). The standard coefficient was small ($\beta = 0.11$), suggesting a weak and non-significant association.

Hypothesis 3 – Moderation by Injury Status

To examine whether injury status moderated the relationship between perceived control and ERP amplitudes, two multiple linear regression analyses were conducted including perceived control, injury status, and their interaction term as predictors of P2 and P300 amplitudes.

For the P2 component, the overall model was not significant, $F(3,17) = 0.37$, $p = .776$, explaining only a small proportion of the variance ($R^2 = .061$). The interaction between perceived control and injury status was also not significant, $b = -0.036$, SE = 0.117, $t(17) = -0.31$, $p = .762$, 95% CI [-0.283, 0.211]. Neither the main effect of perceived control, $b = 0.013$, SE = 0.080, $t(17) = 0.16$, $p = .877$, or injury status, $b = 0.159$, SE = 1.880, $t(17) = 0.09$, $p = .934$, was significant.

For the P300 component, the overall model was not significant, $F(3,17) = 0.33$, $p = .801$, with low variance explained ($R^2 = .056$). The interaction between perceived control and injury status was not significant, $b = 0.226$, SE = 0.444, $t(17) = 0.51$, $p = .618$, 95% CI [-0.711, 1.162]. The main effects of perceived control, $b = 0.004$, SE = 0.304, $t(17) = 0.01$, $p = .991$, and injury status, $b = -2.431$, SE = 7.135, $t(17) = -0.34$, $p = .738$, were also not significant.

Exploratory Analyses – Illness Coherence and Emotional Representations Subscales

Exploratory analyses were conducted to examine whether illness coherence and emotional representations (IPQ-R subscales) were associated with P2 and P300 amplitudes, and whether these relationships were moderated by injury status.

Illness Coherence

Illness coherence was not a significant predictor of P2 amplitude, $b = 0.005$, SE = 0.047, $t(19) = 0.11$, $p = .915$, 95% CI [-0.093, 0.103], $R^2 < .001$, or P300 amplitude, $b = 0.20$, SE = 0.17, $t(19) = 1.15$, $p = .265$, 95% CI [-0.16, 0.56], $R^2 = .065$.

Moderation analyses indicated that injury status did not significantly influence these relationships. The interaction between illness coherence and injury status was not significant for P2, b = -0.002, SE = 0.113, $t(17)$ = -0.02, p = .983, 95% CI [-0.241, 0.236], $F(3,17)$ = 0.40, p = .758, R^2 = .065, or for P300, b = -0.58, SE = 0.40, $t(17)$ = -1.45, p = .166, 95% CI [-1.43, 0.27], $F(3,17)$ = 1.21, p = .337, R^2 = .176.

Emotional Representations

Emotional representations were not a significant predictor of P2 amplitude, b = 0.019, SE = 0.032, $t(19)$ = 0.61, p = .551, 95% CI [-0.048, 0.086], R^2 = .019, or P300 amplitude, b = 0.038, SE = 0.122, $t(19)$ = 0.31, p = .758, 95% CI [-0.217, 0.293], R^2 = .005.

Moderation analyses showed that injury status did not significantly influence these relationships. The interaction between emotional representations and injury status was not significant for P2, b = -0.077, SE = 0.070, $t(17)$ = -1.09, p = .290, 95% CI [-0.225, 0.071], $F(3,17)$ = 0.77, p = .526, R^2 = .120, or for P300, b = 0.097, SE = 0.275, $t(17)$ = 0.35, p = .729, 95% CI [-0.483, 0.676], $F(3,17)$ = 0.34, p = .795, R^2 = .057.

Exploratory Analyses – Group Differences in Self-Report Measures

Independent samples t-tests were conducted to examine group differences between participants with and without a history of mTBI on self-report measures of psychological distress, pain catastrophizing, and illness perceptions.

For depressive symptoms (PHQ-9), the control group (M = 11.14) reported significantly higher scores than the mTBI group (M = 6.84), $t(51.41)$ = 2.70, p = .009, 95% CI [1.11, 7.49], d = 0.72. This suggests that participants without a history of mTBI reported higher levels of depressive symptoms compared to those with mTBI.

Similarly for pain catastrophizing (PCS), the control group (M = 28.55) reported significantly higher scores than the mTBI group (M = 12.92, $t(51.01)$ = 4.92, p < .001, 95% CI

[9.26, 22.00], d = 1.34. This indicates that control participants reported greater maladaptive pain-related thinking compared to those with mTBI.

For perceived control (IPQ-R), no significant group differences were found, $t(49.61) = -0.26$, $p = .795$, 95% CI [-2.19, 1.69], $d = -0.07$.

For illness coherence (IPQ-R), the mTBI group ($M = 15.00$) scored significantly higher than the control group ($M = 11.52$), $t(51.65) = -2.81$, $p = .007$, 95% CI [-5.97, -0.99], $d = -0.76$. This suggests that individuals with mTBI reported a clearer understanding of their injury compared to controls asked to imagine experiencing a brain injury, likely reflecting differences between lived experiences and hypothetical injury representations.

For emotional representations (IPQ-R), the control group ($M = 15.55$) scored significantly higher than the mTBI group ($M = 9.18$), $t(41.20) = 4.35$, $p < .001$, 95% CI [3.42, 9.33], $d = 1.22$. This indicates that control participants reported experiencing more emotional impact or distress related to a health condition compared to individuals with mTBI.

To control for potential Type I error inflation due to multiple comparisons, a Benjamini-Hochberg false discovery rate (FDR) correction was applied to the set of independent samples t-tests conducted on self-report measures. All group differences remained statistically significant following FDR correction (adjusted p-values < .01), indicating that the observed effects were robust to multiple comparisons adjustment.

Exploratory Analyses – PHQ-9 and PCS as Predictors of ERP Amplitudes

Exploratory linear regression analyses were conducted to examine whether depressive symptoms (PHQ-9) and pain catastrophizing (PCS) predicted P2 and P300 amplitudes, and whether these relationships were moderated by injury status. See Table 2.

Main Effects of PHQ-9

Depressive symptoms were not a significant predictor of P2 amplitude, $b = 0.027$, SE = 0.030, $t(19) = 0.91$, $p = .374$, 95% CI [-0.035, 0.089], $R^2 = .042$, or of P300 amplitude, $b = 0.147$, SE = 0.110, $t(19) = 1.34$, $p = .195$, 95% CI [-0.082, 0.376], $R^2 = .087$.

Main Effects of PCS

Pain catastrophizing was also not a significant predictor of P2 amplitude, $b = 0.012$, SE = 0.013, $t(19) = 0.88$, $p = .391$, 95% CI [-0.016, 0.039], $R^2 = .039$, or P300 amplitude, $b = -0.025$, SE = 0.050, $t(19) = -0.50$, $p = .620$, 95% CI [-0.131, 0.080], $R^2 = .013$.

Moderation by Injury Status

Injury status did not significantly moderate the relationship between PHQ-9 or PCS scores and ERP amplitudes. All interaction effects were non-significant for P2 (PHQ-9: $b = -0.075$, $p = .233$; PCS: $b = 0.013$, $p = .720$) and P300 (PHQ-9: $b = -0.031$, $p = .896$; PCS: $b = 0.037$, $p = .795$).

DISCUSSION

The present study examined whether perceived control over post-injury symptoms was associated with neural responses to pain-related mental imagery, indexed by the P2 and P300 ERP components, and whether injury status moderated this association. Overall, the findings did not support the primary hypotheses, as neither perceived control nor injury status predicted P2 or P300 amplitudes and no moderation was observed. Instead, meaningful group differences were found in exploratory analyses of the self-report measures, where the mTBI and control groups differed substantially but in directions that contradict the patterns reported in the mTBI literature.

Contrary to Hypothesis 1, individuals with a history of mTBI did not significantly differ from control participants in P2 or P300 amplitudes. Although small to moderate effect sizes were found for P2, these differences were not statistically significant. This suggests that, within this specific sample, mTBI status alone may not strongly influence early attentional (P2) or later cognitive-evaluative (P300) processing of pain-related imagery or the study was not powered sufficiently to detect effects of this magnitude.

Hypothesis 2 was also not supported, as perceived control did not significantly predict P2 or P300 amplitudes. The observed effects were negligible, indicating that individual differences in perceived control were not meaningfully associated with neural responses to pain-related imagery in this paradigm. Similarly, Hypothesis 3 was not supported, as injury status did not moderate the relationship between perceived control and ERP amplitudes.

An important methodological consideration when interpreting these null ERP findings is the quality and quantity of data used to estimate component amplitudes. Due to preprocessing constraints, the number of artifact-free trials retained for some participants was relatively low, which can reduce signal-to-noise ratio (SNR) and increase variability in ERP amplitude

estimates. Reduced SNR limits the ability to detect subtle neural effects and may have contributed to the absence of significant findings for both the P2 and P300 components. As such, the present results should not be interpreted as definitive evidence of no neural differences, but rather reflecting limited sensitivity to detect such effects under conditions of increased noise and reduced trial counts.

Although the primary hypotheses were not supported, the exploratory analyses produced a distinct set of findings worth examining in detail. Exploratory analyses produced an unexpected pattern of group differences in self-report measures. Control participants scored substantially higher than the mTBI group on the PHQ-9 ($d = 0.72$), the PCS ($d = 1.34$), and the IPQ-R Emotional Representations subscale ($d = 1.22$), while the mTBI group scored higher on Illness Coherence ($d = -0.76$). These effect sizes are large, and three of them invert the direction predicted by the mTBI literature, which generally documents elevated depression, catastrophizing, and emotional reactivity in mTBI populations relative to non-injured peers. This pattern suggests that while psychological differences between groups were evident, these differences did not translate into measurable differences in ERP amplitudes, or this study was not powered enough to detect them with EEG.

One possible interpretation is that self-reported cognitive and emotional processes may not map directly onto the neural processes indexed by P2 and P300. However, an additional and important consideration is that the present pattern may reflect characteristics of the sample rather than true deviations from established literature. University-based control samples are known to overrepresent individuals experiencing elevated psychological distress, including symptoms of depression and anxiety (Karyotaki et al., 2020), which may partially explain the higher-than-expected PHQ-9 and PCS scores observed in the control group. In this context, the control group

may not represent a low-symptom baseline, but rather a relatively high-functioning but psychologically stressed comparison group.

In addition, the mTBI sample in the present study consisted of individuals within approximately 18 months post-injury who were functionally able to attend college and participate in laboratory-based EEG research. This suggests that the sample may represent a higher-functioning subgroup of individuals with mTBI, potentially characterized by greater engagement in academic and daily functioning and increased likelihood of having received psychoeducation, medical care, or psychological support during recovery. Such factors may contribute to improved coping strategies and reduced catastrophizing over time, which could attenuate expected group differences.

It's also important to consider that individuals with mTBI often demonstrate impaired self-awareness and reduced accuracy in reporting post-injury symptoms (Shi et al., 2024). As a result, lower self-reported symptom levels in the mTBI group may not necessarily reflect lower distress, but rather differences in symptom awareness or reporting tendencies relative to controls.

The finding that individuals with mTBI reported greater illness coherence than controls is especially interesting. Because control participants were instructed to respond hypothetically, this difference likely reflects the role of lived experience in shaping injury representations. Those with direct experience of mTBI may develop more coherent and structured understandings of their condition, whereas control participants may rely on abstract or incomplete representations when responding in a simulated context. Additional exploratory analyses examining relationships between ERP amplitudes and additional psychological variables, including depressive symptoms and pain catastrophizing, were also non-significant. This further supports

the notion that neural responses to pain-related imagery may be relatively independent of these self-reported cognitive and emotional factors, at least within the constraints of this study design.

One additional consideration relates to the observed polarity and magnitude of the P300 component. Although the P300 is conventionally characterized as a positive deflection, the mean amplitude values in the present study were slightly negative (e.g., -1.32 μV). This discrepancy may reflect differences between peak-based visual inspection of waveforms and the mean amplitude calculations used in statistical analyses, which can be influenced by the selected time window, baseline correction procedures, and individual variability in waveform morphology. Importantly, inspection of the grand average waveforms indicates a typical positive-going deflection consistent with the P300 component, suggesting that the negative mean values likely reflect analytic and averaging characteristics rather than a reversal of the underlying neural response. Additionally, the relatively low number of artifact-free trials retained for some participants may have introduced additional variability in amplitude estimates, further contributing to small shifts in mean values.

Building on this, an additional methodological consideration is the selection of electrodes used to quantify the P300 component. The electrodes selected for the primary analyses may not have optimally captured the centroparietal distribution typically associated with the P300. To further evaluate this possibility, additional exploratory plotting was conducted using electrodes at Pz and CPz, where P300 activity is most commonly observed. These plots revealed a more typical positive-going deflection consistent with the expected morphology of the P300 component, suggesting that the electrode selection in the primary analyses may have attenuated or distorted the observed amplitude. Taken together, these findings suggest that the observed

P300 characteristics likely reflect a combination of analytic choices, signal variability, and electrode selection, rather than a true absence or reversal of P300-related neural activity.

Limitations

Several limitations should be considered when interpreting these findings. First, the sample size was relatively small, which may have limited statistical power to detect subtle effects, especially in the interaction models. Although small sample sizes are common in ERP research, the ability to detect moderation effects is especially constrained under these conditions. There was also a substantial loss of useable EEG data following artifact rejection. Of the original sample ($N = 54$), only a subset of participants ($N = 21$) contributed useable ERP data after preprocessing, representing a data rejection rate of over 60%.

Second, a key limitation related to this attrition is the low number of artifact-free trials available for ERP estimation in a portion of the retained sample. Although a minimum threshold of six trials was used to retain participants, this is below commonly recommended levels for stable estimation ERP components like the P300, which typically requires about 20-30 or more trials to achieve adequate SNR. As a result, the ERP waveforms reported in the present study should be interpreted as relatively low SNR estimates of neural activity rather than fully stabilized component measures. This increased variability likely reduced statistical power and sensitivity to detect true effects.

This level of data loss also raises the possibility of selection bias. Participants with usable EEG data may represent a subset characterized by lower movement artifacts, greater task compliance, or reduced symptom burden. In the context of mTBI, where symptoms such as headache, discomfort, and fatigue are common, this may result in a final ERP sample that is not fully representative of the broader population. Consequently, group differences may be attenuated, and findings may be biased toward null effects.

Third, the use of hypothetical responding in the control group introduces a potential confound. While this approach allowed for comparison across groups on injury perception measures, it also means their responses may reflect simulated or abstract representations rather than lived experience. This difference in response may limit the interpretability of group comparisons, particularly for constructs like illness coherence.

Fourth, the ERP components examined may not have been sensitive enough to detect the cognitive and emotional processes of interest. While these components are commonly associated with attentional and evaluative processing, they may not capture more nuanced or sustained aspects of pain-related cognition, such as rumination or emotional appraisal. Additionally, this study relied on averaged ERP amplitudes across trials which, while increasing variability, may obscure trial-level variability or condition-specific effects. It is possible that more complex analyses could reveal patterns not captured in the current approach. Plus, the cross-sectional design limits the ability to draw conclusions about causal relationships between perceived control, psychological factors, and neural responses.

Lastly, an additional limitation concerns the selection of electrode sites used to quantify ERP components. Although standard midline and frontal electrodes were included in the primary analyses, the chosen electrodes may not have optimally captured the spatial distribution of the P300 component, which is typically maximal over centroparietal sites. As a result, amplitude estimates may be sensitive to electrode specification, potentially influencing the magnitude of observed effects.

Future Directions

Future research should aim to address these limitations and further clarify the relationship between psychological factors and neural responses to pain-related imagery. Increasing sample size would improve statistical power and allow for more robust testing of interaction effects.

Future studies may also benefit from implementing procedures designed to reduce EEG data loss, such as increasing the number of trials, improving participant preparation procedures, or using more advanced artifact correction methods. Additionally, future studies should consider alternative approaches to control group comparisons, such as including participants with different types of injuries or using designs that do not rely on hypothetical responding. Incorporating more comprehensive screening of psychological and psychiatric variables may also help clarify the extent to which symptom reporting and pain-related cognitive processes are influenced by comorbid emotional factors.

Further work is also needed to identify neural measures that are more sensitive to cognitive and emotional aspects of pain processing. This may include examining other ERP components, time-frequency analyses, or complementary neuroimaging methods. Longitudinal designs could also provide valuable insight into how perceived control and injury representations evolve over time following mTBI and how these changes relate to neural processing. Future research could also explore more ecologically valid or emotionally engaging paradigms for eliciting pain-related responses, which may strengthen the link between self-report measures and neural activity.

Also, there should be careful consideration of ROI selection when examining ERP components, particularly those with well-established spatial distribution such as the P300. Using data-driven or validated centroparietal electrode clusters (e.g., Pz, CPz, and adjacent sites) may improve sensitivity to expected neural responses and reduce measurement variability. In addition, future studies may benefit from complementing mean amplitude analyses with peak-based or time-frequency approaches to better capture condition-specific and spatially distributed effects.

Overall, the present study contributes to a growing body of literature suggesting that psychological factors such as perceived control and injury representations, while important for understanding subjective experience, may not always be directly reflected in neural measures of cognitive processing, highlighting the need for multimodal approaches to studying pain and injury-related outcomes.

Table 1
Descriptive Statistics

Variable	N = 54[1]	mTBI (ERP sample, *n* = 9)	Control (ERP sample, *n* = 12)
Age	21 (3 [18, 33])	19 (0.97 [18, 21])	23 (4 [18, 33])
Gender			
Male	15 (28%)	3 (33%)	4 (33%)
Female	39 (72%)	6 (67%)	8 (67%)
Race			
White	41 (76%)	8 (89%)	8 (67%)
Latino	3 (5.6%)	-	1 (8%)
Asian	4 (7.4%)	-	3 (25%)
Mixed Race	6 (11%)	1 (11%)	-
Year			
Freshman	19 (36%)	5 (56%)	2 (17%)
Sophomore	13 (25%)	2 (22%)	1 (8%)
Junior	11 (21%)	2 (22%)	3 (25%)
Senior	7 (13%)	-	4 (33%)
Graduate Student	3 (5.7%)	-	2 (17%)
Injury Status			
TBI	25 (46%)	9 (43%)	-
Control	29 (54%)	-	12 (57%)
PHQ-9 Total Score	9 (6 [0, 27])	8.9 (6.5 [1, 19])	11 (6.8 [2, 27])
PCS Total Score	21 (14 [0, 52])	11.5 (10.3 [0, 28])	27.8 (14.1 [2, 52])
IPQ-R Personal Control	15.9 (3.5 [6.5, 23.0])	15.5 (3.8 [8.5, 20])	15.9 (3.44 [11, 21])
IPQ-R Illness Coherence	13.1 (4.9 [0.5, 20.0])	14.9 (3.3 [10, 20])	12.4 (4.7 [0.5, 19.5])
IPQ-R Emotional Representation	13 (6 [0, 24])	10.4 (6.6 [0, 18])	15.2 (5.1 [8.5, 24])
P2 Amplitude (μV; n = 21)	0.48 (0.87 [-1.66, 2.26])	0.25 (0.55 [-0.57, 1.13])	0.65 (1.03 [-1.67, 2.26])
P300 Amplitude (μV; n = 21)	-1.32 (3.29 [-9.45, 4.24])	-0.71 (2.29 [-3.21, 4.24])	-1.79 (3.91 [-9.45, 3.15])

[1]Mean (SD [Min, Max]); n (%)

Table 2
Descriptive Statistics for Survey Measures

Measure	*N*	mTBI *M*	mTBI *SD*	Control *M*	Control *SD*	*t*	*df*	*p*	*p* (FDR-corrected)	*d*
PHQ-9	54	6.84	5.08	11.14	6.59	-2.70	51.41	.009	.012	-.72
PCS	54	12.92	11.56	28.55	11.71	-4.92	51.01	<.001	<.001	-1.34
IPQ-R Perceived Control	54	16.08	3.65	15.83	3.41	.26	49.61	.795	.795	.07
IPQ-R Illness Coherence	54	15.00	4.40	11.52	4.72	2.80	51.65	.007	.012	.76
IPQ-R Emotional Representations	54	9.18	6.20	15.55	4.19	-4.35	41.20	<.001	<.001	-1.22

Note. mTBI = mild traumatic brain injury group; Control = non-injured comparison group. False discovery rate (FDR) was applied to adjust for multiple comparisons in exploratory analyses. P-values are presented both uncorrected and FDR-adjusted.

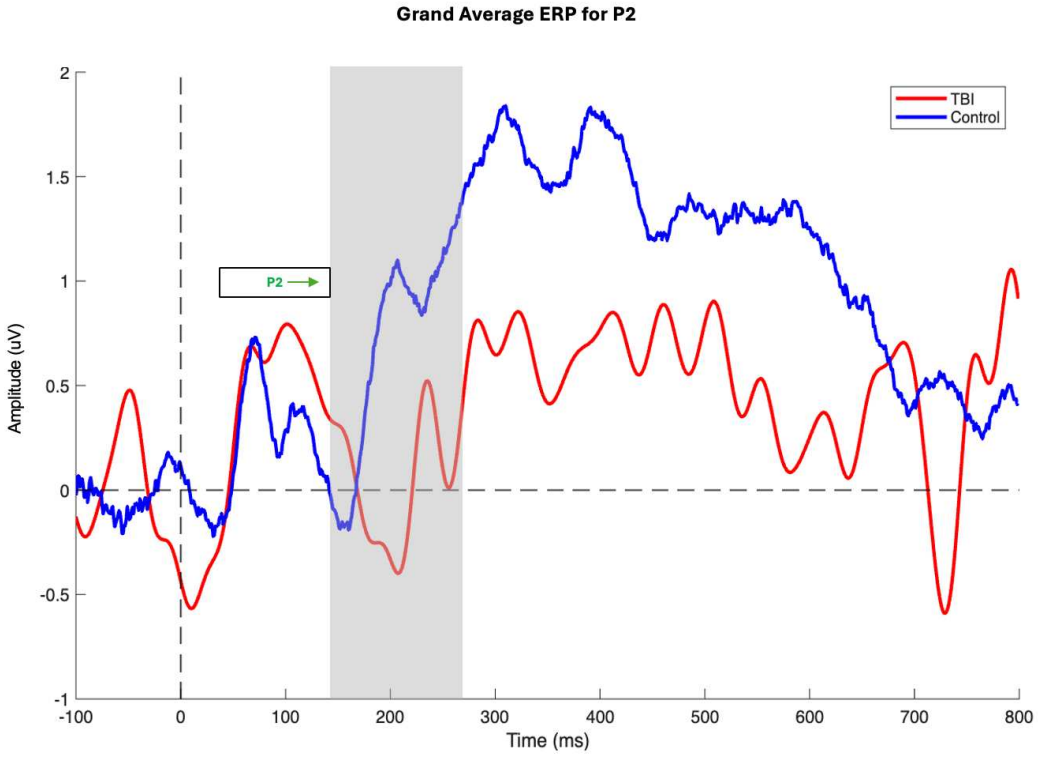


Figure 1. Grand average ERP waveforms for the P2 component, averaged across particpants and electrodes within a centroparietal region of interest (C1, C2, P3, P4). Only participants with usable ERP data at all electrodes in the P2 ROI were included. Waveforms reflect mean amplitudes collapsed across trials and ROI electrodes. The shaded region represents the P2 time window used for analysis (150-250 ms).

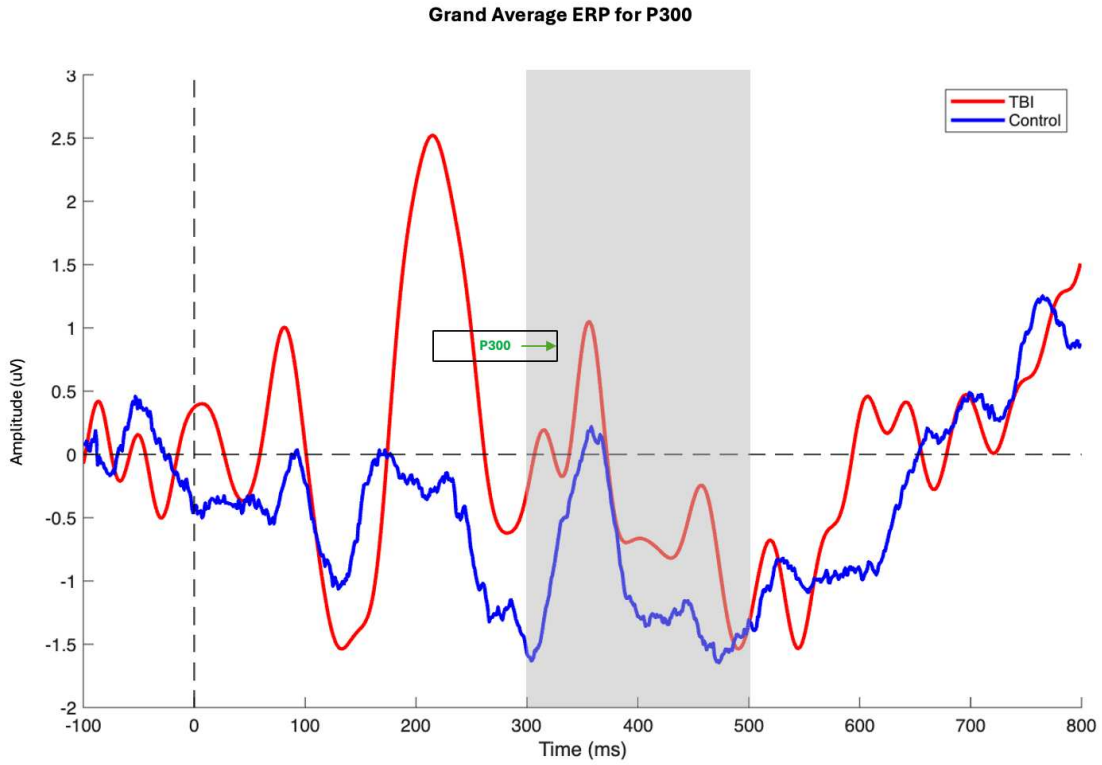


Figure 2. Grand average ERP waveforms for the P300 components, averaged across participants and electrodes within a frontocentral region of interest (AF3, AF4, FC1, FC2, F3, F4, Fz). Only participants with usable ERP data at all electrodes in the P300 ROI were included. Waveforms reflect mean amplitudes collapsed across trials and ROI electrodes. The shaded region represents the P300 time window used for analysis. (300-500 ms).

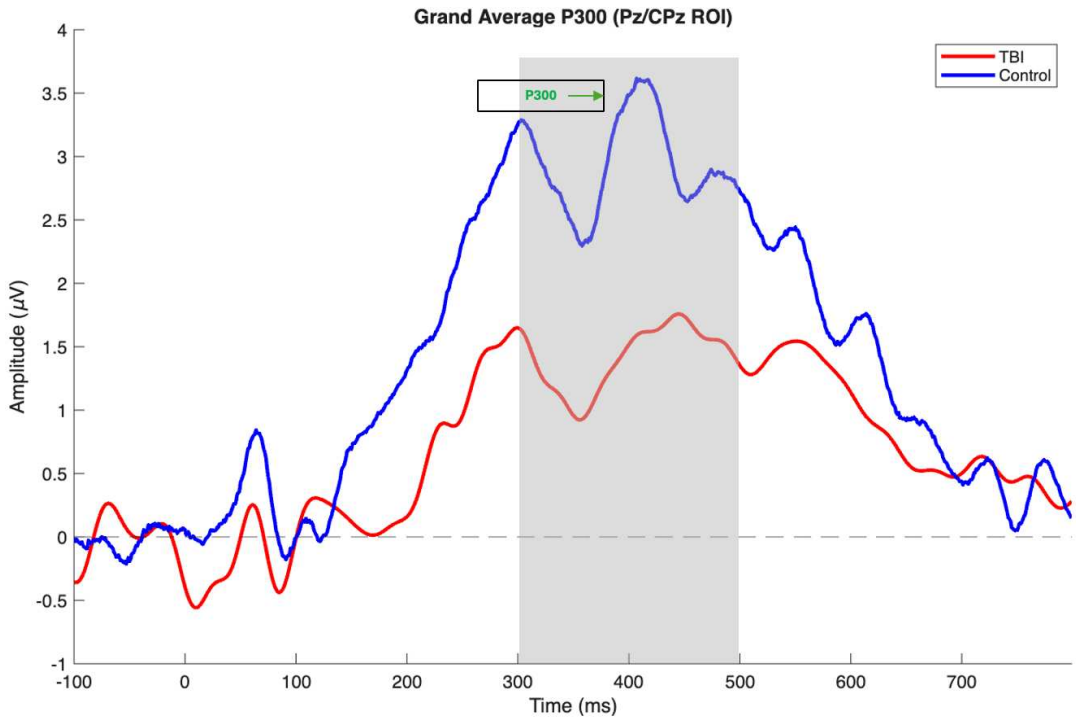


Figure 3. Exploratory grand average P300 waveform at Pz and CPz electrodes. This figure reflects a re-analysis of P300 activity using centroparietal electrodes, which are typically associated with P300 generation.

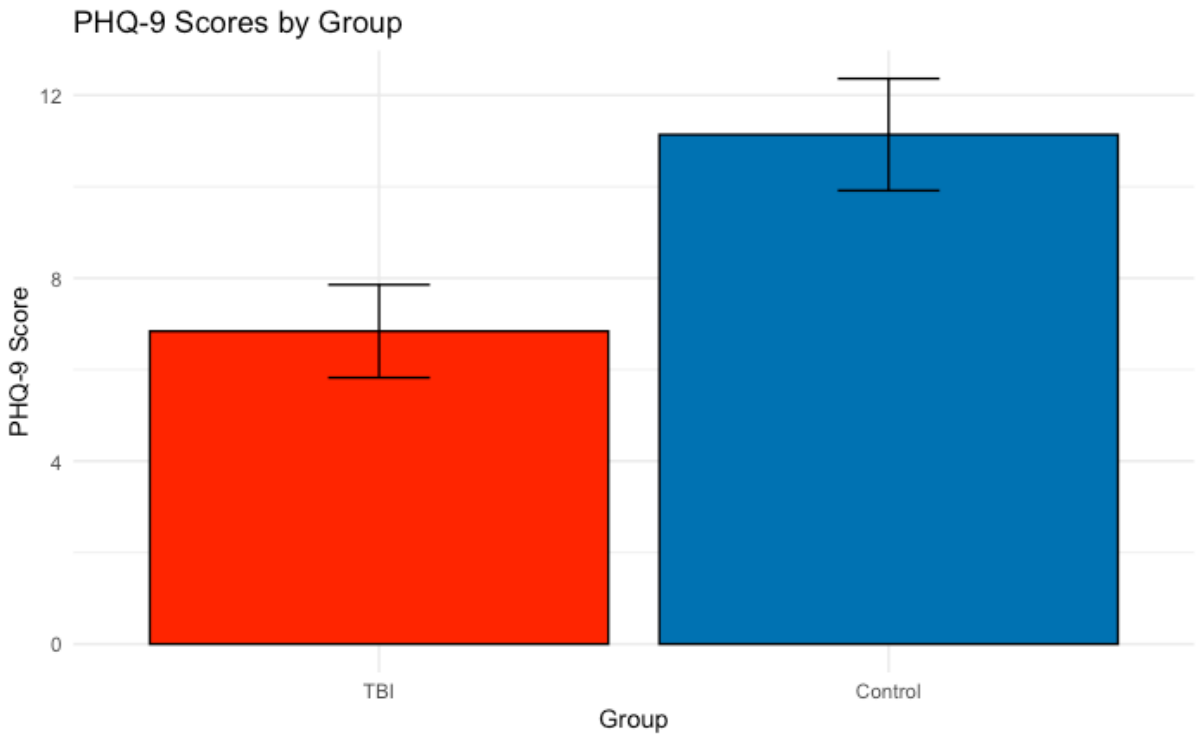


Figure 4. Group differences in PHQ-9 total scores. Bars represent group means, with error bars indicating standard error. The Control group (n = 29) showed higher mean PHQ-9 scores than the TBI group (n = 25).

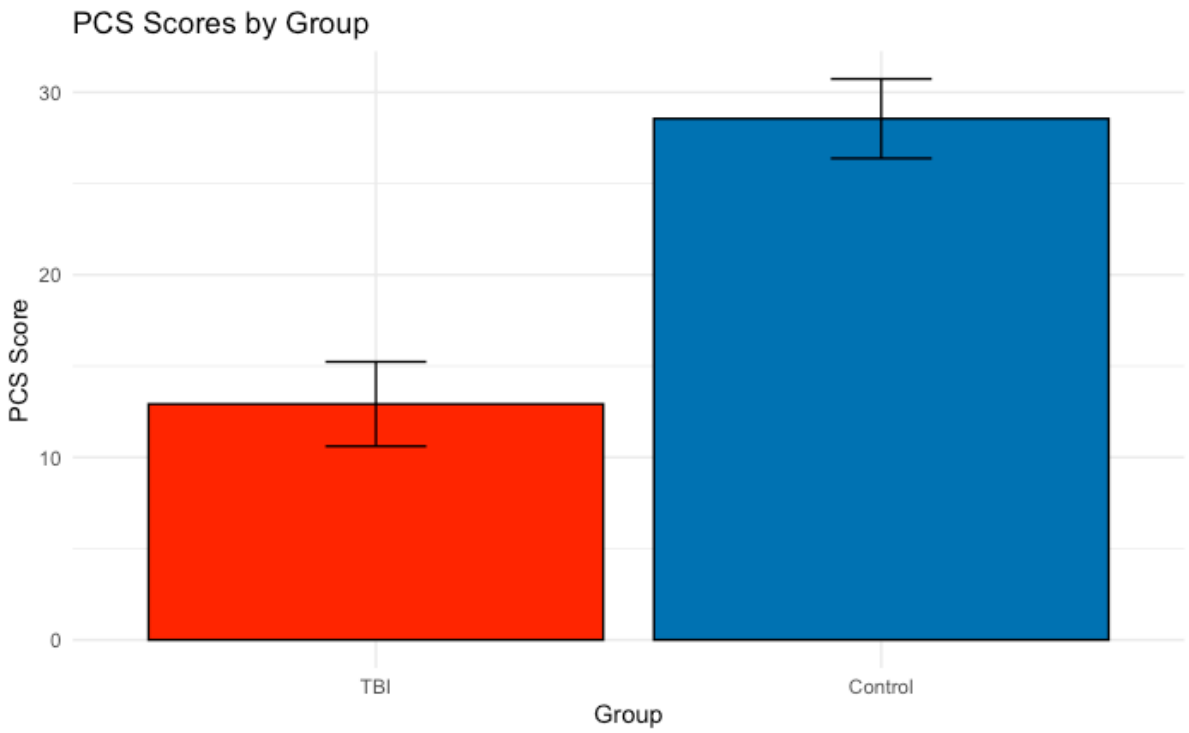


Figure 5. Group differences in PCS total scores. Bars represent group means, with error bars indicating standard error. The Control group (n = 29) exhibited higher mean PCS scores than the TBI group (n = 25).

REFERENCES

Arciniega, H., Shires, J., Furlong, S., Kilgore-Gomez, A., Cerreta, A., Murray, N. G., & Berryhill, M. E. (2021). Impaired visual working memory and reduced connectivity in undergraduates with a history of mild traumatic brain injury. *Scientific Reports, 11*(1). https://doi.org/10.1038/s41598-021-80995-1

Bartolomeo, P., Bourgeois, A., Bourlon, C., & Migliaccio, R. (2012). Visual and motor mental imagery after brain damage. *Multisensory Imagery*, 249–269. https://doi.org/10.1007/978-1-4614-5879-1_13

Bell, J. M., Master, C. L., & Lionbarger, M. R. (2017). The clinical implications of Youth Sports Concussion Laws: A Review. *American Journal of Lifestyle Medicine*, *13*(2), 172–181. https://doi.org/10.1177/1559827616688883

Brain Injury Association of America. (2025, March 31). https://biausa.org/

Carroll, L. J., Cassidy, J. D., Cancelliere, C., Côté, P., Hincapié, C. A., Kristman, V. L., Holm, L. W., Borg, J., Nygren-de Boussard, C., & Hartvigsen, J. (2014). Systematic review of the prognosis after mild traumatic brain injury in adults: Cognitive, psychiatric, and mortality outcomes: Results of the international collaboration on Mild Traumatic Brain Injury Prognosis. *Archives of Physical Medicine and Rehabilitation*, *95*(3). https://doi.org/10.1016/j.apmr.2013.08.300

Centers for Disease Control and Prevention. (2024, October 29). *TBI Data*. Centers for Disease Control and Prevention. https://www.cdc.gov/traumatic-brain-injury/data-research/index.html

Chaput, G., Lajoie, S. P., Naismith, L. M., & Lavigne, G. (2016). Pain catastrophizing correlates with early mild traumatic brain injury outcome. *Pain Research and Management*, *2016*, 1–7. https://doi.org/10.1155/2016/2825856

Christian, B. M., Parkinson, C., Macrae, C. N., Miles, L. K., & Wheatley, T. (2015). When imagining yourself in pain, visual perspective matters: The neural and behavioral correlates of simulated sensory experiences. *Journal of Cognitive Neuroscience, 27*(5), 866–875. https://doi.org/10.1162/jocn_a_00754

Eierud, C., Craddock, R. C., Fletcher, S., Aulakh, M., King-Casas, B., Kuehl, D., & LaConte, S. M. (2014). Neuroimaging after mild traumatic brain injury: Review and meta-analysis. *NeuroImage: Clinical*, *4*, 283–294. https://doi.org/10.1016/j.nicl.2013.12.009

Fann, J. R., Bombardier, C. H., Dikmen, S., Esselman, P., Warms, C. A., Pelzer, E., Rau, H., & Temkin, N. (2005). Validity of the patient health questionnaire-9 in assessing depression following traumatic brain injury. *Journal of Head Trauma Rehabilitation*, *20*(6), 501–511. https://doi.org/10.1097/00001199-200511000-00003

Fardo, F., Allen, M., Jegindø, E.-M. E., Angrilli, A., & Roepstorff, A. (2015). Neurocognitive evidence for mental imagery-driven hypoalgesic and hyperalgesic pain regulation. *NeuroImage*, *120*, 350–361. https://doi.org/10.1016/j.neuroimage.2015.07.008

Ferdek, M. A., Oosterman, J. M., Adamczyk, A. K., van Aken, M., Woudsma, K. J., Peeters, B. W. M. M., Nap, A., Wyczesany, M., & van Rijn, C. M. (2019). Effective connectivity of beta oscillations in endometriosis-related chronic pain during rest and pain-related mental imagery. *The Journal of Pain*, *20*(12), 1446–1458. https://doi.org/10.1016/j.jpain.2019.05.011

Gagnon, I., Grilli, L., Friedman, D., & Iverson, G. L. (2015). A pilot study of active rehabilitation for adolescents who are slow to recover from sport-related concussion. *Scandinavian Journal of Medicine & Science in Sports*, *26*(3), 299–306. https://doi.org/10.1111/sms.12441

Gaw, C. E., & Zonfrillo, M. R. (2016). Emergency department visits for head trauma in the United States. *BMC Emergency Medicine*, *16*(1). https://doi.org/10.1186/s12873-016-0071-8

Gombay, N., & Andrews, G. J. (2021). Living with embodied vibrations: Sensory experiences following a traumatic brain injury. *Social Science & Medicine*, *284*, 114233. https://doi.org/10.1016/j.socscimed.2021.114233

Gosden, T., Morris, P. G., Ferreira, N. B., Grady, C., & Gillanders, D. T. (2013). Mental imagery in chronic pain: Prevalence and characteristics. *European Journal of Pain*, *18*(5), 721–728. https://doi.org/10.1002/j.1532-2149.2013.00409.x

Gosselin, N., Bottari, C., Chen, J.-K., Petrides, M., Tinawi, S., de Guise, É., & Ptito, A. (2011). Electrophysiology and functional MRI in post-acute mild traumatic brain injury. *Journal of Neurotrauma*, *28*(3), 329–341. https://doi.org/10.1089/neu.2010.1493

Granot, M., & Ferber, S. G. (2005). The roles of pain catastrophizing and anxiety in the prediction of postoperative pain intensity. *The Clinical Journal of Pain*, *21*(5), 439–445. https://doi.org/10.1097/01.ajp.0000135236.12705.2d

Grilli, M. D., & Glisky, E. L. (2010). Self-imagining enhances recognition memory in memory-impaired individuals with neurological damage. *Neuropsychology*, *24*(6), 698–710. https://doi.org/10.1037/a0020318

Harrison-Felix, C., Sevigny, M., Beaulieu, C. L., Callender, L., Dams-O'Connor, K., Hammond, F. M., Hanks, R., Ketchum, J. M., Martin, A. M., Marwitz, J. H., Peckham, M., Rabinowitz, A. R., Sander, A. M., Sterling, A., Walker, W. C., Nakase-Richardson, R., & Hoffman, J. M. (2024). Characterization and treatment of chronic pain after traumatic brain injury—comparison of characteristics between individuals with current pain, past pain, and no pain: A NIDILRR and VA TBI Model Systems Collaborative Project. *Journal of Head Trauma Rehabilitation*, *39*(1), 5–17. https://doi.org/10.1097/htr.0000000000000910

Haneef, Z., Levin, H. S., Frost, J. D., & Mizrahi, E. M. (2013). Electroencephalography and quantitative electroencephalography in mild traumatic brain injury. *Journal of Neurotrauma*, *30*(8), 653–656. https://doi.org/10.1089/neu.2012.2585

Hill, S. (2010). The Illness Perceptions Questionnaire-Revised (IPQ-R). *Journal of Physiotherapy*, *56*(4), 280. https://doi.org/10.1016/s1836-9553(10)70062-x

Ianof, J. N., & Anghinah, R. (2017). Traumatic brain injury: An EEG point of View. *Dementia & Neuropsychologia*, *11*(1), 3–5. https://doi.org/10.1590/1980-57642016dn11-010002

Jones, K. M., Theadom, A., Barker-Collo, S., Broadbent, E., Feigin, V. L., Feigin, V., Barker-Collo, S., McPherson, K., Theadom, A., Jones, K. M., Jones, A., Ao, B. T., Kydd, R., Barber, P. A., Parag, V., Ameratunga, S., Starkey, N., Dowell, A., Kahan, M., … Brown, P. (2017). Associations between brain drawings following mild traumatic brain injury and negative illness perceptions and post-concussion symptoms at 4 years. *Journal of Health Psychology*, *24*(10), 1448–1458. https://doi.org/10.1177/1359105317695430

Karamian, A., Lucke-Wold, B., & Seifi, A. (2024). Prevalence of traumatic brain injury in the general adult population of the USA: A meta-analysis. *Neuroepidemiology*, 1–10. https://doi.org/10.1159/000540676

Karr, J. E., Williams, M. W., Iverson, G. L., Huang, S.-J., & Yang, C.-C. (2022). Pre-injury headache and post-traumatic headache in patients with mild traumatic brain injury: Neuropsychological, Psychiatric, and post-concussion symptom outcomes. *Brain Injury*, *36*(2), 175–182. https://doi.org/10.1080/02699052.2022.2043440

Karyotaki, E., Cuijpers, P., Albor, Y., Alonso, J., Auerbach, R. P., Bantjes, J., Bruffaerts, R., Ebert, D. D., Hasking, P., Kiekens, G., Lee, S., McLafferty, M., Mak, A., Mortier, P., Sampson, N. A., Stein, D. J., Vilagut, G., & Kessler, R. C. (2020). Sources of stress and their associations with mental disorders among college students: Results of the World Health Organization World Mental Health Surveys International College Student initiative. *Frontiers in Psychology*, *11*. https://doi.org/10.3389/fpsyg.2020.01759

Konrad, C., Geburek, A. J., Rist, F., Blumenroth, H., Fischer, B., Husstedt, I., Arolt, V., Schiffbauer, H., & Lohmann, H. (2010). Long-term cognitive and emotional consequences of mild traumatic brain injury. *Psychological Medicine*, *41*(6), 1197–1211. https://doi.org/10.1017/s0033291710001728

Kroenke, K., Spitzer, R. L., & Williams, J. B. (2001). The PHQ-9. *Journal of General Internal Medicine*, *16*(9), 606–613. https://doi.org/10.1046/j.1525-1497.2001.016009606.x

Ku, D., Mao, L., Nikolova, S., Dumkrieger, G. M., Ross, K. B., Huentelman, M., Anderson, T., Porreca, F., Navratilova, E., Starling, A., Wu, T., Li, J., Chong, C. D., & Schwedt, T. J. (2025). Longitudinal analysis of pain-induced brain activations in post-traumatic headache. *Cephalalgia*, *45*(5). https://doi.org/10.1177/03331024251345160

Leventhal, H., Meyer, D., & Nerenz, D. (1980). The common sense model of illness danger. Rachman, S.(ed.) Medical psychology. *Contributions to medical psychology*, 7-30.

Leventhal, H., Phillips, L. A., & Burns, E. (2016). The common-sense model of self-regulation (CSM): A Dynamic Framework for understanding illness self-management. *Journal of Behavioral Medicine*, *39*(6), 935–946. https://doi.org/10.1007/s10865-016-9782-2

Lithwick, A., Lev, S., & Binshtok, A. M. (2013). Chronic pain-related remodeling of cerebral cortex – 'pain memory': A possible target for treatment of chronic pain. *Pain Management*, *3*(1), 35–45. https://doi.org/10.2217/pmt.12.74

Liu, S., Shi, C., Ma, X., Zhao, B., Chen, X., & Tao, L. (2021). Cognitive deficits and rehabilitation mechanisms in mild traumatic brain injury patients revealed by EEG connectivity markers. *Clinical Neurophysiology*, *132*(2), 554–567. https://doi.org/10.1016/j.clinph.2020.11.034

Lonsdale, J. H. (2010). Imagery and emotion in chronic pain. *Edinburgh Medical School Thesis and Dissertation Collection*. http://hdl.handle.net/1842/5517

Luo, J., Zhu, H.-Q., Gou, B., & Wang, X.-Q. (2022). Neuroimaging assessment of pain. *Neurotherapeutics*, *19*(5), 1467–1488. https://doi.org/10.1007/s13311-022-01274-z

Lyby, P. S., Aslaksen, P. M., & Flaten, M. A. (2011). Variability in placebo analgesia and the role of fear of pain—an ERP study. *Pain*, *152*(10), 2405–2412. https://doi.org/10.1016/j.pain.2011.07.010

Marks, D. F. (1973). Visual imagery differences in the recall of pictures. *British Journal of Psychology*, *64*(1), 17–24. https://doi.org/10.1111/j.2044-8295.1973.tb01322.x

McCrea, M., Broglio, S., McAllister, T., Zhou, W., Zhao, S., Katz, B., Kudela, M., Harezlak, J., Nelson, L., Meier, T., Marshall, S. W., & Guskiewicz, K. M. (2019). Return to play and

risk of repeat concussion in collegiate football players: Comparative Analysis from the NCAA Concussion Study (1999–2001) and Care Consortium (2014–2017). *British Journal of Sports Medicine*, *54*(2), 102–109. https://doi.org/10.1136/bjsports-2019-100579

McInnes, K., Friesen, C. L., MacKenzie, D. E., Westwood, D. A., & Boe, S. G. (2019). Correction: Mild traumatic brain injury (mtbi) and chronic cognitive impairment: A scoping review. *PLOS ONE*, *14*(6). https://doi.org/10.1371/journal.pone.0218423

Medley, A. R., Powell, T., Worthington, A., Chohan, G., & Jones, C. (2010). Brain Injury Beliefs, self-awareness, and coping: A preliminary cluster analytic study based within the self-regulatory model. *Neuropsychological Rehabilitation*, *20*(6), 899–921. https://doi.org/10.1080/09602011.2010.517688

Melzack, R. (2001). Pain and the neuromatrix in the brain. *Journal of Dental Education*, *65*(12), 1378–1382. https://doi.org/10.1002/j.0022-0337.2001.65.12.tb03497.x

Meske, S., Hazzard, J. B., Ni, M., Hanson, T., Van Horn, L., & Smith, J. (2019). The prevalence of traumatic brain injury and on-campus service utilization among undergraduate students. *Journal of Head Trauma Rehabilitation*, *34*(1). https://doi.org/10.1097/htr.0000000000000407

Moro, V., Berlucchi, G., Lerch, J., Tomaiuolo, F., & Aglioti, S. M. (2008). Selective deficit of mental visual imagery with intact primary visual cortex and visual perception. *Cortex*, *44*(2), 109–118. https://doi.org/10.1016/j.cortex.2006.06.004

Moss-Morris, R., Weinman, J., Petrie, K., Horne, R., Cameron, L., & Buick, D. (2002). The Revised Illness Perception Questionnaire (IPQ-R). *Psychology & Health*, *17*(1), 1–16. https://doi.org/10.1080/08870440290001494

Nampiaparampil, D. E. (2008). Prevalence of chronic pain after traumatic brain injury: A systematic review. *JAMA*, *300*(6), 711–719. https://doi.org/10.1001/jama.300.6.711

Nandrajog, P., Idris, Z., Azlen, W., Liyana, A., & Abdullah, J. (2017). The use of event-related potential (p300) and neuropsychological testing to evaluate cognitive impairment in mild traumatic brain injury patients. *Asian Journal of Neurosurgery*, *12*(03), 447–453. https://doi.org/10.4103/1793-5482.180921

National Academies of Sciences, Engineering, and Medicine. (2022). The scope and burden of traumatic brain injury. In C. Matney, K. Bowman, & D. Berwick (Eds.), *Traumatic brain injury: A roadmap for accelerating progress* (Chapter 2). National Academies Press. https://www.ncbi.nlm.nih.gov/books/NBK580076/

Naugle, K. M., Corrona, S., Smith, J. A., Nguyen, T., Saxe, J., & White, F. A. (2021). Physical activity behavior in the first month after mild traumatic brain injury is associated with physiological and psychological risk factors for chronic pain. *PAIN Reports*, *6*(4). https://doi.org/10.1097/pr9.0000000000000969

Niemeier, J. P., Cifu, D. X., & Kishore, R. (2001). The lighthouse strategy: Improving the functional status of patients with unilateral neglect after stroke and brain injury using a visual imagery intervention. *Topics in Stroke Rehabilitation*, *8*(2), 10–18. https://doi.org/10.1310/7ukk-hj0f-gdwf-hhm8

Oldenburg, C., Lundin, A., Edman, G., Nygren-de Boussard, C., & Bartfai, A. (2015). Cognitive reserve and persistent post-concussion symptoms—a prospective mild traumatic brain injury (mtbi) cohort study. *Brain Injury*, *30*(2), 146–155. https://doi.org/10.3109/02699052.2015.1089598

Oostra, K. M., Vereecke, A., Jones, K., Vanderstraeten, G., & Vingerhoets, G. (2012). Motor imagery ability in patients with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, *93*(5), 828–833. https://doi.org/10.1016/j.apmr.2011.11.018

Pavlovic, D., Pekic, S., Stojanovic, M., & Popovic, V. (2019). Traumatic brain injury: Neuropathological, neurocognitive and neurobehavioral sequelae. *Pituitary*, *22*(3), 270–282. https://doi.org/10.1007/s11102-019-00957-9

Peterson, A. B., & Thomas, K. E. (2021). Incidence of nonfatal traumatic brain injury–related hospitalizations — United States, 2018. *MMWR. Morbidity and Mortality Weekly Report*, *70*(48), 1664–1668. https://doi.org/10.15585/mmwr.mm7048a3

Philips, H. C. (2013). Imagery and likelihood cognitive bias in pain. *Behavioural and Cognitive Psychotherapy*, *43*(3), 270–284. https://doi.org/10.1017/s1352465813000982

Philips, H. C. (2011). Imagery and pain: The prevalence, characteristics, and potency of imagery associated with pain. *Behavioural and Cognitive Psychotherapy*, *39*(5), 523–540. https://doi.org/10.1017/s1352465811000282

Piolino, P., Desgranges, B., Manning, L., North, P., Jokic, C., & Eustache, F. (2007). Autobiographical memory, the sense of recollection and executive functions after severe traumatic brain injury. *Cortex*, *43*(2), 176–195. https://doi.org/10.1016/s0010-9452(08)70474-x

Polinder, S., Cnossen, M. C., Real, R. G., Covic, A., Gorbunova, A., Voormolen, D. C., Master, C. L., Haagsma, J. A., Diaz-Arrastia, R., & von Steinbuechel, N. (2018). A multidimensional approach to post-concussion symptoms in mild traumatic brain injury. *Frontiers in Neurology*, *9*. https://doi.org/10.3389/fneur.2018.01113

Proverbio, A. M., Tacchini, M., & Jiang, K. (2023). What do you have in mind? ERP markers of visual and auditory imagery. *Brain and Cognition*, *166*, 105954. https://doi.org/10.1016/j.bandc.2023.105954

Rivera, E., Levoy, K., Park, C., Villalobos, A., Martin, P., Jung Kim, M., & Hirschman, K. B. (2024). Internal consistency reliability of the Revised Illness Perceptions Questionnaire: A systematic review and reliability generalization meta-analysis. *Journal of Health Psychology*, *29*(7), 734–746. https://doi.org/10.1177/13591053231221351

Robertson, K., & Schmitter-Edgecombe, M. (2015). Self-awareness and traumatic brain injury outcome. *Brain Injury*, *29*(7–8), 848–858. https://doi.org/10.3109/02699052.2015.1005135

Shi, S., Picon, E. L., Rioux, M., Panenka, W. J., & Silverberg, N. D. (2024). Catastrophizing is associated with excess cognitive symptom reporting after mild traumatic brain injury. *Neuropsychology*, *38*(2), 126–133. https://doi.org/10.1037/neu0000930

Skaansar, O., Tverdal, C., Rønning, P. A., Skogen, K., Brommeland, T., Røise, O., Aarhus, M., Andelic, N., & Helseth, E. (2020). Traumatic brain injury—the effects of patient age on treatment intensity and mortality. *BMC Neurology*, *20*(1). https://doi.org/10.1186/s12883-020-01943-6

Snell, D. L., Siegert, R. J., Hay-Smith, E. J., & Surgenor, L. J. (2010). An examination of the factor structure of the Revised Illness Perception Questionnaire modified for adults with mild traumatic brain injury. *Brain Injury*, *24*(13–14), 1595–1605. https://doi.org/10.3109/02699052.2010.523048

Strigo, I. A., Spadoni, A. D., Lohr, J., & Simmons, A. N. (2014). Too hard to control: Compromised pain anticipation and modulation in mild traumatic brain injury. *Translational Psychiatry*, *4*(1). https://doi.org/10.1038/tp.2013.116

Sullivan, M. J., Bishop, S. R., & Pivik, J. (1995). The pain catastrophizing scale: Development and validation. *Psychological Assessment*, *7*(4), 524–532. https://doi.org/10.1037//1040-3590.7.4.524

Sun, G., Wen, Z., Ok, D., Doan, L., Wang, J., & Chen, Z. S. (2022). Detecting acute pain signals from human EEG. *Journal of Neuroscience Methods*, *347*, 108964. https://doi.org/10.1016/j.jneumeth.2020.108964

Sun, H., Luo, C., Chen, X., & Tao, L. (2017). Assessment of cognitive dysfunction in Traumatic Brain Injury Patients: A Review. *Forensic Sciences Research*, *2*(4), 174–179. https://doi.org/10.1080/20961790.2017.1390836

Uomoto, J. M., & Esselman, P. C. (1993). Traumatic Brain Injury and Chronic Pain: Differential Types and Rates by Head Injury Severity. *Archives of Physical Medicine and Rehabilitation*, *74*(1), 61–64.

Vynorius, K. C., Paquin, A. M., & Seichepine, D. R. (2016). Lifetime multiple mild traumatic brain injuries are associated with cognitive and mood symptoms in young healthy college students. *Frontiers in Neurology*, *7*. https://doi.org/10.3389/fneur.2016.00188

Wheeler, C. H. B., Williams, A. C., & Morley, S. J. (2019). Meta-analysis of the psychometric properties of the pain catastrophizing scale and associations with participant characteristics. *Pain*, *160*(9), 1946–1953. https://doi.org/10.1097/j.pain.0000000000001494

Woodman, G. F. (2010). A brief introduction to the use of event-related potentials in studies of perception and attention. *Attention, Perception, & Psychophysics*, *72*(8), 2031–2046. https://doi.org/10.3758/bf03196680

Yoshida, K., Sawamura, D., Ogawa, K., Mototani, T., Ikoma, K., & Sakai, S. (2023). Prospective and retrospective metacognitive abilities and their association with impaired self-awareness in patients with traumatic brain injury. *Journal of Cognitive Neuroscience*, *35*(12), 1960–1971. https://doi.org/10.1162/jocn_a_02064

Zis, P., Liampas, A., Artemiadis, A., Tsalamandris, G., Neophytou, P., Unwin, Z., Kimiskidis, V. K., Hadjigeorgiou, G. M., Varrassi, G., Zhao, Y., & Sarrigiannis, P. G. (2022). EEG recordings as biomarkers of pain perception: Where do we stand and where to go? *Pain and Therapy*, *11*(2), 369–380. https://doi.org/10.1007/s40122-022-00372-2

Appendix A

Self-Report Measures

Vividness of Visual Imagery Questionnaire

For items 1-4, think of some relative or friend whom you frequently see (but who is not with you at present) and consider carefully the picture that comes before your mind's eye.

1. The exact contour of face, head, shoulders and body.
2. Characteristic poses of head, attitudes of body, etc.
3. The precise carriage, length of step, etc., in walking.
4. The different colors worn in some familiar clothes.

Visualize a rising sun. Consider carefully the picture that comes before your mind's eye.

5. The sun is rising above the horizon into a hazy sky.
6. The sky clears and surrounds the sun with blueness.
7. Clouds. A storm blows up, with flashes of lightning.
8. A rainbow appears.

Think of the front of a shop which you often go to. Consider the picture that comes before your mind's eye.

9. The overall appearance of the shop from the opposite side of the road.
10. A window display including colors, shapes and details of individual items for sale.
11. You are near the entrance. The color, shape and details of the door.
12. You enter the shop and go to the counter. The counter assistant serves you. Money changes hands.

Finally, think of a country scene which involves trees, mountains and a lake. Consider the picture that comes before your mind's eye.

13. The contours of the landscape.
14. The color and shape of the trees.
15. The color and shape of the lake.
16. A strong wind blows on the trees and on the lake causing waves.

Patient Health Questionnaire

Over the last 2 weeks, how often have you been bothered by any of the following problems? Answer on a scale from 0-3; 0 = Not at all, 1 = Several days, 2 = More than half the days, 3 = Nearly every day

1. Little interest or pleasure in doing things
2. Feeling down, depressed, or hopeless
3. Trouble falling or staying asleep, or sleeping too much
4. Feeling tired or having little energy
5. Poor appetite or overeating

6. Feeling bad about yourself – or that you are a failure or have let yourself or your family down
7. Trouble concentrating on things, such as reading the newspaper or watching television
8. Moving or speaking so slowly that other people could have noticed. Or the opposite being so fidgety or restless that you have been moving around a lot more than usual
9. Thoughts that you would be better off dead, or of hurting yourself
   Add all columns =
   Total =
10. If you checked off any problems, how difficult have these problems made it for you to do your work, take care of things at home, or get along with other people?
    a. Not difficult at all
    b. Somewhat difficult
    c. Very difficult
    d. Extremely difficult

Pain Catastrophizing Scale

Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery. We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

Answer on a scale of 0-4; 0 = Not at all, 1 = To a slight degree, 2 = To a moderate degree, 3 = To a great degree, 4 = All the time

1. I worry all the time about whether the pain will end
2. I feel I can't go on
3. It's terrible and I think it's never going to get any better
4. It's awful and I feel that it overwhelms me
5. I feel I can't stand it anymore
6. I become afraid that the pain will get worse
7. I keep thinking of other painful events
8. I anxiously want the pain to go away
9. I can't seem to keep it out of my mind
10. I keep thinking about how much it hurts
11. I keep thinking about how badly I want the pain to stop
12. There's nothing I can do to reduce the intensity of the pain
13. I wonder whether something serious may happen

Revised Illness Perception Questionnaire (Modified for this study)

YOUR VIEWS ABOUT YOUR INJURY

Listed below are a number of symptoms that you may or may not have experienced since your injury. Please indicate by circling Yes or No, whether you have experienced any of these symptoms since your injury, and whether you believe that these symptoms are related to your injury.

1. Pain

2. Sore Throat
3. Nausea
4. Breathlessness
5. Weight Loss
6. Fatigue
7. Stiff Joints
8. Sore Eyes
9. Wheeziness
10. Headaches
11. Upset Stomach
12. Sleep Difficulties
13. Dizziness
14. Loss of Strength

We are interested in your own personal views of how you now see your current injury.
Please indicate how much you agree or disagree with the following statements about your injury by ticking the appropriate box.
Answer by ticking one of five choices; Strongly Disagree, Disagree, Neither Agree nor Disagree, Agree, Strongly Agree

1. My injury will last a short time
2. My injury is likely to be permanent rather than temporary
3. My injury will last for a long time
4. This injury will pass quickly
5. I expect to have this injury for the rest of my life
6. My injury is a serious condition
7. My injury has major consequences on my life
8. My injury does not have much effect on my life
9. My injury strongly affects the way others see me
10. My injury has serious financial consequences
11. My injury causes difficulties for those who are close to me
12. There is a lot which I can do to control my symptoms
13. What I do can determine whether my injury gets better or worse
14. The course of my injury depends on me
15. Nothing I do will affect my injury
16. I have the power to influence my injury
17. My actions will have no effect on the outcome of my injury
18. My injury will improve in time
19. There is very little that can be done to improve my injury
20. My treatment will be effective in curing my injury
21. The negative effects of my injury can be prevented (avoided) by my treatment
22. My treatment can control my injury
23. There is nothing which can help my condition
24. The symptoms of my condition are puzzling to me
25. My injury is a mystery to me
26. I don’t understand my injury
27. My injury doesn’t make any sense to me

28. I have a clear picture or understanding of my condition
29. The symptoms of my injury change a great deal from day to day
30. My symptoms come and go in cycles
31. My injury is very unpredictable
32. I go through cycles in which my injury gets better and worse.
33. I get depressed when I think about my injury
34. When I think about my injury I get upset
35. My injury makes me feel angry
36. My injury does not worry me
37. Having this injury makes me feel anxious
38. My injury makes me feel afraid

CAUSES OF MY INJURY

We are interested in what you consider may have been the cause of your injury. As people are very different, there is no correct answer for this question. We are most interested in your own views about the factors that caused your injury rather than what others including doctors or family may have suggested to you. Below is a list of possible causes for your injury. Please indicate how much you agree or disagree that they were causes for you by ticking the appropriate box.

Answer by ticking one of five choices; Strongly Disagree, Disagree, Neither Agree nor Disagree, Agree, Strongly Agree

1. Stress or worry
2. Hereditary - it runs in my family
3. A Germ or virus
4. Diet or eating habits
5. Chance or bad luck
6. Poor medical care in my past
7. Pollution in the environment
8. My own behaviour
9. My mental attitude e.g. thinking about life negatively
10. Family problems or worries caused my injury
11. Overwork
12. My emotional state e.g. feeling down, lonely, anxious, empty
13. Ageing
14. Alcohol
15. Smoking
16. Accident or injury
17. My personality
18. Altered immunity

In the table below, please list in rank-order the three most important factors that you now believe caused YOUR injury. You may use any of the items from the box above, or you may have additional ideas of your own.

The most important causes for me:

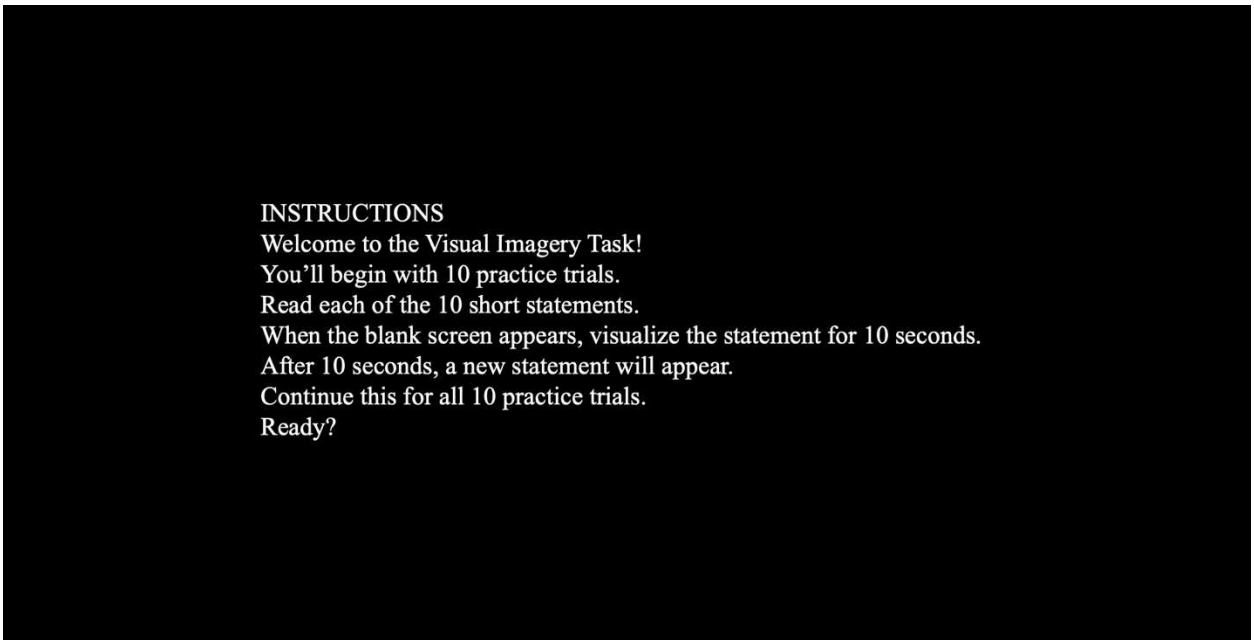
1. _____
2. _____
3. _____

Appendix B

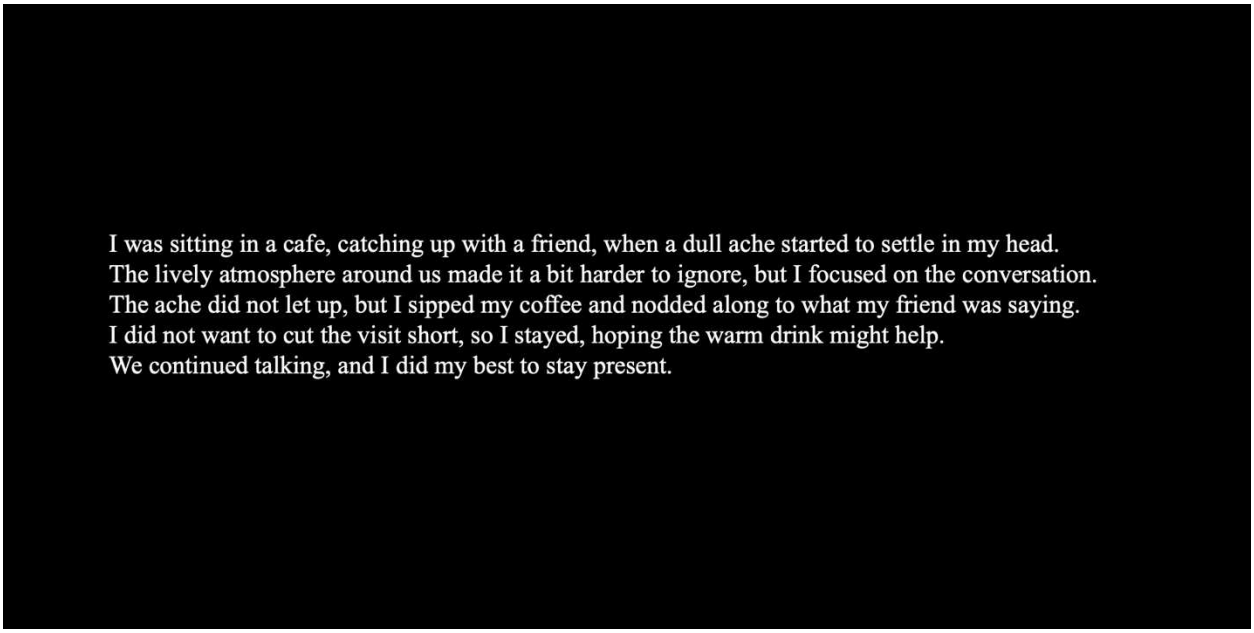
Visual Imagery Task

Task Stimuli

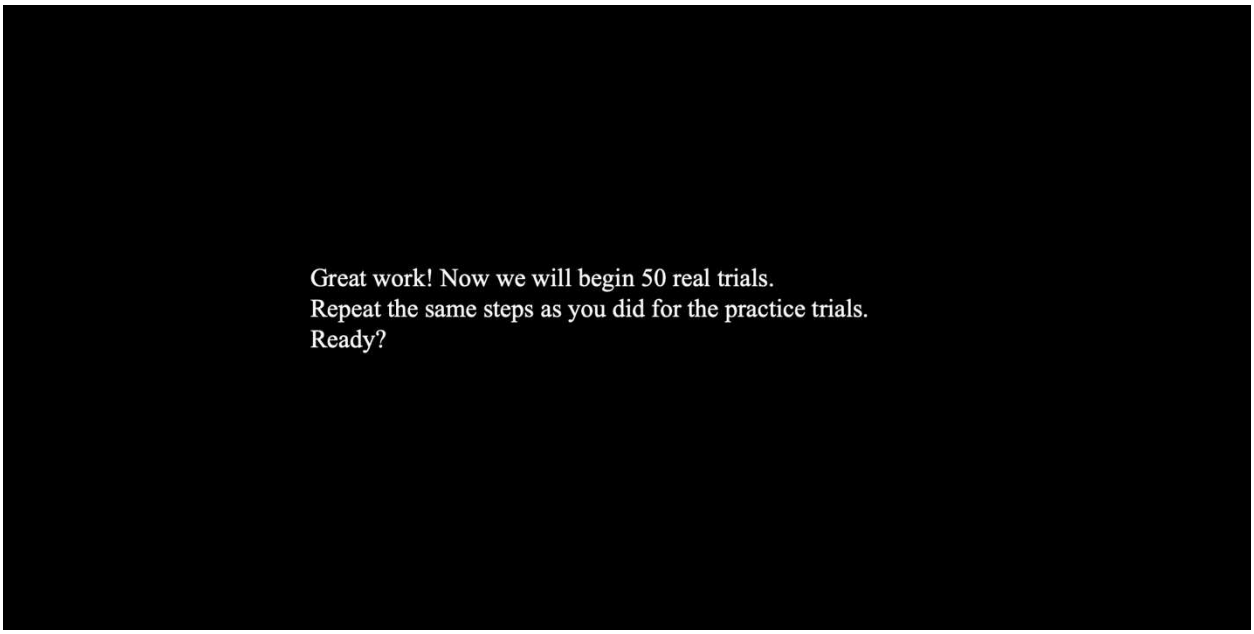
Instructions for visual imagery task:



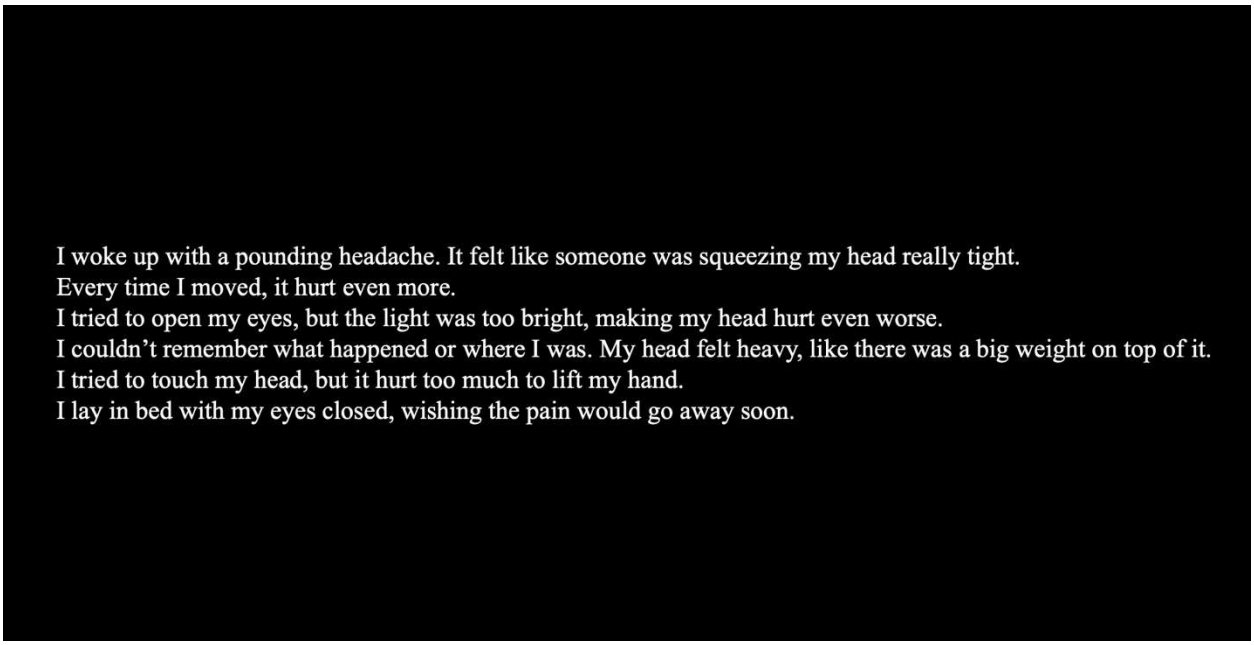
Practice Trial:



Task Instructions (After Practice Trials):



Real Trial:



Visual Imagery Task Instructions (read by researchers):

During the task, you'll be asked to read several short paragraphs that describe a scenario of experiencing pain following a brain injury. You'll have 15 seconds to read the paragraph. After the 15 seconds, a blank screen will appear. This is your cue to begin visualizing what you just

read. Don’t worry if you’re unable to finish reading before the screen switches. You are not being timed on how fast you read. You have 10 seconds to visualize what you read and then another paragraph will appear. You will repeat these steps for 10 practice trials and 50 real trials. You will receive these same instructions again on screen when the task begins. Do you have any questions?

Appendix C

EEG Cap Layout with Electrodes of Interest