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The Association between Self-Reported Psychiatric Symptoms and Performance Validity Test Failure across Brain Injury Severity Levels

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By

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ABSTRACT

Performance validity testing is a routine procedure used in neuropsychological assessment to ensure an examinee is putting forth a performance that is an accurate representation of their ability. There is increasing evidence that individuals reporting high levels of psychiatric symptoms as well as those with mild traumatic brain injury (mTBI) fail performance validity tests (PVTs) more often. This research sought to determine if the high base rate of failure (BR_{Fail}) in the mTBI sample were related to an increase of self-reported psychiatric elevations. An archival dataset of 480 individuals either with an orthopedic injury, mTBI, or moderate-severe traumatic brain injury (msTBI) diagnosis (Dx) referred for neuropsychological testing was retrospectively analyzed. Of the sample, 70.4% were male and the average age was 39.3 years (*SD* = 11.9). Participants took part of a routine neuropsychological assessment which included the Word Memory Test (WMT). Those in the mTBI Dx group failed the WMT more often than those with msTBI. There was no interaction between Dx and failing PVTs on psychiatric symptom reporting. Results did show a relationship between Dx and PVT failure on some psychiatric symptom scales independently. Failing the WMT was related to poorer outcome on neurocognitive tests. Overall, no interaction between PVT outcome and Dx was found on self-reported psychiatric symptoms, each factor independently explained a significant amount of variance. Results suggest clinicians should continue to be aware of factors that may contribute to PVT failure in their practice, such as elevated psychiatric symptom reporting and diagnostic group membership.

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CHAPTER 1

Introduction

Traumatic Brain Injury

Traumatic brain injuries (TBIs) are a leading cause of death and disability worldwide. It has been estimated that 69 million people suffer from them each year (Dewan et al., 2019) and in the United States alone it is estimated that 1.7 million TBIs occur each year with approximately 52,000 resulting in death (Faul et al., 2010). In Canada, about 0.5% of the 2014 population, or approximately 175,000 individuals experienced a TBI each year and that between 2005-2014 there was a incidence of TBI more than doubled (Rao et al., 2017). The increased incidence of TBI throughout the years was thought to be because of increased awareness and reporting of the condition. Thus, both the incidence and awareness of this debilitating injury is high and increasing over time highlighting the importance of research in this area.

TBIs are a complex and heterogenous because of the range of mechanisms of injury as well as sequalae that can result. The current definition of TBI is an alteration of brain function or other evidence of brain pathology due to an external force to the head (Menon et al., 2010). An alteration of brain function includes post-traumatic amnesia, confusion, or disorientation after injury, loss of consciousness (LOC) as well as any other neurological deficits such as changes in sensory perception, loss of balance, and aphasia. In more severe cases, TBI could also result in neuroradiologically confirmed damage to the brain, such as evidence of edema, hemorrhage, or skull fracture in the acute stage. Long-term residual effects include atrophy, encephalomalacia and disruption in white matter connectivity. The cause of TBI can range from blunt force to the head, rapid acceleration-deceleration resulting in coup-countercoup injuries, projectiles penetrating the brain, or acceleration/deceleration of the head causing damage. Therefore,

although often grouped into one diagnostic category, TBIs include an expansive range of causes, mechanisms, and outcomes.

Due to the wide range of TBI presentations and methods of injury, these injuries are commonly classified into four severity groups: mild TBI (mTBI), mild complicated TBI (mcTBI), moderate TBI (MTBI), and severe TBI (STBI). There are different criteria by which severity classifications are made. One of the most widely used criteria is the Glasgow Coma Scale (GCS) (see Table 1). Introduced in 1974 (Teasdale & Jennett), the GCS provides a behaviorally based assessment of TBI. The observer provides rating on motor responsiveness, verbal performance, and eye opening. Brain injury severity is indicated by a score between 3 and 15 with lower scores indicating lower responsiveness (STBI $\leq$8, MTBI 9-12, mTBI $\geq$ 13). Other commonly used criteria include the duration of LOC or post-traumatic amnesia (PTA) (see Table 2). The differentiation between mTBI and mcTBI is the presence of positive neuroimaging findings (bleeding, swelling or skull fractures) in the latter category (Iverson, 2005). However, outcomes from mcTBI tend to more closely resemble those of MTBI (Kashluba et al., 2008).

The sequalae of TBI are heterogenous and expansive as they influence many broad domains of cognitive and emotional functioning that typically vary based on the severity of injury. For example, in mTBI, which encompasses between 70-90% of TBIs (Maas et al., 2017), individuals often make a full recovery after experiencing negative symptoms for a few weeks. A subset of mTBI patients continue to report symptoms beyond the normative window of recovery, and utilize associated services (Bigler, 2008; King, Wade, & Beehler, 2014). This condition is referred to as Post Concussive Syndrome (PCS). Patients with MTBI or STBI often have long-term cognitive, vestibular, behavioural and emotional repercussions that individuals with mTBI only face short-term (if at all). The cognitive sequelae of TBI often include long-term and

working memory impairment, difficulty with attentional/concentration and slowed processing speed (Azouvi et al., 2017). Individuals often experience headaches and occasionally, other post-traumatic neurological disorders such as seizures (Annegers et al., 1998; Brown et al., 2015). The emotional sequelae includes impairments with social cognition (Babbage et al., 2011), mood regulation (Hanks et al., 1999) and increase of both diagnosed psychiatric/ mood disorders as well as self-reported symptoms (Perry et al., 2016; Osborn et al., 2014; Osborn et al., 2016).

TBI and the Incidence of Self-Reported Psychiatric Problems

The relationship between TBI and psychiatric symptom reporting is especially important because of the common increase of psychiatric problems seen after injury – either exacerbated pre-existing conditions or newly onset. These psychiatric problems are evident in both formal diagnosis (Perry et al., 2016) and in self-reported symptoms (Osborn et al., 2014; Osborn et al., 2016). Patients with mTBI tend to report higher levels of psychiatric symptoms than those with moderate and severe TBI (msTBI) (Osborn et al., 2014; Osborn et al., 2016).

This counterintuitive, but well-replicated finding is attributed to patients with mTBI having more insight, less focus on cognitive repercussions, and fewer resources available to help them than those with msTBI. Additionally, patients with TBI have a history of displaying different psychiatric symptom self-reporting at different injury severity levels. On the Minnesota Multiphasic Personality Inventory (Second Edition) (MMPI-2), a comprehensive self-report inventory of personality and psychological functioning, patients with mTBI often report higher elevations on the Hysteria (Hs), Hypochondriasis (Hy), Depression (De), and Psychasthenia (Pa) scales than those with more severe brain injuries (Gass & Russell, 1991;Youngjohn, Davis, & Wolf, 1997). This reverse dose-response relationship between injury severity and psychiatric

symptoms runs counter to expectations. The leading explanations are the presence of genuinely elevated psychopathology in the mTBI group or the PCS being similar to somatization disorder.

Neuropsychological Assessment

Due to the complex cognitive and emotional changes post TBI, individuals are often referred for neuropsychological assessment to determine their level of functioning and estimate the extent of residual deficits. Neuropsychological assessments contribute unique information about functioning, above and beyond what current neuroimaging technologies provide, because they determine how the injury affects the patient's ability to perform cognitive tasks and by extension, occupational and adaptive functioning. Neuropsychology is a diverse field of study based on analysis of the brain-behaviour relationship. By utilizing psychometric tests, an individual's performance in various cognitive domains (attention, processing speed and memory) is evaluated across sensory modalities (auditory, visual, tactile). Test behavior is quantified (i.e., number of correct answers, time taken to complete a task) and then compared to a normative sample to determine the individual's relative standing. The pattern of performance across different tests provides insight on the examinee's overall level of functioning, and severity of neurocognitive deficits. This information is used in a broader context for diagnostic consideration, to optimize the clinical management of the underlying condition and injuries such as TBI, and provide suggestion for helpful treatments and therapies.

TBIs often involve changes in emotional functioning as well. Therefore, neuropsychologists also assess for psychiatric symptoms, using structured symptom inventories and clinical interviewing of both the patient and collateral informant. A comprehensive neuropsychological evaluation includes assessments of psychopathology because there is research suggesting emotional functioning can influence cognitive outcomes (Eysenck et al.,

2007; Owens et al., 2008; Qureshi et al., 2011; Rock et al., 2014; Ross et al., 2003; Samuelson et al., 2006; Scott et al., 2015; Snyder, 2013), neurological conditions have emotional sequalae, and psychopathology can affect participation in rehabilitation therapies, therefore the assessment of psychopathology is relevant to rehabilitation efforts.

Performance Validity

Throughout neuropsychological assessment, interpretation of test results rests on the assumption that the examinee's test performance is an accurate representation of their true ability level (Larrabee, 2012; Bigler, 2015). The importance of valid responding is typically conveyed at the beginning of the assessment, when examinees are instructed to "perform at the best of their abilities." This request has long been interpreted as an implicit verbal contract that ensured valid performance. This posed an issue as there are many reasons why one may not be able or willing to perform to the best of their abilities. Although the underlying reasons for non-credible presentation are often unknown to the clinician and ultimately irrelevant (Merten & Merckelbach, 2013), implementation of objective measures of symptom and performance validity monitoring have gained use in recent years.

Performance Validity Tests

The uncertainty of performance validity within neuropsychological assessment poses a risk to the interpretation of results. Determining the credibility of the patient's presentation during the assessment based on clinical judgement alone has long proven to be ineffective (Heaton et al., 1978). Therefore, performance validity tests (PVTs), which are objective measures of the credibility of a given response set, have been endorsed by major professional organizations (Committee on Psychological Testing, 2015). PVTs are tests calibrated so that

patients with severe *bona fide* neurologic and psychiatric problems (Heilbronner et al., 2009) can pass them.

PVTs can either be free-standing or embedded instruments. Free-standing PVTs are measures that were created with the intended purpose of monitoring performance validity, providing little to no information on cognitive ability. Embedded validity indicators (EVIs) are instruments derived from existing neuropsychological tests that are then subsequently co-opted to give a measurement of performance validity (Committee on Psychological Testing, 2015). PVT failures indicate that the overall neurocognitive profile likely underestimates the examinee's true ability level (Larrabee, 2012), without providing any information about the underlying reason (Boone, 2013). Currently, PVTs have gained use as objective empirical methods to evaluate performance validity and are now considered standard use in clinical practice (Chafetz et al., 2015).

Symptom Validity Tests

Symptom validity tests (SVTs) are measures designed to monitor the validity one's symptom reports. Individuals self-reporting psychiatric symptoms may not be presenting themselves in an accurate manner, and personality inventories on are often unhelpful on their own in separating genuine and exaggerated symptoms (Boone, 2007). For this reason, SVTs have been implemented in self-report symptom scales in order to assess for response bias.

There is conflicting evidence on the relationship between SVTs and PVTs. Some studies suggest that they are independent of one another (Demakis, Gervais, & Rohling, 2008; Nelson, Sweet, Berry, Bryant, & Granacher, 2007; Ruocco et al., 2008), while others found that patients who fail PVTs are more likely to over-endorse symptoms (Haggerty, Frazier, Busch, & Naugle, 2007; Mathias et al., 2002; Whiteside, Dunbar-Mayer, & Waters, 2009; Jones et al., 2012). A

confirmatory factor analysis performed by Van Dyke and colleagues (2013) to determine the relationship between performance validity and symptom validity tests found that symptom validity, performance validity and cognitive abilities were independent factors.

TBI and PVT Outcome

Due to the medicolegal context of TBIs (i.e., personal injury litigation, disability claims, somatization), PVTs are essential during neuropsychological assessment. Paradoxically, base rates of PVT failure rate (BR_{Fail}) has a reverse dose-response relationship to TBI severity, much like psychiatric symptom reporting: mTBI is associated with higher BR_{Fail} than more severe TBI (Donders, Lefebre & Goldsworthy, 2019; Abeare et al., 2019; Carone, 2008; Erdodi & Rai, 2017; Green, Flaro, & Courtney, 2009; Green, Iverson & Allen, 1999). This finding is counterintuitive, as severely injured individuals typically perform worse on measures of cognitive ability. PVTs, however, are known to be insensitive to genuine impairment (Critchfield et al., 2019) so the true mechanism of this reverse dose-response relationship is poorly understood. The issue is further complicated by the ubiquity of this phenomenon: it is observed in both medico-legal and clinical contexts, suggesting that external incentive to appear impaired does not fully explain the elevated BR_{Fail} in mTBI.

Factors Influencing PVT Outcome

Effort and engagement during neuropsychological assessment is a dynamic process and many factors can contribute to one's ability to demonstrate valid performance. Malingering (intentional suppression of test performance in the presence of external incentives to appear impaired; American Psychiatric Association, 2013) is the most researched etiology for PVT failure. Genuine and severe impairment can also account for PVT failures, such as dementia and intellectual disabilities (Dean et al., 2008; Dean et al., 2009; Smith et al., 2014). These diagnostic

categories are considered exempt from PVTs, although recent investigations identified specific instruments with low false positive rates even in these populations (Messa et al., 2020).

In addition to neurological diagnoses, there are other reported influences on performance validity in the literature. For example, individuals with limited English proficiency (LEP) and patients with aphasia have higher failure rates on PVTs with high verbal mediation, likely due to focal impairment in language (Ali et al., 2020; Bodner, Merten & Benke, 2019; Erdodi et al., 2017). Language dominance was able to explain between 6-31% of the variance in passing or failing PVTs with high verbal mediation (Erdodi et al., 2017).

In undergraduate populations, it is surprisingly common to see PVT failures because there is a lack of incentive to demonstrate their full ability (An et al., 2017; Roye et al., 2019). Additionally, in this population, there is motivation to finish testing as quickly as possible which could also result in PVT failure (DeRight & Jorgensen, 2015). Even when incentivized to perform well, university students fail PVTs (Abeare et al., 2021) at rates comparable to clinical and even forensic settings (Young, 2015; Larrabee, Millis, & Meyers, 2009) – a phenomenon that lacks a coherent explanation.

Somatic disorders, where emotional distress is expressed as physical symptoms that cause disruption in daily life, have also shown to inflate BR_{Fail}. Patients with medically unexplained symptoms also often fail PVTs (Kemp et al., 2008), although results diverge across studies, instruments and populations (Tyson et al., 2018).

Psychiatric Symptoms and PVTs

Another key factor explored in the literature is the influence of a psychiatric symptoms on performance validity. It has been documented in the literature that specific disorders such as depression (Rock et al., 2014; Snyder, 2013), anxiety (Owens et al., 2008; Eysenck et al., 2007),

PTSD (Scott et al., 2015; Qureshi et al., 2011; Samuelson et al., 2006), schizophrenia (Heinrichs & Zakzanis, 1998; Heaton et al., 2001) influence cognitive test scores. These psychiatric disorders may interfere with cognitive processes that can lead to lower overall test scores. Additionally, individuals can have a psychiatric condition that manifests in a way that it presents itself as a neurodegenerative disease otherwise referred to as "pseudodementia" (Heaton et al., 2001). The literature has not been consistent on whether or not these symptoms influence the outcome of performance validity testing.

Recent research has found evidence that psychiatric symptoms are related to PVT outcomes. Erdodi et al. (2017) showed in a mixed clinical sample there were moderate associations between failures on the Grooved Pegboard Test as a PVT and self-reported depression and anxiety. Miskey et al. (2019) found that failure on memory-based (but not attention-based) PVTs was related to PTSD severity. Marcopulos et al. (2014) found that a sizeable proportion (11%) of their psychiatric sample failed PVTs in the absence of external incentive to underperform. These individuals were more likely to be female, have less education and have a diagnosis of schizophrenia.

Past research has not always supported this notion as, for example, Ashendorf et al., (2004) found that those that self-reported depression and anxiety was unrelated to PVT outcome across both trials. Similarly, Considine et al., (2011) found there was no difference between those diagnosed with Major Depressive Disorder (MDD) and healthy controls on either trials of the Test of Memory Malingering (TOMM). In different severity levels of depressed older adults (mild, moderate, severe as diagnosed by clinical interview), Lee et al. (2000) did not find any differences on the Dot Count Test or the Rey-15 item test.

Merten and Merckelbach's 2013 paper argued that attributing PVT failures to psychiatric symptoms is weakly supported because these psychiatric symptoms usually cause minimal interference in the person's daily life. They stated that any PVT failure that cannot be explained by outright malingering should just be considered poor cooperation as there is no convincing evidence the disorder itself can cause failure and is likely linked with external incentives. Merten and Merckelbach suggest psychogenic interference as an exculpatory explanation for PVT failures, "poor cooperation" is an ill-defined construct. However, Wise & Smith (2011) outline that engagement in testing is made up of many intrinsic and extrinsic factors that can contribute to one's effort during assessment. Generalizing all of these factors into poor cooperation is overly reductionistic, as it discounts the complexity of human behaviour and specific reasons an individual may not be fully engaged in testing.

Merten & Merckelbach's claim that an individual's psychiatric symptoms create minimal interference in their daily lives may be true, but that does not indicate it never plays a factor in their functioning. It is well documented that in some psychiatric conditions, including anxiety, PTSD, and depression, that individuals practice avoidance behaviours in their daily lives to minimize their ongoing symptoms (Badour et al., 2012; Ottenbreit & Dobson, 2004; Aderka et al., 2013). By tailoring their daily life to adapt to the hardship of living with these symptoms, individuals are often creating less interference of their symptoms day-to-day. Neuropsychological assessment, however, could be a more stressful and anxiety provoking environment where the individual is confronted with new cognitive demands, must follow a rigid set of rules in order to complete the assessments. Therefore, what is minimally invasive in their daily lives might not be in this unique setting designed to evaluate one's global neuropsychological functioning. Of course, evidence to the contrary has also been widely

mentioned in the clinical literature. Namely, laboratory-based tests have limited ecological validity because of the structured, distraction-free, and supportive environment in which they are administered. Essentially, one cannot be sure if the individual being assessed would perform better or worse in real-life situations. Therefore, the argument that there is minimal interference in the testing environment from psychological symptoms may be overly simplistic.

Lastly, their argument does not address specific cases such as individuals with TBI that often display a complex relationship between cognitive and emotional functioning. A key factor in the aforementioned studies that found no link between psychiatric and performance on PVTs is that they were performed in otherwise healthy populations. In TBI populations, individuals are uniquely dealing with a combination of cognitive and emotional symptomatology that can complicate their ability to perform on neuropsychological assessments. One study on veterans with mTBI and PTSD found that they performed significantly worse on cognitive measures than those with mTBI or PTSD alone (Combs et al., 2015). A similar effect is often reported with depression, as they work in an additive fashion with TBI and can compound severity over and above a typical TBI profile (Schultz et al., 2018). This indicates there may be some form of additive effect of psychiatric symptomatology with TBI that causes greater levels of impairment than expected based on their neurological injury alone. Additional support for this was found in Stenberg (2020) where mTBI patient's self-reported cognitive symptoms were highly related to their emotional and somatic symptoms and unrelated to their actual cognitive performance. This underscores the point that in a brain injury sample, a complex interaction between cognition and emotional functioning can influence perceived recovery. Nonetheless, it is unclear why this additive effect occurs, but emotional factors such as all-or-none thinking, anxiety, depression and

stress have been found in those who have a prolonged recovery from PCS (Hou et al., 2012; Ponsford et al., 2012).

The Influence of Psychiatric Symptoms on PVT Outcomes in TBI Populations

The interaction between cognitive and emotional functioning after brain injury has been shown to influence test scores. In fact, in a TBI sample Udala et al. (2019) discovered specific domains of cognitive functioning that were correlated to increased self-reported symptoms on the MMPI-2. Higher scores on Psychasthenia, Schizophrenia, Obsessions, and Bizarre Mentation predicted lower scores on list learning measures. A higher rating of the Fears subscale predicted a lower working memory score. Higher endorsement of Schizophrenia scale was related to lower verbal memory test scores. Lastly, higher scores on the Depression scale predicted a lower visuographic memory score. This study uniquely demonstrates that within a TBI population, specific psychiatric self-reported symptoms is predictive of lower cognitive functioning, however, does not answer if these low scores translate to PVT failure.

In 2018, Erdodi and colleagues found that a TBI sample that failed a forced-choice recognition PVT reported higher levels of psychiatric symptoms. Another study by Erdodi et al., (2019) found that failing the Word Memory Test (WMT), a free-standing PVT, was associated with self-reported level of depression in a mTBI sample. In both studies however, they failed to account for the validity of reported psychiatric symptoms. As such, it is unclear whether elevations reflect genuine distress or exaggerated symptom report.

Another study demonstrated that a dual diagnosis of TBI and PTSD-was associated with higher BR_{Fail} than TBI and subsyndromal PTSD. It is thought that their complex trauma may have caused more risk of failure (Greiffenstein & Baker, 2008). Due to the fact that there was no PTSD-only group, it is uncertain if this effect would occur in the absence of TBI as well. Clark et

al., (2014) also found that veterans who failed PVTs had increased rates of clinical diagnoses of TBI, PTSD, or comorbid TBI/PTSD, along with depression and substance use. Additionally, they discovered that those who reported more severe symptoms on self-report rating symptom scales also had higher BR_{Fail}. It is important to note that in both these studies those who failed SVTs were not excluded. Overlap between PVT and SVT failure was identified therefore PVT failure cannot be attributed to the dual diagnosis alone (Clark et al., 2014).

Erdodi et al. (2016) investigated the BDAE Complex Ideation Material as a PVT and showed that not every group that displayed a complex psychiatric history or emotional distress had increased PVT failure rates. In particular, those that were categorized as having PCS (cognitive complaints past the 3-month recovery window) which included individuals with comorbid psychiatric conditions showed a much higher PVT failure rate. BR_{Fail} was higher for the PCS group than those with non-PCS psychiatric features such as those with psychogenic non-epileptic seizures. These results, again, demonstrate a unique relation between those with TBI and comorbid psychiatric symptoms showing invalid performance even in comparison to other psychiatric groups.

A study by Jurick et al. (2019) reported on PVT failure rates in 100 veterans with comorbid PTSD and TBI. The goal was to determine if individuals with PTSD symptoms that failed PVTs benefited from psychotherapeutic intervention for those symptoms and improved PVT failure outcome. The authors found that both PVT pass and fail groups had reduced PTSD symptoms after intervention. BR_{Fail} was lower after treatment, suggesting a link between emotional distress and PVT failure.

Another similar study reported on a sample of 411 veterans with mTBI with the goal of characterizing the cognitive concerns of veterans after mTBI and to inspect their

neuropsychological profiles (Jak et al., 2019). They found that a third of the sample failed PVTs; these veterans were more likely to have a psychiatric diagnosis such as PTSD and depression. Additionally, self-reported PTSD, depression, anxiety and neurobehavioral symptoms were also higher in the PVT failure group. This work is important as it further supports the argument that individuals that fail PVTs with TBIs are more likely to have elevations on self-reported psychiatric symptom scales.

Sherer et al., (2020) utilized cluster analysis to inspect clinically meaningful subgroups of those who failed PVTs in a TBI population. They identified three clusters: subgroup 1 had problems with memory but no excess complaints, subgroup 2 had impaired memory and processing speed with cognitive and emotional complaints and subgroup 3 had impairment in all domains and elevated emotional and cognitive complaints. It is apparent that in a majority of the groups, there was significant self-reported emotional/psychiatric distress. This study, however, did not control for litigation status or utilize symptom validity tests. This means that it is unknown if there are other factors involved in the PVT failure such as external incentive or if the distress they are reporting is credible. Also, this study did not offer a non-TBI control group, meaning these clusters may not be unique to the TBI population.

Cogniphobia

In addition to the literature on self-reports of general psychiatric symptoms (depression, anxiety, PTSD etc.) other forms of emotional distress, such as cogniphobia, have also been shown to be linked to PVT failure. The phenomenon occurs when individuals that have a history of headaches (either migraines or headaches after TBI) do not exert the cognitive effort necessary to perform to the best of their abilities due to fear of bringing on or exacerbating their headaches. This phenomenon was based off of a similar phenomenon that occurs with general

bodily injury called “kinesophobia” where an individual has a fear of moving because they do not want to make their physical pain worse. Kinesophobia has been shown to decrease performance on physical tasks (Trost, France, & Thomas, 2011) and the effect can be present whether or not pain actually comes from the activity. Martelli et al., (1999) were the first to expand the phenomenon to cognitive performance and individuals with headaches. This fear of headaches could be a motivating factor in withholding some cognitive effort, resulting in lower neuropsychological test performance.

Suhr & Spickard, (2012) found that fear of headache onset was associated with lower scores on a measure of sustained attention in a population of undergraduates that experience frequent headaches. Those who failed the WMT were more likely to have high scores on the fear/avoidance measure of cogniphobia. However, the authors were unconvinced these findings were accurately showing that those with cogniphobia failed PVTs more often. This was because when removing those who failed the WMT, there was still an effect of fear/avoidance on cognitive performance. Silverberg, Iverson & Panenka (2017) found that failing the Medical Symptom Validity Test (MSVT) a PVT was associated with fear/ avoidance of exerting mental effort. The effect was also found for other memory based cognitive measures. Those that reported avoiding mental exertion also avoided physical activity and traumatic stressors, which suggests that cogniphobia may be linked to a broader style of avoidant coping.

Henry and colleagues (2018) identified three factors that influenced performance validity, particularly in their TBI population: cogniphobia, attribution of their symptoms to their injury, and forensic context. This research regarding the complex relationship between the sequalae of brain injury and fear of exacerbating symptoms through mental exertion, further underscores the point that many factors involving an individual’s own distress, beliefs and attributions, and

context can influence performance on neuropsychological assessment as well as their outcomes on PVTs.

Somatization/ Cogniform Disorders

In addition to cogniphobia there is another phenomenon, termed cogniform disorder, that occurs when cognitive impairment not explained by medical findings appears due to emotional distress. The phenomenon is similar to somatic symptom disorder within the DSM-5 where individuals have an extreme focus on physical symptoms (commonly pain or fatigue) causing distress and dysfunction in their daily lives. Somatic symptom disorder however, focuses on physical symptoms and anxiety related to those physical manifestations and dismisses any cognitive manifestations. Due to the limitation of that diagnosis, Delis and Wetter (2007) coined the term cogniform disorder (and the less pervasive subtype, cogniform condition) to encompass the cognitive presentation of the disorder. Cogniform disorder occurs when an individual that reports cognitive complaints and also shows low scores on cognitive testing which is not otherwise accounted for by medical or psychiatric conditions. Their main primary complaint is of cognitive changes, and in addition to their low cognitive scores they also have some kind of impairment in their daily life. As well, they exhibit an abnormal degree of worry about their cognitive impairment and there is lack of evidence that their low scores were produced intentionally. Therefore, it is thought that the individual's cognitive complaints and poor performance are an unconscious conversion reaction that manifest as cognitive problems (Kosaka, 2003). Cogniform disorders can also occur alongside neurological disorders such as TBI. In these cases, however, the impairment must be severe over and above what could be expected from the brain injury to be considered as cogniform disorder. For someone with an mTBI this could be, for example, loss of the ability to perform over-learned skills such as

reading, spelling or simple math (when the individual was able to do so prior and there is no history of a learning disorder). This disorder captures these phenomena that although one's injury may not be pervasive enough to cause impairment, there are other psychological mechanisms that can lead to impaired presentation in neuropsychological test data. Within the symptoms of this condition, failing PVTs is a criterion for the disorder therefore, when investigating the failure rate of those with mTBI, cogniform disorder warrants consideration.

Psychogenic Interference

The combination of psychological factors discussed above that are related to BR_{Fail} are heterogenous ranging from formal diagnoses of a disorder (Greiffenstein & Baker, 2008), self-report of psychiatric symptoms (Erdodi et al., 2018), fear of cognitive exertion bringing on physical symptoms (Suhr & Spickard, 2012), and distress about one's cognitive symptoms over-and-above what is to be expected from injury (Delis & Wetter, 2007). This results in a non-credible pattern of deficits, meaning the individual would be identified as giving an invalid performance which may mean they are presenting as more impaired than what is to be expected based on their cognitive abilities. The concept of emotional distress influencing neuropsychological testing (i.e. PVT outcomes) was explored in 2016 when Erdodi and colleagues found that individuals with a heterogenous display of psychiatric concerns (complex trauma, somatic concerns) failed PVTs at a very high rate (61-93%) in comparison to the expected 30-50% failure rate in a forensic setting (Martin et al., 2015). This phenomenon of psychological distress interfering with mental performance was referred to as "psychogenic interference" by the authors. Instead of focusing on exact psychiatric symptoms that potentially are interfering with neurocognitive performance, this term encompasses all episodes of emotional disturbance that could potentially be linked to PVT outcomes. Also, instead of

hypothesizing the reason for why each set of symptoms increase could potentially be linked to PVT failure just the knowledge that if there broadly is some form of distress it could be a factor in PVT failure holds clinical utility.

The Current Study

This research seeks to determine if the high BR_{Fail} in the mTBI sample is related to an increase of self-reported psychiatric elevations. The goal is to determine if there is a link between injury severity, psychiatric elevations, and PVT failure. The results of this study will provide important information on how factors outside of those already discovered (LEP, motivation) may influence outcomes on PVTs. It will also give insight into the perplexing phenomenon of the "reverse-dose response" relationship of TBI severity and PVT failure. The preceding literature review highlights a gap in the current neuropsychological literature and offers no explanation to the question of why individuals with mTBI have higher BR_{Fail}. The literature also suggests that patients with mTBI often report higher levels of psychiatric symptomatology than those with msTBI. Therefore, research is warranted to examine if there is a link between PVT failure and self-reported psychiatric symptoms and if this differs across injury severity levels.

In previous work there are inconsistencies that make results difficult to interpret. In many cases, researchers are not utilizing symptom validity testing, meaning they cannot be sure if respondents are accurately representing their self-reported symptoms. When utilizing self-reported measures of psychiatric symptomatology it is important to validate whether the claims of the respondent are valid. It has been repeatedly shown that individuals who are seeking compensation following mTBI engage in symptom over-reporting (Greiffenstein & Baker, 2008). To ensure we are getting an accurate estimation of their psychiatric pathology those who

fail SVTs will be grouped separately for analysis of the relationship between psychiatric symptoms and PVT outcome.

Also, much of the previous research focuses on linking PVT failures with full diagnoses of psychiatric disorders or other metrics such as previous history of psychiatric treatment (Donders et al., 2019). In this research, we will focus on self-report of broad psychiatric symptoms that can be classified under multiple disorders by using the MMPI-2. This classification of psychiatric symptoms is being used because it assesses what the individual is feeling close to the assessment time and it will capture psychiatric elevations that may otherwise be dismissed as "subclinical" or not meeting full criteria if basing our research on clinical diagnosis.

Although similar work has shown validity and clinical scales on the MMPI-2 correlate with PVT outcomes and SVT failure was more common in mTBI (Thomas & Youngjohn, 2009), the proposed study improves on this finding in multiple ways. In addition to having a substantially larger sample size, this research uses a comparison group of orthopedic controls. This control group is important because it will determine if the phenomenon is unique to individuals with TBIs including some form of alteration of cognitive and emotional functioning, or if increases of self-reported psychiatric symptoms is correlated with PVT failure even without head injury. This will indicate whether the findings are specifically related to TBI or to injury in general. We will also inspect cognitive functioning on a range of neuropsychological measures between PVT groups. This will inform us if cognitive tests scores are also different between the diagnostic categories and PVT failure groups.

The first research question is whether those with mTBI have a higher BR_{Fail} than those with msTBI or orthopedic patients. This control group adds a unique opportunity to match

participants on their level of distress from a previous injury but compares between those with TBI and those without. It is expected that similar to previous research findings, those with mTBI will fail PVTs more often than those with msTBI. It is hypothesized that because individuals within the orthopedic control group should be cognitively intact and have less psychiatric symptoms, they should have the lowest BR_{Fail}.

The second research question is whether individuals with higher self-reported psychiatric symptoms have higher BR_{Fail} than those with lower reported symptoms. It is expected from previous research that those who fail PVTs will have higher self-reported psychiatric symptoms on the MMPI-2.

The third research question is whether BR_{Fail} on PVTs across injury severity is related to self-reported psychiatric symptoms. Based on research examining each phenomenon separately, it is hypothesized that individuals who fail PVTs will be more likely to be in the mTBI group and also have higher levels of self-reported psychiatric elevations.

CHAPTER 2

Method

Participants

A dataset of 480 total individuals referred for neuropsychological or psychiatric assessment was collected by a clinical neuropsychologist within their private practice in Alberta, Canada. Participants were referred from a range of sources including Workers Compensation Board, Insurance, Legal, Doctors, Private or other specific occupational referrals. Individuals were separated into three diagnostic groups, orthopedic injuries (n=41), mTBI (n=346), and msTBI (n=93), based on their injury severity characteristics (GCS, PTA). The resultant sample

size was deemed to be sufficient based on G*Power analysis and studies on similar previously published data undertaking similar analyses across TBI severity (Abeare et al., 2019).

Measures

Minnesota Multiphasic Personality Inventory- 2 (Hathaway & McKinley, 1989) This measure was used for both the Clinical Scales and the validity scales embedded within the measure. The notable Clinical Scales used were outlined by Gass et al., (2017) as Hypochondriasis (Hs), Hysteria (Hy), Schizophrenia (Sc), Hypomania (Ma), Psychopathic Deviate (Pd), Paranoia (Pa), and Depression (De). The F scale was utilized as a proxy for symptom validity due to accessibility of the data. Although some reports indicate that the F scale is not as effective of SVT as the Fake Bad Scale (specifically in TBI populations due to the lack of somatic related items in the scale), other resources have found that it was able to identify TBI patients considered to be malingering under the Slick, Sherman and Iverson criteria (1999) with AUC of 0.79 and positive predictive powers between 0.78-0.88 (Greve et al., 2006). Therefore, for purposes of this research it was utilized as the main SVT.

Beck Depression Inventory- II (Beck, 1996) This questionnaire includes questions related to the affective, cognitive and somatic symptoms of depression and was used as a measure of self-reported depression.

Word Memory Test (Green, 1995) This measure is a free-standing performance validity measure that utilizes recall of previously seen words to determine if individuals are giving a valid performance. It has been independently validated in TBI populations (Green et al., 1999).

California Verbal Learning Test (Delis, Kramer, Kaplan & Ober, 1987) The California Verbal Learning Test (CVLT) is a list learning task that assesses an individual's learning abilities

over multiple trials. The scores that were utilized in this study are acquisition trial scores (CVLT 1-5), Long Delay free recall (CVLT LD) and recognition trial (CVLT RH).

Grooved Pegboard Test (Matthews & Klove, 1964) The Grooved Pegboard Test is a measure of manual dexterity, specifically fine motor speed. The participant is asked to put grooved pegs into angled slots as quickly as possible one at a time, incorporating a heavy spatial component along with the fine motor skills.

Finger Tapping Test (Reitan, 1969) The Finger Tapping Test (FTT) is a test of motor functioning that requires participants to tap a lever attached to a mechanical counter using their extended index finger for ten seconds at a time, over at least five consecutive trials. This procedure is then repeated for the non-dominant hand. Once the FTT is completed, the trials are averaged, which is then used as the summary score each hand (dominant and non-dominant) hands.

Trail Making Test (Reitan,1955) The Trail Making Test (TMT) is a test of attention, visuomotor and executive functioning. It has two parts, the first where the participant must connect numbers in sequence on a page. The second where the participant must connect numbers and letters in order on the page which has a greater focus on executive functioning skills and set-shifting skills.

Procedure

Participants were referred for neuropsychological assessment at a private practice in Alberta, Canada. Sessions were completed by a trained psychometrist in either one session or multiple sessions based on the individual needs of the patient. Assessment sessions consistent of different measures, depending on the clinical objective of the assessment. Data was compiled

over a 9-year span and retrieved by researchers at the University of Windsor for retrospective analyses.

Data Analysis

This was a case control design, as those with mTBI, msTBI, and an orthopedic control were compared with regard to psychiatric elevations and PVT failure. Independent *t*-test was used to analyze PVT failure group differences in neurocognitive test outcomes. ANOVA was conducted to test for diagnosis group differences on neurocognitive test scores to demonstrate external validity of previous results. Scores on the GPB, FTT, TMT were corrected using demographically adjusted norms by Heaton et al. (2004). When relevant, the base rate of failure (BR_{Fail}) were used convey the percent of the sample that failed the PVT.

For the analysis of scores on psychiatric self-report scales (MMPI-2 or BDI-II) a two-way ANOVA (Analysis of Variance) with pairwise *post hoc* contrasts was used. This method allowed both the main effects of diagnosis (Dx) and WMT outcome on psychiatric symptom report to be analyzed as well as the interaction between these two independent variables. Independent sample *t*-tests were used to compare the effect of WMT outcome on psychiatric symptom reporting at each diagnosis level. To determine if there was a difference on cognitive test performance (CVLT, TMT, GPB, FTT) between the diagnostic groups, a one-way between subjects ANOVAs were performed. All *post hoc* pairwise contrasts were uncorrected as emphasis was put on effect size allowing readers to evaluate the clinical significance of group differences without relying on p-values.

Effect size estimates are expressed in partial eta squared (η^2_p) and Cohen's d (*d)*. Independent sample *t*-tests (two-tailed) were used to analyze the effect of WMT outcome on cognitive test scores. A chi-squared test of independence was utilized to inspect of

passing/failing PVTs was related to passing/failing of SVT (MMPI-2 F Scale) with effect size estimates expressed in phi squared (Φ^2). Prior to conducting the ANOVA, the assumptions of normality, and homogeneity of variance were evaluated. The assumption of normality was violated for the tests, however, homogeneity of variance was not. Due to ANOVA being robust to violations of normality, especially when sample size is large and homogeneity of variance is not violated (Tabachnick & Fidell, 2013), the analyses were continued.

CHAPTER 3

Results

Demographics

Data for a total of 480 participants was received from an archival dataset. Clients were administered different psychiatric and neuropsychological measures depending on their referrals, resulting in small fluctuations in group size between measures. Overall, 70.4% of the participants were male, 90.9 % were right handed, the mean age was 39.3 years (*SD* = 11.9), and the mean number of years of education was 12.0 (*SD* = 2.6). In this group, 91.0% of participants identified as having English as their first language. There were no significant age differences among the diagnostic groups: orthopedic (*M* = 40.0, *SD* = 11.7), mTBI (*M* = 39.6, *SD* = 11.7), msTBI (*M* = 37.7, *SD* = 12.4), $F(2) = 1.06$, $p = .374$, η^2_p=.004 [small effect]). There were also no education difference between groups: orthopedic (*M* = 12.1, *SD* = 2.0), mTBI (*M* = 12.1, *SD* = 2.7), msTBI (*M* = 11.8, *SD* = 2.5), $F(2) = 2.45$. $p = .700$, $\eta^2_p = .001$ [small effect]). There was a significant difference for age between those who passed (*M* = 38.2, *SD* = 11.7) and failed (*M* = 41.9, *SD* = 12.0) the WMT $t(477) = 3.06$, $p = .002$, $d = 0.31$ (small effect) as well as a significant education difference between those who passed (*M* = 12.3, *SD* = 2.6) and failed (*M* = 11.5, *SD* = 2.6) PVTs $t(477) = 2.89$, $p = .004$, $d = 0.31$ (small effect).

Psychiatric Symptom Report

Analysis using a 2-way factorial ANOVA was conducted to determine the influence of the two independent variables (Dx and Pass/ Fail status on the WMT) on psychiatric symptom reporting. Significant main effects for Dx were found for BDI-II, MMPI-2 Hs, Hy scales (Tables 1 to 8). Significant main effects for WMT outcome were found for MMPI-2 Hs, De, Hy, and Ma scales. There was no interaction between the WMT and Dx for any of the psychiatric symptom report scales (Tables 1 to 8). Independent sample *t*-test revealed higher levels of psychiatric symptom reporting on all scales for those who failed the WMT in the mTBI group (*d*: 0.25-0.58), except for the Ma scale. In the orthopedic group there were higher levels of symptom reporting on the Hy, Hs, and Ma scales for those who failed the WMT.

Post hoc tests revealed significant differences only on the BDI-II between the mTBI and msTBI groups that failed the WMT ($d = 0.80$; large effect). On the MMPI-Hs scale, a significant difference was observed between mTBI and msTBI groups that passed the WMT ($d = 0.36$; small effect). On the MMPI-Hy scale, there were significant *post hoc* contrasts for some levels of Dx and WMT pass/fail status (*d*: 0.46- 1.46; medium-large effect). This excluded the orthopedic and mTBI group that passed the WMT as well as the mTBI and msTBI group that failed the WMT.

Analyses were recomputed after removing patients who had failed the MMPI F Scale in order to determine if non-credible psychiatric symptom reporting contaminated the analyses. The resulting outcomes remained the same when removing those who failed SVTs, except the main effect of Dx on the BDI-II outcome was no longer present.

The BR_{Fail} on the WMT differed across Dx on both the BDI-II [χ^2 (2, $N = 414$) = 7.50, $p = .023$, $\Phi^2 = .018$ (small effect; Table 1)], and the MMPI-2 [χ^2 (2, $N = 462$) = 8.27, $p = .016$, $\Phi^2 =$

.018 (small effect; Tables 2-7)]. Patients in the mTBI group failed the WMT at a higher rate (31.5 %) compared to msTBI group (19.6 %) but not when compared to the orthopedic group (12.9 %): χ^2 (1, N = 439) = 4.88, p = .027, Φ^2 = .011 (small effect).

Table 1

Factorial ANOVA Examining the Effect of Diagnostic Group and WMT Outcome on Scores on the Beck Depression Inventory-2

	Diagnostic Groups							Outcome Measure					
	1 ORTHO		2 *m*TBI		3 *ms*TBI			BDI-II					
	n = 22		*n* = 308		*n* = 84			ANOVA			Sig. *post*	Main Effect Pass	Main Effect Fail
WMT	M	*SD*	M	*SD*	M	*SD*	Effect	*F*	*p*	η^2_p	*hoc*s	*d*	*d*
Pass	12.8	*8.6*	15.9	*11.3*	14.9	*10.5*	Dx	4.70	.011	.023	1-2	--	--
Fail	17.0	*2.8*	22.1	*10.0*	14.1	*9.5*	WMT	1.35	.252	.003	1-3	--	--
							Interaction	2.62	.072	.013	2-3	--	0.80
t	0.68		4.77		0.29								
p	.503		<.01		.771								
d	--		0.58		--								
BR_{Fail}	9.1%		31.5%		21.4%								

Note. WMT: Word Memory Test (dichotomous outcome based on standard cutoffs); ORTHO: Orthopedic injury; *m*TBI: Mild traumatic brain injury; *ms*TBI: Moderate-to-severe traumatic brain injury; Dx: Diagnosis of ORTHO, *m*TBI or *ms*TBI group; BR_{Fail}: Base rate of failure (% of the subsample that scored in the failing range); Dx: Diagnostic group; BDI-II: Beck Depression Inventory– Second Edition; Sig. *post hoc*s: Significant *post hoc* contrasts (uncorrected *t*-tests, *p*-value <.05).

Table 2

Factorial ANOVA Examining the Effect of Diagnostic Group and WMT Outcome on Scores on the MMPI-II Hs-Scale

	Diagnostic Groups							Outcome Measure					
	1 ORTHO		2 *m*TBI		3 *ms*TBI			MMPI-2 Hs					
	n = 31		*n* = 339		*n* = 92			ANOVA			Sig. *post*	Main Effect Pass	Fail
WMT	M	*SD*	M	*SD*	M	*SD*	Effect	*F*	*p*	η^2_p	*hoc*s	*d*	*d*
Pass	72.1	*12.1*	72.4	*12.9*	67.7	*13.1*	Dx	4.34	.014	.019	1-2	--	--
Fail	86.3	*12.3*	77.9	*12.6*	72.9	*13.0*	WMT	10.01	.002	.022	1-3	--	--
							Interaction	0.76	.466	.003	2-3	0.36	--
t	2.19		3.64		1.47								
p	.037		<.001		.146								
d	1.16		0.43		--								
BR_{Fail}	12.9%		31.0%		19.6%								

Note. WMT: Word Memory Test (dichotomous outcome based on standard cutoffs); ORTHO: Orthopedic injury; *m*TBI: Mild traumatic brain injury; *ms*TBI: Moderate-to-severe traumatic brain injury; Dx: Diagnosis of ORTHO, *m*TBI or *ms*TBI group; BR_{Fail}: Base rate of failure (% of the subsample that scored in the failing range); Dx: Diagnostic group; MMPI-2: Minnesota Multiphasic Personality Inventory – Second Edition; Sig. *post hoc*s: Signficant *post hoc* contrasts (uncorrected *t*-tests, *p*-value <.05).

Table 3

Factorial ANOVA Examining the Effect of Diagnostic Group and WMT Outcome on Scores on the MMPI-II De-Scale

	Diagnostic Groups							Outcome Measure					
	1 ORTHO		2 *m*TBI		3 *ms*TBI			MMPI-2 De					
	n = 31		*n* = 339		*n* = 92			ANOVA			Sig. *post*	Main Effect Pass	Fail
WMT	M	*SD*	M	*SD*	M	*SD*	Effect	*F*	*p*	η^2_p	*hoc*s	*d*	*d*
Pass	69.6	*15.6*	68.6	*14.5*	67.7	*13.7*	Dx	1.34	.263	.006	1-2	--	--
Fail	76.5	*18.9*	75.9	*12.9*	70.3	*13.3*	WMT	3.93	.050	.009	1-3	--	--
							Interaction	0.68	.507	.003	2-3	--	--
t	0.81		4.48		0.74								
p	.424		.010		.461								
d	--		0.46		--								
BR_{Fail}	12.9%		31.0%		19.6%								

Note. WMT: Word Memory Test (dichotomous outcome based on standard cutoffs); ORTHO: Orthopedic injury; *m*TBI: Mild traumatic brain injury; *ms*TBI: Moderate-to-severe traumatic brain injury; Diagnosis of ORTHO, *m*TBI or *ms*TBI group; BR_{Fail}: Base rate of failure (% of the subsample that scored in the failing range); Dx: Diagnostic group; MMPI-2: Minnesota Multiphasic Personality Inventory – Second Edition; Sig. *post hoc*s: Signficant *post hoc* contrasts (uncorrected *t*-tests, *p*-value <.05).

Table 4

Factorial ANOVA Examining the Effect of Diagnostic Group and WMT Outcome on Scores on the MMPI-II Hy-Scale

	Diagnostic Groups							Outcome Measure					
	1 ORTHO		2 *m*TBI		3 *ms*TBI			MMPI-2 Hy					
	n = 31		*n* = 339		*n* = 92			ANOVA			Sig. *post*	Main Effect Pass	Fail
WMT	M	*SD*	M	*SD*	M	*SD*	Effect	*F*	*p*	η^2_p	*hoc*s	*d*	*d*
Pass	72.0	*14.5*	71.4	*14.9*	64.5	*14.9*	Dx	7.06	.001	.030	1-2	--	1.27
Fail	94.8	*10.7*	77.1	*16.5*	71.2	*20.2*	WMT	14.0	<.001	.030	1-3	0.51	1.46
							Interaction	2.03	.132	.009	2-3	0.46	--
t	3.00		3.14		1.61								
p	.005		.002		.112								
d	1.79		0.36		--								
BR_{Fail}	12.9%		31.0%		19.6%								

Note. WMT: Word Memory Test (dichotomous outcome based on standard cutoffs); ORTHO: Orthopedic injury; *m*TBI: Mild traumatic brain injury; *ms*TBI: Moderate-to-severe traumatic brain injury; Diagnosis of ORTHO, *m*TBI or *ms*TBI group; BR_{Fail}: Base rate of failure (% of the subsample that scored in the failing range); Dx: Diagnostic group; MMPI-2: Minnesota Multiphasic Personality Inventory – Second Edition; Sig. *post hoc*s: Signficant *post hoc* contrasts (uncorrected *t*-tests, *p*-value <.05).

Table 5

Factorial ANOVA Examining the Effect of Diagnostic Group and WMT Outcome on Scores on the MMPI-II Pd-Scale

	Diagnostic Groups							Outcome Measure					
	1		2		3			MMPI-Pd					
	ORTHO		*m*TBI		*ms*TBI						Sig.	Main Effect	
	n = 31		*n* = 339		*n* = 92			ANOVA			*post*	Pass	Fail
WMT	M	*SD*	M	*SD*	M	*SD*	Effect	*F*	*p*	η^2_p	*hoc*s	*d*	*d*
Pass	58.2	*14.3*	56.0	*12.3*	58.5	*14.0*	Dx	0.08	.924	<.001	1-2	--	--
Fail	54.8	*8.5*	59.7	*11.3*	57.1	*11.7*	WMT	0.02	.872	<.001	1-3	--	--
							Interaction	1.44	.239	.006	2-3	--	--
t	.471		2.61		.396								
p	.641		.010		.693								
d	--		0.31		--								
BR_{Fail}	12.9%		31.0%		19.6%								

Note. WMT: Word Memory Test (dichotomous outcome based on standard cutoffs); ORTHO: Orthopedic injury; *m*TBI: Mild traumatic brain injury; *ms*TBI: Moderate-to-severe traumatic brain injury; Diagnosis of ORTHO, *m*TBI or *ms*TBI group; BR_{Fail}: Base rate of failure (% of the subsample that scored in the failing range); Dx: Diagnostic group; MMPI-2: Minnesota Multiphasic Personality Inventory – Second Edition; Sig. *post hoc*s: Signficant *post hoc* contrasts (uncorrected *t*-tests, *p*-value <.05).

Table 6

Factorial ANOVA Examining the Effect of Diagnostic Group and WMT Outcome on Scores on the MMPI-2-Pa- Scale

	Diagnostic Groups							Outcome Measure					
	1		2		3			MMPI-Pa					
	ORTHO		*m*TBI		*ms*TBI						Sig.	Main Effect	
	n = 31		*n* = 339		*n* = 92			ANOVA			*post*	Pass	Fail
WMT	M	*SD*	M	*SD*	M	*SD*	Effect	*F*	*p*	η^2_p	*hoc*s	*d*	*d*
Pass	56.7	*17.5*	57.0	*14.1*	57.9	*15.7*	Dx	0.52	.594	.002	1-2	--	--
Fail	56.0	*10.0*	60.7	*15.1*	63.1	*15.6*	WMT	0.84	.359	.002	1-3	--	--
							Interaction	0.22	.800	.001	2-3	--	--
t	0.07		2.20		1.27								
p	.942		.028		.206								
d	--		0.25		--								
BR_{Fail}	12.9%		31.0%		19.6%								

Note. WMT: Word Memory Test (dichotomous outcome based on standard cutoffs); ORTHO: Orthopedic injury; *m*TBI: Mild traumatic brain injury; *ms*TBI: Moderate-to-severe traumatic brain injury; Diagnosis of ORTHO, *m*TBI or *ms*TBI group; BR_{Fail}: Base rate of failure (% of the subsample that scored in the failing range); Dx: Diagnostic group; MMPI-2: Minnesota Multiphasic Personality Inventory – Second Edition; Sig. *post hoc*s: Signficant *post hoc* contrasts (uncorrected *t*-tests, *p*-value <.05).

Table 7

Factorial ANOVA Examining the Effect of Diagnostic Group and WMT Outcome on Scores on the MMPI-2 Sc Scale

	Diagnostic Groups							Outcome Measure					
	1		2		3			MMPI-Sc					
	ORTHO		*m*TBI		*ms*TBI						Sig.	Main Effect	
	n = 31		*n* = 339		*n* = 92			ANOVA			*post*	Pass	Fail
WMT	M	*SD*	M	*SD*	M	*SD*	Effect	*F*	*p*	η^2_p	*hoc*s	*d*	*d*
Pass	65.2	*15.2*	64.6	*15.5*	65.8	*16.0*	Dx	0.53	.589	.002	1-2	--	--
Fail	64.3	*7.8*	72.7	*13.4*	69.2	*17.0*	WMT	1.31	.253	.003	1-3	--	--
							Interaction	1.09	.338	.005	2-3	--	--
t	0.12		4.64		0.79								
p	.902		<.001		.427								
d	--		0.56		--								
BR_{Fail}	12.9%		31.0%		19.6%								

Note. WMT: Word Memory Test (dichotomous outcome based on standard cutoffs); ORTHO: Orthopedic injury; *m*TBI: Mild traumatic brain injury; *ms*TBI: Moderate-to-severe traumatic brain injury; Diagnosis of ORTHO, *m*TBI or *ms*TBI group; BR_{Fail}: Base rate of failure (% of the subsample that scored in the failing range); Dx: Diagnostic group; MMPI-2: Minnesota Multiphasic Personality Inventory – Second Edition; Sig. *post hoc*s: Signficant *post hoc* contrasts (uncorrected *t*-tests, *p*-value <.05).

Table 8

Factorial ANOVA Examining the Effect of Diagnostic Group and WMT Outcome on Scores on the MMPI-II Ma-Scale

	Diagnostic Groups							Outcome Measure					
	1 ORTHO		2 *mi*TBI		3 *ms*TBI			MMPI-2 Ma					
	n = 31		*n* = 339		*n* = 92			ANOVA			Sig. *post*	Main Effect Pass	Fail
WMT	M	*SD*	M	*SD*	M	*SD*	Effect	*F*	*p*	η^2_p	*hoc*s	*d*	*d*
Pass	55.0	*11.1*	53.1	*10.7*	53.7	*11.6*	Dx	1.08	.341	.005	1-2	--	--
Fail	42.5	*4.43*	52.2	*9.9*	49.4	*9.7*	WMT	7.44	.007	.016	1-3	--	--
							Interaction	2.39	.093	.010	2-3	--	--
t	2.19		.745		1.43								
p	.037		.457		.156								
d	1.48		--		--								
BR_{Fail}	12.9%		31.0%		19.6%								

Note. WMT: Word Memory Test (dichotomous outcome based on standard cutoffs); ORTHO: Orthopedic injury; *mi*TBI: Mild traumatic brain injury; *ms*TBI: Moderate-to-severe traumatic brain injury; Dx: Diagnosis of ORTHO, *mi*TBI or *ms*TBI group; BR_{Fail}: Base rate of failure (% of the subsample that scored in the failing range); Dx: Diagnostic group; MMPI-2: Minnesota Multiphasic Personality Inventory – Second Edition; Sig. *post hoc*s: Signficant *post hoc* contrasts (uncorrected *t*-tests, *p*-value <.05).

Cognitive Performance

Scores on neurocognitive measures were compared separately using one-way ANOVAs to test for Dx and WMT group differences. A one-way ANOVA was used instead a 2- way ANOVA given the limited sample size due to the inconsistently administered tests. Performing factorial ANOVAs created cells with no participants. Performance on the CVLT 1-5 and CVLT RH were significantly different between Dx (Table 9). *Post hoc* analyses revealed that the msTBI group performed significantly worse on the CVLT 1-5 than the mTBI and orthopedic groups (*d:* 0.31-0.50; small-medium effect). On the CVLT RH those in the mTBI and msTBI group did significantly worse than the orthopedic group (*d* = 0.56; medium effect). There were no other group differences on cognitive measures. Failing the WMT was associated with lower scores on all neurocognitive tests (*d*: 0.68-0.98; medium-large effect) (Table 10).

Table 9

One Way ANOVA Examining the Effect of Diagnostic Group on Scores on Cognitive Measures

	Diagnostic Groups											
	1 ORTHO		2 *m*TBI		3 *ms*TBI					Sig. post hocs		
	n = 38		*n* = 336		*n* = 89		ANOVA					
	M	*SD*	M	*SD*	M	*SD*	*F*	*p*	η^2_p	*1-2*	*1-3*	*2-3*
TMTA	47.1	*12.5*	46.8	*11.8*	43.7	*12.0*	2.31	.100	.120	--	--	--
TMTB	47.1	*8.7*	47.7	*11.7*	45.0	*12.5*	1.61	.202	.008	--	--	--
FTT	45.9	*16.2*	48.2	*12.3*	45.6	*12.5*	1.42	.243	.009	--	--	--
GPB	41.1	*14.1*	45.5	*12.7*	42.6	*11.5*	2.38	.094	.012	--	--	--
CVLT 1-5	51.0	*9.9*	49.3	*11.8*	45.7	*11.2*	4.30	.014	.018	--	0.50	0.31
CVLT LD-FR	10.4	*3.1*	10.1	*3.5*	9.4	*3.7*	1.64	.194	.007	--	--	--
CVLT RH	14.9	*1.2*	13.8	*2.5*	13.9	*2.2*	4.06	.018	.017	0.56	0.56	--

Note. ORTHO: Orthopedic injury; *m*TBI: Mild traumatic brain injury; *ms*TBI: Moderate-to-severe traumatic brain injury; Diagnosis of ORTHO, *m*TBI or *ms*TBI group; Sample size fluctuated across measures (orthopedic: 11-38; mTBI: 235- 336; msTBI: 79-89 *n* size fluctuate between tests; TMT Trail Making Test demographically adjusted T-score (Abeare et al., 2019; Erdodi & Lichtenstein, 2020) based on norms by Heaton et al. (2004); FTT DH Finger Tapping Test dominant hand demographically adjusted T-score (Erdodi et al., 2019b) based on norms by Heaton et al. (2004); GPB DH Grooved Pegboard Test dominant hand demographically adjusted T-score (Erdodi et al., 2018c; Erdodi et al., 2017) based on norms by Heaton et al. (2004); CVLT California Verbal Learning Test, 1–5: acquisition trials (sum of raw scores across trials 1 through 5; Curtis et al., 2006; Millis et al., 1995; Shura et al., 2016; Trueblood, 1994); RH: Yes/No Recognition hits (true positives) raw score (Persinger et al., 2018; Sweet et al., 2000; Trueblood, 1994; Wolfe et al., 2010); Sig. *post hoc*s: Signficant *post hoc* contrasts (uncorrected *t*-tests, *p*-value <.05).

Table 10

Examining the Effect of Word Memory Test Pass/Fail on Cognitive Measures

	Word Memory Test						
	Pass		Fail				
	n = 335		*n* = 127				
	M	*SD*	*M*	*SD*	*t*	*p*	*d*
TMTA	48.3	*11.2*	40.5	*11.7*	6.14	<.001	0.68
TMTB	49.2	*10.5*	40.9	*12.8*	6.62	<.001	0.71
FTT	49.5	*11.1*	41.6	*12.5*	5.33	<.001	0.67
GPB	47.0	*12.4*	38.7	*10.8*	5.97	<.001	0.71
CVLT 1-5	51.5	*10.5*	41.5	*11.4*	8.90	<.001	0.91
CVLT LD-FR	10.9	*3.2*	7.7	*3.3*	9.26	<.001	0.98
CVLT RH	14.5	*1.7*	12.2	*3.1*	10.2	<.001	0.92

Note. WMT: Word Memory Test (dichotomous outcome based on standard cutoffs); Sample size fluctuated across measures (pass: 246-335; fail: 79-127); TMT Trail Making Test demographically adjusted T-score (Abeare et al., 2019; Erdodi & Lichtenstein, 2020) based on norms by Heaton et al. (2004); FTT DH Finger Tapping Test dominant hand demographically adjusted T-score (Erdodi et al., 2019b) based on norms by Heaton et al. (2004); GPB DH Grooved Pegboard Test dominant hand demographically adjusted T-score (Erdodi et al., 2018c; Erdodi et al., 2017c) based on norms by Heaton et al. (2004); CVLT California Verbal Learning Test, 1–5: acquisition trials (sum of raw scores across trials 1 through 5; Curtis et al., 2006; Millis et al., 1995; Shura et al., 2016; Trueblood, 1994); RH: Yes/No Recognition hits (true positives) raw score (Persinger et al., 2018; Sweet et al., 2000; Trueblood, 1994; Wolfe et al., 2010);

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A chi-squared test of independence was calculated to compare group membership across PVT and SVT outcomes (*Pass/Fail* status). Failing the MMPI F scale and the WMT were unrelated in the overall sample (Table 11). As previously mentioned, failing the PVT was related to Dx and those with mTBI group failed the WMT at a higher rate compared to msTBI group but not when compared to the orthopedic group. MMPI F scale *Pass/Fail* status was unrelated to group membership χ^2 (2, N = 480) = 1.63, p = .444, Φ^2 = .003 (small effect). However, there was a significant relationship between passing and failing the MMPI F scale within the mTBI group (small effect).

Table 11

Examining Word Memory Test and Minnesota Multiphasic Personality Inventory F- Scale Across Injury Severity

	Diagnostic Groups								
	ORTHO		*m*TBI		*mod-sev*TBI				
	n = 41		*n* = 345		*n* = 93				
	MMPI-2 F						Contrasts		
WMT	Pass	Fail	Pass	Fail	Pass	Fail	χ^2	*p*	Φ^2
Pass	28	5	188	68	55	20	1.99	.370	.005
Fail	6	2	50	39	13	5	.858	.651	.006
χ^2	.441		9.19		.009				
p	.507		.002		.924				
Φ^2	.011		.027		<.001				
RR	1.14		0.83		0.72				

Note. WMT: Word Memory Test (standard cutoffs); MMPI-2: Minnesota Multiphasic Personality Inventory – Second Edition; F: F Scale (*Fail* defined as T > 70); ORTH: Orthopedic injury; *m*TBI: Mild traumatic brain injury; *mod-sev*TBI: Moderate-to-severe traumatic brain injury; RR: Risk ratio.

CHAPTER 4

Discussion

PVT failure has previously been shown to vary based on injury severity level, with mTBI paradoxically having the highest BR_{Fail} (Donders, Lefebre & Goldsworthy, 2019; Abeare et al., 2019; Carone, 2008; Erdodi & Rai, 2017; Green, Flaro, & Courtney, 2009; Green, Iverson & Allen, 1999). There is accumulating evidence of a positive relationship between BR_{Fail} and self-reported psychiatric symptoms (Erdodi et al., 2017; Miskey et al., 2019). The objective of the current study was to determine if patients with mTBI who fail PVTs have higher psychiatric symptom reporting than those in other Dx groups, taking symptom validity into account, and thus offer a potential explanation for the inverse dose-response relationship between Dx and BR_{Fail}.

As predicted, WMT failure differed across Dx groups with those in the mTBI group failing more than those with msTBI. Consistent with the hypotheses, BR_{Fail} differed across Dx, driven by the significantly higher BR_{Fail} within the mTBI sample. Participants who failed the WMT had significantly higher levels of self-reported psychopathology on the MMPI-2 Hy, De, Hs, Ma scales. This finding is consistent with previous reports of a link between-psychiatric symptom reporting and PVT failure (Erdodi et al., 2017; Marcopulos et al., 2014; Miskey et al., 2019). In particular, the Hy, De, and Hs scales on the MMPI-2 have been cited as related to PVT failure in the context of the "conversion V pattern" (Boone & Lu, 1999). This pattern indicates elevations on Hy and Hs scales with a lower De score, interpreted as a tendency to express emotional difficulties as somatic symptoms. In the current findings, however, the De scale was also elevated along with the Hy and Hs scales. It is unclear in the literature if a lower De scale is necessary for this link between psychiatric symptoms and PVT failure. Some authors suggest any source of evidence that PVT failure is linked with higher levels of self-reported symptoms is supportive of the psychogenic interference hypothesis (Erdodi et al., 2016; Erdodi et al., 2018). Therefore these findings suggest that having all three scales elevated may also be linked to PVT failure.

There was also support for a relationship between Dx and psychiatric symptom reporting. Significant main effects for Dx emerged for BDI-II, MMPI-2 Hy and Hs scales. Much of this effect appeared to be driven by the differences between mTBI and msTBI groups. Notably, there was no effect of Dx on the De or Pa scales which has been shown between mTBI and msTBI groups in previous research (Gass & Russell, 1991;Youngjohn, Davis, & Wolf, 1997). However, the orthopedic group was significantly higher on the Hy scale than the other diagnostic groups. Although non-significant in *post hoc* contrasts, patients who failed WMT within the orthopedic

group also scored higher on MMPI-Hy. It has been shown that individuals seeking treatment for medical diagnoses (i.e., orthopedic injuries) are often elevated on these two MMPI-2 scales, and this high elevation can be indicative of functional factors maintaining somatic symptoms (Graham, 2000). Characteristics of those with orthopedic injuries who require psychiatric/ neuropsychological assessment may contribute to an increased levels of distress, or increased risk of non-credible presentation in this sample as compared to those with TBI.

Interestingly, there was no evidence of an interaction effect of Dx and PVT on psychiatric symptom elevations. This finding is counter to the support provided by previous studies that have linked increased PVT failure to those with higher psychiatric symptoms and TBI (Erdodi et al., 2016; Erdodi et al., 2018; Greiffenstein & Baker, 2008; Clark et al., 2014). However, there are notable design differences between the present study and previous literature on the topic. For example, in this sample self-reported symptom elevation was used instead of diagnosed disorders which may have not have been pervasive enough to interfere with PVT outcome. In addition, only one PVT was used. Although it is a highly sensitive PVT, in isolation it likely has poorer sensitivity than studies that used multiple PVTs.

There were also differences between two scales that measure symptoms of depression, the BDI-II and the MMPI-2 De scale. Previous research has shown that the MMPI-2 De scale is much more sensitive to depressive symptoms than the BDI-II (Burneo et al., 2016). This sensitivity could have conflated depressive symptomatology reporting across the groups, potentially minimizing any organic group effects in depression reporting. Alternatively, when removing those who failed the SVT (MMPI-F scale) the main effect of Dx disappeared for the BDI-II. This finding is consistent with literature comparing the two depression scales that has shown the BDI-II scores are often predicted by over-reporting scales, meaning that BDI-II scores

may be contaminated by symptom exaggeration (Burneo et al., 2016) and should be interpreted in the context of SVT.

Cognitive Performance

Another surprising finding was that neurocognitive test scores did not follow the predicted Dx gradient. The only measures that showed a significant difference among Dx were CVLT 1-5 and RH trials. This finding is counter to what has been demonstrated in previous studies (Green, 2007; Rohling et al., 2003) which may be due to sample-specific findings. For instance, in this study there was no distinction made between mild uncomplicated TBI and a more severe injury mild complicated TBI. Given that the sequalae of mcTBI more closely resemble those of MTBI, the lack of distinction could have deflated the mean and inflated SDs on cognitive test scores in this group, thereby obscuring the expected Dx group effect. Additionally, the MTBI and STBI were also combined, washing away between-group differences that could have otherwise been detected if there were greater distinction made between injury severity level. Also, BR_{Fail} in the orthopedic group did not differ significantly from the mTBI group which may explain why their cognitive test performance was poorer overall.

Contrary to our assumption that the orthopedic group in this sample would be a suitable control, the subset of individuals within the sample had some unexpected characteristics. For example, they often reported higher levels of some psychiatric symptoms or performed poorly on cognitive measures compared to those in the mTBI or msTBI groups. This finding highlights a potential selection bias: patients with orthopedic injuries were likely referred for neuropsychological testing due to complicated/atypical recovery. This suggests that these

patients were likely experiencing some sort of psychiatric and/or cognitive which may have influenced their cognitive, psychiatric, and PVT outcomes.

There was a significant difference in cognitive measures as a function of PVT outcome. Those who failed the WMT performed significantly worse on every cognitive measure. This relationship was expected as previous research has shown those who perform poorly on PVTs often have poorer outcomes across a broad range of cognitive tests (Constantinou et al., 2005; Meyers et al., 2011; West et al., 2011). Performance validity often serves as a transdiagnostic factors as it explains a higher percentage of cognitive test scores regardless of clinical characteristics.

Clinical Implications

Although no interaction between Dx and WMT failure emerged, PVT failure was related to increased levels of self-reported psychopathology on some MMPI-2 subscales. Patients with mTBI reported more symptoms of depression, hypochondriasis, and hysteria than those in the msTBI group. However, other MMPI-2 subscales did not show a link and the Dx groups did not always behave in the predicted manner as the orthopedic group was higher than both mTBI and msTBI groups on the Hy scale. The unpredicted outcome regarding the orthopedic group serves as an important reminder that "reason for referral" or "main Dx" are heterogeneous categories with highly variable clinical characteristics.

The psychogenic interference hypothesis was possibly supported as failing the PVT was related to higher symptom reporting on the Hs, De, Hy, and Ma scales. This pattern of findings may be useful to determine potential mechanisms behind PVT failure. Elevations on the these scales for those who failed the WMT may suggest that certain clinical characteristics such as being overly concerned with their health, anxiety and stress, low mood, or mania may be linked

to PVT failure. Whether this is a causal relationship or not is an important detail that is beyond the scope of the present investigation.

Results for Dx and WMT *Pass/Fail* outcomes were consistent even after removing those who failed SVTs, except for the effect of Dx on BDI-II. Due to the high transparency of the test items on the BDI-II, it is possible that patients found it easier to overreport symptoms on this scale. In other words, it is likely that the effect of PVT failure on BDI-II symptom reporting was due to symptom over-reporting and not genuine psychopathology. Lastly, PVT and SVT outcomes were unrelated, which further supports the concept of domain specificity and the need to independently assess both cognitive performance validity as well as self-reported symptom validity (Armistead-Jehle et al., 2020).

Overall, these findings are important for clinicians to consider in their practice when designing their test batteries and interpreting test scores. Knowing that some individuals with certain cluster of psychiatric symptoms are more likely to fail PVTs (and therefore, produce lower scores on measures of cognitive ability) may encourage the clinician to be aware of these factors prior to neuropsychological assessment to get the most accurate performance from the patient. Also, these results should encourage clinicians to include PVTs and SVTs in their batteries even when the patient does not necessarily belong to a clinical population that has been previously shown to be susceptible to PVT or SVT failure.

Limitations and Future Direction

The most significant limitations of this study stem from the lack of critical information due to the use of archival data. Multiple variables that would have helped the analyses by controlling for them were not available for analysis. For example, information on litigation status/external incentive was not available. This information is valuable as litigation status is

often related to PVT failure which could offer another potential link between mTBI and high PVT failure rates. Additionally, information on participant's premorbid psychiatric history was unavailable which has been more directly linked to elevated PVT failure in patients with TBI in previous studies (Armistead-Jehle, 2010; Miskey et al., 2020). Future research should control for these confounding factors to isolate their influence on PVT outcome. Another limitation of this dataset was the classification of TBI severity groups. Previous research investigating the MMPI-2 scales across brain injury severity indicates that mTBI and MTBI groups often have higher elevations on the Hy, De, and Hs scales than those with severe TBI (Ferguson & Mittenberg, 1995; Mittenberg et al., 1992; Youngjohn et al., 1997). In the archival dataset utilized in this study there was no distinction made between moderate and severe TBI meaning that some of the distinctions between those groups may have been underreported because of this. Also, traditionally STBI patients are less likely to require medicolegal evaluations which may have further obscured this Dx group differences by creating unrepresentative samples of Dx. Future research should increase the purity of criterion groups in order to tease apart these differences. This sample was also taken from one Canadian city and their psychiatric diagnoses were not known. Therefore, these results may not generalize to different geographic (Lichtenstein et al., 2019) or clinical (Glassmire et al., 2019) populations. Additionally, limitations of sample size underpowered the interaction analyses due to small group size – particularly the orthopedic PVT failure group. Lastly, only one PVT and SVT were used for classification which may have created issues with instrument artifacts. Future research should inspect this phenomenon using multiple different PVTs.

In summary, this study evaluated psychiatric symptom reporting across PVT outcome and TBI injury severity levels. Although no link between PVT outcome and Dx was found in

terms of self-reported psychiatric symptoms, each factor independently explained a significant amount of variance of psychiatric self-reporting. Future research expanding this study with different PVTs, incorporating information on premorbid psychiatric history, and distinguishing between moderate and severe TBI is warranted.

References

Abeare, C., Sabelli, A., Taylor, B., Holcomb, M., Dumitrescu, C., Kirsch, N., & Erdodi, L. (2019). The importance of demographically adjusted cutoffs: Age and education bias in raw score cutoffs within the Trail Making Test. *Psychological Injury and Law, 12*(2), 170-182.

Abeare, K., Razvi, P., Sirianni, C. D., Giromini, L., Holcomb, M., Cutler, L., ... & Erdodi, L. A. (2021). Introducing alternative validity cutoffs to improve the detection of non-credible symptom report on the BRIEF. *Psychological Injury and Law*, *14*(1), 2-16.

Aderka, I. M., McLean, C. P., Huppert, J. D., Davidson, J. R. T., & Foa, E. B. (2013). Fear, avoidance and physiological symptoms during cognitive-behavioral therapy for social anxiety disorder. *Behaviour Research and Therapy*, *51*(7), 352–358. https://doi.org/10.1016/j.brat.2013.03.007

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.

American Congress of Rehabilitation Medicine, Head Injury Interdisciplinary Special Interest Group.(1993). Definition of mild traumatic brain injury. *The Journal of Head Trauma Rehabilitation, 8*(3), 86-87.

An, K. Y., Kaploun, K., Erdodi, L. A., & Abeare, C. A. (2017). Performance validity in undergraduate research participants: A comparison of failure rates across tests and cutoffs. *The Clinical Neuropsychologist, 31*(1), 193-206.

Annegers, J. F., Hauser, W. A., Coan, S. P., & Rocca, W. A. (1998). A population-based study of seizures after traumatic brain injuries. *New England Journal of Medicine, 338*(1), 20-24.

Armistead-Jehle, P. (2010). Symptom validity test performance in US veterans referred for evaluation of mild TBI. *Applied Neuropsychology, 17*(1), 52-59.

Armistead-Jehle, P., Ingram, P. B., & Morris, N. M. (2020). Personality assessment inventory cognitive bias scale: Validation in a military sample. *Archives of Clinical Neuropsychology, 3*5(7), 1154-1161.

Ashendorf, L., Constantinou, M., & McCaffrey, R. J. (2004). The effect of depression and anxiety on the TOMM in community-dwelling older adults. *Archives of Clinical Neuropsychology*, 6.

Azouvi, P., Arnould, A., Dromer, E., & Vallat-Azouvi, C. (2017). Neuropsychology of traumatic brain injury: An expert overview. *Revue Neurologique, 173*(7-8), 461-472.

Babbage DR, Yim J, Zupan B, Neumann D, Tomita MR, Willer B. (2011). Meta-analysis of facial affect recognition difficulties after traumatic brain injury. *Neuropsychology,* 25, 277–285. doi:10.1037/a0021908

Badour, C. L., Blonigen, D. M., Boden, M. T., Feldner, M. T., & Bonn-Miller, M. O. (2012). A longitudinal test of the bi-directional relations between avoidance coping and PTSD severity during and after PTSD treatment. *Behaviour Research and Therapy*, *50*(10), 610–616. https://doi.org/10.1016/j.brat.2012.06.006

Beck, A. (1996). *Beck Depression Inventory-II Manual.* San Antonio, Tex. : Psychological Corp.

Bigler, E. D. (2008). Neuropsychology and clinical neuroscience of persistent post-concussive syndrome. *Journal of the International Neuropsychological Society, 14*(1), 1-22.

Bigler, E. D. (2014). Effort, symptom validity testing, performance validity testing and traumatic brain injury. *Brain Injury*, *28*(13-14), 1623-1638.

Bigler, E. D. (2015). Structural image analysis of the brain in neuropsychology using magnetic resonance imaging (MRI) techniques. *Neuropsychology Review*, *25*(3), 224-249.

Bodner, T., Merten, T., & Benke, T. (2019). Performance validity measures in clinical patients with aphasia. *Journal of Clinical and Experimental Neuropsychology*, *41*(5), 476-483.

Boone, K. B., & Lu, P. (1999). Impact of somatoform symptomatology on credibility of cognitive performance. *The Clinical Neuropsychologist, 13*, 414–419.

Boone, K. B. (Ed.). (2007). *Assessment of feigned cognitive impairment: A neuropsychological perspective.* The Guilford Press.

Boone, K. B. (2013). Clinical practice of forensic neuropsychology. New York, NY: Guilford.

Brodaty, H., & Connors, M. H. (2020). Pseudodementia, pseudo-pseudodementia, and pseudodepression. *Alzheimer's & Dementia : Diagnosis, Assessment & Disease Monitoring*, *12*(1). doi.org/10.1002/dad2.12027

Brown, A. W., Watanabe, T. K., Hoffman, J. M., Bell, K. R., Lucas, S., & Dikmen, S. (2015). Headache after traumatic brain injury: a national survey of clinical practices and treatment approaches. *The Journal of Injury, Function, and Rehabilitation, 7*(1), 3-8.

Burneo, D. C. S., Bowden, S. C., & Simpson, L. C. (2016). Incremental Validity of the Minnesota Multiphasic Personality Inventory, (MMPI-2) Relative to the Beck Depression Inventory-Second Edition (BDI-II) in the Detection of Depressive Symptoms. *Australian Psychologist, 5*(51), 389-399.

Carone, D. A. (2008). Children with moderate/severe brain damage/dysfunction outperform adults with mild-to-no brain damage on the Medical Symptom Validity Test. *Brain Injury*, *22*(12), 960-971.

Chafetz, M. D., Williams, M. A., Ben-Porath, Y. S., Bianchini, K. J., Boone, K. B., Kirkwood, M. W., ... & Ord, J. S. (2015). Official position of the American Academy of Clinical Neuropsychology Social Security Administration policy on validity testing: Guidance and recommendations for change. *The Clinical Neuropsychologist, 29*(6), 723-740.

Clark, A. L., Amick, M. M., Fortier, C., Milberg, W. P., & McGlinchey, R. E. (2014). Poor performance validity predicts clinical characteristics and cognitive test performance of OEF/OIF/OND Veterans in a research setting. *The Clinical Neuropsychologist, 28*(5), 802-825.

Combs, H. L., Berry, D. T. R., Pape, T., Babcock-Parziale, J., Smith, B., Schleenbaker, R., Shandera-Ochsner, A., Harp, J. P., & High, W. M. (2015). The Effects of Mild Traumatic Brain Injury, Post-Traumatic Stress Disorder, and Combined Mild Traumatic Brain Injury/Post-Traumatic Stress Disorder on Returning Veterans. *Journal of Neurotrauma, 32*(13), 956–966. https://doi.org/10.1089/neu.2014.3585

Committee on Psychological Testing, I. V. (2015). Psychological Testing in the Service of Disability Determination.

Considine, C. M., Weisenbach, S. L., Walker, S. J., McFadden, E. M., Franti, L. M., Bieliauskas, L. A., ... & Langenecker, S. A. (2011). Auditory memory decrements, without dissimulation, among patients with major depressive disorder. *Archives of Clinical Neuropsychology*, *26*(5), 445-453.

Constantinou, M., Bauer, L., Ashendorf, L., Fisher, J. M., & McCaffrey, R. J. (2005). Is poor performance on recognition memory effort measures indicative of generalized poor performance on neuropsychological tests? *Archives of Clinical Neuropsychology, 20*, 191–198.

Critchfield, E., Soble, J. R., Marceaux, J. C., Bain, K. M., Chase Bailey, K., Webber, T. A., ... & O'Rourke, J. J. (2019). Cognitive impairment does not cause invalid performance: Analyzing performance patterns among cognitively unimpaired, impaired, and noncredible participants across six performance validity tests. *The Clinical Neuropsychologist, 33*(6), 1083-1101.

Dean, A. C., Victor, T. L., Boone, K. B., & Arnold, G. (2008). The Relationship of IQ to Effort Test Performance. *The Clinical Neuropsychologist*, *22*(4), 705–722. doi.org/10.1080/13854040701440493

Dean, A. C., Victor, T. L., Boone, K. B., Philpott, L. M., & Hess, R. A. (2009). Dementia and effort test performance. *The Clinical Neuropsychologist*, *23*(1), 133–152. doi.org/10.1080/13854040701819050

Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (1987). *CVLT, California Verbal Learning Test: Adult Version: Manual*. Psychological Corporation.

Delis, D. C., & Wetter, S. R. (2007). Cogniform disorder and cogniform condition: Proposed diagnoses for excessive cognitive symptoms. *Archives of Clinical Neuropsychology, 22*(5), 589-604.

Demakis, G. J., Gervais, R. O., & Rohling, M. L. (2008). The effect of failure on cognitive and psychological symptom validity tests in litigants with symptoms of post-traumatic stress disorder. *The Clinical Neuropsychologist, 22*(5), 879-895.

DeRight, J., & Jorgensen, R. S. (2015). I just want my research credit: Frequency of suboptimal effort in a non-clinical healthy undergraduate sample. *The Clinical Neuropsychologist, 29*(1), 101-117.

Dewan, M. C., Rattani, A., Gupta, S., Baticulon, R. E., Hung, Y.-C., Punchak, M., Agrawal, A., Adeleye, A. O., Shrime, M. G., Rubiano, A. M., Rosenfeld, J. V., & Park, K. B. (2019). Estimating the global incidence of traumatic brain injury. *Journal of Neurosurgery, 130*(4), 1080–1097. doi.org/10.3171/2017.10.JNS17352

Donders, J., Lefebre, N., & Goldsworthy, R. (2019). Patterns of performance and symptom validity test findings after mild traumatic brain injury. *Archives of Clinical Neuropsychology,* 1-9

Erdodi, L. A., Nussbaum, S., Sagar, S., Abeare, C. A., & Schwartz, E. S. (2017). Limited English Proficiency Increases Failure Rates on Performance Validity Tests with High Verbal Mediation. *Psychological Injury and Law*, *10*(1), 96–103. doi.org/10.1007/s12207-017-9282-x

Erdodi, L. A., Seke, K. R., Shahein, A., Tyson, B. T., Sagar, S., & Roth, R. M. (2017). Low scores on the Grooved Pegboard Test are associated with invalid responding and psychiatric symptoms. *Psychology & Neuroscience, 10*(3), 325.

Erdodi, L. A., & Rai, J. K. (2017). A single error is one too many: Examining alternative cutoffs on Trial 2 of the TOMM. *Brain Injury, 31*(10), 1362-1368.

Erdodi, L. A., Abeare, C. A., Medoff, B., Seke, K. R., Sagar, S., & Kirsch, N. L. (2018). A single error is one too many: The Forced Choice Recognition Trial of the CVLT-II as a measure of performance validity in adults with TBI. *Archives of Clinical Neuropsychology, 33*(7), 845-860.

Erdodi, L. A., Green, P., Sirianni, C. D., & Abeare, C. A. (2019). The myth of high false-positive rates on the word memory test in mild TBI. *Psychological Injury and Law, 12*(2), 155-169.

Erdodi, L. A., Tyson, B. T., Abeare, C. A., Lichtenstein, J. D., Pelletier, C. L., Rai, J. K., & Roth, R. M. (2016). The BDAE Complex Ideational Material—A measure of receptive language or performance validity? *Psychological Injury and Law, 9*(2), 112-120.

Eysenck, M. W., Derakshan, N., Santos, R., & Calvo, M. G. (2007). Anxiety and cognitive performance: Attentional control theory. *Emotion, 7*(2), 336–353. doi.org/10.1037/1528-3542.7.2.336

Faul, M., Wald, M. M., Xu, L., & Coronado, V. G. (2010). Traumatic brain injury in the United States; emergency department visits, hospitalizations, and deaths, 2002-2006.

Gass, C. S., & Russell, E. W. (1991). MMPI profiles of closed head trauma patients: Impact of neurologic complaints. *Journal of Clinical Psychology, 47*(2), 253-260.

Gass, C. S., Rogers, D., & Kinne, E. (2017). Psychological characteristics in acute mild traumatic brain injury: An MMPI-2 study. *Applied Neuropsychology: Adult, 24*(2), 108-115.

Glassmire, D. M., Wood, M. E., Ta, M. T., Kinney, D. I., & Nitch, S. R. (2019). Examining false-positive rates of Wechsler Adult Intelligence Scale (WAIS-IV) processing speed based embedded validity indicators among individuals with schizophrenia spectrum disorders. *Psychological Assessment*, *31*(1), 120–125.

Graham, J. R. (2000). *MMPI-2: Assessing personality and psychopathology (3rd ed.).* New York: Oxford University Press

Green, P., & Astner, K. (1995). *Manual for the oral word memory tes*t. North Carolina: Cognisyst.

Green, P. (2007). The pervasive influence of effort on neuropsychological tests. *Physical medicine and rehabilitation clinics of North America, 18*(1), 43-68.

Green, P., Flaro, L., & Courtney, J. (2009). Examining false positives on the Word Memory Test in adults with mild traumatic brain injury. *Brain Injury, 23*(9), 741-750.

Green, P., Iverson, G. L., & Allen, L. (1999). Detecting malingering in head injury litigation with the Word Memory Test. *Brain Injury, 13*(10), 813-819.

Greiffenstein, M. F., & Baker, W. J. (2008). Validity Testing in Dually Diagnosed Post-Traumatic Stress Disorder and Mild Closed Head Injury. *The Clinical Neuropsychologist, 22*(3), 565–582. doi.org/10.1080/13854040701377810

Greve, K. W., Bianchini, K. J., Love, J. M., Brennan, A., & Heinly, M. T. (2006). Sensitivity and specificity of MMPI-2 validity scales and indicators to malingered neurocognitive dysfunction in traumatic brain injury. *The Clinical Neuropsychologist, 20*(3), 491-512.

Hanks, R. A., Temkin, N., Machamer, J., & Dikmen, S. S. (1999). Emotional and behavioral adjustment after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 80*(9), 991-997.

Haggerty, K. A., Frazier, T. W., Busch, R. M., & Naugle, R. I. (2007). Relationships among Victoria Symptom Validity Test indices and Personality Assessment Inventory validity scales in a large clinical sample. *The Clinical Neuropsychologist, 21*(6), 917-928.

Hathaway, S.R., & McKinley, J.C. (1989). *MMPI-2 Minnesota Multiphasic Personality Inventory-2*. Minneapolis: University of Minnesota Press.

Heaton, R. K., Gladsjo, J. A., Palmer, B. W., Kuck, J., Marcotte, T. D., & Jeste, D. V. (2001). Stability and Course of Neuropsychological Deficits in Schizophrenia. *Archives of General Psychiatry, 58*(1), 24. doi.org/10.1001/archpsyc.58.1.24

Heaton, R. K., Smith, H. H., Lehman, R. A., & Vogt, A. T. (1978). Prospects for faking believable deficits on neuropsychological testing. *Journal of Consulting and Clinical Psychology*, *46*(5), 892–900. doi.org/10.1037/0022-006X.46.5.892

Heaton, R. K., Miller, S. W., Taylor, M. J., & Grant, I. (2004). *Revised comprehensive norms for an expanded Halstead-Reitan battery: Demographically adjusted neuropsychological norms for African American and Caucasian adults.* Lutz, FL: Psychological Assessment Resources.

Henry, G. K., Heilbronner, R. L., Suhr, J., Gornbein, J., Wagner, E., & Drane, D. L. (2018). Illness perceptions predict cognitive performance validity. *Journal of the International Neuropsychological Society, 24*(7), 735-745.

Heilbronner, R. L., Sweet, J. J., Morgan, J. E., Larrabee, G. J., Millis, S. R., & Conference Participants 1. (2009). American Academy of Clinical Neuropsychology Consensus Conference Statement on the neuropsychological assessment of effort, response bias, and malingering. *The Clinical Neuropsychologist*, *23*(7), 1093-1129.

Heinrichs, R. W., & Zakzanis, K. K. (n.d.). *Neurocognitive Deficit in Schizophrenia: A Quantitative Review of the Evidence*. 20.

Hou, R., Moss-Morris, R., Peveler, R., Mogg, K., Bradley, B. P., & Belli, A. (2012). When a minor head injury results in enduring symptoms: A prospective investigation of risk factors for postconcussional syndrome after mild traumatic brain injury. *Journal of Neurology, Neurosurgery & Psychiatry*, *83*(2), 217–223. doi.org/10.1136/jnnp-2011-300767

Iverson, G. L. (2005). Outcome from mild traumatic brain injury. *Current Opinion in Psychiatry,* 18(3), 301-317.

Jak, A. J., Jurick, S., Crocker, L. D., Sanderson-Cimino, M., Aupperle, R., Rodgers, C. S., ... & Keller, A. V. (2019). SMART-CPT for veterans with comorbid post-traumatic stress disorder and history of traumatic brain injury: a randomised controlled trial. *Journal of Neurology, Neurosurgery and Psychiatry*, *90*(3), 333-341.

Jones, A., Ingram, M. V., & Ben-Porath, Y. S. (2012). Scores on the MMPI-2-RF scales as a function of increasing levels of failure on cognitive symptom validity tests in a military sample. *The Clinical Neuropsychologist, 26*(5), 790-815.

Jurick, S. M., Crocker, L. D., Keller, A. V., Hoffman, S. N., Bomyea, J., Jacobson, M. W., & Jak, A. J. (2019). The Minnesota Multiphasic Personality Inventory-2-RF in treatment-seeking Veterans with history of mild traumatic brain injury. *Archives of Clinical Neuropsychology, 34*(3), 366-380.

Kashluba, S., Hanks, R. A., Casey, J. E., & Millis, S. R. (2008). Neuropsychologic and functional outcome after complicated mild traumatic brain injury. *Archives of physical medicine and rehabilitation*, *89*(5), 904-911.

Kemp, S., Coughlan, A. K., Rowbottom, C., Wilkinson, K., Teggart, V., & Baker, G. (2008). The base rate of effort test failure in patients with medically unexplained symptoms. *Journal of Psychosomatic Research*, *65*(4), 319–325. doi.org/10.1016/j.jpsychores.2008.02.010

King, P. R., Wade, M. J., & Beehler, G. P. (2014). Health service and medication use among veterans with persistent postconcussive symptoms. *The Journal of Nervous and Mental Disease*, *202*(3), 231-238.

Kosaka, B. (2003). Cogniform disorder. In T. A. Hurwitz & W. T. Lee(Eds.), *Casebook of Neuropsychiatry* (pp. 189-197). American Psychiatric Publishing.

Larrabee, G. J. (2012). Performance validity and symptom validity in neuropsychological assessment. *Journal of the International Neuropsychological Society*, 18(4), 625.

Lee, A., Brauer Boone, K., Lesser, I., Wohl, M., Wilkins, S., & Parks, C. (2000). Performance of older depressed patients on two cognitive malingering tests: False positive rates for the Rey 15-Item Memorization and Dot Counting Tests. *The Clinical Neuropsychologist, 14*(3), 303-308.

Lichtenstein, J. D., Flaro, L., Baldwin, F. S., Rai, J., & Erdodi, L. A. (2019). Further evidence for embedded performance validity tests in children within the Conners' continuous performance test-second edition. *Developmental Neuropsychology*, 44(2), 159–171.

Maas, A. I. R., Menon, D. K., Adelson, P. D., Andelic, N., Bell, M. J., Belli, A., Bragge, P., Brazinova, A., Büki, A., Chesnut, R. M., Citerio, G., Coburn, M., Cooper, D. J., Crowder, A. T., Czeiter, E., Czosnyka, M., Diaz-Arrastia, R., Dreier, J. P., Duhaime, A.-C., & Zumbo, F. (2017). Traumatic brain injury: Integrated approaches to improve prevention, clinical care, and research. *The Lancet Neurology*, *16*(12), 987–1048. doi.org/10.1016/S1474-4422(17)30371-X

Marcopulos, B. A., Caillouet, B. A., Bailey, C. M., Tussey, C., Kent, J.-A., & Frederick, R. (2014). Clinical Decision Making in Response to Performance Validity Test Failure in a Psychiatric Setting. *The Clinical Neuropsychologist*, *28*(4), 633–652. doi.org/10.1080/13854046.2014.896416

Martin, P. K., Schroeder, R. W., & Odland, A. P. (2015). Neuropsychologists' Validity Testing Beliefs and Practices: A Survey of North American Professionals. *The Clinical Neuropsychologist*, *29*(6), 741. doi.org/10.1080/13854046.2015.1087597

Martelli, M. F., Zasler, N. D., Grayson, R., & Liljedahl, E. L. (1999). Kinesiophobia and cogniphobia: Assessment of avoidance conditioned pain related disability (ACPRD). *Neuropsychology, 14*(8), 804.

Mathias, C. W., Greve, K. W., Bianchini, K. J., Houston, R. J., & Crouch, J. A. (2002). Detecting malingered neurocognitive dysfunction using the reliable digit span in traumatic brain injury. *Assessment, 9*(3), 301-308.

Matthews, C. G., & Klove, H. (1964). *Instruction manual for the adult neuropsychology test battery*. Madison, WI: University of Wisconsin Medical School.

Menon, D. K., Schwab, K., Wright, D. W., & Maas, A. I. (2010). Position Statement: Definition of Traumatic Brain Injury. *Archives of Physical Medicine and Rehabilitation*, *91*(11), 1637–1640. doi.org/10.1016/j.apmr.2010.05.017

Meyers, J. E., Volbrecht, M., Axelrod, B. N., & Reinsch-Boothby, L. (2011). Embedded symptom validity tests and overall neuropsychological test performance. *Archives of Clinical Neuropsychology, 26*, 8–15.

Merten, T., & Merckelbach, H. (2013). Symptom validity testing in somatoform and dissociative disorders: A critical review. *Psychological Injury and Law, 6*(2), 122-137.

Messa, I., Korcsog, K., & Abeare, C. (2020). An updated review of the prevalence of invalid performance on the Immediate Post-Concussion and Cognitive Testing (ImPACT). *The Clinical Neuropsychologist,* 1-24.

Miskey, H. M., Martindale, S. L., Shura, R. D., & Taber, K. H. (2020). Distress Tolerance and Symptom Severity as Mediators of Symptom Validity Failure in Veterans With PTSD. *The Journal of Neuropsychiatry and Clinical Neurosciences*, *32*(2), 161-167.

Nelson, N. W., Sweet, J. J., Berry, D. T., Bryant, F. B., & Granacher, R. P. (2007). Response validity in forensic neuropsychology: Exploratory factor analytic evidence of distinct cognitive and psychological constructs. *Journal of the International Neuropsychological Society,13*(3), 440.

Ottenbreit, N. D., & Dobson, K. S. (2004). Avoidance and depression: The construction of the Cognitive–Behavioral Avoidance Scale. *Behaviour Research and Therapy, 42*(3), 293–313. doi.org/10.1016/S0005-7967(03)00140-2

Owens, M., Stevenson, J., Norgate, R., & Hadwin, J. A. (2008). Processing efficiency theory in children: Working memory as a mediator between trait anxiety and academic performance. *Anxiety, Stress & Coping*, *21*(4), 417–430. doi.org/10.1080/10615800701847823

Osborn, A. J., Mathias, J. L., & Fairweather-Schmidt, A. K. (2014). Depression following adult, non-penetrating traumatic brain injury: a meta-analysis examining methodological variables and sample characteristics. *Neuroscience & Biobehavioral Reviews, 47*, 1-15.

Osborn, A. J., Mathias, J. L., & Fairweather-Schmidt, A. K. (2016). Prevalence of anxiety following adult traumatic brain injury: A meta-analysis comparing measures, samples and postinjury intervals. *Neuropsychology*, *30*(2), 247.

Perry, D. C., Sturm, V. E., Peterson, M. J., Pieper, C. F., Bullock, T., Boeve, B. F., & Welsh-Bohmer, K. A. (2016). Association of traumatic brain injury with subsequent neurological and psychiatric disease: a meta-analysis. *Journal of Neurosurgery*, *124*(2), 511-526.

Ponsford, J., Cameron, P., Fitzgerald, M., Grant, M., Mikocka-Walus, A., & Schönberger, M. (2012). Predictors of postconcussive symptoms 3 months after mild traumatic brain injury. *Neuropsychology, 26*(3), 304–313. doi.org/10.1037/a0027888

Qureshi, S. U., Long, M. E., Bradshaw, M. R., Pyne, J. M., Magruder, K. M., Kimbrell, T., Hudson, T. J., Jawaid, A., Schulz, P. E., & Kunik, M. E. (2011). Does PTSD Impair Cognition Beyond the Effect of Trauma? *Journal of Neuropsychiatry*, *23*(1), 16–28. doi.org/10.1176/appi.neuropsych.23.1.16

Rao, D. P., McFaull, S., Thompson, W., & Jayaraman, G. C. (2017). Trends in self-reported traumatic brain injury among Canadians, 2005-2014: A repeated cross-sectional analysis. *Canadian Medical Association Journal*, *5*(2), 301–307. doi.org/10.9778/cmajo.20160115

Reitan, R. M. (1955). Investigation of the validity of Halstead's measures of biological intelligence. *Archives of Neurology and Psychiatry, 73*, 28-35.

Reitan. R M (1969). *Manual for administration of neuropsychological test batteries for adults and children*. Privately published by author.

Rock, P. L., Roiser, J. P., Riedel, W. J., & Blackwell, A. D. (2014). Cognitive impairment in depression: A systematic review and meta-analysis. *Psychological Medicine, 44*(10), 2029–2040. doi.org/10.1017/S0033291713002535

Rohling, M. L., Meyers, J. E., & Millis, S. R. (2003). Neuropsychological impairment following traumatic brain injury: A dose-response analysis. *The Clinical Neuropsychologist, 17*(3), 289-302.

Ross, S. R., Putnam, S. H., Gass, C. S., Bailey, D. E., & Adams, K. M. (2003). MMPI-2 indices of psychological disturbance and attention and memory test performance in head injury. *Archives of Clinical Neuropsychology*, *18*(8), 905-906.

Ruocco, A. C., Swirsky-Sacchetti, T., Chute, D. L., Mandel, S., Platek, S. M., & Zillmer, E. A. (2008). Distinguishing between neuropsychological malingering and exaggerated psychiatric symptoms in a neuropsychological setting. *The Clinical Neuropsychologist, 22*(3), 547-564.

Samuelson, K. W., Neylan, T. C., Metzler, T. J., Lenoci, M., Rothlind, J., Henn-Haase, C., Choucroun, G., Weiner, M. W., & Marmar, C. R. (2006). Neuropsychological functioning in posttraumatic stress disorder and alcohol abuse. *Neuropsychology*, *20*(6), 716–726. doi.org/10.1037/0894-4105.20.6.716

Schultz, I. Z., Sepehry, A. A., & Greer, S. C. (2018). Impact of Common Mental Health Disorders on Cognition: Depression and Posttraumatic Stress Disorder in Forensic Neuropsychology Context. *Psychological Injury and Law*, *11*(2), 139–152. doi.org/10.1007/s12207-018-9322-1

Scott, J. C., Matt, G. E., Wrocklage, K. M., Crnich, C., Jordan, J., Southwick, S. M., Krystal, J. H., & Schweinsburg, B. C. (2015). A quantitative meta-analysis of neurocognitive functioning in posttraumatic stress disorder. *Psychological Bulletin*, *141*(1), 105–140. doi.org/10.1037/a0038039

Sherer, M., Sander, A. M., Ponsford, J., Vos, L., Poritz, J. M., Ngan, E., & Novelo, L. L. (2020). Patterns of Cognitive Test Scores and Symptom Complaints in Persons with TBI Who Failed Performance Validity Testing. *Journal of the International Neuropsychological Society*, 1-7.

Slick, D. J., Sherman, E. M., & Iverson, G. L. (1999). Diagnostic criteria for malingered neurocognitive dysfunction: Proposed standards for clinical practice and research. *The Clinical Neuropsychologist, 13*(4), 545-561.

Silverberg, N. D., Iverson, G. L., & Panenka, W. (2017). Cogniphobia in mild traumatic brain injury. *Journal of Neurotrauma, 34*(13), 2141-2146.

Smith, K., Boone, K., Victor, T., Miora, D., Cottingham, M., Ziegler, E., Zeller, M., & Wright, M. (2014). Comparison of Credible Patients of Very Low Intelligence and Non-Credible Patients on Neurocognitive Performance Validity Indicators. *The Clinical Neuropsychologist, 28*(6), 1048–1070. doi.org/10.1080/13854046.2014.931465

Snyder, H. R. (2013). Major depressive disorder is associated with broad impairments on neuropsychological measures of executive function: A meta-analysis and review. *Psychological Bulletin, 139*(1), 81–132. doi.org/10.1037/a0028727

Stenberg, J., Karr, J. E., Terry, D. P., Håberg, A. K., Vik, A., Skandsen, T., & Iverson, G. L. (2020). Change in self-reported cognitive symptoms after mild traumatic brain injury is associated with changes in emotional and somatic symptoms and not changes in cognitive performance. *Neuropsychology, 34*(5), 560.

Suhr, J., & Spickard, B. (2012). Pain-related Fear is Associated with Cognitive Task Avoidance: Exploration of the Cogniphobia Construct in a Recurrent Headache Sample. *The Clinical Neuropsychologist, 26*(7), 1128–1141. doi.org/10.1080/13854046.2012.713121

Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: a practical scale. *The Lancet, 304*(7872), 81-84.

Trost, Z., France, C. R., & Thomas, J. S. (2011). Pain-related fear and avoidance of physical exertion following delayed-onset muscle soreness. *Pain, 152*(7), 1540-1547.

Udala, M., Clark, J., Miller, H., Leitner, D., & Libben, M. (2019). Psychological disturbance and cognitive functioning in TBI patients. *Applied Neuropsychology: Adult, 26*(6), 581-590.

Van Dyke, S. A., Millis, S. R., Axelrod, B. N., & Hanks, R. A. (2013). Assessing effort: Differentiating performance and symptom validity. *The Clinical Neuropsychologist, 27*(8), 1234-1246.

West, L. K., Curtis, K. L., Greve, K. W., & Bianchini, K. J. (2011). Memory in traumatic brain injury: The effects of injury severity and effort on the Wechsler Memory Scale-III. *Journal of Neuropsychology, 5*, 114–125.

Whiteside, D. M., Dunbar-Mayer, P., & Waters, D. P. (2009). Relationship between TOMM performance and PAI validity scales in a mixed clinical sample. *The Clinical Neuropsychologist, 23*(3), 523-533.

Wise, S. L., and Smith, L. F. (2011). "*A model of examinee test-taking effort," in High-Stakes Testing in Education: Science and Practice in K-12 Settings*, eds J. A. Bovaird, K. F. Geisinger, and C. W. Buckendal (Washington, DC: American Psychological Association), 139–153. doi: 10.1037/12330-009

Youngjohn, J. R., Davis, D., & Wolf, I. (1997). Head injury and the MMPI-2: Paradoxical severity effects and the influence of litigation. *Psychological Assessment, 9*(3), 177.

Appendix

Table 1. Glasgow Coma Scale

Response	Scale	Score
Eye Opening Response	Open spontaneously	4
	Open to verbal command, speech, or shout	3
	Open to pain (not applied to face)	2
	Do not open	1
Verbal Response	Oriented	5
	Confused conversation, can answer questions	4
	Inappropriate response, words dissemble	3
	Incomprehensible sounds or speech	2
	No verbal response	1
Motor Response	Obeys commands for movement	6
	Purposeful movement to painful stimulus	5
	Withdraws from pain	4
	Abnormal (spastic) flexion; decorticate posture	3
	Extensor (rigid) response; decerebrate posture	2
	No motor response	1

Table 2. Classification of Brain Injury by the American Congress of Rehabilitation Medicine

Injury Severity	LOC	GCS	PTA
Mild	LOC < 30 minutes	13-15	PTA< 24 hours
Moderate	30 minutes > LOC> 24 hours	9-12	24 hours > PTA > 7 days
Severe	LOC> 24 hours	3-8	PTA > 7 days

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