The Effect of Diagnostic Terminology on Cognitive, Emotional, and Somatic Outcomes Following Mild Traumatic Brain Injury

by

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Abstract

Although used interchangeably to represent similar acute injury characteristics, mild traumatic brain injury (mTBI) and concussion may result in different functional outcomes. This investigation sought to determine whether varying diagnostic terminology resulted in differences in perceived cognitive, emotional, somatic symptom sequelae, and expected recovery time. A total of 108 university students were randomly assigned to one of three conditions: concussion (n=31), mTBI (n=44), or undiagnosed injury (n=33), and were instructed to simulate on a battery of cognitive, emotional, and somatic measures. There were no differences between the mTBI and concussion groups on cognitive ($d=.09$), emotional ($d=.09$), and somatic ($d=.09$) variables; however, the mTBI group perceived greater cognitive ($d=.15$), emotional ($d=.33$), and somatic ($d=.28$) consequences compared to the no diagnosis group. These findings provide preliminary support for the potential iatrogenic effects that may arise as a result of providing participants with a list of common brain injury symptoms.
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1 Introduction

Traumatic brain injury (TBI) results from blunt force trauma to the head, and is a leading cause of death and disability (National Center for Injury Prevention and Control, 2003). In Ontario, TBI is estimated to occur at a rate of 623 out of 100,000 individuals per annum (Colantonio et al., 2010). Although the majority (75-80%) of TBI’s are classified as mild in severity (Langlois et al., 2003), this subgroup is, arguably, the least well understood (Ruff & Weyer Jamora, 2009; Shaw, 2002). One explanation for this lack of understanding pertains to that of divergent methodological approaches whereby the absence of consistent terminology and definition across studies is most evident. That is, the terms mild TBI (mTBI) and concussion are often used interchangeably in the literature, sharing diagnostic criteria, acute injury characteristics, symptomatic presentation, and persisting neurocognitive impairments (Bigler, 2008; DeMatteo et al., 2010; Hoge, Goldberg, & Castro, 2009; Iverson & Lange, 2011; Jeter et al., 2013; King, Brughelli, Hume, & Gissane, 2014; Ruff, Iverson, Barth, Bush, & Broshek, 2009; Sharp & Jenkins, 2015; Sullivan, Edmed, & Kempe, 2014; Weber & Edwards, 2010; West & Marion, 2014).

A conceptual definition for mTBI was initially put forth by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (ACRM) in 1993 (American Congress of Rehabilitation Medicine, 1993). Despite the ACRM’s attempt to conceptualize mTBI, discrepancies in diagnostic criteria (e.g., in loss of consciousness, posttraumatic amnesia, and Glasgow Coma Scale (GCS) scale scores) continued to exist among research studies.
Noticing this inconsistency, the World Health Organization (WHO) Collaborative Center Task Force on Mild Traumatic Brain Injury advanced the definition initially created by the ACRM for the purposes of creating a standardized operational definition. This definition reads,

“MTBI is an acute brain injury resulting from mechanical energy to the head from external physical forces. Operational criteria for clinical identification include: (i) 1 or more of the following: confusion or disorientation, loss of consciousness for 30 minutes or less, post-traumatic amnesia for less than 24 hours, and/or other transient neurological abnormalities such as focal signs, seizure, and intracranial lesion not requiring surgery; (ii) Glasgow Coma Scale score of 13-15 after 30 minutes post-injury or later upon presentation for healthcare. These manifestations of MTBI must not be due to drugs, alcohol, medications, caused by other injuries or treatment for other injuries (e.g. systemic injuries, facial injuries, or intubation), caused by other problems (e.g. psychological trauma, language barrier or coexisting medical conditions) or caused by penetrating craniocerebral injury” (Carroll, Cassidy, Holm, Kraus, & Coronado, 2004, p. 115).

It must be noted that although this definition is accepted by most neuropsychologists (Ruff et al., 2009), it has limitations. While the upper boundaries of mTBI are relatively clear, the minimum thresholds are ambiguous, and create challenges relating to specificity. According to this standardized definition, an individual can experience no loss of consciousness, and no posttraumatic amnesia, but can still meet the criteria for mTBI. It is unclear whether the terms mTBI and concussion are considered as the same or separate entities.
The definition of concussion is vague, and falls under the umbrella term for mTBI. According to West and Marion (2014), a concussion is “a traumatically, or biomechanically, induced alteration of brain function. Emphasis is placed on a pathophysiological process, or functional disruption, as opposed to an anatomic, structural, or tissue injury” (p. 166). Though some researchers (e.g., Harmon et al., 2013; Mccrory et al., 2013) posit that concussion is a subset of mTBI on the less severe end of the spectrum, there are no clear boundaries for when an mTBI would fall outside of the concussion diagnosis. In other words, it is unknown where concussion ends and where mTBI begins. The American Medical Society for Sport Medicine states that “while all concussions are mTBI’s, not all mTBI’s are concussions” (Harmon et al., 2013, p. 3). Since the diagnostic criteria do not fully distinguish an mTBI from a concussion, researchers began investigating the effects of the brain injury (e.g., symptom presentation, pathophysiology) as possible avenues for distinction. Thus, the focus shifted from determining whether or not a concussion or mTBI occurred, to exploring the patterns of symptom presentations and persisting neurocognitive impairments.

The similarity of symptom presentation following mTBI and concussion provide support for the interchangeable use of the two terms. The common symptoms associated with each condition are mostly subjective and non-specific, including headaches, fatigue, memory impairment, difficulty concentrating, slowed responses, irritability, and depression. Not only are these symptoms nonspecific to mTBI and concussion, but they also have significant overlap with a myriad of disorders, including depression, sleep dysfunction, chronic pain, and somatoform disorders and importantly, those who have never sustained a mTBI or concussion (Chan, 2001; Iverson & Lange, 2003; Wang, Chan, & Deng, 2006; Zakzanis & Yeung, 2011). In addition, the literature suggests that neurocognitive impairment following
both mTBI and concussion is evident across all cognitive domains, including delayed memory recall, verbal fluency, executive functioning, attention, and concentration (Belanger, Curtiss, Demery, Lebowitz, & Vanderploeg, 2005; Binder, 1997; Hartikainen et al., 2010; Iverson, 2005; King et al., 2014). Because of the vague defining criteria, non-specific symptomatology, and indistinct neurocognitive impairment, the diagnosis of both mTBI and concussion largely rely on acute injury characteristics, as defined by Glasgow Coma Scale score, loss of consciousness, posttraumatic amnesia, and altered mental status, not on objective measures such as symptom checklists and standardized test measures administered post facto.

If mTBI and concussion are considered as the same entities in that they are defined by acute injury characteristics, one would expect functional outcome to be similar across injuries. Yet, the literature suggests that the functional outcome of these two entities is vastly different. This is where the mTBI and concussion literatures are divided; while most individuals diagnosed with mTBI are able to resume activities within 30-90 days following mTBI (van der Naalt, van Zomeren, Sluiter, & Minderhoud, 1999; Wäljas et al., 2014), those diagnosed with concussion resume activities within 7-25 days (Macciocchi, Barth, Alves, Rimel, & Jane, 1996; Mainwaring, Hutchison, Bisschop, Comper, & Richards, 2010; West & Marion, 2014). Accordingly, in the context of identical acute injury characteristics, problematic as they may be to reliably garner, there exists significant discrepancy in recovery following mTBI and concussion coupled with the absence of any physiological/ “organic” explanation.
1.1 Physiological Frameworks Related to Acute Symptomatology

Historically, the leading theoretical approaches to understanding mild brain injury were physiological (McCrory & Berkovic, 2001; Shaw, 2002). However, these physiological frameworks have been challenged by several findings, including the persistence of symptoms after physiological deficits have restored, the variable prognosis of persistent symptoms after equivalent severity of head injury, and the presence of symptoms in those without any physiological deficits (Jacobson, 1995). Thus, though ostensibly a straightforward form of brain trauma, mild brain injury is complicated by this lack of explanation for persisting symptoms at the physiological level. While these physiological factors are involved in the initial effects of mild brain injury, their role lessens over time, and other factors are needed to explain symptoms that continue to exist following the post-acute phase of recovery. For this reason, researchers began deviating from theoretical models that emphasize physiological processes, and moving towards theoretical models that highlight functional and psychogenic processes. Today, identifying factors, which predict who or why individuals are failing to recover is an ongoing challenge, and more researchers are adopting a psychosocial framework to study mild brain injury. It is largely through this theoretical lens that persistent symptoms are being explored (Bigler, 2008; Kempe, Sullivan, & Edmed, 2013; Mulhern & McMillan, 2006; Snell, Surgenor, Hay-Smith, Williman, & Siegert, 2015; Whittaker, Kemp, & House, 2007).

1.2 Psychosocial Frameworks Related to Persisting Symptomatology

Extending beyond physiological frameworks, several psychosocial frameworks can be used to explain persisting symptomatology following mild brain injury. These include Weiner’s (1985) theory of attribution, Bandura’s (1986) theory of self-efficacy, Mittenberg’s
(1992) expectation as etiology theory, and Steele and Aronson’s (1995) theory of stereotype threat, with an extension to diagnosis threat (Gunstad & Suhr, 2002). When considered together, these theories explain how individuals may internalize negative expectations and use them to inform perceptions of their own abilities following mTBI.

1.2.1 Theory of Attribution and Theory of Self-Efficacy

Weiner’s theory of attribution is one branch of research that has explored the idea of expectations informing abilities, and therefore affecting test performance (Weiner, 1985). This theory suggests that expectations are influenced by three components: a perceived locus of causality, stability, and controllability. Locus of causality refers to the perception that the cause of an event is internal (i.e., individual characteristics) or external (i.e., environmental factors). Stability refers to whether the outcome of an event remains steady (i.e., is difficult to change). Controllability refers to causes that can either be generalizations across situations (i.e., global; apply to many domains of an individual’s life), or specific to the event. Weiner suggests that negative expectations, and in turn unsuccessful performance, are more likely to occur in individuals making internal, stable, and global attributions for failure.

It is possible that interactions with healthcare professionals in the early stages of the injury can influence these three attribution components, which may ultimately contribute to persisting symptoms following mild brain injury, and impede recovery. Following onset, individuals that sustain brain injuries are often exposed to a variety of healthcare professionals, including physicians, psychiatrists, and others with invested interest in outcome including family members and friends, along with personal injury lawyers and triers of fact. Each of these individuals may ask distinct questions about the patients’ symptoms and have varying agendas and interpretations of the reported symptoms. Leading questions
and conflicting messages can cause the individual to be unsure of what they are truly experiencing, leading to *iatrogenic reactions*. Iatrogenic reactions can be defined as symptoms or injuries caused by medical attention and can arise during the post-acute period of recovery. This is pertinent to the development of persistent symptoms, as the symptoms are already ambiguous and non-specific to head injury. Wood (2004) suggests that the first point of contact following hospital discharge is usually the most critical in terms of developing persisting symptomatology. Commonly used terms such as ‘brain damage’ or ‘diffuse axonal injury’ may reinforce the patients’ perception that symptoms are permanent and out of their control. Similarly, conferring a diagnostic label gives legitimacy to the patients’ symptoms, providing entry into the sick role and stability in attributions (Bender & Matusewicz, 2013). Thus, the persistent questions and conflicting messages of various healthcare professionals can cause individuals to make internal, stable, and global attributions.

Bandura’s self-efficacy theory is another branch of research that has explored the idea of expectations informing abilities (Bandura, 1986). Bandura argues that an individuals’ performance on a task partly depends on their beliefs about how well they will be able to organize their knowledge, skills, and cognitive abilities, and how much effort they will put forth. As such, individuals with high self-efficacy are likely to attribute failure to a lack of effort, and have positive beliefs about their abilities. In attribution theory, these individuals would likely be making internal and controllable judgments about their test performance. Alternatively, individuals with low self-efficacy would attribute failure to a lack of ability, leading these individuals to approach situations anxiously, and this experience of anxiety may lower their expectation that they will be able to perform well. Self-efficacy is a domain-
specific construct. That is, individuals may have high levels of self-efficacy for one area (e.g., math ability), and low self-efficacy for other areas (e.g., memory functioning). Individuals who have sustained an mTBI may suffer from low self-efficacy in certain domains. For example, Kit and colleagues found that memory self-efficacy was lower in those with a history of mTBI than in a control group (Kit, Mateer, & Graves, 2007). These findings suggest that attribution and self-efficacy theories may add to our current understanding of the persistent symptoms following mTBI.

1.2.2 Expectation as Etiology

Mittenberg’s expectation as etiology theory has strong ties to both the attribution and self-efficacy models, and was developed to explain why post concussive symptoms (PCS) exist following head injury in the absence of impairment on neuropsychological examination (Mittenberg, DiGiulio, Perrin, & Bass, 1992). Mittenberg, DiGiulio, Perrin, and Bass (1992) found 67% shared variance between symptoms expected on an everyday basis by controls and those experienced by individuals with PCS. Yet, those with head injury reported pre-injury symptoms at a lower rate than that currently experienced by healthy controls, suggesting a re-attribution of these symptoms to the trauma. Mittenberg and his colleagues hypothesized that this re-attribution resulted in a selective attention to symptoms that are congruent with the trauma and, subsequently, anxiety regarding their significance. Overall, this research suggests that individuals share a set of negative beliefs about the effects of mTBI.

This theory has been supported by several studies finding that negative expectations are associated with brain injury in the general population (McLellan, Bishop, & McKinlay, 2010; Mulhern & McMillan, 2006), athletes (Ferguson, Mittenberg, Barone, & Schneider,
1999; Weber & Edwards, 2010), mTBI patients (Whittaker et al., 2007), pediatric clinicians (DeMatteo et al., 2010), parents (Gordon, Dooley, Fitzpatrick, Wren, & Wood, 2010), and university staff and students (Sullivan et al., 2014). These studies support the idea that negative expectations could have a role in poor recovery following mTBI. For example, Whittaker et al. (2007) found that the perceived long-term consequences in the post-acute stages of the injury were the best predictors of actual symptoms 3 months after the injury. As such, Whittaker et al. (2007) conclude that researchers and clinicians should consider patients’ perceptions of their head injury when assessing risk of developing persisting symptoms. Additionally, negative expectations have been found to alter both psychological outcomes (Mulhern & McMillan, 2006) and neuropsychological test performance (Suhr & Gunstad, 2002, 2005). Collectively, these studies suggest that expectations indirectly play a causal role in recovery following mTBI, with the perception of long-term consequences ultimately leading to poorer recovery.

The effect of expectations on symptomatology is widely supported in the medical literature and has been demonstrated to cause symptoms related to drug allergy (Lombardi, Gargioni, Canonica, & Passalacqua, 2008), chronic back pain (Pfingsten et al., 2001), cancer-related fatigue (De La Cruz, Hui, Parsons, & Bruera, 2010), headaches (Stovner, Oftedal, Straume, & Johnsson, 2008), lactose intolerance (Vernia, Di Camillo, Foglietta, Avallone, & De Carolis, 2010), pseudoseizures (Lancman, Asconapé, Craven, Howard, & Penry, 1994), and coronary heart disease (Cocco, 2009). In medicine, this causation of illness by expectation of illness has been termed the nocebo phenomenon. Contradictory to placebo, where a positive expectation results in a reduction of symptoms, nocebo is defined as a harmful effect on health caused by psychological or psychosomatic factors (i.e., a negative
expectation has a negative outcome) (Hahn, 1997). In the context of mild brain injury, nocebo effects might explain why some patients develop persistent symptoms because negative symptom expectations can impede recovery (Vanderploeg, Belanger, & Kaufmann, 2014). To summarize, the literature suggests that while physiological factors contribute to acute symptoms, attributions and expectations may explain the persisting symptomatology following mild brain injury.

1.2.3 Stereotype Threat and Diagnosis Threat
A third framework that has explored the idea of expectations informing abilities is stereotype threat. Stereotype threat is one of the most comprehensively studied phenomena in social psychology, suggesting that members of a social group (e.g., African Americans, women) “must deal with the possibility of being judged or treated stereotypically, or of doing something that would confirm the stereotype” (Steele & Aronson, 1995, p.401). For example, when African American participants were told that African Americans performed worse on intelligence tests, the participants standardized test performance dramatically decreased (Steele, 1997; Steele & Aronson, 1995). Since Steele and Aronson’s seminal paper, empirical evidence supporting stereotype threat has steadily grown, and has been extended to other stigmatized social groups, including cognitive testing in individuals from low socioeconomic backgrounds, mathematical skills in women, and memory in older adults (Nguyen & Ryan, 2008).

Diagnosis threat is an extension of stereotype threat, specific to head injury. Suhr and Gunstad (2002) refer to diagnosis threat as a negative impact on cognitive functioning as a result of calling attention to a personal history of head injury and its potential effects on cognitive outcome. Diagnosis threat has been found to impact cognitive performance
following both concussion (Suhr & Gunstad, 2002, 2005) and PCS (Ozen & Fernandes, 2011). Consistent with attribution and expectation effects, these findings suggest that the information provided to individuals following mTBI may influence their experience of symptoms that they then attribute to the injury itself (Suhr & Gunstad, 2002).

Overall, based on the literature reviewed above, it is conceivable that the theories of attribution, self-efficacy, expectations of etiology, and stereotype threat are relevant to a mild brain injured population, and may be contributing to persistent symptomatology.

1.3 Diagnostic Terminology

To date, diagnostic terminology has not been recognized as a part of the psychosocial frameworks. However, it is possible that diagnostic terminology may play a role in these psychosocial theories. At present, multiple terms are used interchangeably to describe mild brain injury, despite the ambiguity this creates for researchers and clinicians. There is a lack of consensus regarding appropriateness of diagnostic terminology, with some researchers arguing for the adoption of the term concussion, and others advocating for the use of the term mTBI. Those that argue to discard mTBI suggest that this term may reinforce illness perception, as individuals given this diagnosis likely attribute their symptoms to the biological consequences of brain injury (Wood, 2004). Instead, concussion is generally more easily understood and less alarming, making it less likely to have an adverse psychological effect on the individual when they learn about their injury (DeMatteo et al., 2010; King et al., 2014). On the other hand, the authors that advocate for the term mTBI do so because concussion does not have a consistent definition, leading to an increase in diagnostic confusion (Mccrory et al., 2013; Sharp & Jenkins, 2015). In addition, the term mTBI already falls under a standardized classification system (i.e., mild, moderate and severe),
characterized by GCS score, posttraumatic amnesia duration and loss of consciousness. Furthermore, using the term concussion might increase the likelihood that recovery advice is ignored (Weber & Edwards, 2010). In summary, concussion seems to promote health and recovery, and mTBI seems to be associated with an increase in perceived impairment.

Several studies have examined whether this variation in diagnostic terminology can influence the perception of long-term consequences following mild brain injury. In a simulation study using contact-sport players, Edmed and Sullivan (2015) found no differences between perceptions of symptoms between mTBI, concussion or an unlabeled injury. Kempe et al. (2013) found individuals who received advice regarding concussion expected more PCS symptoms overall, particularly in the domains of cognition and sensory symptoms. However, no differences emerged in terms of recovery timeline and injury consequences. In contrast, Weber and Edwards (2010) found university athletes expect worse outcomes following mTBI versus concussion. In addition, DeMatteo et al. (2010) found that pediatric clinicians prefer to retire the term mTBI because it is more alarming. Finally, in a simulation study using university staff and students, Sullivan et al. (2014) concluded that an unlabeled injury or an injury that is labeled as mTBI is worse than an injury that is labeled as concussion. Taken together, the effects observed across studies are not uniform, with some studies suggest that we adopt the term concussion, while others suggesting the term mTBI be adopted, and still others argue that terminology may not be a potent influence on a myriad of outcomes (see Table 1). The reasons for this variability in the literature are not well understood. As such, the effect of these diagnostic terminologies must be explored further.

To the best of my knowledge, only two other studies have investigated the effect of diagnostic terminology on persistent symptoms following mTBI using healthy university
students who received a simulation design (Kempe et al., 2013; Sullivan et al., 2014).

However, these studies examined expected PCS using subjective self-report symptom checklists. The goal of this study was to extend these findings to the perception of cognitive, emotional, and somatic consequences, as well as expected recovery time, using objective performance based measures in conjunction with self-report measures, while maintaining the high level of experimental control described in these studies.
<table>
<thead>
<tr>
<th>Study</th>
<th>Sample</th>
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<th>Outcome Measures</th>
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<tr>
<td>Weber &amp; Edwards (2010)</td>
<td>- N=224 - University student-athletes</td>
<td>- Concussion - MTBI - Minor HI</td>
<td>- 29-item questionnaire with variation in diagnostic terminology</td>
<td>- 29-item questionnaire - Hospital Anxiety and Depression Scales - Positive and Negative Affectivity Scale - RPQ</td>
<td>- MTBI conceptualized as more negative and least familiar - MTBI was expected to be longer lasting and might leave the athlete with depressive symptoms and learning difficulties - Groups did not differ on actual symptom reporting</td>
</tr>
<tr>
<td>DeMatteo et al. (2010)</td>
<td>- N=268 - Children with HI</td>
<td>- Mild concussion - Moderate or severe injury</td>
<td>- N/A</td>
<td>- Clinical data extracted from medical chart (e.g., hospital discharge, return to school, GCS, computed tomography scan results)</td>
<td>- Concussion was predictive of earlier hospital discharge - Normal computed tomography scan was predictive of receiving a concussion diagnosis</td>
</tr>
<tr>
<td>Sullivan, Edmed &amp; Kempe (2014)</td>
<td>- N=204 - Healthy university staff and students¹</td>
<td>- Concussion - MTBI - Minor HI - No diagnosis</td>
<td>- MVA vignette with variation in diagnostic terminology</td>
<td>- Neurobehavioral Symptom Inventory - PTSD Checklist-Civilian - Mild Brain Injury Atypical Symptoms Scale - Illness Perception Questionnaire - Revised - Perceived desirability item</td>
<td>- MTBI and no diagnoses expected worse PTSD symptoms, were higher on undesirability, and had greater symptoms than minor HI and concussion groups</td>
</tr>
<tr>
<td>Kempe, Sullivan &amp; Edmed (2013)</td>
<td>- N=135 - Healthy university staff and students¹</td>
<td>- Concussion - MTBI</td>
<td>- MVA vignette with variation in diagnostic terminology - 2-page discharge advice sheet with variation in diagnostic terminology</td>
<td>- Neurobehavioral Symptom Inventory - PTSD Checklist-Civilian - Mild Brain Injury Atypical Symptoms Scale - Illness Perception Questionnaire - Revised</td>
<td>- No differences in expected recovery timeline and injury consequences - Concussion group expected greater PCS symptoms on cognitive, sensory and affective domains - Most outcomes were not influenced by the change in diagnostic terminology</td>
</tr>
<tr>
<td>Edmed &amp; Sullivan (2015)</td>
<td>- N=122 - University student-athletes (contact sports only)</td>
<td>- Concussion - MTBI - No diagnosis</td>
<td>- Sports-related vignette with variation in diagnostic terminology</td>
<td>- Neurobehavioral Symptom Inventory - PTSD Checklist-Civilian - Illness Perception Questionnaire - Revised - Perceived desirability item</td>
<td>- No differences in injury perceptions - No differences in symptom disturbance between mTBI and concussion groups - Concussion and mTBI expected greater symptom disturbance than no diagnosis</td>
</tr>
</tbody>
</table>

*Note.* ¹No history of head injury or psychiatric/neurological disorder. PCS= Post Concussive Symptoms; PTSD=Post-Traumatic Stress Disorder; RPQ=Rivermead Post Concussion Symptoms Questionnaire.
1.4 Current Study

I sought to determine if an effect of diagnostic terminology is present on several key outcomes that have not been studied in past research. To date, the existing literature has examined a limited number of outcome variables (see Table 1). Accordingly, I examined the effect of varied diagnostic terminology (mTBI, concussion, or no diagnosis) on the perception of cognitive, emotional, and somatic consequences, as well as expected recovery time.

1.4.1 Hypotheses

The literature suggests that significant differences exist between individuals diagnosed with mTBI and those diagnosed with concussion in terms of resumption of daily living activities. Since there is no clear physiological explanation to distinguish concussion from mTBI, psychosocial factors must be considered. Thus, this study sought to determine whether diagnostic terminology influences the perceived cognitive, emotional and somatic consequences, as well as expected recovery times, of mild brain injury. Consistent with the literature, I believed that mTBI is viewed as being more severe than concussion, and that this negative expectation may help explain the longer recovery times associated with mTBI. Thus, it was hypothesized that the persisting deficits associated with mTBI are influenced by diagnostic terminology, with individuals perceiving greater cognitive, emotional and somatic consequences when diagnosed with mTBI versus concussion or no diagnosis.

I also examined whether diagnostic terminology influences functional ability at the global level (e.g., cognitive functioning), or whether it is a domain-specific construct (e.g., attention, executive functioning, spatial). In other words, does diagnostic terminology influence the perception of cognitive difficulties differently between cognitive domains? Or are all cognitive domains expected to be influenced by diagnostic terminology in a similar manner? In the
literature, attention and memory difficulties are expected at higher levels than executive functioning, language, and spatial difficulties (Gunstad & Suhr, 2002; Mittenberg et al., 1992; Sullivan & Edmed, 2012; Sullivan et al., 2014). Thus, I hypothesized that the mTBI group would expect the memory and attention domains to be more greatly impaired than the executive functioning, language, and spatial domains. In terms of emotional functioning, the literature does not clearly differentiate between the expectation of depressive and anxious symptomatology following mTBI (Gunstad & Suhr, 2001; Mittenberg et al., 1992). Consequently, I hypothesized that depressive and anxious symptomatology would be endorsed in a similar manner. In the context of somatic symptomatology, headaches, fatigue, and dizziness are expected at higher levels than other somatic symptoms, including blurred vision, light sensitivity, and restlessness (Gunstad & Suhr, 2002; Mittenberg et al., 1992; Sullivan & Edmed, 2012; Sullivan et al., 2014; Weber & Edwards, 2010). Therefore, I hypothesized that headaches, fatigue, and dizziness would be the most frequently endorsed somatic symptoms. Finally, the literature suggests that those diagnosed with an mTBI have slower recovery times when compared to those diagnosed with a concussion. Thus, I hypothesized that those in the mTBI group would expect longer recovery times than the concussion and no diagnosis groups.

2 Method

2.1 Participants

A power analysis was conducted which indicated that the minimum required sample for the proposed analysis is 99 (33 participants per group), with an anticipated $d = .50$, $\alpha = .05$, and power $= .80$. Participants were undergraduate students recruited through the SONA system at the University of Toronto Scarborough. Those that consented were randomly allocated to one of three conditions: no diagnosis ($n=51$), concussion, ($n=51$), or mTBI ($n=52$). Several exclusion
criteria were applied to the data prior to analysis. Of the total 154 participants, two participants had incomplete data and were excluded. Participants were also excluded if they endorsed at least one of the following items: (a) history of previous head injury; (b) history of previous motor vehicle accident; and (c) history of neuropsychological or psychological assessment. These criteria were included to limit confounding variables (e.g., if they had previously sustained a head injury and were placed in the control group). Results indicated that 28 individuals endorsed a history of previous head injury; 5 individuals endorsed a history of previous motor vehicle accident; and 20 individuals endorsed a history of neuropsychological or psychological assessment. In addition, the use of the Post-Experimental Questionnaire (described below) assessed participant comprehension of the simulation task. Results indicated that six individuals were excluded for not understanding the instructions provided in the study or for forgetting to simulate the accident, and 14 individuals were excluded because their qualitative responses indicated that they had not understood the task instructions (e.g., “no idea”, “to remember a lot of stuff”, “to test my attention”). Furthermore, the use of the Test of Memory Malingering (described below) was employed to measure effort level and hence, the credibility of test performance. An additional three individuals were excluded for performing below the recommended cutoff score on this measure. A selection of our sample is detailed in Figure 1. In total, 108 participants were retained for analysis, with 33 participants in the no diagnosis group, 31 participants in the concussion group, and 44 participants in the mTBI group.
Figure 1. Participant selection by group

*Note. MTBI = mild traumatic brain injury; HI = head injury; MVA = motor vehicle accident; Prior Ax = prior psychological or neuropsychological assessment; PEQ = Post-Experimental Questionnaire; TOMM = Test of Memory Malingering. The total number excluded from each group does not equal the sum of those who failed the exclusion criteria because some individuals failed multiple criteria.

2.2 Measures

2.2.1 Demographics Questionnaire

A demographics questionnaire was used to assess sample demographics and to verify that participants met the inclusion criteria mentioned above. This questionnaire included items related to age, sex, handedness, language, ethnicity, and relevant history (e.g., have you ever suffered a head injury).
2.2.2 Simulation Scenario

Participants read a vignette that depicted an injury that was sustained during a motor vehicle accident (see Appendix A). The injury described in the vignette is consistent with a diagnosis of an mTBI defined according to the WHO standards (Carroll et al., 2004) and a diagnosis of a concussion defined according to West & Marion (2014). In this study, the experimental groups (i.e., mTBI or concussion) had an additional sentence conveying the relevant diagnosis, and were given a list of common symptoms following mTBI/concussion to help simulate the scenario (e.g., frequent headaches, fatigue, memory impairment). The list of common symptoms were identical in the mTBI and concussion groups. The control group (i.e., undiagnosed injury) did not receive a diagnosis, nor did they receive a list of common symptoms.

2.2.3 Simulation Reminder

Throughout the study, participants received four simulation reminders to help them remember the purposes of the study (see Appendix B). For the experimental groups, the participant was reminded of their relevant diagnosis and common impairments. The control group was reminded that they were simulating a motor vehicle accident.

2.2.4 Assessment

The perceived consequences of mild brain injury were measured using the following tests. A performance validity measure was employed to ensure adequate effort was put forth by participants (see An, Zakzanis, & Joordens, 2012). In addition, a post-experimental questionnaire was administered to assess compliance with the vignette.
2.2.4.1 Cognitive Functioning

2.2.4.1.1 Neuropsychological Assessment Battery – Screening Module (NAB-S)

The Neuropsychological Assessment Battery-Screening Module (Stern & White, 2003) is a set of brief neuropsychological tests which measure the examinee’s functioning across various cognitive domains including attention, language, memory, spatial ability and executive functioning. This battery is composed of 15 brief tests, 14 of which contribute to the five index scores, which map onto the five cognitive domains. A total index score can also be interpreted.

2.2.4.2 Emotional Functioning

2.2.4.2.1 Beck Anxiety Inventory (BAI)

The Beck Anxiety Inventory (Beck, Steer, & Brown, 1993) is a 21-question measure used to determine the severity of an examinee’s anxiety. Items are in polytomous format to assist the examinee in rank ordering the severity of levels of anxiety. Items are ranked from 0 (no anxiety) to 3 (high levels of anxiety).

2.2.4.2.2 Beck Depression Inventory-II (BDI-II)

The Beck Depression Inventory-II (Beck, Steer, & Brown, 1996) is a 21-question measure used to determine the severity of an examinee’s depressive symptoms. Items are in polytomous format to assist the examinee in rank ordering the severity of levels of depression. Items are ranked from 0 (no depressive ideation) to 3 (high levels of depressive ideation). Beck et al. (1996) reported a two-factor solution to the data. These factors corresponded to somatic-affective and cognitive dimensions.
2.2.4.3 Somatic Symptomatology

2.2.4.3.1 Rivermead Post-Concussion Symptoms Questionnaire (RPQ)

The Rivermead Post-Concussion Symptoms Questionnaire (King, Crawford, Wenden, Moss, & Wade, 1995) is an 18-question measure used to determine the severity of symptoms following a TBI (e.g., headaches, fatigue, irritability). Items are in polytomous format to assist the examinee in rank ordering the severity of symptoms. Items 1-16 are ranked from 0 (symptom not experienced at all) to 4 (symptom is severely problematic). Items 17 and 18 are open ended responses, where the examinee is asked to list if they are experiencing any other difficulties.

2.2.5 Performance Validity

2.2.5.1 Test of Memory Malingering (TOMM)

The Test of Memory Malingering (Tombaugh, 1996) is a 50-item, forced choice recognition test of visual memory and is composed of two learning trials and a retention trial. After presentation of the 50 pictures during the first learning trial, participants are shown 50 two-choice recognition panels consisting of a previously presented picture and a distractor picture. Participants are asked to identify the picture previously presented. After each response, feedback is given. The participant is then shown the 50 pictures again in the second learning trial, and again asked to choose which picture he/she has seen before. In the retention trial, participants are not shown the 50 pictures again, but are simply asked to choose which pictures they have seen before in a forced choice design. To differentiate between participants who exert poor effort and those who exert normal effort, a cut off score of less than 45 was used for Trial 2 and the Retention Trial, as is suggested by Tombaugh (1996).
2.2.6 Post-Experimental Check

2.2.6.1 Post-Experimental Questionnaire

The Post-Experimental Questionnaire was administered to assess compliance with study instructions and vignette comprehension. This assessment is recommended for studies that use experimental designs where participants must follow instructions, such as this one (Oppenheimer, Meyvis, & Davidenko, 2009). The following questions were asked: (1) Did you understand the instructions provided in the study? (2) Did you forget to put yourself in the position of the character described in the accident while answering any of the symptom items? (3) Briefly explain what you were required to do in the experiment. Participants were excluded from this study if they answered ‘No’ to one of the first two questions, and were not able to accurately respond to the third question (i.e., did not recall the simulation or the instructions of the study).

2.3 Procedure

The Research Ethics Board at the University of Toronto granted ethical approval for this study (REB # 34633). Participants were recruited from the University of Toronto online research participant pool (SONA). A standard battery and procedure was utilized for this study. Participants completed the study in exchange for course credit. Consenting participants completed the demographics questionnaire, and were randomly assigned to one of the three experimental conditions. Participants both read and listened to one of three vignettes (mTBI, concussion, or no diagnosis). Participants in the mTBI and concussion conditions were presented with the vignette and the additional diagnostic sentence and common impairments. Participants in the no diagnosis condition were only presented with the vignette.
Participants were then administered the TOMM Trial 1, followed by the TOMM Trial 2, and a simulation reminder. Subsequently, participants were given the BAI, BDI-II, and RPQ. If participants failed Trial 2 (<45), they were administered the TOMM Retention Trial after the RPQ. Alternatively, if the participant received a score of above 45 on the TOMM Trial 2, they were deemed as having credible performance, and were not administered the TOMM Retention Trial. Subsequently, participants were given a simulation reminder, followed by administration of the NAB-S. Next, participants were given another simulation reminder. Upon completion of these instruments, participants were instructed to answer the following question: “How long do you think it will take you to return to your daily activities after this car accident?” Finally, participants were administered the Post-Experimental Questionnaire and were debriefed on the purposes of the study.

2.4 Analysis
A one-way analysis of variance (ANOVA) and three chi-squared analyses were undertaken to determine whether differences exist between groups on demographic variables. To determine if diagnostic terminology affects scores on cognitive, emotional and somatic symptom domains, I examined mean-level differences between groups for these domains. This included means, standard deviations, and tests of statistical significance with effect size estimates.

Preliminary analyses were conducted to ensure no violation of the normality assumption. If the normality assumption was violated (i.e., emotional variables), non-parametric analyses were run. If the normality assumption was not violated (i.e., cognitive and somatic variables), parametric analyses were run. To determine if diagnostic terminology affects expected recovery time, we examined mean-level differences for the three groups. Again, this included means, standard deviations, and tests of statistical significance with effect size estimates. Preliminary analyses
were conducted to ensure no violation of the normality assumption. This variable was found to be significantly non-normal. Therefore, non-parametric analyses were run. Significance was evaluated against a Bonferroni-corrected alpha level.

3 Results

3.1 Demographic Information

The demographic characteristics of our sample are shown in Table 2. Using a Bonferroni adjusted alpha level of .0125 per test (.05/4), results indicated that there were no significant differences between groups on age \( F(2,103)=1.125, p=.329 \), ethnicity \( \chi^2(2)=10.100, p=.039 \), sex \( \chi^2(2)=1.974, p=.373 \), or handedness \( \chi^2(4)=3.347, p=.501 \).

Table 2. Demographic characteristics by group

<table>
<thead>
<tr>
<th>Variable</th>
<th>No Diagnosis</th>
<th>Concussion</th>
<th>MTBI</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>18.88</td>
<td>19.17</td>
<td>18.39</td>
<td>.329</td>
</tr>
<tr>
<td>SD</td>
<td>1.58</td>
<td>3.80</td>
<td>1.02</td>
<td></td>
</tr>
<tr>
<td>N (%)</td>
<td>N (%)</td>
<td>N (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
<td>.039</td>
</tr>
<tr>
<td>South Asian</td>
<td>17 (53.1)</td>
<td>5 (17.2)</td>
<td>16 (37.2)</td>
<td></td>
</tr>
<tr>
<td>East and Southeast Asian</td>
<td>5 (15.6)</td>
<td>10 (34.5)</td>
<td>7 (16.3)</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>10 (31.1)</td>
<td>14 (48.3)</td>
<td>20 (46.5)</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
<td>.373</td>
</tr>
<tr>
<td>Female</td>
<td>21 (63.6)</td>
<td>22 (75.9)</td>
<td>36 (81.8)</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>10 (30.3)</td>
<td>7 (24.1)</td>
<td>8 (18.2)</td>
<td></td>
</tr>
<tr>
<td>Handedness</td>
<td></td>
<td></td>
<td></td>
<td>.501</td>
</tr>
<tr>
<td>Right</td>
<td>28 (84.8)</td>
<td>25 (86.2)</td>
<td>41 (93.2)</td>
<td></td>
</tr>
<tr>
<td>Left</td>
<td>4 (12.1)</td>
<td>4 (13.8)</td>
<td>3 (6.8)</td>
<td></td>
</tr>
<tr>
<td>Ambidextrous</td>
<td>1 (3.0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td></td>
</tr>
</tbody>
</table>
3.2 Effect of Diagnostic Terminology on Cognitive Functioning

Descriptive statistics for the perceived cognitive consequences of mild brain injury are shown in Table 3. This table illustrates the cognitive difficulties that participants expected after exposure to one of the three experimental conditions. To determine if there was a significant difference on the memory and attention cognitive domains between diagnostic terminology groups, a multivariate analysis of variance (MANOVA) was conducted using the attention and memory scores as dependent variables. The data were screened to determine suitability for parametric analyses and none of the assumptions were violated. Using Wilk’s Lambda, there was no significant effect of diagnostic terminology on the memory and attention cognitive domains, Wilk’s $\Lambda=0.933$, $F(4,176)=1.549$, $p=0.19$.

Table 3. Mean scores and standard deviations for cognitive variables in no diagnosis, concussion, and mTBI groups

<table>
<thead>
<tr>
<th></th>
<th>No Diagnosis</th>
<th></th>
<th>Concussion</th>
<th></th>
<th>MTBI</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Attention</td>
<td>88.52</td>
<td>19.02</td>
<td>84.78</td>
<td>17.79</td>
<td>85.38</td>
<td>19.34</td>
</tr>
<tr>
<td>Executive Functioning</td>
<td>84.28</td>
<td>18.60</td>
<td>88.63</td>
<td>18.29</td>
<td>89.03</td>
<td>17.60</td>
</tr>
<tr>
<td>Language</td>
<td>85.48</td>
<td>15.02</td>
<td>78.85</td>
<td>18.47</td>
<td>84.70</td>
<td>13.75</td>
</tr>
<tr>
<td>Memory</td>
<td>99.00</td>
<td>11.80</td>
<td>91.48</td>
<td>17.44</td>
<td>90.92</td>
<td>12.24</td>
</tr>
<tr>
<td>Spatial</td>
<td>92.34</td>
<td>17.25</td>
<td>91.70</td>
<td>16.94</td>
<td>94.00</td>
<td>18.03</td>
</tr>
<tr>
<td>NAB-S Total</td>
<td>85.61</td>
<td>13.62</td>
<td>81.59</td>
<td>15.73</td>
<td>82.39</td>
<td>20.18</td>
</tr>
</tbody>
</table>

*Note. A low score on the NAB-S indicates a greater level of impairment.

Cohen’s point estimate effect size $d$ was calculated to articulate the magnitude of difference between the three groups for each of the cognitive variables (Cohen, 1988). These are presented in Table 4. This table also includes the hypothetical overlap percentage (OL%) based on the amount of overlap in the distribution of test measure scores between samples (Zakzanis, 2001). This OL% is meant to reflect the degree of overlap in sample score dispersion associated
with Cohen’s $d$. Results suggest that worse scores were expected on the attention, language, memory, and spatial domains among those in the concussion and mTBI groups compared to the no diagnosis group, with Cohen’s $d$ effect sizes ranging from .04 to .67 ($M=0.15$). Cohen’s guidelines for interpretation indicate an effect size of 0.2 to be “small”, 0.5 to be “medium”, and 0.8 to be “large”. However, an effect size greater than 3.0 (OL%<5) is necessary for a variable to be interpreted as a clinical marker that reliably differentiates the two groups (Zakzanis, 1998, 2001). As seen in Table 4, the largest effect size between the no diagnosis and concussion groups was for the memory domain ($d=.51$, OL%=67). Between the no diagnosis and mTBI groups, the largest effect size was also for the memory domain ($d=.67$, OL%=59). Between the mTBI and concussion groups, the largest effect size was for the language domain ($d=.36$, OL%=76). At least one comparison in each domain generated larger effect sizes than did the NAB-S Total score, with the exception of the visuospatial domain.

Table 4. Effect sizes and score overlap percentage of cognitive variables by group comparison

<table>
<thead>
<tr>
<th></th>
<th>No diagnosis vs. concussion $^a$</th>
<th>No diagnosis vs. mTBI $^b$</th>
<th>Concussion vs. mTBI $^c$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$d$</td>
<td>OL%</td>
<td>$d$</td>
</tr>
<tr>
<td>Attention</td>
<td>.20</td>
<td>85</td>
<td>.17</td>
</tr>
<tr>
<td>Executive Functioning</td>
<td>.23</td>
<td>83</td>
<td>.26</td>
</tr>
<tr>
<td>Language</td>
<td>.39</td>
<td>73</td>
<td>.05</td>
</tr>
<tr>
<td>Memory</td>
<td>.51</td>
<td>67</td>
<td>.67</td>
</tr>
<tr>
<td>Spatial</td>
<td>.04</td>
<td>96</td>
<td>.09</td>
</tr>
<tr>
<td>NAB-S Total</td>
<td>.03</td>
<td>97</td>
<td>.19</td>
</tr>
<tr>
<td>Mean</td>
<td>0.16</td>
<td></td>
<td>0.15</td>
</tr>
</tbody>
</table>

3.3 Effect of Diagnostic terminology on Emotional Functioning

Descriptive statistics for the perceived emotional consequences of mild brain injury are shown in Table 5. This table illustrates the anxious and depressive symptoms that participants
expected after exposure to one of the three experimental conditions. To determine if there was a significant difference in severity of symptoms by group, a MANOVA was conducted using BAI and BDI-II Total scores as the dependent variables. The data was screened to determine suitability for parametric analyses. The homogeneity of covariance matrix was violated, thus Pillai’s trace was used. There was no significant effect of diagnostic terminology on anxious and depressive symptoms, $V=.046$, $F(4,206)=1.213$, $p=.306$.

Table 5. Mean scores and standard deviations for emotional variables in no diagnosis, concussion, and mTBI groups

<table>
<thead>
<tr>
<th></th>
<th>No Diagnosis</th>
<th>Concussion</th>
<th>MTBI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>BDI-II</td>
<td>17.30</td>
<td>11.90</td>
<td>23.10</td>
</tr>
</tbody>
</table>

*Note. A high score on both the BAI and the BDI-II indicate a greater level of impairment.

Cohen’s point estimate effect size $d$ was calculated to articulate the magnitude of difference between the three groups for both the depression and anxiety variables (see Table 6). The results suggest that compared to the no diagnosis group, participants in the concussion and mTBI groups expected higher levels of anxious and depressive symptoms, with Cohen’s $d$ effect sizes ranging from .21 to .45. The largest effect size between the no diagnosis and concussion groups was for depressive symptoms ($d=.45$, OL%=70). Between the no diagnosis and mTBI groups, the largest effect size was for depressive symptoms ($d=.44$, OL%=71). Between the mTBI and concussion groups, the largest effect size was for anxious symptoms ($d=.15$, OL%=89).

Table 6. Effect sizes and score overlap percentage of emotional variables by group comparison

<table>
<thead>
<tr>
<th></th>
<th>No diagnosis vs. concussion $^a$</th>
<th>No diagnosis vs. mTBI $^b$</th>
<th>Concussion vs. mTBI $^c$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$d$</td>
<td>OL%</td>
<td>$d$</td>
<td>OL%</td>
</tr>
</tbody>
</table>
Descriptive statistics for the perceived somatic consequences of mild brain injury are shown in Table 7. This table illustrates the somatic symptoms that participants expected after exposure to one of the three experimental conditions. Among the no diagnosis group, feeling frustrated, poor concentration, and fatigue were the most frequently endorsed somatic symptoms. On the other hand, among the concussion group, fatigue, poor concentration, and forgetfulness were the most frequently endorsed somatic symptoms. Finally, among the mTBI group, fatigue, poor concentration, and headaches were the most frequently endorsed somatic symptoms. These results suggest a similarity in expected somatic symptoms among the three groups.

To determine if there was a significant difference in somatic symptoms by group, a one-way ANOVA was conducted using RPQ Total as the dependent variable. The data was screened to determine suitability for parametric analyses, and none of the assumptions were violated. No significant differences exist between the diagnostic terminology groups on somatic symptoms, $F(2,103)=1.912, p=.153$.

### Table 7. Mean scores and standard deviations for somatic variables in no diagnosis, concussion, and mTBI groups

<table>
<thead>
<tr>
<th></th>
<th>No Diagnosis</th>
<th></th>
<th>Concussion</th>
<th></th>
<th>MTBI</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td>Headaches</td>
<td>2.06</td>
<td>1.14</td>
<td>2.66</td>
<td>1.11</td>
<td>2.50</td>
<td>1.13</td>
</tr>
<tr>
<td>Dizziness</td>
<td>1.91</td>
<td>1.16</td>
<td>2.14</td>
<td>1.25</td>
<td>2.25</td>
<td>1.28</td>
</tr>
<tr>
<td>Nausea/vomiting</td>
<td>1.24</td>
<td>1.30</td>
<td>1.50</td>
<td>1.11</td>
<td>1.58</td>
<td>1.48</td>
</tr>
<tr>
<td>Noise sensitivity</td>
<td>2.15</td>
<td>1.56</td>
<td>2.62</td>
<td>1.24</td>
<td>2.09</td>
<td>1.34</td>
</tr>
<tr>
<td></td>
<td>Group 1</td>
<td>Group 2</td>
<td>Group 3</td>
<td>Group 4</td>
<td>Group 5</td>
<td>Group 6</td>
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<td>---------------------</td>
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<td>---------</td>
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<td>---------</td>
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</tr>
<tr>
<td>Sleep disturbance</td>
<td>1.94</td>
<td>1.50</td>
<td>2.29</td>
<td>1.27</td>
<td>2.34</td>
<td>1.38</td>
</tr>
<tr>
<td>Fatigue</td>
<td>2.21</td>
<td>1.47</td>
<td>2.93</td>
<td>0.88</td>
<td>2.68</td>
<td>1.24</td>
</tr>
<tr>
<td>Irritability</td>
<td>1.82</td>
<td>1.29</td>
<td>2.34</td>
<td>1.17</td>
<td>2.14</td>
<td>1.32</td>
</tr>
<tr>
<td>Feeling depressed/tearful</td>
<td>1.85</td>
<td>1.37</td>
<td>2.34</td>
<td>1.20</td>
<td>2.50</td>
<td>1.25</td>
</tr>
<tr>
<td>Feeling frustrated</td>
<td>2.24</td>
<td>1.32</td>
<td>2.25</td>
<td>1.30</td>
<td>2.30</td>
<td>1.23</td>
</tr>
<tr>
<td>Forgetfulness</td>
<td>1.73</td>
<td>1.49</td>
<td>2.69</td>
<td>1.14</td>
<td>2.18</td>
<td>1.32</td>
</tr>
<tr>
<td>Poor concentration</td>
<td>2.21</td>
<td>1.43</td>
<td>2.93</td>
<td>1.13</td>
<td>2.55</td>
<td>1.17</td>
</tr>
<tr>
<td>Slowed cognition</td>
<td>1.91</td>
<td>1.46</td>
<td>2.59</td>
<td>1.24</td>
<td>2.41</td>
<td>1.28</td>
</tr>
<tr>
<td>Blurred vision</td>
<td>1.18</td>
<td>1.21</td>
<td>1.48</td>
<td>1.21</td>
<td>1.75</td>
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<tr>
<td>Light sensitivity</td>
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<td>1.34</td>
<td>1.72</td>
<td>1.16</td>
<td>1.84</td>
<td>1.43</td>
</tr>
<tr>
<td>Double vision</td>
<td>0.85</td>
<td>1.03</td>
<td>1.04</td>
<td>1.00</td>
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</tr>
<tr>
<td>Restlessness</td>
<td>1.76</td>
<td>1.32</td>
<td>2.28</td>
<td>1.28</td>
<td>2.20</td>
<td>1.44</td>
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<tr>
<td>RPQ Total</td>
<td>28.70</td>
<td>16.32</td>
<td>35.55</td>
<td>12.38</td>
<td>34.34</td>
<td>15.88</td>
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</tbody>
</table>

*Note. A high score on the RPQ indicates a greater level of impairment.

Cohen’s point estimate effect size $d$ was calculated to articulate the magnitude of difference between the three groups on somatic symptoms (see Table 8). The results suggest that compared to the no diagnosis group, participants in the concussion and mTBI groups expected higher levels of somatic symptoms, with Cohen’s $d$ effect sizes ranging from .01 to .72. The largest effect size between the no diagnosis and concussion groups was for fatigue ($d=.59$, OL%=61). Between the no diagnosis and mTBI groups, the largest effect size was for feeling depressed/tearful ($d=.50$, OL%=67). Between the mTBI and concussion groups, the largest effect size was for noise sensitivity and forgetfulness ($d=.41$, OL%=73). The RPQ Total score generated larger effect sizes than each somatic symptom, with the exception of headaches, fatigue, irritability, feeling depressed/tearful, forgetfulness, poor concentration, and slowed cognition.

Table 8. Effect sizes and score overlap percentage of somatic variables by group comparison
<table>
<thead>
<tr>
<th></th>
<th>concussion (^a)</th>
<th>mTBI (^b)</th>
<th>mTBI (^c)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(d)</td>
<td>OL%</td>
<td>(d)</td>
</tr>
<tr>
<td>Headaches</td>
<td>.53</td>
<td>64</td>
<td>.39</td>
</tr>
<tr>
<td>Dizziness</td>
<td>.19</td>
<td>85</td>
<td>.28</td>
</tr>
<tr>
<td>Nausea/ vomiting</td>
<td>.21</td>
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<td>.24</td>
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<tr>
<td>Noise sensitivity</td>
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<td>Sleep disturbance</td>
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<td>.28</td>
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<tr>
<td>Fatigue</td>
<td>.59</td>
<td>61</td>
<td>.35</td>
</tr>
<tr>
<td>Irritability</td>
<td>.42</td>
<td>73</td>
<td>.24</td>
</tr>
<tr>
<td>Feeling depressed/tearful</td>
<td>.38</td>
<td>74</td>
<td>.50</td>
</tr>
<tr>
<td>Feeling frustrated</td>
<td>.01</td>
<td>99</td>
<td>.05</td>
</tr>
<tr>
<td>Forgetfulness</td>
<td>.72</td>
<td>58</td>
<td>.32</td>
</tr>
<tr>
<td>Poor concentration</td>
<td>.56</td>
<td>64</td>
<td>.26</td>
</tr>
<tr>
<td>Slowed cognition</td>
<td>.50</td>
<td>67</td>
<td>.36</td>
</tr>
<tr>
<td>Blurred vision</td>
<td>.25</td>
<td>82</td>
<td>.46</td>
</tr>
<tr>
<td>Light sensitivity</td>
<td>.06</td>
<td>95</td>
<td>.14</td>
</tr>
<tr>
<td>Double vision</td>
<td>.19</td>
<td>85</td>
<td>.20</td>
</tr>
<tr>
<td>Restlessness</td>
<td>.40</td>
<td>73</td>
<td>.32</td>
</tr>
<tr>
<td>RPQ Total</td>
<td>.47</td>
<td>68</td>
<td>.35</td>
</tr>
<tr>
<td>Mean</td>
<td>0.36</td>
<td>0.28</td>
<td>0.09</td>
</tr>
</tbody>
</table>

3.5 Effect of Diagnostic terminology on Expected Recovery Time

Descriptive statistics for expected recovery times by diagnostic terminology group are shown in Table 9. To determine if there was a significant difference in expected recovery time by group, a one-way ANOVA was conducted after transforming the variable into standardized scores. The data were screened to determine suitability for parametric analyses. The assumptions were violated; therefore, a Kruskal-Wallis test was conducted. Expected recovery time was significantly affected by diagnostic terminology, \(H(2)=3.286, p=.042\). Pairwise comparisons with adjusted \(p\)-values showed that there were no significant differences between expected
recovery time between mTBI and no diagnosis groups ($p=.08$), mTBI and concussion groups ($p=1.00$), or no diagnosis and concussion groups ($p=.89$).

Table 9. Mean scores and standard deviators for expected recovery time

<table>
<thead>
<tr>
<th></th>
<th>No Diagnosis</th>
<th>Concussion</th>
<th>MTBI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Recovery</td>
<td>4.15</td>
<td>9.38</td>
<td>9.79</td>
</tr>
<tr>
<td>SD</td>
<td>4.63</td>
<td>10.40</td>
<td>10.96</td>
</tr>
</tbody>
</table>

Cohen’s point estimate effect size $d$ was calculated to articulate the magnitude of difference between the three groups on expected recovery time. Compared to the no diagnosis group, the concussion group excepted longer recovery times ($d=0.66$, OL%=59). Compared to the no diagnosis group, the mTBI group also expected longer recovery times ($d=.62$, OL%=62). On the other hand, the concussion and mTBI groups were similar, with the mTBI group expecting slightly longer recovery times ($d=.04$, OL%=96).

4 Discussion

The present study investigated the effect of a systematic variation of diagnostic terminology (i.e., mTBI, concussion, undiagnosed injury) following a standardized injury description as it pertains to perceptions of cognitive, emotional and somatic consequences following mild brain injury. The underlying premise and hypothesis driving the current study is that the term mTBI is associated with negative expectations, which, in turn, impedes upon neuropsychological and psychological test performance, and ultimately, recovery post-injury. Thus, I set out to address the following questions via an experimental cueing of these negative expectations: (1) does the diagnosis of mTBI generate the perception of greater cognitive consequences compared to a diagnosis of concussion or an undiagnosed injury; (2) does diagnostic terminology influence cognitive ability at a global level, or is it a domain-specific construct; (3) does the diagnosis of mTBI generate the perception of greater depressive and
anxious symptomatology compared to a diagnosis of concussion or an undiagnosed injury; (4) does the diagnosis of mTBI generate the perception of greater somatic consequences compared to a diagnosis of concussion or an undiagnosed injury; and (5) does the diagnosis of mTBI generate the expectation of longer recovery times compared to a diagnosis of concussion or an undiagnosed injury? It was hypothesized that participants would perceive greater cognitive, emotional and somatic consequences following a brain injury that was diagnosed as an mTBI, compared to a concussion or an undiagnosed injury. In addition, it was hypothesized that diagnostic terminology would influence cognitive ability at a domain-specific level. Finally, it was hypothesized that participants exposed to the mTBI vignette would expect longer recovery times compared to those exposed to the concussion and undiagnosed injury vignettes. Overall, my hypotheses were partially supported. While no differences existed among the concussion and mTBI groups, our findings suggest that individuals in the mTBI group perceived greater cognitive, emotional, and somatic consequences when compared to the individuals in the no diagnosis group.

4.1 Cognitive Functioning

My hypotheses related to cognitive functioning were partially supported. While no significant differences emerged, effect size analyses indicated that the mTBI group perceived greater cognitive consequences compared to the no diagnosis group. Specifically, my findings demonstrate that memory performance may provide a more discriminative index of perceived cognitive impairment than the NAB-S Total score, differentiating approximately 41% of participants in the mTBI condition from participants in the no diagnosis condition. This may be because memory difficulty is often listed as a symptom that is commonly experienced following mild brain injury in psychoeducational handouts. Thus, individuals are more familiar with
memory impairment following mild brain injury when compared with other cognitive domains. This familiarity could increase the rate at which they are expected (Gunstad & Suhr, 2002; Mittenberg et al., 1992; Sullivan & Edmed, 2012; Sullivan et al., 2014). On the other hand, my findings demonstrate that performance on attention tasks may not provide a more discriminative index of expected cognitive impairment, differentiating just 11% of participants in the mTBI condition from participants in the no diagnosis condition. While the attention domain did not produce large enough effect sizes to infer clinical meaning, my overall hypothesis was supported in that individuals expect worse outcomes in some domains (e.g., memory), and expect better outcomes in other domains (e.g., language). Thus, my findings suggest that the effect diagnostic terminology has on cognitive functioning is domain-specific.

On the other hand, contrary to my initial hypothesis, no meaningful inferences can be made between the mTBI and concussion groups. While effect size analyses suggest that the concussion group expected to experience greater cognitive difficulty among attention, executive functioning, language, and spatial domains, these effect sizes were too small to infer clinical meaning ($d=-.02$ to -.36). That is, the variation of the diagnostic terms mTBI and concussion is not sufficient to cause a difference in expected cognitive impairment.

### 4.2 Emotional Functioning

My hypotheses related to emotional functioning were partially supported. No significant differences emerged among the groups. However, effect size analyses suggested that greater symptom severity were expected in the mTBI group when compared to the no diagnosis group. Specifically, my findings demonstrate that depressive symptoms differentiated approximately 29% of participants in the mTBI condition from participants in the no diagnosis condition. In addition, anxious symptoms differentiated approximately 15% of participants in the mTBI
condition from participants in the no diagnosis condition. However, contrary to my hypothesis, no meaningful inferences can be made between the mTBI and concussion groups on depressive and anxious symptomatology. While effect size analyses suggest that the concussion group expected to experience greater difficulty in both depressive and anxious symptomatology, these effect sizes were too small to infer clinical meaning ($d=.03$ and $.15$, respectively). In summary, my findings suggest that the variation of the diagnostic terms mTBI and concussion is not sufficient to cause a difference in expected symptom severity.

### 4.3 Somatic Symptomatology

My hypotheses related to the perception of somatic consequences following mild brain injury were partially supported. While no significant differences existed among the three groups, effect size analyses suggested that greater amounts of somatic symptoms are expected among participants exposed to the term mTBI versus those exposed to an undiagnosed injury. Specifically, my findings demonstrate that total somatic symptoms differentiated approximately 24% of participants in the mTBI condition from participants in the no diagnosis condition. However, my findings did not support my hypothesis in that no meaningful differences emerged between the mTBI and concussion groups on somatic symptomatology. While effect size analyses suggest that the concussion group expected to experience greater somatic symptoms, these effect sizes were too small to infer clinical meaning ($d=-.03$ to $.33$). That is, the variation of the diagnostic terms mTBI and concussion is not sufficient to cause a difference in expected somatic symptoms.

My hypothesis regarding the prevalence of somatic symptom endorsement among groups was partially supported. While I hypothesized that headaches, fatigue, and dizziness would be the most frequently endorsed somatic symptoms, my findings suggest that all three groups
endorsed symptoms relating to fatigue and concentration difficulties. The consistency of these symptoms across diagnostic terminology groups suggests that variation in diagnostic terminology produces small differences in the type of somatic symptoms that are expected.

4.4 Expected Recovery Time

My hypothesis related to expected recovery time was partially supported. A significant difference existed among the three groups, which seems to reflect that an undiagnosed injury lowers the amount of time an individual expects to recover after an accident. Specifically, my findings demonstrate that expected recovery time differentiated approximately 38% of participants in the mTBI condition from participants in the no diagnosis condition. However, my findings did not support our hypothesis in that no meaningful differences emerged between the mTBI and concussion groups on expected recovery time, differentiating approximately 4%.

Overall, these results suggest that diagnostic terminology is not sufficient to cause differences in expected recovery times, such that all participants exposed to the mTBI group will not expect greater recovery times compared to the concussion group.

4.5 General Discussion

Overall, my findings suggest that some but not all outcomes were susceptible to our experimental manipulation. While none of the cognitive, emotional, and somatic variables indicated significant differences among the three groups, the effect size analyses indicated a consistent pattern in the data. My findings suggest that individuals in the mTBI group perceived greater cognitive, emotional, and somatic consequences when compared to the individuals in the no diagnosis group. However, no meaningful inferences can be made when comparing those that were exposed to the mTBI and concussion vignettes on cognitive, emotional, or somatic
domains. That is, both the mTBI and concussion groups, which did not differ from each other, produced worse expectations than the no diagnosis group.

To date, very few studies have analyzed the effects of diagnostic terminology on expected symptoms, and no studies have analyzed cognitive, emotional, and somatic symptoms collectively. With respect to the existing research literature, the finding that there were no meaningful differences in expected outcomes between the terms mTBI and concussion is relatively consistent with Kempe et al.’s (2013) study, which examined the differences between university staff and students’ expected symptoms and illness perceptions as a function of diagnostic terminology (i.e., mTBI and concussion). Overall, most of the variables tested by Kempe et al. were not influenced by diagnostic terminology, and, similar to the present study, the authors concluded that “changing the embedded term from concussion to mTBI does not have clinically significant consequences” (p. 9). However, discrepancies between the current study and Kempe et al.’s study exist. For example, Kempe et al. (2013) found that individuals who were exposed to the term concussion expected more PCS symptoms in the cognitive, affective, and sensory domains when compared to those exposed to mTBI. There are a number of different methodological differences between Kempe et al.’s (2013) study and the current study that may account for these different findings. For example, the current study may not have found significant differences among groups on cognitive and affective symptoms because of the different outcome measures used in the study. While Kempe et al. (2013) used the 22-item Neurobehavioral Symptom Inventory to measure cognitive and affective symptoms, the present study employed the NAB-S, the BAI, and the BDI-II. Hence, I employed performance based measures regarding cognition in addition to self-report measures and symptom checklists. In addition, Kempe et al. (2013) provided participants with a two-page discharge advice sheet,
which contained the experimental manipulation (i.e., varying terminology), whereas this study used a list of common symptoms which was read to the participant in the experimental conditions. Finally, Kempe et al. (2013) did not use a control (i.e., no diagnosis) group to compare to the mTBI and concussion groups. Thus, a comparison between this study and Kempe et al.’s (2013) study on no diagnosis and mTBI/concussion groups is not directly possible.

The finding that cognitive, emotional, and somatic variables were not affected by diagnostic terminology is inconsistent with Sullivan, Edmed & Kempe’s (2014) study, which examined the influence of diagnostic terminology on illness perception and expected PCS and post-traumatic stress disorder (PTSD) symptoms using a sample of healthy university staff and students. Sullivan and colleagues (2014) found that participants expected worse outcomes when simulating an mTBI diagnosis or an undiagnosed injury, than simulating the same injury diagnosed as a concussion. The current study may not have found significant differences among diagnostic terminology groups because of the different outcome measures used in the study. While Sullivan et al. (2014) analyzed somatic symptoms, they used the Neurobehavioral Symptom Inventory, whereas this study used the RPQ. In addition, Sullivan et al. (2014) included variables related to PTSD symptoms, but did not include cognitive and emotional variables in their study. Another difference between these studies was their geographical location. Both Kempe et al. (2013) and Sullivan et al. (2014) collected data in Australia, whereas the current study was conducted in Canada. Edmed & Sullivan (2015) suggest that variability in brain injury management policy and media coverage between countries could contribute to differences in expectations, and could account for the different findings between studies.

In keeping with my findings that demonstrate a difference between mTBI and no diagnosis groups, it may be that providing participants with a list of common brain injury
symptoms can help explain the persisting symptoms following brain injury. Drawing on research documenting misattribution, self-efficacy, and expectancy effects, my findings suggest that symptom lists presented injudiciously may lead to iatrogenic reactions, reinforcing the individuals’ perception that symptoms are permanent and out of their control, and causing the individual to make internal, stable, and global attributions (Mulhern & McMillan, 2006; Rohling, Larrabee, & Millis, 2012; Vanderploeg et al., 2014; Whittaker et al., 2007; Wood, 2004). Thus, if an individual is involved in an accident and is subsequently given a handout with a list of symptoms that are commonly experienced after a brain injury, then they may begin to expect these symptoms, ultimately playing a causal role in recovery. This could be damaging to the individual’s health as the expectation of poor recovery could lead to poor recovery itself.

While these negative consequences exist, several studies have suggested that negative expectations can be offset by brief psychoeducational interventions, significantly reducing the severity of outcomes and duration of PCS (Miller & Mittenberg, 1998; Mittenberg, Tremont, Zielinski, Fichera, & Rayls, 1996; Mittenberg, Zielinski, & Fichera, 1993). In addition, the literature indicates that reassuring individuals of a positive outcome in the post-acute period may minimize potential iatrogenic effects, and facilitate resilience and recovery after mTBI (Bryant, 2008; Rohling et al., 2012; Vanderploeg et al., 2014). That is, a positive message can be communicated to brain injury patients, with potential beneficial effects. For instance, Vanderploeg and colleagues (2014) suggest that the following message should be conveyed to increase self-efficacy and promote healthy behavior:

“You may well have some normal, expected symptoms for a few days or so, but we can help you deal with them during this transitional period. There are also things that you can
to do minimize or cope with these symptoms. We will work together to get through this
transitional period as you recover” (p. 250).

If brain injury patients are provided with accurate information of expected symptomatology and recovery, as well as headache, pain, sleep, and stress management techniques, these negative expectations, and in turn persistent symptoms, may be offset. Thus, it is evident that the information provided to individuals in the post-acute period of their injury can produce both harmful and beneficial effects. Clinicians must be cautious of how a diagnosis is presented, regardless of whether the individual is diagnosed with a concussion or an mTBI. The negative expectations that are exacerbated by negative clinician-patient interactions should be targeted in the attempt to promote full recovery following brain injury (Greville-Harris & Dieppe, 2015).

5 Limitations, Future Directions, and Conclusions

Although this study has the strength of experimental manipulation and control, there are several limitations that should be kept in mind when drawing conclusions. First, the participants were primarily healthy females recruited from a Canadian university population. The extent to which these results can generalize to other samples is unknown, and should be replicated in other populations (e.g., clinical populations, males, cross-culturally). Second, while I attempted to remove the potential confound of pre-existing expectations about brain injury, it is unlikely that the participants’ expectations were only influenced by the vignette that they were exposed to in this study. It is possible that factors such as exposure to or knowledge about brain injury would mediate the expected cognitive, emotional and somatic outcomes in our study. Further research should be conducted to elucidate the relationship between pre-existing conceptualizations and post-injury expectations. Third, while the total number of participants met adequate power, there was a disparity in the sizes of the three groups, and the concussion group was slightly
underpowered. Thus, future research conducted with each group meeting adequate power is needed. Next, the use of the simulation design may have underestimated the effect of variation in diagnostic terminology. For example, for an individual’s test scores to be influenced by diagnosis threat, the individual must have a strong belief that they are a part of the group being tested (Suhr & Gunstad, 2002, 2005). It is unlikely that this was captured in our simulation.

Despite these limitations, this study suggests that perceptions of cognitive, emotional, and somatic consequences, as well as expected recovery times, are relatively unchanged by the variation of the terms concussion and mTBI. However, my findings indicate that providing the individual with a list of symptoms commonly experienced following brain injury affects several important injury-related outcomes. By demonstrating that individuals exposed to a diagnosed injury with a list of common symptoms perceive greater negative outcomes than individuals exposed to an undiagnosed injury without a list of common symptoms, this study provides indirect support for psychosocial models that are used to explain persisting symptoms following mTBI. Clinicians must be aware of the effect that non-neurological variables such as symptom lists can have on recovery, and must be cautious when deciding what information is provided to patients in the post-acute stages of mild brain injury.

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Appendix A: Simulation Scenarios

MTBI Simulation Scenario
“Imagine that you were in a car accident that involved being hit by another car. You were briefly knocked unconscious, and your first memory after the impact was of waking in the hospital about 45 minutes later. You were kept overnight for observation. The doctors told you that you experienced a mild Traumatic Brain Injury. Pretend it is the day after the accident and you are thinking about the long-term effects of mild Traumatic Brain Injury. Now we want you to imagine that a year later you are undergoing testing to determine your emotional and cognitive state. You are about to take a series of cognitive and emotional tests that would be used in such a situation. We are about to give you these tests to see how you believe you would be doing.

Common problems following a mild Traumatic Brain Injury, which may help you in your simulation include:
- frequent headaches
- being easily fatigued
- problems with memory
- difficulty paying attention and concentrating
- slowed responses
- irritability
- anxiety
- depression”

Concussion Simulation Scenario
“Imagine that you were in a car accident that involved being hit by another car. You were briefly knocked unconscious, and your first memory after the impact was of waking in the hospital about 45 minutes later. You were kept overnight for observation. The doctors told you that you experienced a concussion. Pretend it is the day after the accident and you are thinking about the long-term effects of concussion. Now we want you to imagine that a year later you are undergoing testing to determine your emotional and cognitive state. You are about to take a series of cognitive and emotional tests that would be used in such a situation. We are about to give you these tests to see how you believe you would be doing.

Common problems following a concussion, which may help you in your simulation include:
- frequent headaches
- being easily fatigued
- problems with memory
- difficulty paying attention and concentrating
- slowed responses
- irritability
- anxiety
- depression.”

No Diagnosis Simulation Scenario
“Imagine that you were in a car accident that involved being hit by another car. You were briefly knocked unconscious, and your first memory after the impact was of waking in the
hospital about 45 minutes later. You were kept overnight for observation. Pretend it is the day after the accident and you are thinking about the long-term effects of the accident. Now we want you to imagine that a year later you are undergoing testing to determine your emotional and cognitive state. You are about to take a series of cognitive and emotional tests that would be used in such a situation. We are about to give you these tests to see how you believe you would be doing.”
Appendix B: Simulation Reminders

MTBI Simulation Reminder
“Remember that you are pretending to have been in a car accident and were diagnosed with a mild traumatic brain injury. Again, common problems following a mild traumatic brain injury include:
- frequent headaches
- being easily fatigued
- problems with memory
- difficulty paying attention and concentrating
- slowed responses
- irritability
- anxiety
- depression”

Concussion Simulation Reminder
“Remember that you are pretending to have been in a car accident and were diagnosed with a concussion. Again, common problems following a concussion include:
- frequent headaches
- being easily fatigued
- problems with memory
- difficulty paying attention and concentrating
- slowed responses
- irritability
- anxiety
- depression”

No Diagnosis Simulation Reminder
“Remember that you are pretending to have been in a car accident”.