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# Language Dysfunction in Traumatic Brain Injury While Controlling for Effort

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Language Dysfunction in Traumatic Brain Injury While Controlling for Effort

A Dissertation

Submitted to the Graduate Faculty of the  
University of New Orleans  
in partial fulfillment of the  
requirements for the degree of

Doctor of Philosophy  
In  
Applied Biopsychology

by

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## Abstract

The present study included three traumatic brain injury (TBI) groups (good effort mild TBI, poor effort mild TBI, and good effort moderate/severe TBI) and two neurologic control groups (dementia and unilateral left hemisphere stroke). Language impairment was examined using the following measures: Wechsler Adult Intelligence Scale-III Verbal Comprehension Index and the Vocabulary, Similarities, Information, and Comprehension subtests; the Boston Naming Test; the Phonemic and Semantic cue conditions of the Controlled Oral Word Association Test; the Auditory Comprehension subtest of the Cognistat; Wide Range Achievement Test-3 Reading subtest; and the Peabody Picture Vocabulary Test. When effort was controlled, there was a significant effect of injury severity on language impairment. Poor effort and diagnosable malingering were responsible for most of the neuropsychological test evidence of language impairment in mild TBI.

Keywords: traumatic brain injury, effort, language, malingering



# LANGUAGE DYSFUNCTION IN TRAUMATIC BRAIN INJURY WHILE CONTROLLING FOR EFFORT

## Introduction

Traumatic brain injury has a documented course of recovery that follows a temporal and severity gradient, which has been seen in animal models at the neural level and in humans on neuropsychological evaluations. Patients with the mildest injuries recover within the first three months post-injury and should not have persistent cognitive problems, while residual problems may be present in moderate-severe TBI patients.

Some clinicians have accepted symptom presentation as evidence of impairment. The nature of neuropsychological measures is such that it requires the patient to have intact capacities, but also relies on the patient to put forth effort. This means that if someone intentionally or unintentionally puts forth poor effort, the results of the evaluation are not a true indication of that patient's capacities. As a response to this problem, measures and techniques have been developed to assess effort. Some hold the position that if effort is not controlled in clinical and research settings, it is impossible to truly understand the findings.

There have been some studies that have put this into practice in traumatic brain injury and revealed that when effort is controlled, cognitive impairment is directly associated with injury severity in a dose-response relationship. Despite the mounting evidence in support of this position, some have reported persisting language impairment in the mildest of traumatic brain injuries. Therefore, it is the purpose of the present

study to examine the effect of traumatic brain injury on language impairment while controlling for effort.

### *Traumatic Brain Injury*

Thurman, Alverson, Dunn, Guerrero, and Sniezek (1999) define traumatic brain injury as: “an occurrence of injury to the head (arising from blunt or penetrating trauma or from acceleration-deceleration forces) that is associated with symptoms or signs attributable to the injury: decreased level of consciousness, amnesia, other neurological or neuropsychological abnormalities, skull fracture, diagnosed intracranial lesions—or death” (p. 603). In penetrating head injuries (PHI) some sort of high velocity missile or sharp object has penetrated the skull and meninges and directly damaged the brain itself (for a review on PHI see Hannay, Howieson, Loring, Fischer, & Lezak, 2004). It has been reported that 41% of survivors die within 48 hours in medical care (Zafonte, Wood, Harrison-Felix, Valena, & Black, 2001). Penetrating injuries are relatively rare in civilian populations where brain trauma is usually considered “closed” and is due to blunt force.

Unlike brain injuries that have penetrated the cranial cavity, closed traumatic brain injuries are caused by some blunt force that has been applied to the head of an individual by way of contact forces, which are direct impacts to a relatively non-moving head, or inertial forces, which are related to a moving head that impacts a stationary object (Hannay et al., 2004). For the remainder of the paper, all references to traumatic brain injury will be closed TBI, unless specified. Contact forces cause trauma directly to the brain if the skull is molded inward and there may also be damage related to rebounding of brain tissue in adjacent areas. If the skull fractures, some of the

traumatic force may be dissipated, but the fracture itself may result in distributed damage and a higher risk for infection. Inertial forces include translational acceleration forces, which relates to linear movement of the head and brain, rotational acceleration forces, which describe the rotational movement of the brain around its center of gravity, and angular acceleration, which is a combination of translational and rotational acceleration, and is most often seen in impacts of the head, when the head and neck are moving, which is typical in motor vehicle accidents (Hannay et al., 2004).

### *Neuropathology*

There is evidence for both focal and diffuse injuries as a result of traumatic brain injuries. Focal injuries will be discussed first. The primary injury that may occur as a result of the application of traumatic forces to the skull is usually in the form of a contusion (Gaetz, 2004; Hannay et al., 2004). Another type of direct damage is in the form of a laceration, which may tear the pia mater or arachnoid. The two basic types of contusions are: the coup, which is located at the point of impact, and the contrecoup, which involves nonadjacent tissue and does not necessarily have to be opposite to the coup injury (Gaetz, 2004). These contusions are most often seen at the crests of the gyri, and the location of these contusions are most frequently found in the temporal poles, inferior and lateral surfaces of the temporal lobes, cortex above the Sylvian fissure, frontal poles, and the orbitofrontal cortex (Gennarelli & Graham, 1998; Hannay et al., 2004; Levin, Williams, Eisenberg, High & Guinto, 1992). These focal contusions are usually the result of the brain striking the bony protuberances of the interior of the skull during rapid deceleration injuries like those often seen in motor vehicle accidents and falls.

Direct physical trauma can also disrupt or damage blood vasculature in the brain. The hemorrhagic effects may cause either a tumor-like mass, which may exert pressure on surrounding brain tissue (Hannay et al., 2004), or it may lead to ischemic effects due to a lack of blood flow (Gennarelli & Graham, 1998).

The secondary effects of the injury are the resultant physiological processes, which may do more harm than the primary trauma (Hannay et al., 2004). The two secondary effects considered to be the most dangerous are ischemia and edema (Gaetz, 2004). Ischemia is reduced cerebral blood flow, which is not sufficient enough to meet the acute metabolic needs of brain tissue with resulting neuronal necrosis (Bullock, Maxwell, Graham, Teasdale & Adams, 1991; Hannay et al., 2004; Muizelaar, 1996; Obrist & Marion, 1996). Graham, Adams, and Doyle (1978) had illustrated the severity of ischemia, when they reported that 91% of fatal severe TBI patients had histological evidence of ischemia. Ischemia has an indirect effect in adjacent brain tissue. It leads to hypoxia-related depolarizations, which increase glutamate to neurotoxic levels due to excessive release, decreased uptake, and a positive feedback loop that is fed by an increase in the influx of calcium, which leads to further glutamate release (Choi, 1988; Gaetz, 2004; Gennarelli, 1993). Edema is also potentially fatal. If the swelling is great enough to cause a brainstem level herniation, life functions would cease (Gaetz, 2004). In 1975, Fishman delineated two primary types of edema: vasogenic, which is related to failures of the blood brain barrier; and cytotoxic, which involves intracellular swelling as a result of hypoxia (Bullock et al., 1991; Fishman, 1975), increases in extracellular excitatory neurotransmitters like glutamate and glycine (Choi, 1988), and sometimes the result of direct physical trauma, which can cause

deformations of the neuronal membrane. This last cause of cytotoxic edema leads to a massive efflux of intracellular potassium ions, which causes subsequent astrocyte swelling and damage, because the supportive glial cells are trying to maintain the appropriate levels of ion concentrations in their vicinity (Schroder, Muizelaar, Bullock, Salvant & Povlishock, 1995).

The same acceleration/deceleration forces responsible for the focal lesions (both primary and secondary effects) described above have long been associated with white matter degenerative changes (Oppenheimer, 1968; Strich, 1956, 1961). The degenerative changes are often called diffuse axonal injury (DAI) and it has been described as torn axons, retraction balls (extruded axoplasm that has leaked out of the severed segments of an axon), and swelling of the damaged axons is often reported (Hannay et al., 2004; Strich, 1961). Iverson (2005) stated that the term DAI is being phased out for the more favorable term, traumatic axonal injury (TAI).

Research over the past two decades has accumulated, which suggests that the original conception of DAI or traumatic shearing of neurons (primary axotomy) is not complete. This new research has introduced the idea that most damage is technically secondary axotomy, which is a process that occurs over hours and days after the injury and is not unlike the physiological processes tied to the secondary effects of focal injuries (Maxwell, Watt, Graham & Gennarelli, 1993). Gaetz (2004) provides a thorough review describing the time-course and the process, which is initiated by the acceleration/deceleration forces of an analogously mild injury. In this review, the author describes how secondary axotomy creates the same retraction balls, swelling, and torn axons that had been previously described in the primary axotomy literature. These

changes were related to a time-course demonstrated in animal models and human cadavers. One other interesting contribution from this research following the effects of brain injury over time is the fact that torn axons do not necessarily lead to dead neurons (Singleton, Zhu, Stone & Povlishock, 2002), and some axonal separation has been followed by regenerative actions (Mandolesi, Madeddu, Bozzi, Maffei & Ratto, 2004; Povlishock & Becker, 1985; Yaghmai & Povlishock, 1992).

All of the previous literature accepts the fact that mechanical strain is the cause of axonal injury, but the question arises as to how much injury. Gennarelli (1996) addressed this directly when he varied the amount of mechanical strain on neurons, and noted that there were four stages of traumatic axonal injury. Mild strain forces lead to Stage I damage, which involves transient ionic changes, which lead to imbalances that impair the neuron's ability to create and maintain action potentials. This is a temporary effect that resolves within minutes. Strains of slightly greater force lead to Stage II TAI, which initiates an extreme level of ionic imbalances, which is also temporary, but may lead to secondary axotomy in very few cases. Even greater mechanical strain leads to Stage III damage, which also has ionic imbalances, but it involves an irreversible influx of calcium, whose negative effects were described above. Mechanical strains, that are greater than the previous three levels, cause Stage IV damage, which is actual primary axotomy.

Gaetz (2004) summarized the literature supporting the classic view that the brainstem is often damaged leading to posttraumatic losses of consciousness. An opposing view that is receiving increasing support was first posited by Ommaya and Gennarelli (1974). These authors proposed that loss of consciousness was not

necessarily tied to damage or disruption of the brainstem alone. They felt that alterations or loss of consciousness could be explained in a graded fashion with mild traumatic forces causing mechanical strains only at the cortical level and more severe forces affecting deeper structures. Kallakuri, Cavanaugh, Özaktay and Takebayashi (2003) and Saatman, Graham, and McIntosh (1998) both demonstrated in rat models of TBI that the level of impact force is directly related to the depth of neuronal injury. Thus, a mild injury might result in a mechanical strain that may cause a transient disruption of cortical areas, which would result in an alteration or short loss of consciousness, but definitely not affect the brainstem. A severe injury could potentially cause damage as deep as the brainstem, and, hence, would result in a coma and irreversible damage. According to this model, Ommaya and Gennarelli (1974) made a particularly interesting prediction, that someone without a loss of consciousness may have cognitive symptoms like confusion and disruption of memory processes, but no one could have a loss of consciousness without acute cognitive symptoms.

In summary, the amount of force applied to the skull, whether it is in the form of an acceleration injury or a deceleration injury, is related to the severity of the injury. Greater forces lead to greater pathology. Mild forces affect cortical areas only with minimal disruption of consciousness, and may cause enough mechanical strain to disrupt neuronal processes temporarily with rapid resolution of functioning as the norm. Greater forces that may lead to more severe traumatic brain injuries are more likely to create lesions that are greater in size, and are more likely to have ischemia and edema. These forces affect cortex and underlying structures with the possibility of disruption of brainstem functioning, which leads to extended LOC. The mechanical strain on

neurons is also much greater and may result in long-term temporary changes or necrotic processes.

### *Diagnosis*

The severity of a traumatic brain injury is defined by its acute neurological characteristics. These are usually recorded by ambulance or other emergency personnel (Alexander, 1995). Dikmen, Machamer, Winn and Temkin (1995) summarized these acute characteristics: depth of coma (Alexandre, Colombo, Nertempi, & Benedetti, 1983; Dikmen, McLean, Temkin, & Wyler, 1986; Levin et al., 1990), length of coma (Dacey et al., 1991; Dikmen, Machamer, Temkin, & McLean, 1990; Dikmen, McLean, & Temkin, 1986; Levin et al., 1990), mass lesions (Alexandre et al., 1983), non-reactive pupils (Levin et al., 1990), and conditions indicative of central nervous system complications, such as, posttraumatic hydrocephalus or cardiac arrest (Dikmen et al., 1995). Posttraumatic amnesia is also another acute indicator of injury severity (Hannay et al., 2004). The Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (1993) specifically addressed criteria for the diagnosis of mild TBI, which included some of the above criteria and the addition of loss of consciousness.

The depth of coma is most commonly assessed using the Glasgow Coma Scale (GCS; Teasdale & Jennett, 1974), which measures the depth of coma by assigning a numerical value to a patient's level of responding in three different modalities: eye opening, motor activity, and verbal communication. GCS scores range from three to 15 (lower scores are indicative of a deeper coma). Patients who obtain a GCS of seven or



less are in a coma, and over 50% of all patients with a GCS of eight are also in a coma (Jennett & Teasdale, 1977). GCS scores greater than 12 are considered to reflect mild injury.

Posttraumatic amnesia (PTA) occurs in the acute phase following a traumatic brain injury, which starts with the onset of the injury, includes the coma state (if present), and usually lasts approximately four times as long as the coma itself (Brooks, 1989, as cited in Hannay et al., 2004). During PTA, the patient may be responsive, but he is confused and disoriented with an inability to encode new memories (Sherer & Madison, 2005). PTA is over when the patient exhibits continuous registration and encoding, which may be particularly difficult to determine in patients with aphasic features (Gronwall & Wrightson, 1980). In acute care and inpatient settings, patients may be continuously monitored and given serial assessments of orientation (Sherer & Madison, 2005).

Using these characteristics, brain injury severity has been divided roughly into three levels. Mild TBI or concussion has been defined by: 1) posttraumatic amnesia (PTA) not greater than 24 hours; 2) after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15; 3) loss of consciousness of 30 minutes or less [See Table 1]. Just over a decade later, the WHO Collaborating Centre for Neurotrauma Task Force on Mild Traumatic Brain Injury provided an operational definition for mild TBI, that is basically the same as the preexisting definition with the following addition: "These manifestations of MTBI must not be due to drugs, alcohol, medications, caused by other injuries or treatment for other injuries (e.g. systemic injuries, facial injuries or intubation), caused by other problems (e.g. psychological trauma, language barrier or coexisting medical

conditions) or caused by penetrating craniocerebral injury” (Carroll, Cassidy, Holm, Kraus, & Coronado, 2004, p. 115). It has been reported that 72-80% of all TBIs are of a mild severity (Kraus & Arzemanian, 1989; Kraus & Nourjah, 1988).

Table 1 (TBI severity criteria).

<i>Severity Level</i>	<i>Acute Characteristics</i>	<i>Source</i>
<i>Mild TBI/concussion</i>	GCS 13-15 PTA < 24 hours LOC ≤ 30 minutes	Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine, 1993
<i>Grade I concussion</i>	No LOC Transient confusion Concussion symptoms or mental status abnormalities that last < 15 minutes	American Academy of Neurology, 1997
<i>Grade II concussion</i>	No LOC Transient confusion Concussion symptoms or mental status abnormalities that last > 15 minutes	
<i>Grade III concussion</i>	LOC of seconds or minutes	
<i>Mild Complicated TBI</i>	GCS 13-15 PTA < 24 hours LOC ≤ 30 minutes Positive radiological findings (depressed skull fracture or intracranial lesions)	Williams, Levin & Eisenberg, 1990
<i>Moderate TBI</i>	GCS 9-12 PTA 1-7 days LOC > 30 minutes	Rimel, Giordani, Barth, & Jane, 1982
<i>Severe TBI</i>	GCS 3-8 PTA > 7 days	Sherer & Madison, 2005

Mild TBI or concussion has been further subdivided into different grades (American Academy of Neurology [AAN], 1997). The AAN defined three different grades of concussion: Grade I is defined by transient confusion but no LOC and symptoms of concussion or other mental status abnormalities that resolve within 15 minutes of the injury; Grade II is defined by transient confusion but no LOC and the concussion symptoms or mental status abnormalities last more than 15 minutes; and Grade III involves any loss of consciousness that lasts seconds or minutes.

A subset of TBI patients meet the criteria for mTBI but have acute positive radiological findings such as depressed skull fracture or intracranial lesions (Borgaro, Prigatano, Kwasnica, & Rexer, 2003; Dikmen, Machamer & Temkin, 2003; Williams, Levin & Eisenberg, 1990) [See Table 1]. These cases have been referred to as mild complicated (Williams et al, 1990). There is some evidence that this group has a neuropsychological outcome more like moderate TBI (Dikmen et al., 2003; Williams et al., 1990).

Moderate TBI is characterized by GCS scores of 9-12 (Sherer & Madison, 2005), LOC greater than 30 minutes, and/or PTA of 1 to 7 days. Severe TBI is characterized by GCS scores of 3-8 (coma), a period of unconsciousness of greater than one hour, and/or PTA of 7 or more days. Moderate and severe TBIs each comprise approximately 10% of all traumatic brain injuries (Hannay et al., 2004; Kraus, McArthur, Silverman, & Jayaraman, 1996). In the extant literature it is common for these two severity levels to be combined into one moderate/severe TBI group because researchers often have a limited number of severe cases. See Table 1 for details of the various classification schemes.

### *Cognitive and Neuropsychological Effects*

The last several years have seen the publication of several large-scale qualitative and meta-analytic reviews of the literature that have served to consolidate the findings from the best science on the cognitive and behavioral effects of TBI, particularly mild TBI. The large literature on cognitive, neuropsychological and behavioral effects of TBI was summarized by Schretlen and Shapiro (2003) in their extensive meta-analysis. Although they examined over 2000 published articles on TBI, most had significant methodological flaws and only 39 met inclusion criteria for the meta-analysis. They found no significant effect of mTBI at 90 days post-injury (effect size =  $-.04$ ), but the effect of moderate-severe TBI two years post-injury was still large (effect size =  $.84$ ).

More review articles have focused specifically on mild traumatic brain injury. In their qualitative review of prognosis in mild TBI, the World Health Organization's Mild TBI Task Force (Carroll, Cassidy, Peloso, et al., 2004) consistently found that although cognitive deficits may be present within the first few days following the mTBI, these deficits are largely resolved within the first three months post-injury. These conclusions are consistent with the nearly 20 year old findings of Binder (1986).

Two meta-analytic studies have examined the neuropsychological and cognitive effects of mild traumatic brain injury and those results are consistent with the qualitative reviews. Binder, Rohling, and Larrabee (1997) demonstrated that mild TBI did not produce a clinically significant effect on neuropsychological measures at three months post-injury. Binder et al. (1997) concluded that "the clinician assessing a case of MHT (mild head trauma) is more likely to be correct when diagnosing no brain injury and less likely to be correct when diagnosing brain injury" (Binder et al., 1997, p. 428).

Belanger, Curtiss, Demery, Lebowitz and Vanderploeg (2005) conducted a more recent meta-analysis, breaking down their results by cognitive domain and the particular type of sample. Like the other reviews, the results for unselected sample studies demonstrated no significant effect of mTBI on neuropsychological tests after 3 months. They found larger effect sizes for samples that included patients in litigation and determined that when neuropsychological impairment was present beyond three months, it was most likely due to litigation, poor effort, and/or malingering.

Rees (2003) stated that neuropsychological evaluation may be of value to objectively assess subjective cognitive complaints and warned that deficits observed on neuropsychological assessments are not specific to mTBI. He pointed out that injury-related factors (e.g. pain, stress, lack of sleep, and mood disturbances), pre-morbid psychological problems, learning difficulties, previous head injuries, and motivation/effort issues could also produce similar profiles. Iverson (2005) integrated data from a number of studies and demonstrated the relative effect sizes related to TBI (different severities and time since injury) compared to the effects of psychiatric illness, medications/drugs of abuse, and litigation/effort/malingering. The effect of mild TBI was often the smallest reported.

Regarding the methodology of many mTBI studies, Larrabee (2005) stated that the weakest designs involve clinical samples of convenience. He illustrated the strength of a prospective TBI vs. orthopedic control study (Dikmen et al., 1995) by contrasting it to two non-prospective studies that employed TBI vs. normal control designs (Guilmette & Rasile, 1995; Leininger, Gramling, Farrell, Kreutzer, & Peck, 1990). Larrabee (2005) used effect sizes to compare the results of these three studies, and found that the two

non-prospective studies would be interpreted as significant effects of mild traumatic brain injury, while the prospective study demonstrates no effect at all for mTBI.

Larrabee (2005) implies that due to the strength of the methodology, the Dikmen et al. (1995) results are more plausible, and it is possible that the effect sizes found for the other two studies may be explained by other factors such as motivational factors.

Larrabee (2005) further notes that the Leininger et al. (1990) study had very unequal numbers of non-litigating vs. litigating subjects and did not report mean and standard deviation information for these two groups, so a pattern cannot be discerned. In the Guilmette and Rasille (1995) study, Larrabee (2005) points out that they had employed symptom validity tests (described below) to screen the subjects, but did not use the symptom validity test performance to control for any effects seen between patients and normal controls.

The Dikmen et al.'s (1995) study is important due to the strength of the methodology. The independent variables in their study were measures that determine the level of severity of injury. The primary independent variable was length of coma, which was defined as the time it took to follow commands post-injury. This variable divided the TBI group into six different groups. Dikmen et al. (1995) administered a test battery that included the Halstead-Reitan Neuropsychological Test Battery and other neuropsychological measures, which served as the dependent variables. The cognitive domains assessed included: motor functioning, attention/concentration, flexibility, quickness, memory, and global verbal and performance skills (verbal and performance IQs). The relationship between these different groups and performance on the dependent measures at one year post-injury was significant for all measures. They

found a dose-response relationship between time to follow command (length of coma), which is related to injury severity, and the performance on the following neuropsychological measures: the Halstead-Reitan Neuropsychological Test Battery (Reitan & Wolfson, 1993), the Wechsler Adult Intelligence Scale-Revised (Wechsler, 1981) and the Wechsler Memory Scale-Revised (Wechsler, 1987).

In summary, the cognitive effects of a moderate to severe TBI will improve over the first two years, but, in many of these patients there will still be lingering effects that differentiate their neuropsychological test performance from normative groups. In contrast, there is consistent agreement that the brain impairment related cognitive effects of mild traumatic brain injury should be resolved by three months post-injury at the latest. The only exception for mild TBI is the subset of mild complicated injuries. Thus, with mild TBI there is an acute period wherein brain impairment related symptoms are seen and after this period persistent brain related impairment is not expected. Symptoms and deficits present after a year are not reasonably attributed to the direct neurological effects of mild TBI.

### *Effort*

#### *Assessing Effort*

Neuropsychological tests are measures of capacity and thus require full effort for accurate assessment of cognitive capacity (Bianchini, Mathias, & Greve, 2001). Clinicians are only slightly better than chance at identifying persons asked to fake neurological impairment on neuropsychological tests (Heaton, Smith, Lehman, & Vogt, 1978), and the same is true for identifying children instructed to fake neurological impairment (Faust, Hart, & Guilmette, 1988). Thus, neuropsychological patients who

wish to appear more impaired can simply not perform at their best on neuropsychological testing. In response to these concerns, neuropsychologists developed specific measures to detect effort.

Pankratz (1983) adapted a technique that had originally been used to detect patients with suspicious symptom presentations to use with persons suspected of exaggerated memory deficits. This type of measure is a forced-choice measure that presents a stimulus to the subject, followed by a delay, which is then followed by presentation of 2 stimuli that the subject will decide which of these was previously presented. These tests are usually termed symptom validity tests (SVT). The strength of SVTs is that they require cognitive effort but not cognitive ability, so failure on them is not a reflection of cognitive deficits but of poor effort (Bianchini et al., 2001). A recent study demonstrated that poor performance on recognition memory SVTs was indicative of poor performance on neuropsychological measures in general, as opposed to the idea that results on recognition memory effort tests may only be applied to other memory measures (Constantinou, Bauer, Ashendorf, Fisher, & McCaffrey, 2005).

Bianchini et al. (2001) provided a history of symptom validity testing, a comprehensive list of the literature on SVTs, and a methodology to improve the strength of SVTs in the context of medico-legal cases. Lynch (2004) summarized measures that are suitable for determining effort level within the context of neuropsychological assessments, and specifically focused on measures that were created for the purpose of determining effort (SVTs). Lynch concluded that all neuropsychological assessments should include some measure of effort. This sentiment has been reflected in a position paper published by the National Academy of Neuropsychology (Bush et al, 2005) and



another recent article (Iverson, 2006), which both discuss the importance of the inclusion of SVTs and symptom validity assessment, more broadly, as an ethical necessity within the context of neuropsychological assessment.

Some of the SVTs with the best validation and empirical support are the Portland Digit Recognition Test (PDRT; Binder, 1993a, b; Binder & Willis, 1991), the Test of Memory Malingering (TOMM; Tombaugh, 1996, 1997), the Computerized Assessment of Response Bias (CARB; Allen, Conder, Green, & Cox, 1997), and the Word Memory Test (WMT; Green, Allen, & Astner, 1996). Bianchini et al. (2001) summarized the empirical support for these and other SVTs. Two new papers provide a more current review and classification accuracy of the PDRT (Greve & Bianchini, 2006a) and the TOMM (Greve, Bianchini, & Doane, 2006). Both of these papers demonstrated that the PDRT and TOMM are not affected by more severe TBIs, and that the PDRT may be the more sensitive of the two tests to poor effort.

### *Controlling for Effort*

Green (2003) proposed that there must be a paradigm shift in neuropsychological research that addresses the issue of effort. He suggested that instead of focusing attention squarely on biological variables only, non-biological variables must also be considered. Green offered the possibility that poor effort may be responsible for impaired scores in mild TBI patients. He made an important assertion that if effort is not considered and controlled for systematically it will contaminate test data at both the single clinical case level and in group studies. This assertion gets at the heart of our understanding of traumatic brain injury and functional deficits because it

seems we know one thing, but then when effort is controlled, what we thought we knew was proven false.

The effect of controlling for effort has been investigated in sensory impairment. Green and Iverson (2001a) investigated olfactory discrimination in traumatic brain injury patients with different levels of injury severity while controlling for effort by administering two SVTs designed to detect exaggeration of cognitive functioning, and used them to divide the TBI sample into a good effort group and a poor effort group. In the poor effort group there was no significant correlation between smell test scores and injury severity level. However, in the good effort group, there was a significant correlation between injury severity and olfactory impairment.

Green, Rohling, Iverson, and Gervais (2003) also investigated olfactory test scores in TBI patients. Patients that demonstrated poor effort on the effort tests were taken out of the data, leaving a good effort only sample. In this study they demonstrated that olfactory test scores correlated highly with injury severity, and also that olfactory test scores correlated better with acute injury characteristics than neuropsychological test scores. In these two studies where effort is controlled for, there is support for the notion that there is a dose-response relationship between injury severity and amount of dysfunction, which is reminiscent of Dikmen et al.'s (1995) findings.

The effect of effort on neuropsychological test performance has also been investigated. Green and colleagues investigated the effect of effort on a neuropsychological test battery (Green, Rohling, Lees-Haley, & Allen, 2001). When the mild TBI data included those patients that had failed the effort tests, the performance of

this mild TBI group was not better than the severe TBI and neurological diseases groups. When the poor effort patient data were removed, the severe TBI and neurological disease groups performed worse than the mild TBI group (as expected). In this study the authors found that effort explained 53% of the variance in the test battery data versus 1% each for the acute characteristics (PTA, GCS, and loss of consciousness).

Binder, Kelly, Villanueva, & Winslow (2003) compared neuropsychological test performance of three groups of traumatic brain injury patients: mild TBI, financial incentive, good effort; mild TBI, financial incentive, poor effort; and moderate-severe TBI, good effort. The poor effort mild TBI group performed worse than the other two groups on tests of tactile sensory function and recognition memory. The poor effort mild TBI group was not significantly different from the moderate-severe TBI group on tests of learning and memory, tests of sensory and motor abilities, and problem solving. The findings of this study reflect those of the previous study, because they also found that effort had a stronger effect on some measures than the injury severity level.

Moss, Jones, Fokias, & Quinn (2003) controlled for effort using the TOMM and correlated the injury severity defined by the length of the PTA with patients performance on the Wechsler Memory Scale, 3<sup>rd</sup> edition (WMS-III, Wechsler, 1997a) and the IQ scores from the Wechsler Adult Intelligence Scale, 3<sup>rd</sup> edition (WAIS-III, Wechsler, 1997b). Moss et al. found that when effort was controlled a dose response relationship existed between injury severity and impairment on the administered tests, but no such relationship existed in the poor effort group. One weakness with this study is that persons passing the second trial of the TOMM were not administered the Retention trial,

thus eliminating the possibility of detecting persons that had gone on to fail the retention trial only (Greve & Bianchini, 2006b).

Green (2007) investigated the effect of effort on several common neuropsychological tests. He stratified his sample based on the level of effort given on the WMT, a computer-based SVT. He found that there was a dose-response relationship between the amount of poor effort and the level of impairment on the various neuropsychological tests.

The effect of controlling for effort has even been investigated in chronic pain. In a recent study, attention and memory was assessed in pain patients and TBI patients while controlling for effort (Curtis, Greve, & Bianchini, 2006). Effort measures were utilized to divide the pain patients into good effort and poor effort groups. The effort measures were also used to ensure that the TBI group was demonstrating good effort. The authors found that the good effort pain and good effort TBI groups did not score significantly different from each other or normative data on most of the measures of attention and memory. However, the poor effort pain group scored worse than the normative data, the good effort pain group, and even the good effort TBI group on measures assessing cognitive domains.

In summary, the SVTs described above are necessary to carry forward the suggestion of Green (2003) by controlling for effort to elucidate hidden injury severity-dysfunction correlations. This was demonstrated in a number of studies across different response domains (olfaction, general cognition, and pain). In the studies described here, it is clear that by removing or controlling effort, the level of impairment is indicative of injury severity. This raises the serious issue of what do findings in group studies of

TBI that don't control for effort demonstrate? It is possible that the findings up to this point are not accurate and need to be reexamined. There are many cognitive domains that deserve attention: memory, attention and concentration, language, problem-solving, etc. It is not practical to study all of them, so the focus of this particular study will be on language, which has the greatest historical significance in relation to brain and behavior.

### *Language Deficits*

Language disturbances are commonly reported as a result of neurological damage. Evidence of this dates back to the Edwin Smith Papyrus in the second millennium B.C. (Minagar, Ragheb, & Kelley, 2003), which describes a man who suffered a head injury and lost the ability to speak without paralysis of the tongue. Thousands of years later, patients are still reporting language deficits as a result of strokes, traumatic brain injuries (TBI), and other neurological insults. Within the past century and a half there has been a better understanding of language impairments and the neurological mechanisms behind them.

### *Language and Traumatic Brain Injury*

Most of the early aphasia literature was concerned with stroke patients investigated post-mortem. Aphasia is sometimes found in cases of PHI (Ludlow et al., 1986; Mohr et al., 1980). In fact, the earliest reports of aphasia and other language disorders resulting from traumatic brain injury included cases of penetrating head wounds (Goldstein, 1948; Luria, 1970; Russell & Espir, 1961; Schiller, 1947). Like strokes, a penetrating missile wound produces a fairly focal lesion. Luria (1970) compared language disturbances in penetrating head injuries and closed traumatic brain injuries noting little difference between the two groups immediately after the

accident. In 1983, Groher reanalyzed Luria's (1970) results and found that the open head injuries were associated with greater language deficits that lasted longer than did those in closed head injured. The focal nature of PHI may be one reason for the higher prevalence of aphasia in PHI compared to the more diffuse injuries of closed TBI that have been described above (Marquardt, Stoll, & Sussman, 1988; Murdoch, 1990).

There have been two perspectives on the nature of language impairments in closed TBI: 1) deficits seen in TBI are aphasia; 2) language impairment following TBI is caused by general cognitive disorganization as a result of the diffuse nature of these injuries (Marquardt, Stoll, & Sussman, 1988). Hagen (1984) described the language deficits in TBI as a consequence of impairment of both linguistic and basal nonlinguistic cognitive processes.

Language is a cognitive domain like those that have been described in the context of mild TBI in the sections above. Language as a cognitive domain was included in one of the previously described meta-analyses. In their meta-analysis, Belanger et al. (2005) found a significant language effect size (.64) when testing was done at less than 90 days post-injury but not for greater than 90 days (.20). These findings reflect what is known about the effect of mild TBI on cognitive domains, namely that there may be impairment post-acutely, but resolution of problems is expected within the first few months (Binder et al., 1997; Carroll et al., 2004; Iverson, 2005; Schretlen & Shapiro, 2003). Belanger et al. (2005) also found that the most commonly used measures to assess language functioning included the Controlled Oral Word Association Test (COWAT; Spreen & Strauss, 1998) and the Boston Naming Test (BNT, Kaplan, Goodglass, & Weintraub, 1983).

It is also worth mentioning that the Dikmen et al. (1995) study that was praised earlier for its methodology assessed several different neuropsychological areas in TBI patients at one year post-injury. Unfortunately, the only measure that assessed language functioning in this set of tests was a global measure of verbal abilities, the Verbal IQ score (VIQ) from the WAIS-R (Wechsler, 1981). Like many of the other measures in this study, the VIQ scores also demonstrated the characteristic dose-response curve when considered across the different severity levels based on time to follow commands.

Despite this evidence that the resolution of language impairment takes place within the first few months post-injury, there have been some sources that have reported language impairment in cases of mild TBI beyond the first few months. The first study worth mentioning was a study that investigated TBI patients across a broad range of severity as defined by the range of coma lasting from 15 minutes, which would be considered a mild TBI, to six months (Sarno, 1984). In this study Sarno found that all of the patients that had been diagnosed using neuropsychological test data were impaired on another global language measure. Raskin and Rearick (1996) found that mTBI patients were more impaired on tests of verbal fluency when compared to normal controls. This finding was replicated in Mathias and Coats (1999) study. In a widely-used neuropsychology text (Hannay et al., 2004), there is a brief case report of a patient with language impairment following a mild TBI (GCS of 14). More recently there has been a case study of a patient who suffered a mild TBI defined by a GCS of 14 and LOC of 5 minutes that had multiple impaired scores on neuropsychological language measures compared to an age and education matched normal control group (Whelan,

Murdoch, & Bellamy, 2007). All of these studies reporting impairment in mTBI did not assess effort or mention the possibility of alternative explanations.

In summary, language deficits occur as a result of focal neurological damage. Early on, aphasia was commonly studied in stroke patients. In traumatic brain injury, penetrating head injuries have been associated with patterns of language disturbances similar to the aphasias seen in stroke patients. Some have described aphasias and aphasia-like symptoms in TBI with anomia being the most frequently reported symptom. A meta-analytic study that investigated the cognitive effects of mTBI included language as one of its dependent variables, and found that language impairments may be seen early after an injury, but like other cognitive domains lasting language impairments are not expected. There have been some contrary reports of persistent language impairments in mild TBI after the acute phase. One striking feature about all of these contrary reports is that none of them had accounted for effort. Thus the purpose of this study is to address this apparent contradiction between the overall literature on the cognitive effects of TBI and the specific literature on the effect of TBI on language. Specifically, this study will examine the effect of TBI as well as other forms of neuropathology (i.e., stroke and dementia) on performance on clinical measures of language ability while controlling for the effects of effort in the TBI sample.

#### Hypotheses

1) In traumatic brain injury patients putting forth good effort, there will be a dose-response relationship between the injury severity and the amount of impairment on the language measures; this dose-response relationship will extend to the neurological control patients.



- a) Good effort mild TBI patients will score better than moderate/severe TBI patients on the language measures.
  - b) Good effort mild TBI patients will also score better on the language measures than the neurologic control patients that have been diagnosed with a unilateral stroke of the left-hemisphere or dementia.
- 2) Effort will account for more variance than severity.
- a) Poor effort mild TBI patients will perform worse than the Good effort mild TBI patients on the language measures.
  - b) Poor effort mild TBI patients will perform the same as or worse than moderate/severe TBI patients and neurologic control patients.

## Methods

### *Participants*

#### *Traumatic Brain Injury*

This sample consisted of 71 native English-speaking persons who were older than 18 years of age, but less than 50 years old, and had experienced a blunt force trauma to the head at least one year prior to the neuropsychological evaluation. Patients with less than ten years of education or more than 13 years of education were excluded. Due to the lack of normative data for other races on some of the dependent variables, only Caucasians and African-American patients were included in the study. Patients were classified into one of three groups based on injury severity and effort.

*Mild TBI groups.* Patients were included in the mild TBI groups if they suffered an independently documented blow to the head (whiplash injuries were not sufficient) and met the criteria set by the Mild Traumatic Brain Injury Committee of the Head Injury

Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine (1993): 1) posttraumatic amnesia (PTA) not greater than 24 hours; 2) after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15; 3) loss of consciousness of approximately 30 minutes or less. Patients with linear skull fractures but no intracranial findings were included; whereas, patients with intracranial findings or a depressed skull fracture were excluded. A total of 51 patients met these entry criteria. This mild TBI group was then classified into Good and Poor effort groups based on the Portland Digit Recognition Test (PDRT; Binder & Willis, 1991; Binder, 1993) and Test of Memory Malingering (TOMM; Tombaugh, 1996, 1997) [see sections below for details of this procedure]. Good effort patients passed both of these tests, while Poor effort patients failed at least one. This process resulted in a Good effort sample of 30 patients and a Poor effort sample of 21 patients.

*Moderate/Severe TBI group.* Patients were included in the moderate/severe TBI group (m/s TBI), if they suffered an independently documented blow to the head and met the following criteria for moderate and severe TBI: an initial GCS score less than or equal to 12, PTA greater than 24 hours, and/or LOC greater than 30 minutes (Rimel, Giordani, Barth, & Jane, 1982). Twenty patients met these inclusion criteria. All of these patients were determined to be giving good effort based on the PDRT and TOMM. A Poor effort m/s TBI group was considered, but only eight m/s TBI patients that met all other criteria were identified as Poor effort.

#### *Neurologic Control Groups*

Two control groups consisting of 57 patients with neurologic conditions (stroke and dementia) were included. Due to the lack of normative data for other races on

some of the variables, only native English-speaking Caucasians and African-American patients were included in the study. Patients with less than ten years of education were excluded.

*Left Hemisphere Stroke Group.* The left hemisphere stroke group (LHD) consisted of right-handed persons who were referred for neuropsychological evaluation after a unilateral left forebrain cerebral vascular accident (CVA). Patients were excluded if they had incentive, such as pursuing a disability claim. Twenty-five patients met these inclusion criteria.

*Dementia Group.* The dementia (DEM) group consisted of persons who were able to be diagnosed with dementia via neuropsychological assessment independent of their scores on language measures. Patients were included only if their diagnosis was either probable Alzheimer's disease or fronto-temporal dementia. Patients with vascular-type dementia or subcortical dementias were excluded. Thirty-two patients met these inclusion criteria.

### *Tests/Variables*

The tests were divided into three types of variables: independent (classification) variables, validation variables, and dependent variables.

#### *Classification Variables*

The following tests were used to determine whether mTBI patients were included in the Good or Poor effort groups. They were also used to determine if m/s TBI patients exhibited good effort.

*Portland Digit Recognition Test* (PDRT; Binder & Willis, 1991; Binder, 1993a, b) is a forced-choice symptom validity test that employs visual recognition of auditorily-

presented five-digit number strings. The PDRT has 72 items, and is divided into two sets of 36 items each. The first 36 items are considered the “Easy” items, and the second 36 items are considered the “Hard” items based on the apparent level of difficulty. An abbreviated version of the PDRT was sometimes administered, if the patient demonstrated mastery of the test (Binder, 1993c; Doane, Greve, & Bianchini, 2005). An individual had to obtain a score of 19 or greater on the “Easy” items in order to qualify for the abbreviated version. If this criterion was met, a score of seven out of nine or 12 of 18 on the “Hard” items had to be achieved in order to pass the abbreviated form. Since some of the patients met the abbreviated form criteria, all PDRT administrations included the “Easy” items but not all of them included every “Hard” item. Using the published cut-offs of Binder and Kelly (1996), patients with scores of less than 19 on the “Easy”, less than 18 on the “Hard”, or less than 39 on the Total score were classified as giving poor effort.

*Test of Memory Malingering* (TOMM; Tombaugh, 1996, 1997) is a three-trial forced-choice symptom validity test that employs visual recognition of line drawings of common objects. In the first two trials, 50 items were presented followed by a two-choice recognition test for each trial. After a delay of 15 minutes, a retention trial was administered without further training. Patients scoring <45 on Trial 2 or the Retention Trial were classified as giving poor effort according to the published recommendations (Tombaugh, 1996).

#### *Dependent Variables*

*Effort Validation Tests.* Reliable Digit Span (RDS; Greiffenstein, Baker, & Gola, 1994) and the Fake Bad Scale (FBS; Lees-Haley, English, & Glenn, 1991) of the

Minnesota Multiphasic Personality Inventory-2 (MMPI-2; Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989) were used to assess whether the TBI patients were appropriately assigned to the good or poor effort groups.

The *Reliable Digit Span* is derived from the WAIS-III Digit Span subtest and it is calculated by summing the last forward and backward digit strings in which both trials were completed without error. RDS is an embedded validity indicator that capitalizes on being perceived as a memory test, however, research has shown that it is unaffected by brain trauma (Greiffenstein et al., 1994; Heinly, Greve, Bianchini, Love, & Brennan, 2005; Iverson & Franzen, 1996; Iverson & Tulsky, 2003). A score of six or less on RDS is associated with 39% sensitivity to malingering and a false-positive rate of only 4% (Heinly et al., 2005). These findings have been replicated in chronic pain (Etherton, Bianchini, Greve & Heinly, 2005) and toxic exposure (Greve et al., 2007).

The *Fake Bad Scale* is comprised of 43 MMPI-2 items and is useful in detecting exaggerated claims of disability particularly in forensic settings. In a meta-analysis of FBS, Nelson, Sweet, and Demakis (2006) found that FBS had the largest effect size (.96) of all the MMPI-2 validity scales in groups over-reporting symptoms compared to comparison groups. The recommended cut-offs for FBS are: > 22 is an indicator of threatened validity; and >28 is an indicator of invalidity (Ben-Porath & Tellegen, 2007).

*Language Measures.* The following tests were chosen because they address different language capacities, such as: comprehension, naming, and reading. They were also chosen, because of their frequency of use by many neuropsychologists in clinical and research settings (Rabin, Barr, & Burton, 2005). The most frequently used test in neuropsychological clinics and research was the Wechsler Adult Intelligence

Scale, 3<sup>rd</sup> edition (WAIS-III, Wechsler, 1997b), which is where the first five of the following language measures originate.

1. *Verbal Comprehension Index (VCI)* is an index score of the WAIS-III, and it is a general language measure that encompasses verbal conceptualization, expression, and knowledge. VCI is comprised of the three subsequent subtests.

2. *Vocabulary (Voc)* subtest assesses the patient's recall vocabulary. Patients were visually and verbally presented words and were asked to define them.

3. *Information (Inf)* subtest assesses the patient's ability to answer some general knowledge questions. Patients were presented with questions that address learned knowledge.

4. *Similarities (Sim)* subtest assesses the patient's ability to describe how two things are alike. Patients were verbally presented with two-word items that increase in difficulty from concrete to abstract and were asked how they are similar.

5. *Comprehension (Comp)* subtest assesses verbal reasoning regarding socially relevant questions and understanding of proverbs. Patients were verbally presented these different questions.

6. *Boston Naming Test 2<sup>nd</sup> edition (BNT; Kaplan, Goodglass & Weintraub, 1983)* is a confrontation naming test that includes 60 line drawings of objects. If the participant could not recall the object name directly, the participant was given a semantic cue followed by a phonemic cue. When phonemic cues were presented, the item was coded as incorrect, whether the response was correct or not.

7. The *Phonemic cue condition (Phon)* of the *Controlled Oral Word Association Test (COWAT; Spreen & Strauss, 1998)* is a word fluency test. Patients were

presented three different trials, where they were given 60 seconds to produce as many words that begin with a particular letter with the exception of proper words (ones that begin with a capital letter) and different forms of the same word (e.g. have, has, had).

8. The *Semantic cue condition* (Sem) of the *COWAT* is also a word fluency test. Patients were administered one trial where they were required to name as many animals as they could in 60 seconds.

9. The *Auditory Comprehension* (AC) subtest of the *Neurobehavioral Cognitive Status Examination* (Cognistat; Kiernan, Mueller, & Langston, 1995) assesses the patients' ability to comprehend and carry out commands. During this subtest, the examinee was presented with the screening item, which was a simple three-step command. If the examinee passed the screen a perfect score was recorded. If the examinee did not pass the screen, the examiner proceeded to read commands ranging from one to three steps.

10. The *Reading* subtest of the *Wide Range Achievement Test, 3<sup>rd</sup> edition* (WRAT-3; Wilkinson, 1993) assesses reading recognition ability. Patients were presented with a card that has 42 words on it, and they were asked to read each word. The WRAT-3 Reading subtest is often used as an estimate of pre-morbid intellectual functioning (Ball, Hart, Stutts, Turf, & Barth, 2007; Orme, Johnstone, Hanks, & Novack, 2004).

11. *Peabody Picture Vocabulary Test-III* (PPVT-III; Dunn & Dunn, 1997) is a measure that assesses receptive vocabulary. The PPVT-III consists of 204 picture plates, with four pictures per plate. The examiner read from a word list and the participant was asked to indicate which of the four pictures best characterized the word

that was read. The PPVT-III is often used to estimate verbal intelligence (Bell, Lassiter, Matthew, & Hutchinson, 2001; Smith, 1997).

T-scores that were corrected for age, race, sex, and education were calculated for the WAIS-III scores, the BNT, Phon and Sem of the COWAT. T-scores that were corrected only for age were calculated for AC using normative data. Standard scores were calculated for the WRAT-3 Reading subtest and the PPVT using the normative data that only corrected for age and not for other demographic factors, the most important of which is education. All of the sources of the normative data used for these score calculations are listed in Table 2.

Table 2 (Sources of normative data for the language measures).

Test	Source
WAIS-III	Taylor and Heaton, 2001
BNT Phonemic and Semantic cues of the COWAT	Heaton, Miller, Taylor, and Grant, 2004
Cognistat Auditory Comprehension	The Northern California Neurobehavioral Group, Inc, 1988
WRAT-3 Reading	Wilkinson, 1993
PPVT	Dunn & Dunn, 1997

### *Procedure*

The data were retrospectively collected from patients seen for neuropsychological evaluations at a Southeastern United States Psychology/Neuropsychology clinic. The tests examined in this study are part of the routine neuropsychological evaluation for which these patients were referred. With the exception of the TOMM administered to some dementia patients, these tests were not included specifically for research purposes. The TOMM was administered to the dementia patients as part of an earlier study (Greve, Bianchini, & Doane, 2006). TBI



patients were referred by attorneys, physicians, rehabilitation professionals, or worker's compensation case managers. The neurologic controls were referred by physicians or rehabilitation professionals. Only the TBI patients had external incentive (i.e. worker's compensation claims, personal injury lawsuits, etc.). Approximately 500 medical records were reviewed for the purposes of selecting individuals that met inclusion criteria and determining the appropriate group assignment.

### *Analysis*

Nominal variables (i.e. gender and race) were analyzed with a Chi-square ( $\chi^2$ ) analysis. Group effects for continuous variables were examined with univariate analyses of variance (ANOVAs), and post-hoc comparisons were performed using the Tukey B procedure. The assumption of homogeneity of variance was tested for all ANOVAs that were conducted. If the assumption was not met, ANOVAs were still run, since the ANOVA is robust to violations, and they were followed by a non-parametric Kruskal-Wallis test to confirm the results of the ANOVA. The ANOVA results were considered accurate if the group differences were maintained in the non-parametric procedure. The post-hoc analyses for these analyses were performed using the Dunnet's C procedure, because this procedure does not assume homogeneity of variance.

The dependent language variables were not analyzed with a multivariate analysis of variance (MANOVA) due to the unequal group sizes across variables. The use of a MANOVA would have resulted in the exclusion of some of the patient's test scores, so multiple univariate ANOVAs were conducted instead to ensure that all scores would be included. A statistical method was used to account for the increased possibility of a

Type 1 error that could occur as a result of conducting multiple ANOVAs. The method maintained experiment-wise error rates at the desired alpha level while not affecting the power of the study (Jaccard & Guilamo-Ramos, 2002). The Holm (1979) method is a step-down Bonferroni-based approach to setting the alpha level. The  $p$  values obtained from the analyses were ordered from smallest to largest. Then, the smallest  $p$  value was compared to an alpha level of  $.05/k$ , where  $k$  was the number of analyses (11 for the present study). Each subsequently higher  $p$  value was compared to an alpha level of  $0.5/k-1$ ,  $0.5/k-2$ , etc. until there was a non-significant finding.

## Results

### *Demographics*

The sample included 30 Good mTBI, 21 Poor mTBI, 20 m/s TBI, 25 LHD, and 32 DEM patients. Some of the LHD and DEM patients were not administered all of the measures. Table 3 provides the number of patients per group for each test, and Table 4 presents the demographic data.

Demographic variables were statistically analyzed to verify that the groups were sufficiently matched on these variables. There was a significant group effect for age ( $F [4,123] = 109.30, p < .001, partial\ eta^2 = .78$ ) with the m/s TBI group significantly younger than the Good and the Poor mTBI groups and the LHD group significantly older and the DEM group older still. There was also a significant group effect for time since injury ( $F [3, 92] = 9.28, p < .001, partial\ eta^2 = .23$ ) and education ( $F [4,123] = 8.37, p < .001, partial\ eta^2 = .21$ ). On time since injury, the LHD group was seen significantly earlier post-injury than the TBI groups, and were often seen post-acutely on the hospital rehabilitation unit. The LHD and DEM groups had significantly more education than all

Table 3 (Number of patients per group that were administered each measure).

	Good	m/s	Poor	LHD	Dem
PDRT	30	20	21	0	0
TOMM	30	20	21	0	17
RDS	30	20	21	23	32
FBS	30	20	21	12	13
WAIS-R	0	0	0	10	0
WAIS-III	30	20	21	12	25
BNT	30	20	21	23	30
Phon	30	20	21	25	31
Sem	30	20	21	24	31
Cognistat	30	19	21	23	28
WRAT-3	30	20	21	22	30
PPVT	28	19	19	7	20

*Note:* Good = good effort mild TBI, m/s = moderate/severe TBI, Poor = poor effort mild TBI, LHD = Left-hemisphere damaged CVA, Dem = dementia; PDRT = Portland Digit Recognition Test, TOMM = Test of Memory and Malinger, RDS = Reliable Digit Span, FBS = Fake Bad Scale, WAIS-R = Wechsler Adult Intelligence Scale-Revised, WAIS-III = Wechsler Adult Intelligence Scale-3<sup>rd</sup> ed., BNT = Boston Naming Test, Phon = Phonemic cue condition of COWAT, Sem = Semantic cue condition of COWAT, WRAT-3 = Wide Range Achievement Test-3<sup>rd</sup> ed., PPVT = Peabody Picture Vocabulary Test.

of the TBI groups, which did not differ. The sexes were unequally represented in the sample ( $X^2 [4] = 15.61, p < .01$ ) and the same is true of race ( $X^2 [4] = 12.58, p < .05$ ).

The same inequality was seen for all groups on both variables with the exception of the dementia group countering the trend of more males (only 34.4% males). Therefore, the only demographic variable that the TBI patients differed on was age, and the neurologic control groups differed in that they were older, more educated, and the LHD patients were seen earlier in their recovery.

### *Injury Characteristics*

It is also important to verify that the mTBI cases (Good and Poor) differ from the m/s TBI cases, but do not differ from each other on variables related to the severity of

Table 4 (Demographic variables).

	Good	m/s	Poor	LHD	DEM
	m (sd)	m (sd)	m (sd)	m (sd)	m (sd)
Age	37.9 (9.0) <sup>a</sup>	25.5 (6.1) <sup>b</sup>	37.9 (8.4) <sup>a</sup>	54.0 (10.8) <sup>c</sup>	71.3 (8.3) <sup>d</sup>
Education	11.8 (.8) <sup>a</sup>	11.7 (1.0) <sup>a</sup>	11.7 (.8) <sup>a</sup>	13.8 (3.3) <sup>b</sup>	13.8 (2.6) <sup>b</sup>
Time Since Injury (months)	33.8 (16.8) <sup>a</sup>	38.1 (15.3) <sup>a</sup>	27.9 (13.3) <sup>a</sup>	10.0 (29.3) <sup>b</sup>	n/a
% male	70.0	75.0	71.4	76.0	34.4
% Caucasian	76.7	70.0	76.2	92.0	100
GCS*	14.9 <sup>a</sup> (.3)	5.4 <sup>b</sup> (3.1)	14.8 <sup>a</sup> (.6)	-	-

Note: Good = good effort mild TBI, m/s = moderate/severe TBI, Poor = poor effort mild TBI, LHD = Left-hemisphere damaged CVA, Dem = dementia; m = mean, sd = standard deviation; GCS = Glasgow Coma Scale.

<sup>abc</sup> row means with the same letter are not significantly different from each other.

\* Good n = 19, m/s n = 16, Poor n = 14.

the injury. GCS is a measure of depth of coma, that was not reported for all of the TBI patients, but it was used to define most of the TBI patients into the appropriate severity groups (mild vs. m/s). There was a significant group effect for GCS ( $F [2, 46] = 144.54$ ,  $p < .001$ ,  $partial\ eta^2 = .86$ ), but the Good mTBI and Poor mTBI groups did not differ from each other (see Table 4). The two mTBI groups did not differ in the frequency of reported loss of consciousness (LOC; Good mTBI = 38%, Poor mTBI = 27%) or post-traumatic amnesia (PTA; Good mTBI = 10%, Poor mTBI = 5%). The m/s TBI patients had 90% of the group with reported LOC and 80% with a PTA (see Table 5).

Besides acute characteristics, there are other sources of evidence of potential neuropathology. Table 5 presents a summary of the number of patients per group that had positive findings on these different indicators (brain scan findings, focal neurological

Table 5 (Injury characteristics).

	Good		m/s		Poor		LHD		DEM	
	n	%	n	%	n	%	n	%	n	%
Loss of consciousness	12	(40)	18	(90)	5	(24)	-		-	
Post-traumatic amnesia	3	(10)	16	(80)	1	(5)	-		-	
Brain Scan	0	(0)	18	(90)	0	(0)	23	(92)	12	(38)
Focal Signs	0	(0)	13	(65)	0	(0)	23	(92)	0	(0)
Skull fracture	1	(3)	10	(50)	0	(0)	-		-	
Electroencephalogram	1	(3)	4	(20)	1	(5)	0	(0)	1	(3)
Seizures	0	(0)	5	(25)	0	(0)	0	(0)	0	(0)
Neurosurgery	0	(0)	12	(60)	0	(0)	9	(36)	0	(0)

*Note:* Good = good effort mild TBI, m/s = moderate/severe TBI, Poor = poor effort mild TBI, LHD = Left-hemisphere damaged CVA, Dem = dementia; n = number of patients with positive finding, % = percentage of patients with positive findings signs [e.g. hemiparesis, unilateral pupillary response dysfunction, etc.], skull fracture,

electroencephalogram findings [EEG], seizures, and neurosurgery). The Good and Poor mTBI patients do not have more than 10% of their respective groups with evidence from any of these indicators. These data combined with the lack of differences in the acute characteristics (GCS, LOC, or PTA) indicate that the Poor mTBI group was not injured more severely than the Good mTBI group, but both groups were definitely less severely injured than the m/s TBI, LHD, and DEM groups, which had multiple pieces of evidence indicating neuropathology.

#### *Effort Validation*

Although the Good and Poor effort groups were separated based on TOMM and PDRT scores, the groups were compared to see how much they differed on these

classification variables. Since the Easy items were the only portion of the PDRT that was administered to all that had taken the test, this was the only portion that could be analyzed. A univariate ANOVA demonstrated a significant group effect ( $F [2, 68] = 60.07, p < .001, \text{partial } \eta^2 = .64$ ) with the Poor mTBI group significantly worse than all of the other groups, which did not differ from each other. As noted in Table 3 there was a group of DEM patients that had taken the TOMM, which was part of another study (Greve, Bianchini, & Doane, 2006), so they were included in TOMM analyses. There was a significant group effect for Trial 2 ( $F [3, 84] = 28.02, p < .001, \text{partial } \eta^2 = .51$ ) with the Poor mTBI group scoring significantly worse than the other TBI groups and the DEM group, and the same pattern of performance was seen for the Retention Trial ( $F [3, 83] = 28.96, p < .001, \text{partial } \eta^2 = .51$ ). All of the TOMM ANOVAs did not pass the assumption of homogeneity of variance, so the results of each of these analyses were confirmed with nonparametric analyses. These findings demonstrate that the groups do differ on SVT performance with the Poor mTBI group performing more poorly than the m/s TBI patients and the dementia patients on the TOMM. [see Table 6 for a summary of these variables]

A recent study found that SVTs like the PDRT and TOMM may accurately detect persons putting forth poor effort, but they also miss a substantial portion of persons that were also putting forth poor effort (Greve, Ord, Curtis, Bianchini, & Brennan, in press). Another recent paper suggests the use of multiple validity indicators to increase the probability of detecting persons putting forth poor effort (Larrabee, in press). RDS and FBS are two such validity indicators, and they have been included in the present study

to validate the classification of the TBI groups as Good or Poor effort. In the TBI patients only, there was a significant group effect for RDS ( $F [2, 68] = 6.37, p < .01$ ,  $partial\ eta^2 = .16$ ) with the Poor mTBI group scoring significantly worse than the other TBI groups, and FBS also produced a significant group effect ( $F [2, 68] = 21.79, p < .001, partial\ eta^2 = .39$ ) with the Good mTBI and Poor mTBI groups scoring significantly worse than the m/s TBI group. These validation measures were available for most of the LHD and DEM patients, so their scores were included in analyses to see how they might differ from the TBI groups. After including the control groups, there was a significant group effect for RDS ( $F [4, 121] = 3.31, p < .05, partial\ eta^2 = .10$ ) with the Poor mTBI group performing significantly worse than only the Good mTBI group. There was also a significant group effect for FBS ( $F [4, 91] = 22.44, p < .001, partial\ eta^2 = .50$ ) with the Good mTBI and Poor mTBI groups scoring significantly worse than the m/s TBI, LHD, and DEM groups. Table 6 summarizes these findings, and Table 7 presents the number and percentage of patients per group that scored beyond published cut-offs on the classification variables and effort validation tests. The Poor mTBI group had the highest percentage of patients scoring beyond the cut-offs on all of these measures. These findings indicate that the Poor mTBI patients were correctly classified as not giving good effort. The RDS findings also demonstrated that RDS has utility for detecting poor effort in mTBI, but scores for patients with actual neuropathology fall somewhere between good and poor effort mTBI performance. The Good mTBI patients may have significantly more exaggerated claims of disability than the m/s TBI patients as demonstrated on the FBS, but not at the level of the Poor mTBI patients.

Table 6 (Effort classification and effort validation variables).

	Good	m/s	Poor	LHD	DEM
Classification Variables	m(sd)	m (sd)	m (sd)	m (sd)	m (sd)
PDRT Easy	29.4(3.3) <sup>a</sup>	29.6 (3.1) <sup>a</sup>	17.9 (5.5) <sup>b</sup>	n/a	n/a
TOMM Trial 2	49.7(1.0) <sup>a</sup>	49.60 (1.00) <sup>a</sup>	33.3 (13.8) <sup>b</sup>	n/a	48.0(3.8) <sup>a</sup>
TOMM Retention	49.4(1.1) <sup>a</sup>	49.65 (.81) <sup>a</sup>	32.8 (13.5) <sup>b</sup>	n/a	47.9(5.2) <sup>a</sup>
Effort Validation Tests					
RDS	9.0(2.0) <sup>a</sup>	8.6 (1.5) <sup>ab</sup>	7.1 (2.2) <sup>b</sup>	7.7 (2.2) <sup>ab</sup>	8.5 (2.1) <sup>ab</sup>
FBS	24.9(6.1) <sup>a</sup>	16.0 (6.9) <sup>b</sup>	28.3 (5.6) <sup>a</sup>	14.0 (3.4) <sup>b</sup>	16.0 (5.0) <sup>b</sup>

*Note:* Good = good effort mild TBI, m/s = moderate/severe TBI, Poor = poor effort mild TBI, LHD = Left-hemisphere damaged CVA, Dem = dementia; m = mean, sd = standard deviation; PDRT = Portland Digit Recognition Test, TOMM = Test of Memory and Malinger, RDS = Reliable Digit Span, FBS = Fake Bad Scale.

<sup>ab</sup> row means with the same letter are not significantly different from each other. The significance was determined via ANOVAs that included all groups presented in a row.

### Group Analyses

Significant group effects were found for the WAIS-III VCI ( $F [4, 103] = 4.01, p < .01, partial\ eta^2 = .14$ ), WAIS-III Sim subtest ( $F [4,103] = 4.07, p < .01, partial\ eta^2 = .14$ ), the Phon cue condition of the COWAT ( $F [4,122] = 9.62, p < .001, partial\ eta^2 = .24$ ), the AC subtest of the Cognistat ( $F [4,116] = 6.45, p < .001, partial\ eta^2 = .18$ ), the WRAT-3 Reading subtest ( $F [4,118] = 7.60, p < .001, partial\ eta^2 = .21$ ), and the PPVT ( $F [4,88] = 3.80, p < .01, partial\ eta^2 = .15$ ). On the WAIS-III VCI, the Poor mTBI and DEM groups performed significantly worse than the other groups. On the WAIS-III Sim subtest, the LHD group performed significantly worse than only the Good mTBI group. On the Phon cue condition, the LHD and Poor mTBI group produced significantly less



Table 7 (Accuracy of effort classification and effort validation variables).

Classification Variables	Good # of hits (%)	m/s # of hits (%)	Poor # of hits (%)	LHD # of hits (%)	Dem # of hits (%)
PDRT only	0	0	4 (19)	0	0
TOMM only	0	0	4 (19)	0	1 (6)
Both PDRT & TOMM	0	0	13 (62)	0	0
<b>Effort Validation Tests</b>					
RDS	2 (7)	2 (10)	9 (43)	5 (22)	3 (9)
FBS>22	23 (77)	3(15)	18 (86)	0	0
FBS>28	8 (27)	2 (10)	10 (48)	0	0

*Note:* Good = good effort mild TBI, m/s = moderate/severe TBI, Poor = poor effort mild TBI, LHD = Left-hemisphere damaged CVA, Dem = dementia; m = mean, # of hits = number of patients scoring beyond the cut-off, % = number of hits/number of patients that have taken the test; PDRT = Portland Digit Recognition Test, TOMM = Test of Memory and Malingering, RDS = Reliable Digit Span, FBS = Fake Bad Scale.

words than the other groups. On the AC subtest of the Cognistat, the DEM group performed significantly worse than the Good mTBI and m/s TBI groups, and the Poor mTBI group also performed more poorly than the Good mTBI group. On the WRAT-3 Reading subtest, the DEM group performed significantly better than all of the TBI groups, but not significantly better than the LHD group. On the PPVT, the Poor mTBI group performed significantly worse than the LHD and DEM groups. Table 8 summarizes the findings for all of the language measures. Not all of the analyses were significant, but the pattern of scores for the groups was similar across the different measures with little variation. All of these findings taken together are in accordance with the hypotheses. The groups with neuropathology generally performed worse than the good effort mTBI patients, which supports a dose-response relationship where the

Table 8 (Dependent variables).

	Good	m/s	Poor	LHD	Dem	F	p	eta <sup>2</sup>
	m(sd)	m (sd)	m (sd)	m (sd)	m (sd)			
VCI <sup>1</sup>	43.9(7.8) <sup>a</sup>	42.5 (10.1) <sup>a</sup>	35.1 (11.4) <sup>b</sup>	36.4 (12.4) <sup>a</sup>	35.3 (11.0) <sup>b</sup>	4.01	.01*	.14
Vocabulary <sup>1</sup>	43.6(7.9) <sup>a</sup>	42.7 (9.9) <sup>a</sup>	35.5 (10.8) <sup>a</sup>	35.3 (13.8) <sup>a</sup>	38.7 (11.2) <sup>a</sup>	2.91	.03	.10
Similarities <sup>1</sup>	45.8(8.4) <sup>a</sup>	43.0 (9.5) <sup>ab</sup>	38.0 (10.6) <sup>ab</sup>	34.8 (12.8) <sup>b</sup>	37.0 (12.3) <sup>ab</sup>	4.07	.00*	.14
Information <sup>1</sup>	43.4(8.9) <sup>a</sup>	43.7 (11.0) <sup>a</sup>	37.0 (12.1) <sup>a</sup>	43.8 (8.9) <sup>a</sup>	35.9 (10.1) <sup>a</sup>	3.26	.02	.11
Comprehension <sup>1</sup>	42.8(9.4) <sup>a</sup>	36.2 (9.8) <sup>a</sup>	36.9 (11.6) <sup>a</sup>	37.5 (17.3) <sup>a</sup>	33.9 (12.9) <sup>a</sup>	2.17	ns	.08
BNT <sup>2</sup>	41.4(4.5) <sup>a</sup>	40.3 (8.7) <sup>a</sup>	35.5 (8.9) <sup>a</sup>	38.1 (13.0) <sup>a</sup>	38.7 (10.5) <sup>a</sup>	1.37	ns	.04
Phonemic cue <sup>2</sup>	37.4(7.8) <sup>a</sup>	38.0 (9.8) <sup>a</sup>	26.1 (7.6) <sup>b</sup>	26.6 (10.1) <sup>b</sup>	33.2 (9.3) <sup>a</sup>	9.62	.00*	.24
Semantic cue <sup>2</sup>	30.3(8.9) <sup>a</sup>	28.8 (10.4) <sup>ab</sup>	20.9 (10.3) <sup>b</sup>	24.7 (14.9) <sup>ab</sup>	24.6 (9.2) <sup>ab</sup>	2.89	.03	.09
Cognistat AC <sup>3</sup>	49.9(9.6) <sup>a</sup>	50.4 (12.2) <sup>ab</sup>	32.1 (25.8) <sup>bc</sup>	39.3 (21.2) <sup>abc</sup>	28.4 (25.0) <sup>c</sup>	6.45	.00*	.18
WRAT Reading <sup>4</sup>	88.5(13.2) <sup>a</sup>	88.2 (12.7) <sup>a</sup>	79.2 (18.8) <sup>a</sup>	90.0 (16.8) <sup>ab</sup>	101.1 (10.3) <sup>b</sup>	7.60	.00*	.21
PPVT <sup>4</sup>	91.4(8.8) <sup>ab</sup>	90.8 (15.8) <sup>ab</sup>	79.4 (15.9) <sup>a</sup>	92.7 (11.2) <sup>b</sup>	93.4 (10.7) <sup>b</sup>	3.80	.01*	.15

*Note:* Good = good effort mild TBI, m/s = moderate/severe TBI, Poor = poor effort mild TBI, LHD = Left-hemisphere damaged CVA, Dem = dementia; m = mean, sd = standard deviation; VCI = Wechsler Adult Intelligence Scale Verbal Comprehension Index, BNT = Boston Naming Test; WRAT = Wide Range Achievement Test, PPVT = Peabody Picture Vocabulary Test.

<sup>abc</sup> row means with the same letter are not significantly different from each other.

\* significant using the Holm (1979) method.

<sup>1</sup> T-scores calculated from Taylor and Heaton's (2001) normative data.

<sup>2</sup> T-scores calculated from Heaton, Miller, Taylor, and Grant's (2004) normative data.

<sup>3</sup> T-scores calculated from the Cognistat manual's normative data (The Northern California Neurobehavioral Group, Inc, 1988)

<sup>4</sup> Age corrected standard scores.

good effort mild TBI group has less impairment. The second hypothesis is supported by the results which demonstrate that the Poor mTBI group is performing significantly worse than the Good mTBI group, and also similarly or worse than the m/s TBI group and the neurologically impaired groups on most of the language measures.

### *Effect of Effort vs. Injury Severity*

Some studies have calculated effect sizes as a way to demonstrate the effect of traumatic brain injury on neuropsychological measures (Iverson, 2005; Shretlen & Shapiro, 2003). Effect sizes for the current study, which were reported as Cohen's  $d$ , were calculated using a Microsoft Excel-based macro (Lipsey & Wilson, 2000). The effect of injury severity or objective neuropathology was calculated by comparing the performance of the Good mTBI group to that of the m/s TBI, LHD, and DEM groups. These data represented a gradient of injury severity, which resulted in increasing effect sizes with more pathology (i.e. a dose-response curve for brain dysfunction). Table 9 presents the individual effect sizes. The mean effect sizes of the injury severity groups are as follows: m/s TBI = .14 (sd = .23; -.07 to .69), LHD = .52 (sd = .50; -.14 to 1.21), and DEM = .46 (sd = .65; -1.06 to 1.15) [presented in Figure 1]. The effect of LHD and DEM are nearly four times that of the m/s TBI group.

The effect of effort was based on the comparison of the Good mTBI and Poor mTBI groups, which controls for the effect of severity by comparing two mTBI groups which only differ on effort. The mean effect size of effort was .88 (sd = .26; .56 to 1.46), which is almost eight times greater than the m/s TBI group and almost twice that of the neurologic control groups. The differences in effect sizes were statistically significant ( $F(3, 36) = 4.64, p < .01, partial \eta^2 = .28$ ), with the Poor mTBI group having a

Table 9 (Effect sizes of severity and effort).

	m/s TBI	LHD	DEM	Poor
WAIS Vocab	.10	.84	.51	.88
WAIS Sim	.32	1.12	.85	.83
WAIS Inf	-.03	-.04	.79	.64
WAIS Comp	.69	.44	.80	.56
BNT	.17	.36	.33	.89
Phon	-.07	1.21	.49	1.46
Sem	.16	.47	.63	.99
WRAT Reading	.02	-.10	-1.06	.59
PPVT	.05	-.14	-.21	.99
Cognistat AC	-.05	.68	1.15	.99
mean (sd)	.14(.23)	.52(.50)	.46(.65)	.88(.26)

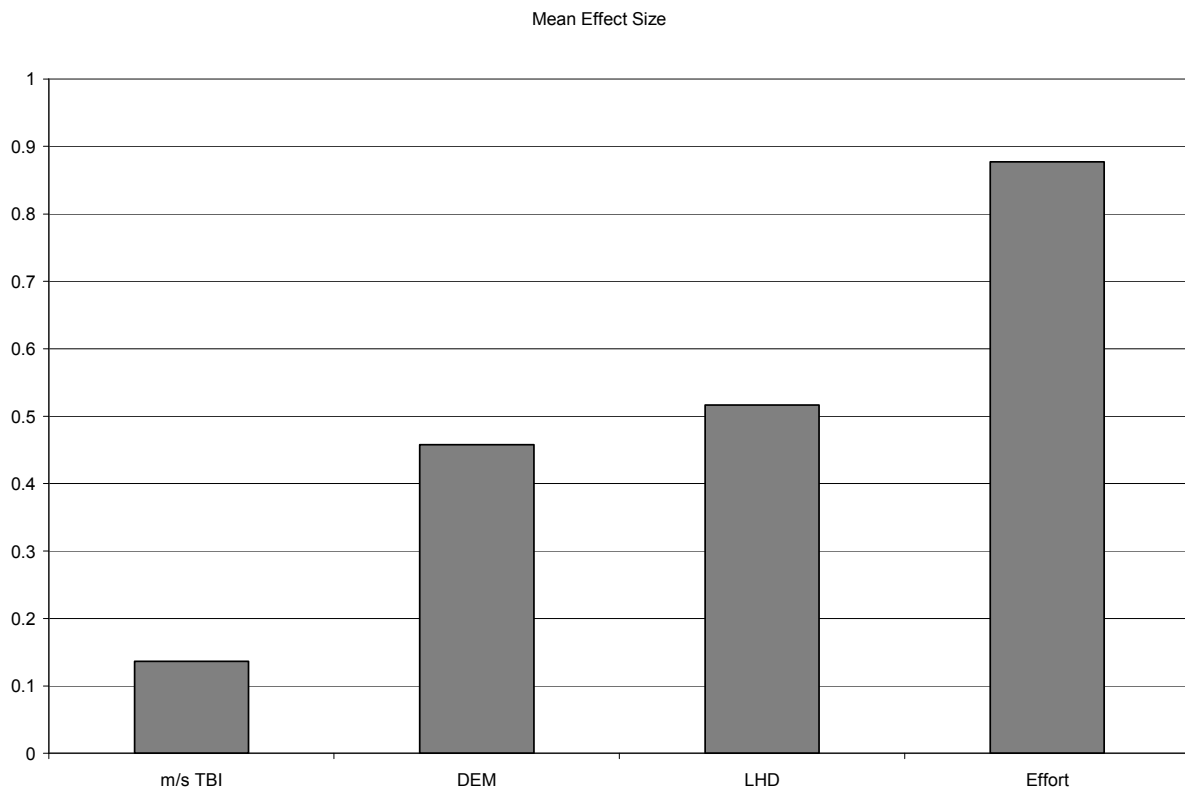
Note: WAIS-III VCI was not included in the calculation of effect sizes; m/s = moderate/severe traumatic brain injury, LHD = Left-hemisphere damaged CVA, Dem = dementia, Poor = poor effort mild TBI; d = Cohen's *d*; WAIS Vocab = Wechsler Adult Intelligence Scale Vocabulary subtest, WAIS Sim = Wechsler Adult Intelligence Scale Similarities subtest, WAIS Inf = Wechsler Adult Intelligence Scale Information subtest, WAIS Comp = Wechsler Adult Intelligence Scale Comprehension subtest, BNT = Boston Naming Test, Phon = Phonemic cue condition of COWAT, Sem = Semantic cue condition of COWAT, WRAT = Wide Range Achievement Test, PPVT = Peabody Picture Vocabulary Test; AC = Auditory Comprehension.

[significantly larger effect size than the m/s TBI group. These data also support the hypotheses, since there was evidence of a dose-response relationship with the moderate/severe group having the weakest effect size relative to the good effort mTBI group, and the neurologic control groups having effect sizes that were approximately four times the moderate/severe effect size. The large effect size of the poor effort mild TBI group, which was significantly larger than the moderate/severe TBI group and almost twice that of the neurologic controls, supports the second hypothesis.

### *Impairment*

To understand the effects of the individuals rather than the groups as a whole, it was necessary to find out how many individuals performed abnormally. Impairment was defined by test scores that were at least 1.5 standard deviations below the

Figure 1 (Mean effect sizes of severity and effort).



published normative means (Table 2 lists the sources of the normative data for each of the measures used in this study). Due to the fact that the WAIS-III VCI incorporates the scores of the Vocab, Sim, and Inf subtests, only the VCI score was included into the total number of tests for the determination of impairment. Since the WAIS-III Comp subtest and the Cognistat AC subtest both assess the patient's ability to comprehend verbal information and the WAIS-III has normative data that take into account age, education, sex, and race, only the WAIS-III Comp subtest was included for the determination of impairment. This leaves a set of seven of the language measures which were used to determine impairment.

Table 10 shows the distributions of the number of impaired scores by group and cumulative frequencies of the impaired scores. The Good mTBI group has most of its

Table 10 (Cumulative percentage of impaired scores across all groups).

Impaired Scores	Mild TBI						Mod/Sev TBI			LHD CVA			Dementia		
	Good Effort			Poor Effort			Good Effort			n per cell	% per cell	cum %	n per cell	% per cell	cum %
	n per cell	% per cell	cum %	n per cell	% per cell	cum %	n per cell	% per cell	cum %						
7	0	0	0	4	19	19	1	5	5	0	0	0	0	0	0
6	0	0	0	2	10	29	2	10	15	1	4	4	3	9	9
5	0	0	0	2	10	39	1	5	20	3	12	16	4	13	22
4	2	7	7	3	14	53	3	15	35	5	20	36	5	15	37
3	5	16	23	5	23	76	2	10	45	3	12	48	4	13	50
2	13	44	67	4	19	95	3	15	60	6	24	72	7	22	72
1	6	20	87	1	5	100	6	30	90	4	16	88	7	22	94
0	4	13	100	0	0		2	10	100	3	12	100	2	6	100

*Note:* Impaired scores refers to the number of language measure scores that are <1.5 standard deviations below normative means. n per cell (% per cell) refers to number (percentage) of cases in the group with x impaired scores. Cum % refers to the percentage of the group with ≥ x impaired scores. Mod/Sev = moderate/severe TBI; TBI = traumatic brain injury; LHD CVA = left-hemisphere stroke

scores distributed around little or no impairment, and the m/s TBI and neurologic control groups have their scores distributed evenly throughout. The Poor mTBI group has the majority of its scores distributed at the upper end of impairment. The skewed nature of the Poor mTBI group is illustrated best by looking at the number of persons with  $\geq 4$  impaired scores: 7% of the Good mTBI patients, 35% of the m/s TBI patients, 36% of the LHD patients, 37% of the DEM patients, and 53% of the Poor mTBI patients. Although the m/s TBI and neurologic control groups have individuals that are impaired, the Poor mTBI patients had more individuals with impaired scores.

#### *Effect of Effort and Severity on Impairment.*

To determine the relationship between injury severity and impairment, a bivariate correlation was run using GCS scores as an indicator of injury severity and the number of impaired language scores represented impairment. When all TBI patients with a reported GCS score including the Poor mTBI group were considered, the correlation between GCS and the number of impaired language scores was not significant ( $r = -.11$ ,  $p = .44$ ). However, when the Poor mTBI group was removed from this analysis, there was a significant negative correlation ( $r = -.43$ ,  $p < .05$ ) between the injury severity of good effort mild and moderate/severe TBI patients and the number of impaired language scores which indicates that the greater injury severity (lower GCS) the greater the amount of impairment. This significant correlation supports the first hypothesis.

#### *Outlier Analyses*

A single score that meets the criteria for an impaired score does not necessarily define someone as impaired or abnormal, because a spurious score may be the product of chance. The more impaired scores that one has, the less likely the impaired scores

were a product of chance alone. The probability of a patient having three or more scores < 1.5 standard deviations from the normative means is less than 5% (Ingraham & Aiken, 1996). This means that patients having three or greater impaired scores on the language measures were considered to be showing a language impairment. Twenty-three percent of the Good mTBI patients have three or more impaired scores, while 45% of the m/s TBI patients, 48% of the LHD, and 50% of the DEM would be interpreted as having a language impairment. The Poor mTBI group had 76% of its patients with three or more impaired scores. A 2 x 2 Chi square analysis of the number of persons identified as impaired (three or more scores of < 1.5 standard deviations from the normative means) in Good vs. Poor mTBI patients revealed that there was a significant difference ( $X^2 [1] = 13.94, p < .001, d = 1.23$ ) suggesting that there was a relationship between the classification of Good or Poor effort and having a language impairment. The effect size reported as Cohen's *d* is a large effect size, which indicates the strength of this relationship. This is more evidence for the second hypothesis, because patients that have been noted as poor effort are more likely to have impaired language measure scores.

*Good Mild TBI.* Seven Good mTBI patients met the above criteria for impairment. Patient RS was a 37 year-old male with 12 years of education and a history a moderate TBI at the age of 12. The patient also reported having lifelong verbal and reading problems, and a significant history of alcohol abuse. RS did not have a GCS recorded acutely, but he had no loss of consciousness or post-traumatic amnesia. He did have a basilar skull fracture with no intracranial findings detected on CT or MRI scans. During his evaluation, there was no evidence of poor effort on cognitive



measures, but there was significant evidence of exaggeration of emotional symptoms on the MMPI-2.

Patient AC was a 39 year-old female with 12 years of education. AC refused treatment at the scene of the accident that caused her injury. She stated that she had a brief loss of consciousness of five minutes or less, and she did not have any findings on CT, MRI, or EEG. Regarding her history, AC reported that she had been on “nerve pills” since the first grade for nervousness and anxiety. In her medical records, there were several different sources that had described or diagnosed psychogenic seizures. AC did not exhibit any evidence of poor effort on cognitive measures, but she did have significant evidence of exaggeration of emotional symptoms and somatization on the MMPI-2.

Patient FL was a 29 year-old man with 11 years of education, and had no loss of consciousness and a GCS of 15. Despite the physical signs of trauma to his head, he did not exhibit any acute signs of concussion, and his neuroradiological scans were considered normal. FL met criteria for a diagnosis of probable Malingered Neurocognitive Dysfunction (MND; Slick, Sherman, & Iverson, 1999) based on a score below a published cut-off (Green & Iverson, 2001b) on the Computerized Assessment of Response Bias (CARB; Allen, Conder, Green, & Cox, 1997), which is a computer-based SVT, a score below a published cut-off on an embedded validity indicator (Millis, Putnam, Adams, & Ricker, 1995), exaggeration of emotional symptoms on the MMPI-2, and he also demonstrated a significant decrease in neuropsychological testing scores from a previous post-injury evaluation, which is considered a compelling inconsistency.

Patient SH was a 49 year-old man with 11 years of education, and had a brief loss of consciousness, and was assigned a GCS score of 15. There was documentation in SH's pre-injury medical records that he was diagnosed as malingering on two separate occasions for previous injuries. During the evaluation used for the present study, the psychometrists documented that SH exerted inconsistent effort throughout the battery of tests, and it was also noted that he exhibited no pain behaviors despite his reports of moderate to severe pain. There was psychometric evidence of physical exaggeration (Larrabee, 2003) on the Pain Disability Index (PDI; Tait, Pollard, Margolis, Duckro, & Krause, 1987) and Modified Somatic Perception Questionnaire (MSPQ; Main, 1983) and emotional exaggeration on the MMPI-2. SH did not meet criteria for MND, but he had enough evidence that there were doubts raised about the validity of his claims.

Patient TO was a forty-one year-old man with ten years of education. TO did not have a loss of consciousness, had a GCS score of 15, and had a normal CT scan. TO indicated that he had been a slow learner all his life, but never had been diagnosed with a learning disability nor did he have any special education classes. In behavioral observation notes the psychometrists had documented that TO was putting forth inconsistent effort throughout the battery of tests. TO met diagnostic criteria for MND based on a score below a published cut-off on an embedded validity indicator (Millis et al., 1995) and there was evidence of emotional exaggeration on the MMPI-2 (Greve, Bianchini, Love, Brennan, & Heinly, 2006).

Patient RZ was a 41 year-old man with 12 years of education. He had a possible brief loss of consciousness and a GCS score of 15 with normal CT scans of the brain.

Behavioral observations during testing noted that RZ was extremely anxious, and he was also diagnosed with an anxiety disorder. Medical records for this patient contained pre-injury test data, and the examiner concluded that RZ's post-injury testing was consistent with his documented pre-injury level.

Patient LK was a 48 year-old man with 12 years of education. He had experienced only a momentary loss of consciousness and had normal neuroradiological scans. LK met diagnostic criteria for MND based on a score below a published cut-off on an embedded validity indicator (Millis et al., 1995) and there was evidence of emotional exaggeration on the MMPI-2 (Greve et al., 2006).

*Moderate/Severe TBI.* There were nine m/s TBI patients that met the above criteria for impairment, and two of them did not have any incentive. Patient RM was a 19 year-old woman with 12 years of education and had no incentive related to the TBI. She did have a significant history of substance abuse including alcohol, marijuana, prolonged ecstasy use (1.5-2 years), and LSD. RM had experienced a severe traumatic brain injury with a skull fracture and left frontal craniotomy for the evacuation of a hematoma. RM was noted to have slowed speech during the evaluation.

Patient LB was a 29 year-old male with 10 years of education and a history of learning disabilities (LD) with remedial classes. Acutely, LB had a GCS of four, a dysconjugate gaze, and was witnessed having post-traumatic seizures. Neuroradiological scans detected multiple bilateral frontal, temporal, and parietal punctuate contusions. Pre-morbid neuropsychological intelligence levels were available due to his history of LD, and his post-injury intelligence was noted as being at nearly exactly the same level as his pre-injury testing.

Patient MS was a 29 year old man with 13 years of education and a history of meningitis as an infant with shunt placement for congenital hydrocephalus. MS was in a coma for eight days, which would be considered a severe TBI. He also had post-traumatic seizures at the scene and in the ER, and a later EEG was documented as abnormal bilaterally with intermittent theta activity over the frontal lobes. During testing it was noted that he complained frequently of being tired and needed to be awakened on several occasions. His performance on the WAIS was noted as possibly not being a true reflection of his ability since he was not consistently paying attention and gave up easily on many items.

Patient RO was a 34 year-old left-handed man with 12 years of education. He had a GCS of four, a right parietal open skull fracture, subsequent right parietal lobectomy, massive edema, and respiratory failure, which raises the possibility of a secondary hypoxic insult to the brain. He had a left hemiparesis and left visual hemifield defect. Despite his documented brain trauma, he had an EEG that was read as normal. During testing he was noted to be very slow to respond and had many anomic and paraphasic errors.

Patient JT was a 43 year-old male with 12 years of education. His acute GCS was a three and he had neuroradiologic evidence for an epidural hematoma, subarachnoid hemorrhage, right frontal hematoma, questionable diffuse shearing injury, hemorrhage in the left lateral ventricle and fourth ventricle, questionable generalized cerebral edema, skull fracture, and a left frontal hemorrhagic contusion. At the time of the evaluation he still had continued right-sided weakness. He was observed to have mild bradykinesia (slowed motor movements), perseveration problems, and also

problems with basic orientation questions, such as, who are you, where are you, and what day/date/time is it.

Patient JK was a 21 year-old man with ten years of education, which was interrupted by his injury. JK had no incentive related to his case. He experienced a severe TBI, since his GCS score was three and he was comatose for six months followed by an altered level of consciousness for another 12 months. His history is significant for visual perceptual problems, ADD, learning problems, and possible Conduct disorder, and he experienced a grand mal seizure at most one year prior to the evaluation. Behaviorally he was noted to be perseverative, impulsive, confabulatory, and socially inappropriate.

Patient CG was a 23 year-old male with 12 years of education and a history of learning disability with remedial classes. CG was also held back one year due to slow reading. CG had a GCS of five as a result of his TBI, and he had a respiration rate of four breaths per minute. His pupils were dilated bilaterally, and he had a skull fracture. It is believed that he had a hypoxic brain injury secondary to his poor post-traumatic respiration. He had neuroradiologic evidence of diffuse cerebral edema, subarachnoid hemorrhage, and a left posterior frontal contusion. During testing he had many circumlocutions, was impulsive, perseverated, and confabulated.

Patient EW was a 23 year-old male with 11 years of education. EW's post-injury GCS was a three, and he had a frontal skull fracture with bifrontal epidural fluid collections. His right pupil was fixed and non-reactive. Brain scans demonstrated that he had bifrontal mass effect, cerebrospinal fluid subdural collection over the right frontal and parietal lobes and midline shift. At three weeks post-injury he still had a GCS of 10

and had a seizure two years post-injury (within one year of the evaluation). EW had an EEG done and it was noted that he had bihemispheric cerebral dysfunction. EW was diagnosed as aphasic, and this was evident during testing where behavioral observations of naming problems and paraphasic errors were common.

Patient CT was a 30 year-old male with 12 years of education and a history of a seizure at the age of seven years old. He had a severe TBI with a GCS of six and had asymmetric pupillary responses. He had bilateral contusions, bifrontal subdural hematoma with minimal midline shift, right frontal-temporal hemorrhagic contusion with mass effect on the right lateral ventricle. Although he did not meet criteria for MND, CT was considered suspicious, because he had evidence of exaggeration on the MMPI-2 validity scales (Greve et al., 2006) and he also demonstrated a decrease in test scores since a previous post-injury evaluation, which is not expected.

#### *Diagnosed Malingering*

One-hundred percent of both the Good mTBI and Poor mTBI groups had some incentive related to their evaluation, while 80% of the m/s TBI group also had incentive. The presence of incentive necessitates a review of the possibility of malingering in the current sample. The Slick, Sherman, and Iverson (1999) criteria for malingered neurocognitive dysfunction (MND) were applied to the sample. LHD and DEM patients were not eligible, since they all lacked external incentive.

The Good mTBI had four patients (13%) that met criteria for MND. Based on impairment scores, noted in the sections above, only three of the four MND diagnosed Good mTBIs were considered impaired. None of the m/s TBI patients met criteria for malingering. The Poor mTBI group had 19 of 21 of its patients meeting criteria for

MND, and fifteen of these 19 Poor mTBI patients diagnosed as MND also met criteria for impairment of language measures. A 2 x 2 Chi square analysis of the number of persons identified as impaired (three or more scores of < 1.5 standard deviations from the normative means) in non-MND vs. MND mTBI patients revealed that there was a significant difference ( $\chi^2 [1] = 23.81, p < .001, d = 1.87$ ) suggesting that there was a relationship between the diagnosis of malingering and having a language impairment. The effect size reported as Cohen's *d* is a very large effect size, which indicates the strength of this relationship.

### Discussion

Traumatic brain injury results in a dose-response relationship between injury severity and cognitive impairment. There is also a temporal gradient in which the effects of the injury decrease or resolve over time, which has been demonstrated in animal models of traumatic brain injury (Gaetz, 2004) and in cognitive and neuropsychological findings in humans with TBI (Shretlen & Shapiro, 2003). At one year or less post injury, mild TBI patients are expected to have no residual impairment due to the direct neurological effects of their injury (Belanger et al., 2005; Binder et al., 1997; Carroll et al., 2004; Shretlen & Shapiro, 2003); in contrast, patients with more severe injuries would be expected to show some residual deficit the magnitude of which should be directly related to the severity of the injury (Shretlen & Shapiro, 2003). It is notable that the results of some studies suggest residual language deficits following mild TBI (Hannay et al., 2004; Mathias & Coats, 1999; Raskin & Rearick, 1996; Sarno, 1984; Whelan, Murdoch, & Bellamy, 2007). The notable characteristic of most of this research is that effort exerted has generally not been addressed or controlled despite

growing evidence that measured effort accounts for a larger proportion of the variance in cognitive ability than does injury severity (Binder et al., 2003; Curtis et al., 2006; Green, 2007; Green & Iverson, 2001a; Green et al., 2001, 2003; Moss et al., 2003). Thus, the purpose of this study was to examine the effect of TBI on language while explicitly measuring and controlling for the effects of effort.

The results of this study demonstrated a near-zero correlation between injury severity as defined by Glasgow Coma Scale score and the number of impaired scores on measures of language ability in the full TBI sample. However, when patients demonstrating poor effort were excluded the correlation became significant, a finding consistent with that of Green et al. (2001). Thus, in the absence of a control for effort, no dose-response relationship was observed. The finding of greater impairment on language testing as a function of injury severity was demonstrated throughout this study in TBI and across the neurological controls. Effort had seven times the effect on language test performance as moderate-severe TBI and nearly twice the effect as stroke and dementia. The finding that the effect of effort dwarfed the effect of objectively defined severe neuropathology is consistent with other reports in the literature (Binder et al., 2003; Green, 2007; Green et al., 2001, 2003). Moreover, these results also indicated that mild TBI patients who exhibited good effort had little or no impairment on language measures. When such impairment was present, it was accounted for by factors unrelated to the concussion itself (e.g., psychological distress, pre-existing cognitive limitations). In contrast, in mild TBI patients who gave poor effort, impairment on language testing was associated with diagnosable malingering.

### *Effect of Severity*



The first hypothesis of this study was that when effort is controlled in traumatic brain injury, there would be a dose-response relationship between injury severity and the amount of language impairment. In the present study, when effort was controlled, a dose-response relationship was seen between the amount of neurologic impairment and language impairment as measured by the dependent variables. In the group analyses there was a very modest effect of severity demonstrated by the moderate/severe TBI group, as evidenced by the small effect size of this group compared to the good effort mTBI group. The neurologic control groups had larger effect sizes, which were considered medium. This progression of more cognitive impairment follows the severity gradient of moderate/severe TBI to the neurologic control groups. There was a significant negative correlation between GCS score and the number of impaired language scores demonstrating that lower GCS scores (greater severity) are associated with a greater number of language impairments which provided even more support for the dose response relationship across injury severity in TBI patients exhibiting good effort.

Despite having significantly more education than the good effort mild TBI group, the neurologic control groups did perform worse on most of the language measures. The only exceptions included the two measures (WRAT-3 Reading and PPVT) that have demonstrated utility as estimates of pre-morbid intelligence, which is largely influenced by education and relatively uninfluenced by neurologic insults (Ball, Hart, Stutts, Turf, & Barth, 2007; Bell, Lassiter, Matthew, & Hutchinson, 2001; Orme, Johnstone, Hanks, & Novack, 2004; Smith, 1997). The stroke group also performed better, but not significantly, than the good effort mild TBI group on the Information

subtest of the WAIS-III. It is possible that the effects of these neurologic control groups were attenuated due to this greater level of education, and it is expected that if the neurologic control groups were better matched to the TBI groups on education, an even greater effect size of these more severely injured groups would be present.

When the individual cases were considered in relation to defined language impairment, the neurologic control groups were comparable to the moderate/severe TBI group. If the stroke and dementia patients had been administered all of the language measures, it is likely that there would be more impaired scores for these groups. Especially considering that some of the stroke patients had been diagnosed with different aphasia and were often seen as in-patients in hospital settings, and many of the dementia patients that had impaired scores had either described language impairments (usually anomia, paraphasias, and/or circumlocutions) in their clinical interview or a family member reported these language impairments during the interview.

The outlier analyses illuminated the fact that the only TBI patients that demonstrated impairment had either a TBI that would be classified as severe based on their acute characteristics (Sherer & Madison, 2005) with residual neurocognitive impairment (one case was documented as having experienced a frank aphasia), or the patients with mild injuries had non-neural contributory factors that affected performance (i.e. exaggeration and/or malingering, somatization, learning disabilities, or other psychiatric conditions). This demonstrates that documented neurologic damage does have an effect on the level of language impairment, and that when neurologic factors are not present, other factors are more likely the cause which supports the conclusions of Green (2003) and Rees (2003). These findings support the hypothesis that a dose-

response relationship would exist between injury severity and the amount of language dysfunction.

### *Effect of Effort*

The second hypothesis of this study was that effort will account for more variance than severity. For the mild TBI groups only, the poor effort group always scored worse than the good effort group, and with the exception of a marginal difference on the WAIS-III Comprehension subtest, the poor effort mild TBI group always scored lower than the moderate/severe TBI group. The poor effort mild TBI group also performed similarly to, or worse than, the other neurologically impaired groups (stroke and dementia). The poor effort mild TBI group performed significantly worse than the dementia group, but not significantly worse than the stroke group on the Semantic cue condition of the COWAT, and the WRAT-3 Reading subtest. The measures that the poor effort mild TBI group performed significantly worse than the stroke group were the WAIS-III Verbal Comprehension Index and the PPVT. These data indicate that the poor effort mild TBI group is performing worse than or equal to patients with objective neuropathology.

This trend can be seen more clearly when comparing the effect sizes calculated from the groups' average performances on the language measures. The poor effort mild TBI group produced a large effect size compared to the small effect of the moderate/severe TBI group and the medium effect sizes of stroke and dementia groups, which supports previous research which found effort to have a larger effect on neuropsychological measures than neuropathology (Binder et al., 2003; Green et al., 2001). The non-significant correlation between GCS scores and number of impaired language scores for all TBI groups including the poor effort mild TBI patients in the

current study also supported prior research (Green & Iverson, 2001a; Green et al., 2001; Moss et al., 2003) that demonstrated that the inclusion of poor effort patients results in no clear dose-response relationship. These findings support Green's (2003) conclusion that by not controlling for effort one cannot be certain that the findings are accurate.

When considered as individual patients, the poor effort group as a whole had more impaired scores than any of the other groups, and most of the poor effort mild TBI group met criteria for language impairment. An effect size that was calculated for the relationship between good vs. poor effort and impairment vs. no impairment was large. Not only did the poor effort mTBI patients perform worse than good effort mTBI patients with comparable injury characteristics and moderate/severe TBI patients with injuries that often had objective neuropathology, they performed worse than stroke patients with unilateral left-hemisphere lesions and dementia patients. These patterns were maintained when these patients were compared as groups and individuals. These findings support the hypothesis that effort will account for more variance than severity of injury.

It is worth noting that, not only were the poor effort mild patients found to be putting forth poor effort, but most of them met diagnostic criteria for malingering. Of the seven good effort mTBI patients that had language impairment, three of them also met diagnostic criteria for malingering, and three more had evidence of exaggeration on a self-report measure (MMPI-2). An effect size that was calculated for the association between malingering vs. non-malingering and impairment vs. no impairment was very large and was comparable to the effect size of malingering on neuropsychological tests

that was presented in Iverson (2005). When severity is controlled, and mild TBI is considered by itself, effort and malingering account for most of the impairment seen on language measures which supports the second hypothesis.

### *Implications of the Findings*

These findings have clinical and research implications. In clinical settings, acute characteristics are very important to appropriately define the severity of the injury, and thorough testing, including SVTs, embedded validity indicators and self-report measures, is necessary to parse out poor effort and assist with differential diagnosis. Impairment on cognitive capacity measures, like language measures, could be the product of actual neurological impairment or non-neural factors, and multiple sources of valid information are necessary to make the correct diagnosis. As stated earlier, neuropathology may result in language or other cognitive impairment, but impaired scores should be interpreted with caution when there is a lack of documented pathology. If confronted with a case of mTBI with language impairment defined by neuropsychological testing, non-neural causes are most likely responsible, so it is important that the clinician assess effort, possible underlying psychological problems, and comprehensively investigate any pre-morbid factors that may have an effect, such as: learning disabilities, poor educational resources, or prior neurological insults.

In research settings, the appropriate severity classification and controlling for effort help to clarify the connection between neural impairment and cognitive impairment. If studies cannot employ the robust prospective design like Dikmen et al. (1995), then the convenience sample may be used if the groups are clearly defined by severity and effort is accounted for. The current study adds to the mounting evidence in

support of the use of effort measures to purify samples that may have some incentive to appear impaired on cognitive tests, such as traumatic brain injury and chronic pain patients (Binder et al., 2003; Curtis et al., 2006; Green & Iverson, 2001a; Green et al., 2001, 2003; Moss et al., 2003). This study also provides support for the use of SVTs to assess effort for cognitive domains outside of memory (Constantinou et al., 2005).

### *Limitations*

One limitation of using retrospective data is the number of cases is limited by the number of clinical cases that have been evaluated and it is not a truly randomized sample. Exclusion and inclusion criteria also reduced the number of available cases, but helped to produce TBI groups that were closely matched on demographic variables. More subjects per group and equal group sizes across all variables would allow one to use more robust statistical procedures and increase the statistical power.

Use of a control group in addition to normative data may also clarify whether good effort mTBI patients perform normally on language or any cognitive measures by accounting for regional differences and/or quality of education in a particular region. Although the average performance of the good effort mTBI group was below the norms on most of the language measures, only seven good effort mild TBI patients were considered impaired on language measures based on the stated criteria and they all had non-neurologic complicating factors.

Three of the seven met diagnostic criteria for malingered neurocognitive dysfunction (Slick, Sherman, & Iverson, 1999) despite scoring above the cut-offs on both of the SVTs used for the purpose of defining good or poor effort in this study. Utilizing false positive error rates of less than or equal to five percent, the PDRT and

TOMM have sensitivities of 52% and 56%, respectively, which leaves false-negative rates of 48% for the PDRT and 44% for the TOMM (Greve, Ord, Curtis, Bianchini, & Brennan, in press). That means that each of these SVTs is missing over 40% of all persons who are intentionally putting forth poor effort. When the two tests are used in tandem and the false positive error rate is held at five percent or better, there is better sensitivity (67%), which leaves 33% of false-negatives undetected (Greve et al., in press). This means that despite the excellent sensitivities of these two SVTs, some patients putting forth poor effort will still slip by. It is important to increase both specificity and sensitivity, which reduces the number of false negatives and the number of false positives. A recent paper (Larrabee, in press) investigated the probability of malingering given one, two, or three scores beyond cut-offs on validity tests that included SVTs embedded indicators, and self-report measures. He found that the use of three scores decreases both false positives and false negatives, and he also found that combinations of three of the various validity indicators produced probabilities of malingering ranging from 93.3% to 99.9%. These findings demonstrate that use of multiple indicators is necessary to more accurately define good vs. poor effort.

### *Summary*

The results of the current study supports the first hypothesis that there is a dose response relationship between severity of neurological impairment and language dysfunction when effort is controlled which replicates the findings across other cognitive domains (Curtis et al., 2006; Green & Iverson, 2001a; Green et al., 2001, 2003; Moss et al., 2003). The second hypothesis which states that effort would account for more variance than severity of injury was also supported and this replicates the findings of

Green et al. (2001) and Binder et al. (2003). Despite a lack of documented neurologic impairment, the poor effort mTBI patients performed more poorly than moderate/severe TBI patients with documented intracranial findings, stroke patients with documented lesions to the language dominant hemisphere, and dementia patients with objectively established memory and cognitive decline. Given the present findings, a mild TBI patient that has evidence of language impairment on neuropsychological testing is more likely to be putting forth poor effort or malingering. If one were to interpret the findings of this study without considering effort, it might be reasonable to conclude that language impairments exist for all traumatic brain injuries and that the severity of the injury has no effect. However, when Green's (2003) advice is taken into consideration and effort is controlled, hidden dose-response relationships between neurologic impairment and cognitive impairment are uncovered.



## References

- Alexander, M.P. (1995). Mild traumatic brain injury: Pathophysiology, natural history, and clinical management. *Neurology*, *45*, 1253-1260.
- Alexandre, A., Colombo, F., Nertempi, P., & Benedetti, A. (1983). Cognitive outcome and early indices of severity of head injury. *Journal of Neurosurgery*, *59*, 751-761.
- Allen, L., Conder, R.L., Green, P., & Cox, D.R. (1997). *CARB '97 Manual for the Computerised Assessment of Response Bias*. Durham, NC: CogniSyst.
- American Academy of Neurology. (1997). Practice parameter: The management of concussion in sports (summary statement). *Neurology*, *48*, 581-585.
- Ball, J.D., Hart, R.P., Stutts, M.L., Turf, E. & Barth, J.T. (2007). Comparative utility of the Barona Formulae, WTAR demographic algorithms, and WRAT-3 Reading for estimating premorbid ability in a diverse research sample. *The Clinical Neuropsychologist*, *21*, 422-433.
- Belanger, H.G., Curtiss, G., Demery, J.A., Lebowitz, B.K., & Vanderploeg, R.D. (2005). Factors moderating neuropsychological outcomes following mild traumatic brain injury: A meta-analysis. *Journal of the International Neuropsychological Society*, *11*, 215-227.
- Bell, N.L., Lassiter, K.S., Matthew, T.D., & Hutchinson, M.B. (2001). Comparison of the Peabody Picture Vocabulary Test-third edition and Wechsler Adult Intelligence Scale-third edition with university students. *Journal of Clinical Psychology*, *57*, 417-422.

- Ben-Porath, Y.S., & Tellegen, A. (2007). *Press Release: FBS added to the MMPI-2 standard scoring materials*. Retrieved July 30, 2007, from <http://www.pearsonassessments.com/resources/fakebadscale.htm>.
- Bianchini, K.J., Mathias, C.W., & Greve, K.W. (2001). Symptom Validity Testing: a critical review. *The Clinical Neuropsychologist*, *15*, 19-45.
- Binder, L.M. (1986). Persisting symptoms after mild head injury: A review of the postconcussive syndrome. *Journal of Clinical and Experimental Neuropsychology*, *8*, 323-346.
- Binder, L. M. (1993a). *Portland Digit Recognition Test manual* (2<sup>nd</sup> ed.) Private Publication: Portland, Oregon, USA.
- Binder, L.M. (1993b). Assessment of malingering after mild head trauma with the Portland Digit Recognition Test. *Journal of Clinical and Experimental Neuropsychology*, *15*, 170-182.
- Binder, L.M. (1993c). An abbreviated form of the Portland Digit Recognition Test. *The Clinical Neuropsychologist*, *7*, 104-107.
- Binder, L.M., & Kelly, M.P. (1996). Portland Digit Recognition Test performance by brain dysfunction patients without financial incentives. *Assessment*, *3*, 403-409.
- Binder, L.M., Kelly, M.P., Villanueva, M.R., & Winslow, M.M. (2003). Motivation and neuropsychological test performance following mild head injury. *Journal of Clinical and Experimental Neuropsychology*, *25*, 420-430.
- Binder, L.M., Rohling, M.L., & Larrabee, G.J. (1997). A review of mild head trauma. Part I: Meta-analytic review of neuropsychological studies. *Journal of Clinical and Experimental Neuropsychology*, *19*, 421-431.

- Binder, L.M., & Willis, S.C. (1991). Assessment of motivation after financially compensable minor head trauma. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, 3, 175-181.
- Borgaro, S.R., Prigatano, G.P., Kwasnica, C., & Rexer, J.L. (2003). Cognitive and affective sequelae in complicated and uncomplicated mild traumatic brain injury. *Brain Injury*, 17, 189-198.
- Brooks, N. (1989). Closed head trauma: Assessing the common cognitive problems. In M.D. Lezak (Ed.), *Assessment of the behavioral consequences of head trauma. Frontiers of clinical neuroscience* (Vol. 7). New York: Alan R. Liss.
- Bullock, R., Maxwell, W., Graham, D., Teasdale, G., & Adams, J. (1991). Glial swelling following human cerebral contusion: an ultrastructural study. *Journal of Neurology, Neurosurgery, and Psychiatry*, 54, 427-434.
- Bush, S.S., Ruff, R.M., Tröster, A.I., Barth, J.T., Koffler, S.P., Pliskin, N.H., Reynolds, C.R., & Silver, C.H. (2005). Symptom validity assessment: Practice issues and medical necessity NAN policy & planning committee. *Archives of Clinical Neuropsychology*, 20, 419-426.
- Butcher, J.N., Dahlstrom, W.G., Graham, J.R., Tellegen, A., & Kaemmer, B. (1989). *MMPI-2: Manual for administration and scoring*. Minneapolis, MN: University of Minnesota Press.
- Carroll, L.J., Cassidy, J.D., Holm, L., Kraus, J., & Coronado, V.G. (2004a). Methodological issues and research recommendations for mild traumatic brain injury: the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine, Suppl 43*, 113-125.

- Carroll, L.J., Cassidy, J.D., Peloso, P.M., Borg, J., von Holst, H., Holm, L., Paniak, C., & Pépin, M. (2004b). Prognosis for mild traumatic brain injury: Results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. *Journal of Rehabilitation Medicine, Suppl 43*, 84-105.
- Choi, D. (1988). Glutamate neurotoxicity and diseases of the nervous system. *Neuron, 1*, 623-634.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2<sup>nd</sup> ed.). Hillsdale, NJ: Lawrence Erlbaum Associates, Publishers.
- 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.
- Curtis, K.L., Greve, K.W., & Bianchini, K.J. (2006). Attention and memory dysfunction in pain patients while controlling for effort on the California Verbal Learning Test-II. *Archives of Clinical Neuropsychology, 21*, 544-545. A poster presented at the 26<sup>th</sup> annual meeting of the National Academy of Neuropsychology, San Antonio, Texas.
- Dacey, R., Dikmen, S., Temkin, N., McLean, A., Jr., Armsden, G., & Winn, H.R. (1991). Relative effects of brain and non-brain injuries on neuropsychological and psychosocial outcome. *Journal of Trauma, 31*, 217-222.
- Dikmen, S., Machamer, J., & Temkin, N. (2003). Mild TBI with or without CT abnormalities. *Journal of the International Neuropsychological Society, 9*, 172-173.

- Dikmen, S., Machamer, J., Temkin, N., & McLean, A. (1990). Neuropsychological recovery in patients with moderate to severe head injury: 2 year follow-up. *Journal of Clinical and Experimental Neuropsychology*, *12*, 507-519.
- Dikmen, S.S., Machamer, J.E., Winn, H.R., & Temkin, N.R. (1995). Neuropsychological outcome at 1-year post head injury. *Neuropsychology*, *9*, 80-90.
- Dikmen, S., McLean, A., & Temkin, N. (1986). Neuropsychological and psychosocial consequences of minor head injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, *49*, 1227-1232.
- Dikmen, S., McLean, A., Temkin, N., & Wyler, A.R. (1986). Neuropsychologic outcome at one-month post injury. *Archives of Physical Medicine and Rehabilitation*, *67*, 507-513.
- Doane, B.M., Greve, K.W., & Bianchini, K.J. (2005). Agreement between the abbreviated and standard Portland Digit Recognition Test. *The Clinical Neuropsychologist*, *19*, 99-104.
- Dunn, L.M., & Dunn, L.M. (1997). *Peabody Picture Vocabulary Test-III*. Circle Pines, MN: American Guidance Service, Inc.
- Etherton, J.L., Bianchini, K.J., Greve, K.W., & Heinly, M.T. (2005). Sensitivity and specificity of reliable digit span in malingered pain-related disability. *Assessment*, *12*, 130-136.
- Faust, D., Hart, K., & Guilmette, T.J. (1988). Pediatric malingering: the capacity of children to fake believable deficits on neuropsychological testing. *Journal of Consulting and Clinical Psychology*, *56*, 578-582.
- Fishman, R. (1975). Brain edema. *New England Journal of Medicine*, *293*, 706-711.

- Gaetz, M. (2004). The neurophysiology of brain injury. *Clinical Neurophysiology*, 115, 4-18.
- Gennarelli, T.A. (1993). Mechanisms of brain injury. *The Journal of Emergency Medicine*, 11, 5-11.
- Gennarelli, T.A. (1996). The spectrum of traumatic injury. *Neuropathology and Applied Neurobiology*, 22, 509-513.
- Gennarelli, T.A., & Graham, D.I. (1998). Neuropathology of head injuries. *Seminars in Clinical Neuropsychiatry*, 3, 160-175.
- Goldstein, K. (1948). *Language and language disturbances*. New York: Grune & Stratton.
- Graham, D.I., Adams, J.H., & Doyle, D. (1978). Ischaemic brain damage in fatal non-missile head injuries. *Journal of the Neurological Sciences*, 39, 213-234.
- Green, P. (2003). Welcoming a paradigm shift in neuropsychology. *Archives of Clinical Neuropsychology*, 18, 625-627.
- Green, P. (2007). The pervasive influence of effort on neuropsychological tests. *Physical Medicine and Rehabilitation Clinics of North America*, 18, 43-68.
- Green, P., Allen, I., & Astner, K. (1996). *Manual for Computerised Word Memory Test*. Durham, NC: CogniSyst.
- Green, P., & Iverson, G.L. (2001a). Effects of injury severity and cognitive exaggeration on olfactory deficits in head injury compensation claims. *NeuroRehabilitation*, 16, 237-243.

- Green, P., & Iverson, G.L. (2001b). Validation of the computerized assessment of response bias in litigating patients with head injuries. *The Clinical Neuropsychologist, 15*, 492-497.
- Green, P., Rohling, M.L., Iverson, G.L., & Gervais, R.O. (2003). Relationships between olfactory discrimination and head injury severity. *Brain Injury, 17*, 479-496.
- Green, P., Rohling, M.L., Lees-Haley, P.R., & Allen, L.M. (2001). Effort has a greater effect on test scores than severe brain injury in compensation claimants. *Brain Injury, 15*, 1045-1060.
- Greiffenstein, M.F., Baker, W.J., & Gola, T. (1994). Validation of malingered amnesic measures with a large clinical sample. *Psychological Assessment, 6*, 218-224.
- Greve, K.W., & Bianchini, K.J. (2006a). Classification accuracy of the Portland Digit Recognition Test in traumatic brain injury: results of a known-groups analysis. *The Clinical Neuropsychologist, 20*, 816-830.
- Greve, K.W., & Bianchini, K.J. (2006b). Should the Retention trial of the Test of Memory Malingering be optional? *Archives of Clinical Neuropsychology, 21*, 117-119.
- Greve, K.W., Bianchini, K.J., & Doane, B.M. (2006). Classification accuracy of the Test of Memory Malingering in traumatic brain injury: results of a known-groups analysis. *Journal of Clinical and Experimental Neuropsychology, 28*, 1176-1190.
- 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*, 491-512.

- Greve, K.W., Ord, J., Curtis, K.L., Bianchini, K.J., & Brennan, A. (in press). Detecting malingering in traumatic brain injury and chronic pain: A comparison of three forced-choice symptom validity tests. *The Clinical Neuropsychologist*.
- Greve, K.W., Springer, S., Bianchini, K.J., Black, F.W., Heinly, M.T., Love, J.M., Swift, D.A., & Ciota, M.A. (2007). Malingering in toxic exposure: classification accuracy of reliable digit span and WAIS-III Digit Span scaled scores. *Assessment*, 14, 12-21.
- Groher, M. (1983). Communication disorders. In M. Rosenthal, E.R. Griffith, M.R. Bond, and J.D. Miller (eds.), *Rehabilitation of the Head Injured Adult*. Philadelphia: F.A. Davis.
- Gronwall, D., & Wrightson, P. (1980). Duration of post-traumatic amnesia after mild head injury. *Journal of Clinical Neuropsychology*, 2, 51-60.
- Guilmette, T.J., & Rasile, D. (1995). Sensitivity, specificity, and diagnostic accuracy of three verbal memory measures in the assessment of mild brain injury. *Neuropsychology*, 9, 338-344.
- Hagan, C. (1984). Language disorders in head trauma. In A. Holland (Ed.), *Language disorders in adults*. San Diego: College-Hill Press.
- Hannay, H.J., Howieson, D.B., Loring, D.W., Fischer, J.S., & Lezak, M.D. (2004). Neuropathology for neuropsychologists. In M.D. Lezak, D.B. Howieson, & D.W. Loring (Eds.), *Neuropsychological Assessment* (4<sup>th</sup> ed.). New York: Oxford University Press.
- 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, Inc.
- Heaton, R.K., Smith, H.H., Lehman, R.A.W., & Vogt, A.T. (1978). Prospects for faking believable deficits on neuropsychological testing. *Journal of Consulting and Clinical Psychology, 46*, 892-900.
- Heinly, M.T., Greve, K.W., Bianchini, K.J., Love, J.M., & Brennan, A. (2005). WAIS digit span–based indicators of malingered neurocognitive dysfunction: classification accuracy in traumatic brain injury. *Assessment, 12*, 429-444.
- Holm, S. (1979). A simple sequentially rejective multiple test procedure. *Scandinavian Journal of Statistics, 6*, 65-70.
- Ingraham, L.J. & Aiken, C.B. (1996). An empirical approach to determining criteria for abnormality in test batteries with multiple measures. *Neuropsychology, 10*, 120-124.
- Iverson, G.L. (2005). Outcome from mild traumatic brain injury. *Current Opinions in Psychiatry, 18*, 301-317.
- Iverson, G.L. (2006). Ethical issues associated with the assessment of exaggeration, poor effort, and malingering. *Applied Neuropsychology, 13*, 77-90.
- Iverson, G. L., & Franzen, M. D. (1996). Using multiple objective memory procedures to detect simulated malingering. *Journal of Clinical and Experimental Neuropsychology, 18*, 38-51.
- Iverson, G. L., & Tulsky, D. S. (2003). Detecting malingering on the WAIS-III Unusual Digit Span performance patterns in the normal population and in clinical groups. *Archives of Clinical Neuropsychology, 18*, 1-9.

- Jaccard, J., & Guilamo-Ramos, V. (2002). Analysis of variance frameworks in clinical child and adolescent psychology: issues and recommendations. *Journal of Clinical Child and Adolescent Psychology, 31*, 130-146.
- Jennett, B., & Teasdale, G. (1977). Aspects of coma after severe head injury. *Lancet, 878-881*.
- Kallakuri, S., Cavanaugh, J.M., Özaktay, A.C., & Takebayashi, T. (2003). The effect of varying impact energy on diffuse axonal injury in the rat brain: a preliminary study. *Experimental Brain Research, 148*, 419-424.
- Kaplan, E.F., Goodglass, H., & Weintraub, S. (1983). *The Boston Naming Test* (2<sup>nd</sup> ed.). Philadelphia: Lea & Febiger.
- Kiernan, R.J., Mueller, J., & Langston, J.W. (1995). COGNISTAT (Neurobehavioral Cognitive Examination). San Francisco: Northern California Neurobehavioral Group, Inc.
- Kraus, J.F., & Arzemanian, S. (1989). Epidemiologic features of mild and moderate brain injury. In J.T. Hoff, T.E. Anderson, & T.M. Cole (Eds.), *Mild to moderate head injury*. Boston, MA: Blackwell.
- Kraus, J.F., McArthur, D.L., Silverman, T.A., & Jayaraman, M. (1996). Epidemiology of brain injury. In R.K. Narayan, J.E. Wilberger, & J.T. Povlishock (Eds.), *Neurotrauma*. New York: McGraw-Hill.
- Kraus, J.F., & Nourjah, P. (1988). The epidemiology of mild, uncomplicated brain injury. *Journal of Trauma, 28*, 1637-1643.
- Larrabee, G.J. (2003). Exaggerated pain report in litigants with malingered neurocognitive dysfunction. *The Clinical Neuropsychologist, 17*, 395-401.

- Larrabee, G.J. (2005). Mild traumatic brain injury. In G.J. Larrabee (Ed.), *Forensic Neuropsychology*. New York: Oxford University Press.
- Larrabee, G.J. (in press). Aggregation across multiple indicators improves the detection of malingering: relationship to likelihood ratios. *The Clinical Neuropsychologist*.
- Lees-Haley, P. R., English, L. T., & Glenn, W. J. (1991). A fake bad scale on the MMPI-2 for personal injury claimants. *Psychological Reports*, 68, 203-210.
- Leininger, B.E., Gramling, S.E., Farrell, A.D., Kreutzer, J.S., & Peck, E.A. (1990). Neuropsychological deficits in symptomatic minor head injury patients after concussion and mild concussion. *Journal of Neurology, Neurosurgery, and Psychiatry*, 53, 293-296.
- Levin, H.S., Gary, H.E., Eisenberg, H.M., Ruff, R.M., Barth, J.T., Kreutzer, J., High, W.M., Jr., Portman, S., Foulkes, M.A., Jane, J.A., Marmarou, A., & Marshall, L.F. (1990). Neurobehavioral outcome 1 year after severe head injury: Experience of the Traumatic Coma Data Bank. *Journal of Neurosurgery*, 73, 699-709.
- Levin, H.S., Williams, D.H., Eisenberg, H.M., High, W.M., & Guinto, F.C. (1992). Serial MRI and neurobehavioural findings after mild to moderate closed head injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, 55, 255-262.
- Lipsey, M.W., & Wilson, D.B. (2000). Practical meta-analysis. *Applied Social Research Methods Series*, 49, Thousand Oaks, CA: Sage.
- Ludlow, C., Rosenberg, J., Fair, C., Buck, D., Schesselman, S., & Salazar, A. (1986). Brain lesions associated with nonfluent aphasia fifteen years following penetrating head injury. *Brain*, 109, 55-80.

- Luria, A. (1970). *Traumatic aphasia: Its syndrome, psychology and treatment*. The Hague: Mouton.
- Lynch, W.J. (2004). Determination of effort level, exaggeration, and malingering in neurocognitive assessment. *Journal of Head Trauma Rehabilitation, 19*, 277-283.
- Main, C.J. (1983). The Modified Somatic Perception Questionnaire (MSPQ). *Journal of Psychosomatic Research, 27*, 503-514.
- Mandolesi, G., Madeddu, F., Bozzi, Y., Maffei, L., & Ratto, G.M. (2004). Acute physiological response of mammalian central neurons to axotomy: ionic regulation and electrical activity. *The FASEB Journal, 18*, 1934-1936.
- Marquardt, T.P., Stoll, J., & Sussman, H. (1988). Disorders of communication in acquired cerebral trauma. *Journal of Learning Disabilities, 21*, 340-351.
- Mathias, J.L., & Coats, J.L. (1999). Emotional and cognitive sequelae to mild traumatic brain injury. *Journal of Clinical and Experimental Neuropsychology, 21*, 200-215.
- Maxwell, W.L., Watt, C., Graham, D.I., & Gennarelli, T.A. (1993). Ultrastructural evidence of axonal shearing as a result of lateral acceleration of the head in non-human primates. *Acta Neuropathologica, 86*, 136-144.
- Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine. (1993). Definition of mild traumatic brain injury. *Journal of Head Trauma Rehabilitation, 8*, 86-87.
- Millis, S.R., Putnam, S.H., Adams, K.M., & Ricker, J.H. (1995). The California Verbal Learning Test in the detection of incomplete effort in neuropsychological evaluation. *Psychological Assessment, 7*, 463-471.

- Minagar, A., Ragheb, J., & Kelley, R.E. (2003). The Edwin Smith surgical papyrus: Description and analysis of the earliest case of aphasia. *Journal of Medical Biography, 11*, 114-117.
- Mohr, J., Weiss, G., Caveness, W., Dillon, J., Kistler, J., Meirowsky, A., & Rish, B. (1980). Language and motor disorders after penetrating head injury in Viet Nam. *Neurology, 30*, 1273-1279.
- Moss, A., Jones, C., Fokias, D., & Quinn, D. (2003). The mediating effects of effort upon the relationship between head injury severity and cognitive functioning. *Brain Injury, 17*, 377-387.
- Muizelaar, J.P. (1996). CBF and patient management. In R.K. Narayan, J.E. Wilberger, & J.T. Povlishock (Eds.), *Neurotrauma*. New York: McGraw-Hill.
- Murdoch, B.E. (1990). *Acquired speech and language disorders: A neuroanatomical and functional neurological approach*. New York: Chapman and Hall.
- Nelson, N.W., Sweet, J.J., & Demakis, G.J. (2006). Meta-analysis of the MMPI-2 Fake Bad Scale: utility in forensic practice. *The Clinical Neuropsychologist, 20*, 39-58.
- Obrist, W.D., & Marion, D.W. (1996). Xenon techniques for CBF measurement in clinical head injury. In R.K. Narayan, J.E. Wilberger, & J.T. Povlishock (Eds.), *Neurotrauma*. New York: McGraw-Hill.
- Ommaya, A., & Gennarelli, T. (1974). Cerebral concussion and traumatic unconsciousness: correlation of experimental and clinical observations on blunt head injuries. *Brain, 97*, 633-654.
- Oppenheimer, D.R. (1968). Microscopic lesions in the brain following head injury. *Journal of Neurology, Neurosurgery, and Psychiatry, 31*, 299-306.

- Orme, D.R., Johnstone, B., Hanks, R., & Novack, T. (2004) The WRAT-3 Reading subtest as a measure of premorbid intelligence among persons with brain injury. *Rehabilitation Psychology, 49*, 250-253.
- Pankratz, L. (1983). A new technique for the assessment and modification of feigned memory deficit. *Perceptual and Motor Skills, 57*, 367-372.
- Povlishock, J.T., & Becker, D.P. (1985). Fate of reactive axonal swellings induced by head injury. *Laboratory Investigation, 52*, 540-552.
- Rabin, L.A., Barr, W.B., & Burton, L.A. (2005). Assessment practices of clinical neuropsychologists in the United States and Canada: A survey of INS, NAN, and APA Division 40 members. *Archives of Clinical Neuropsychology, 20*, 33-65.
- Raskin, S.A., & Rearick, E. (1996). Verbal fluency in individuals with mild traumatic brain injury. *Neuropsychology, 10*, 416-422.
- Rees, P.M. (2003). Contemporary issues in mild traumatic brain injury. *Archives of Physical Medicine and Rehabilitation, 84*, 1885-1894.
- Reitan, R.M., & Wolfson, D. (1993). *The Halstead-Reitan neuropsychological test battery: Theory and clinical interpretation (2<sup>nd</sup> ed.)*. South Tucson, AZ: Neuropsychology Press.
- Rimel, R.W., Giordani, B., Barth, J.T., & Jane, J.A. (1982). Moderate head injury: completing the clinical spectrum of brain trauma. *Neurosurgery, 11*, 344-351.
- Russell, W.R., & Espir, M.L.E. (1961). *Traumatic aphasia: A study of aphasia in war wounds of the brain*. Oxford: Oxford University Press.
- Saatman, K.E., Graham, D.I., & McIntosh, T.K. (1998). The neuronal cytoskeleton is at risk after mild to moderate brain injury. *Journal of Neurotrauma, 15*, 1047-58.

- Sarno, M.T. (1984). Verbal impairment after closed head injury. *The Journal of Nervous and Mental Disease*, 172, 475-479.
- Schiller, F. (1947). Aphasia studied in patients with missile wounds. *Journal of Neurology, Neurosurgery, and Psychiatry*, 10, 183-197.
- Schretlen, D.J., & Shapiro, A.M. (2003). A quantitative review of the effects of traumatic brain injury on cognitive functioning. *International Review of Psychiatry*, 15, 341-349.
- Schroder, M., Muizelaar, P., Bullock, R., Salvant, J., & Povlishock, J. (1995). Focal ischemia due to traumatic contusions documented by stable xenon-CT and ultrastructural studies. *Journal of Neurosurgery*, 82, 966-971.
- Sherer, M., & Madison, C.F. (2005). Moderate and severe traumatic brain injury. In G.J. Larrabee (Ed.), *Forensic Neuropsychology*. New York: Oxford University Press.
- Singleton, R.H., Zhu, J., Stone, J.R., & Povlishock, J.T. (2002). Traumatically induced axotomy adjacent to the soma does not result in acute neuronal death. *The Journal of Neuroscience*, 22, 791-802.
- Slick, D.J., Sherman, E.M.S., & Iverson, G.L. (1999). Diagnostic criteria for malingered neurocognitive dysfunction: Proposed standards for clinical practice and research. *The Clinical Neuropsychologist*, 13, 545-561.
- Smith, A. (1997). Development and course of receptive and expressive vocabulary from infancy to old age: administrations of the Peabody Picture Vocabulary Test, third edition, and the Expressive Vocabulary Test to the same standardization population of 2725 subjects. *International Journal of Neuroscience*, 92, 73-78.

- Spreen, O., & Strauss, E. (1998). *Compendium of neuropsychological tests* (2<sup>nd</sup> ed.). New York: Oxford University Press.
- Strich, S. (1956). Diffuse degeneration of the cerebral white matter in severe dementia following head injury. *Journal of Neurology, Neurosurgery, and Psychiatry*, *19*, 163-185.
- Strich (1961). Shearing of nerve fibres as a cause of brain damage due to head injury. *Lancet*, 443-8.
- Tait, R.C., Pollard, C.A., Margolis, R.B., Duckro, P.N., & Krause, S.J. (1987). The Pain Disability Index: psychometric and validity data. *Archives of Physical Medicine and Rehabilitation*, *68*, 438-441.
- Taylor, M.J., & Heaton, R. K. (2001). Sensitivity and specificity of WAIS-III/WMS-III demographically corrected factor scores in neuropsychological assessment. *Journal of the International Neuropsychological Society*, *7*, 867-874.
- Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: A practical scale. *Lancet*, 81-84.
- The Northern California Neurobehavioral Group, Inc. (1988). *Manual for the Neurobehavioral Cognitive Status Examination*. Fairfax, CA: The Northern California Neurobehavioral Group, Inc.
- Thurman, D.J., Alverson, C., Duna, K.A., Guerrero, J., & Sniezek, J.E. (1999). Traumatic brain injury in the United States: a public health perspective. *Journal of Head Trauma Rehabilitation*, *14*, 602-615.
- Tombaugh, T. (1996). *Test of Memory Malingering manual*. New York: MultiHealth Systems.



- Tombaugh, T.N. (1997). The Test of Memory Malingering (TOMM): Normative data from cognitively intact and cognitively impaired individuals. *Psychological Assessment*, 9, 260-268.
- Wechsler, D. (1981). *Wechsler Adult Intelligence Scale—Revised*. New York: Psychological Corporation.
- Wechsler, D. (1987). *The Wechsler Memory Scale--Revised*. New York: Psychological Corporation.
- Wechsler, D. (1997a). *Wechsler Memory Scale-III*. New York: Psychological Corporation.
- Wechsler, D.A. (1997b). *Wechsler Adult Intelligence Scale-III*. New York: Psychological Corporation.
- Whelan, B.M., Murdoch, B.E., & Bellamy, N. (2007). Delineating communication impairments associated with mild traumatic brain injury: a case report. *The Journal of Head Trauma Rehabilitation*, 22, 192-197.
- Wilkinson, G.S. (1993). *Wide Range Achievement Test 3*. Wilmington, DE: Jastak Associates/Wide Range Inc.
- Williams, D.H., Levin, H.S., & Eisenberg, H.M. (1990). Mild head injury classification. *Neurosurgery*, 27, 422-28.
- Yaghai, A., & Povlishock, J. (1992). Traumatically induced reactive change as visualized through the use of monoclonal antibodies targeted to the neurofilament subunits. *Journal of Neuropathology and Experimental Neurology*, 51, 158-176.

Zafonte, R.D., Wood, D.L., Harrison-Felix, C.L., Valena, N.V., & Black, K. (2001).

Penetrating head injury: A prospective study of outcomes. *Neurological Research*, 23, 219-226.

## Vita

Matthew Heinly was born on September 15, 1973 in Reading, Pennsylvania. He lived in Hamburg, Pennsylvania for 26 years, and is a December, 1996, graduate of Kutztown University of Pennsylvania with a Bachelor of Science degree (cum laude) in psychology. He entered the University of New Orleans in June, 2000, as a graduate student in the Department of Psychology. His area of specialization is Applied Biopsychology. He completed his M.S. degree in December, 2003 in Applied Biopsychology within the Department of Psychology at the University of New Orleans. He and his wife had a prematurely born son on August 10, 2005. His son was discharged from the hospital on August 24, 2005 and the family evacuated the New Orleans area three days later on August 27, 2005. Mr. Heinly relocated back to Pennsylvania after Hurricane Katrina. While in Pennsylvania, he has been working on his dissertation and has been hired as a tenure-track faculty member at his alma mater, Kutztown University of Pennsylvania.