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VERBAL LEARNING ABILITY AFTER TRAUMATIC BRAIN INJURY: ROLES OF WORKING MEMORY AND PROCESSING SPEED

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VERBAL LEARNING ABILITY AFTER TRAUMATIC
BRAIN INJURY: ROLES OF WORKING MEMORY
AND PROCESSING SPEED

by

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Dedication

To my parents, Kip and Carole, who taught me that with persistence

and determination, you can accomplish anything.

Thank you for your unwavering love and support.

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Verbal Learning Ability after Traumatic Brain Injury: Roles of Working Memory and Processing Speed

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Learning and memory impairments are among the most common and enduring cognitive consequences of traumatic brain injury (TBI). Researchers have yet to reach a consensus with regard to the basic cognitive mechanism underlying new learning and memory disturbances after TBI. The purpose of the present study was to investigate the current views regarding the cognitive processes thought to explain impairments in verbal learning and memory subsequent to brain injury. Specifically, this study sought to examine the roles of the central executive component of working memory and processing speed in verbal learning ability following TBI. Latent variable structural equation modeling (SEM) was used to analyze the data of 70 post-acute care TBI patients between the ages of 16 and 65, who completed a full neuropsychological evaluation. Results indicated that verbal learning and memory difficulties following TBI were explained primarily in terms of the central executive aspects of working memory, after accounting for the relative contributions of processing speed in the model. The direct effect of processing speed on verbal learning and memory was not significant when working memory was taken into account in the model. Rather, the effects of processing speed on verbal learning ability were largely indirect through the central executive component of working memory. Results highlight the importance of both working memory and

processing speed in supporting verbal learning and memory processes after TBI. Practical implications for targeting remediation efforts and directing approaches to memory rehabilitation are discussed in light of the study's findings.

Table of Contents

List of Tables

List of Figures

CHAPTER 1

Introduction

Traumatic Brain Injury (TBI), generally as a result of an accident, is the leading

cause of death and disability in young people, with an estimated 1.4 million cases in the

United States each year (King & Tyerman, 2009; Langlois, Rutland-Brown, & Thomas,

2006). Many individuals who survive a TBI are faced with multiple cognitive

impairments that may persist long after their injury. Although the degree of cognitive

impairment typically varies with the severity of brain injury, problems with new learning

and memory represent one of the most common and enduring sequelae following TBI

(Dikmen, Machamer, Winn, & Temkin, 1995; Levin & Hanten, 2004; Sirven &

Malamut, 2008; Vakil, 2005). This finding is not surprising given that human learning

and memory processes rely on the medial-temporal and frontal lobe structures, which are

commonly damaged as a result of brain injury (Wilson, Evans, & Williams, 2009).

Although cognitive functioning can gradually improve over time following TBI,

new learning and memory disturbances have been shown to persist even 10 years post-

injury and rehabilitation (Draper & Ponsford, 2008; Zec et al., 2001). Residual

impairment in the ability to learn and retain information is a devastating consequence of

brain injury and presents numerous challenges for everyday life. Given the prevalence

and persistence of resulting memory impairments as well as their significance in

everyday life, it is important for researchers to gain a better understanding of the basic

cognitive mechanisms that may limit or facilitate new learning processes following TBI.

1

Such advances could provide necessary insights for developing and refining effective approaches to memory rehabilitation.

The ability to learn and remember information reflects a complex process that typically involves three interrelated stages, namely encoding, consolidation, and retrieval. Encoding refers to the initial intake or acquisition of information, consolidation or storage refers to the maintenance and retention of the encoded information, and retrieval involves accessing or recovering the information when it is needed (Wilson, 2009). Research regarding the locus of the memory deficit in patients with TBI has provided variable results, but generally indicates that brain injuries can disrupt any or all of the stages in the learning process (Delis, Kramer, Kaplan, & Ober, 2000; Wilson et al., 2009). To some extent, the process of learning new information may depend on cognitive resources, such as working memory and processing speed, which are also commonly affected in patients with TBI (Luszcz & Bryan, 1999; Rios, Perianez, & Munoz-Cespedes, 2004; Salthouse, 1991).

Researchers studying the effects of TBI have been interested in the cognitive mechanisms that explain the neuropsychological impairments observed following brain injury, including poor verbal learning and memory. However, current views within the TBI literature differ with regard to the specific mechanisms thought to underlie these impairments (see Rios et al., 2004). Specifically, some researchers maintain that cognitive impairments following TBI emerge as a result of deficits in the attentional or executive control aspects of working memory (McAllister, Flashman, Sparling, & Saykin, 2004; McDowell, Whyte, & D'Esposito, 1997; Park, Moscovitch, & Robertson,

1999; Serino et al., 2006), whereas others have attributed the source of the cognitive

impairments to a general slowing of information processing, rather than a specific

functional deficit (Dikmen et al. 1995; Ferraro, 1996; Spikman, van Zomeren, &

Deelman, 1996; Vakil, 2005). Clearly, as indicated by the competing hypotheses, there is

a lack of consensus regarding the basic mechanism underlying new learning impairments

following brain injury. Thus, impairments in complex cognitive processes (i.e., learning)

after TBI could be attributable to a primary working memory deficit or may be secondary

to a broader processing speed impairment.

Working memory generally refers to the temporary storage and manipulation of

information. Impairments in working memory capacity are frequently observed in

individuals who have sustained a TBI. According to the empirically-derived, multiple

component model of working memory (Baddeley, 2000, 2007; Baddeley & Hitch, 1974),

a defining aspect of working memory is the *central executive*, which is responsible for

controlling and allocating attentional resources as well as planning and selecting

strategies for performing more complex tasks. Researchers have investigated the

functioning of the different components of working memory after TBI and their findings

suggest that TBI is specifically associated with an impairment of the central executive

(McDowell et al., 1997; Park et al., 1999; Vallat-Azouvi, Weber, Legrand, & Azouvi,

2007; Vallat-Azouvi, Pradat-Diehl, & Azouvi, 2009). Findings from neuroimaging

studies indicate that tasks known to tap the central executive system tend to activate the

frontal lobes of both hemispheres, and the dorsolateral prefrontal cortex in particular

(Collette & Van der Linden, 2002; Gathercole, 2008). Given that the brain regions that

are critical for working memory are particularly vulnerable to the effects of TBI, it is not surprising that its functioning is frequently impaired as a result of brain injury (McAllister et al., 2004). Accordingly, some authors identify the central executive aspects of working memory as the core deficit in individuals who have sustained a brain injury and propose that this deficit is responsible for a disturbance in many areas of cognitive ability, and learning and memory in particular (McAllister et al., 2004; McDowell et al., 1997; Park et al., 1999; Vallat-Azouvi et al., 2007).

A different interpretation has been proposed by several other researchers, who have attributed the source of impairments across various cognitive domains following TBI to reductions in processing speed, or a general slowing of cognitive subroutines (Evans, 2009; Ferraro, 1996; Vakil, 2005; Zahn & Mirsky, 1999). Processing speed is thought to reflect one's cognitive efficiency in performing simple mental operations. A reduction in speed of processing has been consistently found in patients who have sustained a TBI and is believed to be a major cognitive sequelae of TBI (Kinsella, 2008; Madigan, DeLuca, Diamond, Tramontano, & Averill 2000; Rios et al., 2004). Some researchers have found that processing speed is significantly related to executive processes in patients with TBI (Madigan et al., 2000) and findings from several studies suggest that slowed processing speed accounts for impairments in attention and working memory in TBI patients (van Zomeren & Brouwer, 1994; Willmott, Ponsford, Hocking, & Schonberger, 2009). Further, the effects of slowed processing speed appear to be more evident as task complexity and attentional demands increase (Lezak, 1995). According to studies within the cognitive aging literature, the speed at which information is processed

4

plays a large role in higher cognitive processes (i.e., learning), and in particular the amount of information remembered (Salthouse, 1993). Similarly, research with TBI patients indicates that significant impairments in processing speed can disrupt new learning ability (Chiaravalloti, Christodoulou, Demaree, & DeLuca, 2003). Therefore, adequate processing speed is presumably critical to adequate encoding and later retrieval of newly learned material (Chiaravalloti et al., 2003). Such findings lend support for the hypothesis that specific cognitive deficits observed subsequent to TBI are secondary as they can be attributed to a more general cognitive slowing.

Although researchers have established that TBI frequently disrupts new learning ability, the specific cognitive processes that are responsible for these impairments remain unclear. Specifically, questions remain as to whether new learning difficulties observed following brain injury are primarily generated by an impairment of the central executive component of working memory or a general slowing of processing speed. Thus, more research is needed to clarify the primary limiting factors that explain problems with new learning ability after TBI.

The purpose of this study was to investigate the leading hypotheses regarding the cognitive mechanisms that explain new verbal learning impairments subsequent to brain injury. In particular, this study sought to examine the nature of the relation between processing speed and working memory (i.e. the central executive component), and determine their roles in the ability to learn new verbal information following TBI. Isolating the cognitive mechanisms underlying acquisition and retrieval of newly learned information should contribute to a more advanced understanding of the nature of the

learning difficulties observed after TBI. Furthermore, implications from targeting

component parts could be useful for directing remediation efforts and facilitating

compensatory strategies, potentially leading to more efficient learning.

CHAPTER 2

Review of the Literature

This chapter will provide a review of the literature regarding the nature of

traumatic brain injury (TBI), the characteristics of learning and memory following

cerebral trauma, and the primary cognitive factors underlying learning and memory

impairments. The initial section will briefly describe the epidemiology and

neuropathology of brain injury as well as the cognitive consequences to provide a context

for understanding the clinical importance and rationale for the study. Next, a summary of

the findings on different aspects of memory that are vulnerable to the effects of TBI will

be provided. Research surrounding the leading hypotheses proposed to explain

impairments of learning and memory after TBI will then be presented. The review

concludes with a concise summary of the differing viewpoints and unresolved issues

regarding the basic cognitive mechanisms underlying verbal learning and memory

impairments following TBI.

The Nature of Traumatic Brain Injury

Epidemiology

As a leading cause of death and disability among children and young adults,

traumatic brain injury (TBI) remains a significant area of public health concern (Centers

for Disease Control and Prevention [CDC], 1999; King & Tyerman, 2009). In the United

States alone, it is estimated that around 1.4 million people sustain a Traumatic Brain

Injury (TBI) annually, resulting in approximately 50,000 deaths (Langlois et al., 2006).

Every year, approximately 80,000 to 90,000 Americans will experience permanent

disability as a result of their brain injury. Currently, an estimated 5.3 million men, women, and children are living with a long-term or life-long need for help with activities of daily living as a result of TBI (CDC, 1999).

A traumatic brain injury occurs when sudden trauma from external forces (e.g., blunt blow to the head, moving object, acceleration-deceleration forces) causes damage to the brain. Published studies analyzing prevalence rates indicate that adolescents, young adults, and the elderly are at highest risk of sustaining a TBI (CDC, 1999; Langlois et al., 2006). The rates of TBI are generally higher for males across the ages, with estimates of more than twice as many males as females (Boswell, McErlean, & Verdile, 2002). The leading causes of TBI are due to falls and motor vehicle or traffic-related accidents. Motor vehicle accidents are the primary cause in the younger group whereas falls are the most common cause among the elderly. Other reported TBI causes are attributed to violence, including assaults and being struck by an object.

Types of Brain Injury

Brain injuries can be classified as open or closed (King & Tyerman, 2009). *Open*, or penetrating, brain injuries occur when the skull and protective layers of the brain are damaged and exposed, such as from a gunshot wound to the head. *Closed*, or non-penetrating, brain injuries are associated with damage to the brain within an intact skull, typically arising from a blow to the head or impact from sudden changes in velocity (e.g., acceleration-deceleration). The effects of rapid acceleration-deceleration upon impact can cause unrestricted movement of the head, such that the brain collides with the surrounding surface of the skull (Gennarelli & Graham, 2005). Due to the way the brain

is positioned within the skull as well as the mechanics involved in a typical traumatic brain injury, the frontal and temporal regions of the brain are especially vulnerable to damage (Bigler, 1990).

Damage to the brain as a result of head injury can be broadly classified as either primary or secondary (King & Tyerman, 2009). *Primary* brain damage occurs at the time of injury and includes focal brain lesions (i.e., damage that is localized to a specific area of the brain) and diffuse axonal injury (i.e., damage that is widely distributed throughout the brain). *Secondary* brain damage occurs as an indirect consequence due to complications after the injury, such as hypoxia, hypotension, or increased intracranial pressure from brain tissue swelling.

Focal lesions, including contusions and hematomas, occur more frequently as a result of falls and direct blows, whereas diffuse axonal injury occurs more often as a result of acceleration/deceleration injuries, such as from motor vehicle accidents (Gennarelli & Graham, 2005; King & Tyerman, 2009). Studies have documented that focal lesions are most likely to occur in the inferior surface of the frontal lobe and around the temporal lobe poles due to the anatomy of the brain and skull (Bigler, 1990; Gennarelli & Graham, 2005; King & Tyerman, 2009). In addition, neuropathological studies have demonstrated that the hippocampus is particularly vulnerable to the effects of injury (Levin, Benton, Grossman, 1982; Levin & Hanten, 2004). Diffuse axonal injury (DAI) occurs as a result of the effects of shearing or stretching of neuronal fibers that connect different areas of the brain. The effects of DAI may lead to widespread damage to axons, especially throughout cerebral white matter areas and the brain stem (Bigler,

9

1990; Gennarelli & Graham, 2005). Evidence from neuropathological studies suggests that the typical pattern of brain injury is predominantly of a widespread, diffuse nature (Bigler, 1990; Ommaya & Gennarelli, 1974), in which the most vulnerable cortical regions are the frontal and temporal lobes (Adams, 1975; Draper & Ponsford, 2008; Fork et al., 2005).

Severity of Injury

Although a variety of factors may play a role in the course of recovery and determining long-term outcomes following TBI, some studies have found the severity of brain injury to be the most predictive (Lezak, Howieson, Loring, Hannay, & Fischer, 2004). Severity of injury refers to the degree of brain tissue damage and is typically described as mild, moderate, or severe. Approximately 75% of TBIs that occur each year can be classified as mild (CDC, 2003). Estimates of the percentage of moderate and severe injuries are approximately equal and comprise the remaining TBIs.

The Glasgow Coma Scale (GCS) is a commonly used classification system for determining the level of brain injury severity (Teasdale & Jennett, 1974). The GCS is a standardized measure that evaluates an individual's response to three aspects of consciousness: eye opening, movement, and verbal response. Total scores on the GCS range from 3 to 15, with lower scores indicating more severe injuries. Specifically, a brain injury is considered mild when a patient has a GCS score between 13 and 15, a moderate brain injury is identified with GCS scores from 9 to 12, and a severe brain injury with scores of 8 or lower. One limitation with using the GCS as an indicator of injury severity is that the scores obtained may depend on the timing of the assessment as

they can be quite variable, leading to inaccurate interpretations of injury severity (Lezak et al., 2004). For instance, initial GCS scores obtained at the scene of an accident could differ from scores obtained upon arrival to the emergency department, and the initial scores may not always be the lowest (Yeates, 2000). In addition, GCS scores may be affected by sedating medications and can be spuriously lower for patients requiring intubation since they cannot be assessed on the verbal part of the GCS. In light of these limitations, some clinicians and researchers have relied instead on post-traumatic amnesia (PTA) to indicate the severity of TBI (Lezak et al., 2004). The duration of post-traumatic amnesia (PTA) has been defined as "the period of time between injury and regaining continuous day-to-day memory for events" (King & Tyerman, 2009, pg. 5). Levels of severity associated with the length of PTA include: very mild (less than 5 minutes), mild (5 minutes to 1 hour), moderate (1 to 24 hours), severe (1 to 7 days), and very severe (more than 7 days; Russell, 1971). PTA duration has been shown to be more accurate than depth or duration of coma in predicting recovery of cognitive function (Brooks, Aughton, Bond, Jones, & Rizvi, 1980) and the degree of cognitive impairment long after injury (Draper & Ponsford, 2005).

Cognitive Sequelae

Although medical advances and improved treatments have contributed to an increased survival rate, the nature of TBI and associated damage frequently leaves survivors with a number of neuropsychological consequences, including physical, cognitive, emotional, and behavioral problems. Of particular relevance to this study are the cognitive consequences following TBI. There is a broad range of cognitive

11

consequences that can occur independently or in combination; however, the cognitive functions that are most frequently impaired are in the areas of attention, executive functioning, speed of information processing, and memory (Draper & Ponsford, 2005; King & Tyerman, 2009).

Although the severity of brain injury plays a role in the magnitude and degree of residual cognitive impairments (Dikmen et al., 1995), problems with new learning and memory represent one of the most commonly reported and debilitating cognitive impairments following TBI (Levin & Hanten, 2004; Sirven & Malamut, 2008; Vakil, 2005). Recent research reports that 54% to 84% of patients who have suffered a severe TBI experience memory problems (McKinlay & Watkiss, 1999). The persistence of memory impairments was also shown by Oddy and colleagues (1985), who found that 53% of patients and 79% of their families reported that injury-related deficits in memory functioning were evident seven years post-injury. Significant aspects of daily life, including independent functioning, depend considerably on one's ability to learn and remember new information. For instance, when the integrity of the memory system is compromised, it may manifest as problems with learning new material at school or work, remembering scheduled appointments, new phone numbers, or important conversations with family and friends. Given the high prevalence of persistent memory impairments after sustaining a brain injury and its importance for day-to-day functioning, identifying the mechanisms underlying learning and memory impairments in TBI patients could have vast implications for facilitating the overall process of memory rehabilitation. Before examining such mechanisms proposed to account for verbal learning and memory deficits

after TBI, the next section reviews the research on the basic structure of human memory

and the different aspects of memory that are commonly impaired subsequent to TBI.

<center>*Learning and Memory after TBI*</center>

Types of Memory

From the memory literature, it is evident that memory does not reflect a single,

unitary system, but it is made up of several distinct systems and processes (Schacter &

Tulving, 1994; Squire, 1992; Squire & Shrager, 2008). There are many models and

numerous terms that have been proposed to describe the complex processes that are

involved in memory, which can easily leave a reader confused (Roediger, Zaromb, &

Goode, 2008). To minimize this semantic confusion, the major distinctions and

components that are generally consistent among current conceptualizations are briefly

described in this section, with particular attention given to the subsystems and processes

relevant to this study. Research on memory with patients following TBI will be integrated

throughout this section.

Long-Term Memory

Many models of memory have emphasized the functional and structural

distinction between short-term and long-term storage systems, and substantial evidence

exists in the literature to support their dissociation (Levin & Hanten, 2004; Squire &

Shrager, 2008; Terry, 2003). Whereas short-term memory holds information for a few

seconds, long-term memory refers to the more stable storage of information, lasting

anywhere from minutes to years (Wilson, 2009). Long-term memory can be

conceptualized in terms of several parallel memory systems. The major division,

<center>13</center>

however, is typically between declarative and nondeclarative (or procedural) memory (Squire & Shrager, 2008). The declarative memory system involves conscious recollection of facts or acquired knowledge (i.e., semantic memory) and personal experiences or events (i.e., episodic memory). Nondeclarative memory occurs outside of conscious awareness and is associated with priming, skill learning, and habit formation. In the memory literature, declarative and nondeclarative memory are sometimes referred to as "explicit" and "implicit" memory, respectively, and generally have the same meanings (Squire & Shrager, 2008). Declarative memory is of primary relevance to the current research as it is the memory system generally associated with the ability to learn and remember information.

The impact of TBI on memory functioning has been examined for different modalities in which the information is processed or presented (i.e., verbal, visual, olfactory). Relevant to the present study, however, are the cognitive mechanisms underlying verbal learning and memory impairments after TBI. Accordingly, the research referred to here will highlight the findings on memory functioning within the auditory or verbal domain.

Findings from neuropsychological and neuroimaging studies have provided the foundation for understanding learning and memory functioning after TBI. Neuroimaging studies have documented that declarative memory depends on the hippocampus and related structures within the medial temporal lobe (Levin & Hanten, 2004; Roediger et al., 2008; Squire & Shrager, 2008), which, as previously noted, are highly vulnerable to TBI. The significance of the frontal regions for learning and memory has also been

14

documented, which is thought to reflect the use of more efficient encoding and retrieval strategies in facilitating recall (Baldo & Shimamura, 2002). Evidence from a recent study by Johnson and colleagues (2001) provided support for the involvement of bilateral frontotemporal regions in verbal learning and memory using functional magnetic resonance imaging (fMRI) during performance on a list-learning task.

A number of neuropsychological studies have examined the characteristics of verbal learning and memory after TBI. Typical measures of verbal memory used within these studies rely heavily on one's capacity to acquire and retrieve particular information. These measures are associated with the neuropsychological tests of learning and memory that usually involve recall and recognition of a word list presented in a multi-trial format, such as the California Verbal Learning Test or the Rey-Auditory Verbal Learning Test (Lezak et al., 2004; Strauss, Sherman, & Spreen, 2006). The consistent finding across studies is that both child and adult patients in the post-acute phase following moderate to severe brain injury demonstrate impairments in immediate and delayed memory for auditory-verbal information (Baddeley, Harris, Sunderland, Watts, & Wilson, 1987; Curtiss, Vanderploeg, Spencer, & Salazar, 2001; Levin & Hanten, 2004; Vakil, 2005; Vakil, Arbell, Gozlan, Hoofien, & Blachstein, 1992; Yeates, Blumenstein, Patterson, & Delis, 1995; Zec et al., 2001).

Process of Learning and Remembering

Memory can also be conceptualized in terms of a series of stages involved in the process of learning and remembering. Specifically, information must first be registered and encoded, then consolidated and stored, and finally must be retrieved when the

15

information is needed. Dysfunction at any of these stages can manifest as problems in the ability to learn and remember new information. Deficits in *encoding* reflect problems in the ability to attend to and register incoming information. Such deficits can be identified by a significantly slower rate of learning and may reflect poor use of semantic organization or learning strategies compared to controls (DeLuca, Schultheis, Madigan, Christodoulou, & Averill, 2000). *Consolidation* requires the maintenance, elaboration, and storage of new information that is encoded. When information is not consolidated effectively, it will quickly be forgotten. Thus, problems at this stage can be identified by a more rapid rate of forgetting or poor recognition after a delay (Vanderploeg, Crowell, & Curtiss, 2001). A deficit at the *retrieval* stage implies the preservation of acquisition and retention abilities, but problems with retrieving the stored information from long-term memory. Retrieval deficits are typically identified when individuals perform poorly on delayed free recall tasks with improved performance after some type of retrieval cue, such as with cued recall or recognition tasks (Duchnick, Vanderploeg, & Curtiss, 2002).

The presentation of information in a repeated trials format also allows for the assessment of the rate of learning, defined by the amount of additional information recalled per trial (Vakil, 2005). Several studies examining this aspect of memory have reported that the learning rate of verbal material in TBI patients is significantly impaired relative to controls (Blachstein, Vakil, & Hoofien, 1993; Constantinidou, Neils, Bouman, & Lee, 1996; Gardner & Vrbancic, 1998; Zec et al., 2001). That is, patients who have sustained a TBI tend to acquire verbal information at a disproportionately slower pace. In addition, severe TBI patients exhibit more recall errors, such as intrusions and

16

perseverations, and display poor use of learning strategies to facilitate recall (Carlesimo, Sabbadini, Loasses, & Caltagirone, 1997; Gruen, Frankie, & Schwartz, 1990; Levin & Hanten, 2004; Vakil et al., 1992).

Attempts to identify patterns of performance among TBI patients by analyzing the components of the learning process have generated a range of conclusions, with researchers reporting a specific dysfunction at various stages. In line with results indicating a slower learning rate, some investigators have concluded that compromised memory in TBI patients is a function of specific problems in the initial acquisition of information (DeLuca et al., 2000) and a general failure to apply effective encoding strategies when learning (Curtiss et al., 2001). Other studies, however, purport that memory problems observed after TBI reflect an underlying deficit in the retrieval of stored information (Baum, Vanderploeg, & Curtiss, 1996; Duchnick et al., 2002). In contrast to these findings, verbal learning and memory problems post-TBI have also been found to be attributable to impairments in the consolidation process (Vanderploeg et al., 2001). Some authors have also raised the question of whether memory disorder subtypes exist within TBI (Millis & Ricker, 1994; Vanderploeg et al., 2001) and a recent study found patterns of memory dysfunction corresponding to specific deficits in consolidation, retention, and retrieval processes (Curtiss et al., 2001).

Although more research may be needed to clarify the presence of a specific pattern of memory dysfunction, the general conclusion in the literature suggests that impairments of verbal learning and memory are a likely outcome of TBI. As Levin and Hanten (2004) articulated, "Essential to the eventual development of effective

17

intervention techniques is the elucidation of the specific underlying causes of impairment" (pg. 47). Thus, the following section will review the basic cognitive factors that have been hypothesized to compromise the integrity of learning and memory after TBI.

<div align="center">*Cognitive Variables Contributing to Learning and Memory Impairments*</div>

A number of studies on normal cognitive aging as well as with brain-injured populations have attempted to better understand the cognitive mechanisms underlying deficits in learning and memory. In particular, the literature points toward dominant cognitive resources (e.g., working memory and processing speed) that influence an individual's ability to successfully learn and remember new information. This section provides an overview of the concepts of working memory and processing speed, and reviews the differing perspectives and relevant research examining how these factors contribute to verbal learning and memory impairments following traumatic brain injury.

Role of Working Memory

What is Working Memory? Working memory is the theoretical construct that refers to the limited capacity system used for holding and actively manipulating task-relevant information (Baddeley & Della Sala, 1998; Baddeley & Logie, 1999). Working memory has been shown to play a significant role in a range of complex cognitive processes, including comprehension, problem solving, learning, reasoning, and active listening (Baddeley, 1994; Levin & Hanten, 2004). Briefly, as Shah and Miyake (1999) pointed out, there is much confusion surrounding the concepts of short-term memory and working memory, and even the delineation of the two in many textbooks is often

inconsistent or contradictory. In addition, the relation between these concepts has been defined in different ways by various theoretical frameworks over the years and the terms are often used interchangeably, which undoubtedly adds to the confusion. However, the emphasis in more recent theoretical approaches has been on the distinction between these two concepts, which is increasingly acknowledged in the field (Shah & Miyake, 1999). Findings from Kail and Hall (2001) provided additional evidence for the distinction between working memory and short-term memory using both exploratory and confirmatory factor analyses. Gathercole (2008) clarified that "the term working memory refers to the whole set of cognitive processes that comprise the model, which...includes higher-level attentional and executive processes as well as storage systems specialized for particular information domains" (pg., 150). The concept of working memory, therefore, is thought to subsume short-term memory, which in Baddeley's model involves only the passive, time-limited storage of information (Engle, Tuholski, Laughlin, & Conway, 1999; Kintsch, Healy, Hegarty, Penington, & Salthouse, 1999; Roediger et al., 2008).

The multiple component model of working memory, originally proposed by Baddeley and Hitch (1974) and subsequently revised by Baddeley (2000), is commonly cited as one of the most prominent and widely accepted theoretical models of working memory (Anderson, 2008; Gathercole, 2008; Strauss et al., 2006). This theoretical model, depicted in Figure 1, is considered a well-validated theoretical model derived from extensive research with healthy children and adults as well as patients with brain damage (Anderson, 2008; Baddeley, 2007; Baddeley & Logie, 1999). According to the model's current conceptualization (Baddeley, 2000, 2002, 2003, 2007), working memory is

viewed as a multicomponent system that involves three temporary storage systems (i.e.,

phonological loop, visuospatial sketchpad, and episodic buffer) and a higher-level

attentional controller (i.e., the central executive). The *phonological loop* is responsible

for the temporary storage of auditory-verbal information, whereas the *visuospatial*

sketchpad maintains visual and spatial information. The *episodic buffer*, the most recent

addition to the model, performs a similar function in that it is also a limited-capacity

storage system, but it serves to bind together information from the other subsystems into

integrated episodes (Baddeley, 2000; 2007). At the core of the working memory model

remains the *central executive*, which is responsible for controlling and coordinating the

subsidiary systems by allocating attentional resources and planning and selecting

strategies for performing more complex cognitive tasks.
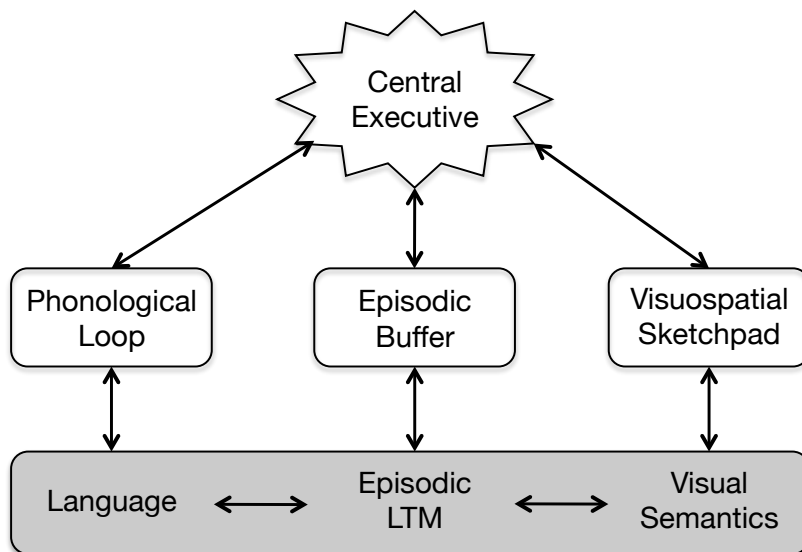
Figure 1. The Multiple Component Model of Working Memory (adapted from Baddeley,
2000).

According to Baddeley (1996, 2000, 2003), there are four main functions of the central executive component. First, the central executive is responsible for the capacity to *focus attention*, which involves exclusively attending to task-relevant information while ignoring distractions and other irrelevant information. A second function includes *divided attention*, which involves the simultaneous processing of multiple tasks or different sources of information at a time. A third role of the central executive involves *switching attention*, which refers to the capacity for shifting attention back and forth between different tasks or sources of information (Baddeley, 2002). Dysfunction in this area can lead to perseverative behavior and manifests as poor cognitive flexibility (Anderson, 2008). The fourth function of the central executive is to engage the long-term memory system temporarily when needed for the selective activation and manipulation of stored information (Anderson, 2008; Collette & Van der Linden, 2002).

Several alternative theoretical models of working memory have been proposed, which have been referred to as attentional-based accounts of working memory (Gathercole, 2008; see Miyake & Shah, 1999 for a thorough review and critique of existing models). Although these conceptualizations have used various terms to describe the key aspects of working memory, the majority of these alternative models also stress the role of a higher-level attentional control function, similar to Baddeley's central executive, as a crucial part of the working memory system (Gathercole, 2008).

Working Memory Hypothesis. As noted previously, working memory impairments are frequently observed after TBI, as the brain regions that are critical for working memory are particularly vulnerable to damage from TBI. Studies examining the

functioning of the different components of working memory after TBI suggest that the more complex attentional and executive processes associated with working memory are preferentially affected, with relative sparing of short-term storage and rehearsal (McDowell et al., 1997; Park et al., 1999; Turner & Levine, 2008; Vakil, 2005; Vallat-Azouvi et al., 2007; Vallat-Azouvi et al., 2009). A number of authors propose that these aspects of working memory represent a core deficit following TBI and further purport that neuropsychological impairments, including verbal memory, emerge as a result of these specific deficits (Azouvi, Jokie, van der Linden, Marlier, & Bussel, 1996; McAllister et al., 2004; McDowell et al., 1997; Park et al., 1999; Serino et al., 2006; Vallat-Azouvi et al., 2007). In other words, according to the working memory hypothesis, individuals who have sustained a TBI have a specific deficit in the executive or attentional control aspects of working memory, which diminishes their ability to learn and retain new information.

The capacity of working memory is thought to play a direct role in the ability to effectively encode and subsequently retrieve information. In particular, impaired working memory capacity may result in fewer associations made among elements of the encoded material, leading to weaker representations in long-term memory and more rapid decay of memory traces (Luszcz & Bryan, 1999). There is some recent evidence to support the hypothesis that learning and memory impairment in TBI patients stems from an attentional or executive working memory deficit. Children who have sustained a TBI have been shown to be less efficient at acquiring new verbal information, which was partially attributed to working memory impairments (Mandalis, Kinsella, Ong, &

22

Anderson, 2007). Another recent study with adults sought to determine the extent to which deficits in attention or executive control after TBI influenced performance on episodic memory tasks (Mangels, Craik, Levine, Schwarz, & Stuss, 2002). Findings from this study provide further support for the notion that learning and memory deficits observed after TBI are secondary to deficits in executive control. Thus, the central executive aspects of working memory are emphasized as playing a critical role in verbal learning and memory functioning after TBI. However, rarely have researchers studying the effects of TBI on memory functioning simultaneously accounted for the relative contributions of both the central executive and processing speed. As highlighted in the next section, processing speed is an important factor outside working memory that may influence how well information is learned and recalled (Salthouse, 1996; Strauss et al., 2006).

Role of Processing Speed

Processing speed is an important cognitive resource that involves the ability to process information efficiently or to perform relatively simple mental operations quickly. Impaired processing speed has been repeatedly observed in patients who have sustained TBI and is often viewed as a major cognitive sequelae of TBI (Kinsella, 2008; Madigan et al., 2000). It has been argued that "...the speed with which elementary cognitive operations are executed places fundamental limits on most aspects of cognition, including remembering" (Luszcz & Bryan, 1999, pg. 3). In this light, processing speed is considered to be a more basic cognitive function, and therefore, must be preserved for other cognitive processes to function properly (Rios et al., 2004).

Processing Speed Hypothesis. In contrast to the working memory hypothesis, a number of other researchers contend that patients who have sustained TBI do not have a specific deficit in the attentional or executive aspects of working memory, but rather a characteristic reduction in cognitive and perceptual speed (Rios et al., 2004). Proponents of this viewpoint claim that the source of TBI-related impairments across various cognitive domains, including learning and memory, can be directly attributed to a more generalized slowing in processing speed (Evans, 2009; Vakil, 2005). As Rios and colleagues (2004) have previously reported, several studies have offered support for the slowed processing hypothesis with TBI patients (Ferraro, 1996; Gronwall & Wrightson, 1981; Stuss et al., 1989; Van Zomeren & Brouwer, 1994; Zahn & Mirsky, 1999).

More specifically, in relation to learning and memory, impaired processing speed is thought to hinder adequate encoding during the learning process since the amount of information that can be processed in the available time is significantly reduced. Indeed, it has been reported that the speed at which an individual processes stimuli significantly contributes to memory performance after TBI (Chiaravalloti et al., 2003). Similarly, some studies have found that memory is significantly impaired in TBI patients relative to controls, but that these effects are eliminated after controlling for processing speed (Timmerman & Brouwer, 1999; Veltman, Brouwer, van Zomeren, & van Wolffelaar, 1996). These results point toward processing speed as the source of the learning and memory impairments in patients with TBI.

Relation between Processing Speed and Working Memory. In general, processing speed is considered to reflect a more basic, automatic function, whereas working memory

is thought to reflect more controlled processing and require more effort. Accordingly, some studies within the TBI literature have investigated the contribution of processing speed to the capacity of working memory. For instance, there is some evidence to suggest that slowed processing speed accounts for impairments in attention and working memory in TBI patients (van Zomeren & Brouwer, 1994). Recently, Willmott and colleagues (2009) used structural equation modeling (SEM) to examine the contribution of impaired processing speed and strategic control of attention to performance of attentional tasks of increasing complexity. The results of this study provided evidence that impaired performance on complex attentional tasks following TBI can be attributed to slowed information processing. Taken together, these studies offer some support for the contribution of processing speed to aspects of working memory in TBI patients.

Within the literature on cognitive aging, Salthouse (1993) has indicated that "with more complex cognitive measures, the speed influence may be largely indirect and perhaps mediated by an impairment in the functioning of working memory" (pp. 735). This would suggest that processing speed influences working memory, which in turn influences verbal learning ability. Given the aforementioned findings from the literature reviewed, it seems plausible that processing speed and working memory could work together in this way to explain verbal learning impairments following TBI. To the best of the author's knowledge, however, published studies within the TBI literature have yet to investigate the potential mechanism (i.e., working memory) through which processing speed exerts its influence on new learning. Thus, whether the influence of processing speed on new learning in TBI patients is primarily indirect or possibly mediated through

the central executive component is a question that has not been completely answered. Furthermore, although the contributions of processing speed and the central executive aspects of working memory have been examined separately in various studies, surprisingly, the relative contributions of these variables to verbal learning ability following TBI have not yet been examined concurrently in a more comprehensive manner. Hence, more research is needed in this area to address the unresolved issues and gain a better understanding of the role that these cognitive variables play in verbal learning after TBI.

Summary of the Problem

To briefly summarize, research has documented that traumatic brain injury frequently results in widespread, diffuse axonal injury, with the frontal and temporal regions being highly susceptible areas to damage. Given the functional significance of these areas, it is not surprising that verbal learning and memory abilities are frequently disrupted following TBI. In fact, there is a considerable amount of research reporting that TBI frequently disrupts an individual's ability to learn and retain new information. Understanding why an individual may perform poorly on measures of learning and memory is especially important for targeting remediation efforts. In addition, knowing what specific factors constrain successful learning could help practitioners identify TBI patients who are likely or unlikely to benefit from particular types of intervention techniques (DeLuca et al., 2000).

A review of the relevant literature reveals differing viewpoints regarding the primary mechanism (i.e., working memory versus processing speed) thought to explain

26

impaired learning and memory after TBI. In particular, there is some evidence to support

a *working memory hypothesis*, which suggests that patients who have suffered a TBI have

a specific deficit in the central executive component of working memory and that this

deficit disrupts their ability to adequately encode and retrieve new information. Other

lines of evidence reviewed, however, provide support for a *processing speed hypothesis*,

which attributes the source of the impairments associated with TBI to a characteristically

slow rate of information processing, rather than a deficit in any particular function. In

examining the literature surrounding these two dominant theoretical accounts, several

unresolved issues have emerged. First, as indicated by the competing hypotheses, a

general consensus regarding the specific mechanism underlying learning and memory

after TBI has not been reached. Therefore, it remains unclear as to whether impairments

in learning and memory after TBI primarily reflect slow processing speed or reduced

working memory capacity. Second, questions regarding the way in which processing

speed may influence verbal learning ability after TBI remain.

The purpose of the present study was to evaluate the dominant theoretical

accounts proposed to explain impairments in verbal learning and memory subsequent to

brain injury: working memory (i.e. the central executive component) and processing

speed. In addition, this study investigated whether processing speed exerts its influence

on verbal learning primarily directly or indirectly through working memory. To allow for

a more accurate evaluation of the competing hypotheses, the relations among the

specified latent variables (processing speed, working memory, and verbal learning) were

analyzed simultaneously using latent variable structural equation modeling (SEM). An

advantage to using a latent variable approach is that it minimizes task invalidity by extracting what is common among the tasks so that a more pure version of the construct of interest is represented (Miyake et al., 2000). Through the application of such statistical techniques, the cognitive mechanisms thought to contribute to learning and memory can be teased apart, and a better understanding of the nature of the learning difficulties observed after TBI can potentially be gained. Such an advanced understanding should have important implications for guiding the development of more effective intervention strategies.

Research Questions and Hypotheses

Research Question 1

Are verbal learning problems after TBI better explained by a specific impairment of the central executive component of working memory or a more general reduction in processing speed?

Hypothesis 1. It is hypothesized that the central executive aspects of working memory will explain verbal learning ability after TBI to a statistically significant degree, even after controlling for processing speed.

Research Question 2

How does processing speed influence verbal learning after TBI? Specifically, does processing speed primarily influence learning directly or indirectly through the central executive component of working memory?

Hypothesis 2. It is hypothesized that processing speed will primarily influence

verbal learning indirectly through the central executive component of working memory.

CHAPTER 3

Method

Participants

This study used an archival neuropsychological dataset of 80 post-acute care

patients, who had sustained an acquired brain injury. Participants were obtained from a

post-acute neurorehabilitation hospital in Texas and were referred for a comprehensive

neuropsychological evaluation as part of their routine clinical care. All participants had

emerged from post-traumatic amnesia (PTA) prior to completing any neuropsychological

testing. Individuals were selected for inclusion in the current study if the following

criteria were met: (a) they had a brain injury resulting from trauma, (b) were between the

ages of 16 and 65 at the time of injury, (c) had functional use of their dominant hand, and

(d) reported English as their primary language. Of the 80 participants, 10 did not meet the

inclusion criteria for the study due to the presence of a non-traumatic acquired brain

injury (e.g., cerebrovascular accident or hypoxic injury). As a result, 70 patients were

selected for inclusion in this study.

Demographic characteristics and injury-related information for the current sample

are presented in Table 1. Participants' ages ranged from 16 to 63, with a mean age of 34.1

years ($SD = 14.76$). Males represented a greater proportion of the sample (80%; $n=56$)

than females (20%; $n=14$), which is largely comparable to percentages reported by other

studies as well as epidemiological data indicating the incidence of TBI to be higher

among males than females. Level of education ranged from 8 to 20 years, with a mean of

13.6 (*SD* = 2.66). The majority of participants were Caucasian (84.3%), followed by

Hispanic (12.9%), Native American (1.4%), and Asian/Pacific Islander (1.4%).

Most of the participants' traumatic brain injuries resulted from motor vehicle

accidents (42%) and falls (21.7%). Remaining causes of injuries were related to

motorcycle accidents (15.9%), struck by/against event (7.2%), cycling (5.8%), gunshot

(2.9%), pedestrian/vehicle accident (1.4%), assault (1.4%), and sports-related accident

(1.4%). Mean duration of post-traumatic amnesia (PTA) was 11.4 days (*SD* = 12.68). The

average time from onset of injury to evaluation was 73 days (*SD* = 135.86).

Table 1

Demographic characteristics and injury-related information

Variable	*M*	SD	Range	*n*	Percentage
Age (years)	34.1	14.76	16 – 63		
Education (years)	13.6	2.66	8 – 20		
Time post-injury (days)	73	135.86	5 – 672		
PTA (days)[a]	11.4	12.68	0 – 49		
Gender					
1 = Males				56	80
2 = Females				14	20
Race					
Caucasian				59	84.3
Hispanic				9	12.9
Native American				1	1.4
Asian/Pacific Islander				1	1.4
Etiology					
MVA				29	42.0
Falls				15	21.7
Motorcycle				11	15.9
Struck by/against event				5	7.2
Cycling				4	5.8
Gunshot				2	2.9
Pedestrian/Vehicle				1	1.4
Assault				1	1.4
Sports				1	1.4
Duration of PTA [a]					
< 5 minutes				8	11.8
5 minutes to 1 hour				2	2.9
1 to 24 hours				7	10.3
1 to 7 days				20	29.4
> 7 days				31	45.6

Note. PTA = post-traumatic amnesia. MVA = motor vehicle accidents.
[a]Estimate of PTA was unavailable for 2 participants.

Measures

A subset of tests were selected from the full neuropsychological test battery to represent the latent constructs of interest in this study and are described in the following section. Table 2 includes a list of the selected subtest indicators organized by theoretical construct. For reference purposes, the complete neuropsychological test battery administered to TBI patients as part of their routine clinical care is listed in Appendix A.

Table 2

Latent Constructs and Associated Subtest Indicators

Latent Construct	Subtest Indicators
Processing Speed	WAIS-IV Symbol Search WAIS-IV Coding Trails A
Central Executive Working Memory	WAIS-IV Digit Span Backward WAIS-IV Digit Span Sequencing Trails B Booklet Category Test
Verbal Learning	CVLT-II: List A Trials 1-5 Total CVLT-II: List B Immediate Recall CVLT-II: Short-Delay Free Recall CVLT-II: Short-Delay Cued Recall CVLT-II: Long-Delay Free Recall CVLT-II: Long-Delay Cued Recall CVLT-II: Total Recognition

Demographics Form

A demographics form (Appendix B) was completed at the time of testing to obtain relevant demographic and injury-related information, including age, gender, education, race/ethnicity, employment status prior to injury, days post-injury, and etiology of trauma (e.g., motor vehicle collision, fall, assault, etc.).

Wechsler Adult Intelligence Scale – Fourth Edition (WAIS-IV)

The Wechsler Adult Intelligence Scale – Fourth Edition (WAIS-IV; Wechsler, 2008) is an individually administered intelligence battery intended for use with adults ages 16 through 90. The battery was normed on a national sample of 2,200 people aged 16 to 90 and stratified based on the 2005 U.S. Census characteristics for age, sex, ethnicity, education level, and geographic region. Three subtests from the WAIS-IV were selected for use in this study: Digit Span, Symbol Search, and Coding. The WAIS-IV manual provides good evidence for internal consistency of these subtests in the standardization sample (Wechsler, 2008). Reliability coefficients for the subtests included in this study were .83 (Digit Span), .86 (Coding), and .81 (Symbol Search). A brief description of the selected subtests is provided below.

Digit Span is a core subtest within the Working Memory scale of the WAIS-IV and is made up of three components: (1) Digit Span Forward, in which the examinee repeats numbers in the same order as they were presented by the examiner, (2) Digit Span Backward, in which the examinee repeats the numbers in the reverse order, and (3) Digit Span Sequencing, in which the examinee repeats the numbers in ascending numerical order. Separate process scores are available on the WAIS-IV for each of the three Digit

Span tasks as they are thought to measure unique mental activities (Lezak et al., 2004; Wechsler, 2008). Specifically, Digit Span Forward measures memory span, and Digit Span Backward and tasks similar to Digit Span Sequencing place additional demands on working memory and require mental manipulation (Wechsler, 2008; Werheid et al., 2002). Memory span refers to the immediate recall of temporal information following a single presentation, whereas working memory describes the ability to temporarily store and perform mental manipulations with information that involves divided attention (McGrew, 2005). Using the multicomponent model of working memory (Baddeley & Hitch, 1974), Digit Span Forward is thought to reflect the phonological loop while Digit Span Backward involves the central executive component (Isaacs & Vargh-Khdem, 1989; Vakil, 2005). Tasks similar to Digit Span Sequencing have also been described as measuring the central executive component of verbal working memory (Hoppe, Muller, Werheid, Thone, & von Cramon, 2000; Werheid et al., 2002). Accordingly, Digit Span Backward and Digit Span Sequencing were included in the present study to assess the latent construct of central executive working memory.

Symbol Search and Coding are the core subtests comprising the Processing Speed scale in the WAIS-IV. In the Symbol Search subtest, the examinee scans a group of symbols and determines whether any of the target symbols are present within the larger group of symbols in a given amount of time. The Coding subtest requires the examinee to copy symbols paired with numbers according to a key within a specified time limit. Scores for each subtest reflect the total number of items completed correctly within the

time limit. Both Symbol Search and Coding were included in this study as measures of the latent construct of processing speed.

Trail Making Test

The adult version of the Trail Making Test (TMT; Reitan & Wolfson, 1985) is designed for ages 15 to 89 years and measures attention, speed, and cognitive flexibility (Straus et al., 2006). The test is composed of two parts, Trails A and Trails B. Trails A consists of an array of encircled numbers scattered on a page, and requires the examinee to draw lines to connect the circles in sequential order as quickly as possible. Trails B consists of a mixed array of encircled numbers and letters, and requires the examinee to draw lines to connect the numbers and letters in an alternating consecutive sequence (e.g., 1–A–2–B–3–C) as quickly as possible. Scores are reported in terms of completion time (in seconds) for each of the two parts, with lower scores indicating better performance. Of the reported reliability coefficients for the TMT, the majority are above .80, with estimates ranging from the .60s to over .90 (Lezak et al., 2004).

Although both parts of the TMT involve visual scanning, sequencing, and speeded processing, part B additionally involves divided attention and cognitive flexibility as it requires the ability to attend to two different task demands simultaneously and shift back and forth between them (Kinsella, 1998; Strauss et al., 2006). Trails A has been denoted as a measure of processing speed, and Trails B has been used as an index of working memory processes associated with executive control (Arbuthnott & Frank, 2000; Kortte, Horner, & Windham, 2002; Nestor, Niznikiewicz, & McCarley, 2010; Oosterman et al., 2010). Because Trails B requires an individual to continuously shift their attention,

it is commonly included as a measure of prefrontal function (Lezak et al., 2004). The additional cognitive demands tapped by Trails B, including working memory, divided attention, and task switching capacity, are thought to represent central executive processes (Baddeley, 2007). Accordingly, the completion time for Trails A was included in this study as an indicator for the processing speed latent variable and the completion time for Trails B was included as an indicator for the central executive working memory latent variable.

Booklet Category Test

The booklet version of the Category Test (BCT; DeFilippis & McCampbell, 1997) continues to be one of the most popular and widely used measures of executive function (Rabin, Barr, & Burton, 2005; Strauss et al., 2006). The BCT is described as a complex measure of concept formation, abstract reasoning, novel problem solving, and cognitive flexibility (Lezak et al., 2004; Strauss et al., 2006). The adult version of the test is intended for ages 15 years and older. The BCT is made up of a series of 208 visually presented items within seven subtests. The initial six subtests are organized by different underlying principles and the seventh and final subtest consists of items previously shown. The examinee's objective is to figure out the principle within each item set and indicate the Roman numeral (1 through 4) provided on the response key that the item suggests. Feedback is given by the examiner (by saying "correct" or "incorrect") after each response, and examinees must use this feedback to infer the organizing principle for each subtest. Therefore, the test requires the ability to deduce the classification rule or principle through response-contingent feedback, maintain the rule while it remains

effective, and abandon the rule when it is no longer effective. The number of errors made on each subtest is recorded and summed to yield a total error score, with lower scores indicating better performance. Several studies have documented very high internal consistency reliability (> .95) for the total score of the Category Test for both normal and brain-injured adults (cf. Strauss et al., 2006). In the present study, the total error score was included as an indicator for the central executive latent variable in the hypothesized model.

California Verbal Learning Test, Second Edition – Adult Version

The California Verbal Learning Test, Second Edition - Adult Version (CVLT-II; Delis et al., 2000) is a multiple-trial, list-learning task that assesses one's ability to learn and remember verbally presented information. The test was designed for individuals between the ages of 16 and 89 years. For the standard form of the CVLT-II, both recall and recognition of two word lists are assessed over immediate and delayed memory trials. Each of the two word lists is made up of 16 items that belong to four different semantic categories. List A is presented for each of the first five trials and the examinee is asked to recall the words immediately after each presentation. This is followed by a single oral presentation of an interference list (List B) for one trial. Directly after the interference trial, the examinee is asked to recall all of the items from List A in a short-delay free-recall trial as well as a short-delay cued-recall trial. Following a delay of 20 minutes, during which only nonverbal tests are administered, the examinee is again asked to recall all of the items from List A in a long-delay free-recall and long-delay cued-recall trial.

Finally, a dichotomous format (e.g., yes/no) recognition trial is completed and includes the 16 List A target words with an additional 32 distracter words.

The standard form of the CVLT-II yields raw and standardized scores that quantify over 50 parameters of learning and memory. The Reliability estimates reported in the CVLT-II manual indicate high internal consistency for the five immediate recall trials as well as high test-retest reliability for the measures assessing overall levels of immediate and delayed recall and recognition (Delis et al., 2000; Strauss et al., 2006). The CVLT-II manual also provided the results from an exploratory factor analysis performed on 19 of these variables in the standardization sample, which revealed a six-factor solution. The six factors were identified as: (1) General Verbal Learning (comprised of 9 variables measuring the level of immediate and delayed recall and recognition), (2) Response Discrimination, (3) Organizational Strategies (4) Primacy-Recency Effects, (5) Recall Efficiency, and (6) Acquisition Rate. Support for the criterion validity of the CVLT-II has been provided with TBI patients (Jacobs & Donders, 2007). A subset of the numerous CVLT-II measures were selected for use in this study to represent key aspects of verbal learning and memory. A description of these measures is included in Table 3.

Table 3

Description of CVLT-II measures included in the analyses

CVLT-II Measures	Description
List A Trials 1-5 Total	Reflects total number of words correctly recalled on trials 1 through 5 of the first list. Provides a global measure of verbal learning ability.
List B Immediate Recall	Represents total number of words correctly recalled from the second (interference) list.
Short-Delay Free Recall (SD Free)	Level of accurate recall following a brief delay interval and exposure to the interference list.
Short-Delay Cued Recall (SD Cued)	Reflects the total number of words recalled from List A according to semantic category.
Long-Delay Free Recall (LD Free)	Level of accurate recall following a 20-minute interval that is free of interference from verbal material.
Long-Delay Cued Recall (LD Cued)	Reflects the total number of words correctly recalled according to semantic cues after the delay interval.
Total Recognition Hits	Ability to identify List A target words among additional distracter items; provides a measure of recognition performance.

Procedure

This study was approved by the Institutional Review Board at The University of Texas at Austin (IRB Protocol # 2010-01-0048), and was conducted in compliance with the ethical principles and standards of research set forth by the American Psychological Association and The University of Texas at Austin.

Data for this study were obtained from an existing dataset of post-acute care patients who were diagnosed with a traumatic brain injury and participated in a full neuropsychological evaluation as part of their routine care at a neurorehabilitation hospital in Texas. At admission, written consent was obtained in order to use the participants' de-identified data for research purposes. As part of a more comprehensive

neuropsychological evaluation, standardized tests measuring central executive aspects of

working memory, processing speed, and verbal learning and memory ability were

administered to each participant individually. All testing was completed by a

neuropsychologist or supervised doctoral students (including the author), trained in

standardized administration and scoring procedures. Test order was invariant across

participants. Demographic data, including age, gender, and years of education, as well as

injury severity were collected and included in the analyses for the purpose of minimizing

potential confounds and accounting for potential common causes. In keeping with

previous research procedures, the severity of brain injury was estimated by using the

duration of post-traumatic amnesia (PTA) in days. Relevant demographic and injury-

related data were obtained through hospital records, clinical interview, and demographics

form.

<p align="center">*Hypothesized Model*</p>

Latent variable structural equation modeling (SEM) was used to analyze the data

and investigate the magnitude of the influence of processing speed and central executive

working memory on verbal learning ability. One of the key advantages of using a latent

variable SEM approach is that it reduces task invalidity by statistically extracting what is

common among the tasks so that a more accurate representation of the construct of

interest is represented (Keith, 2006; Miyake et al., 2000; Weston, Gore, Chan, &

Catalano, 2008). In addition, SEM allows the simultaneous testing of multiple

hypothesized relations among the latent constructs. The hypothesized latent variable

SEM model, illustrated in Figure 2, was developed to assess the theoretically-based

constructs of processing speed, central executive working memory, and verbal learning, and to investigate research-driven hypotheses regarding the relations among these constructs. As shown, relevant demographic characteristics (gender, years of education) as well as injury severity (represented by duration of PTA in days) were controlled statistically in the model. Briefly, for sake of clarity, measured variables (also referred to as indicator, observed, or manifest variables) are depicted graphically in SEM with squares or rectangles. Latent variables (also referred to as factors, constructs, or unobserved variables) are depicted graphically with ovals or circles. In Figure 2, the small circles labeled d1-d3 signify disturbances (also termed residuals), which represent all other sources of influence on the latent variables besides those included in the model. Similarly, the small circles labeled e1-e14 are error terms, representing the combined effect of all other influences on the measured variable other than the latent construct it is intended to measure, including the effects of unreliability and invalidity.
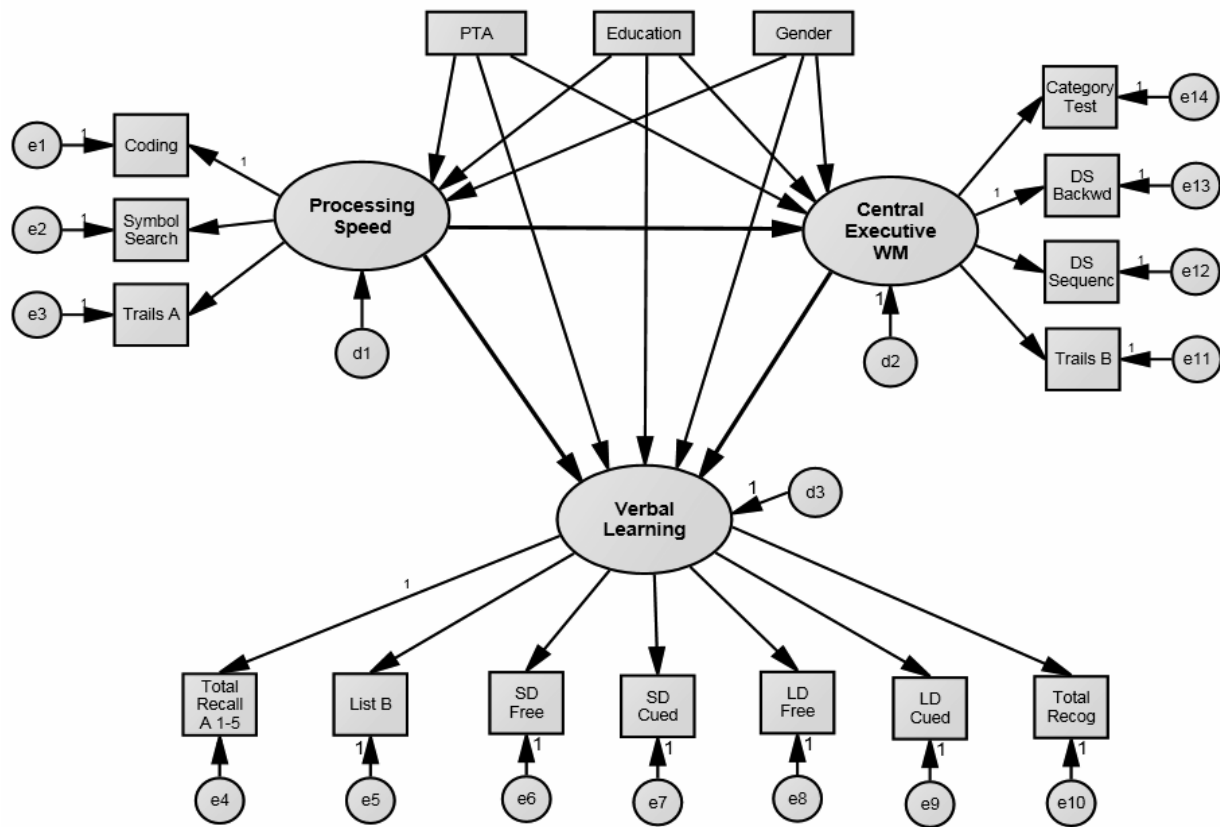
Figure 2. Hypothesized model of the cognitive variables influencing verbal learning ability after TBI.

CHAPTER 4

Results

Preliminary Analyses

Power and Sample Size

An analysis of power was conducted to determine the appropriate sample size for

the present study. Although various guidelines have been recommended as to what

constitutes an adequate sample size for performing confirmatory factor analysis (CFA)

and SEM (e.g., 5 to 10 participants per parameter), researchers have noted these general

recommendations and rules of thumb to be contradictory and lacking an empirical basis

(Guadagnoli & Velicer, 1988; MacCallum, Widaman, Zhang, & Hong, 1999; Weston et

al., 2008). It should be emphasized that recent research has demonstrated that in CFA and

SEM, power is influenced not only by sample size, but also by the degrees of freedom,

which reflect the number of parameters in the model that are constrained to zero (or some

other value) and, thus are not freely estimated (Keith, 2006; MacCallum et al., 1999).

Specifically, models with a higher number of degrees of freedom will result in greater

power. Additionally important in SEM, especially with smaller sample sizes, is the

number of indicators per latent factor, with more indicators producing more stable factors

as well as higher power (Guadagnoli & Velicer, 1988; Keith, 2006). Based on methods

outlined by MacCallum, Browne, and Sugawara (1996), the CSMPOW program software

was used to determine the sample size required with at least .80 power (α = .05) and

with 110 degrees of freedom in the specified model (Figure 2). Based on the power

calculation, a sample size of 58 was needed for the present study. Thus, the current

43

sample size of 70 should result in sufficient power to be able to reject an inadequate model.

Data Screening

To ensure that all variables were normally distributed and reflected their appropriate scales of measurement, the data were checked by examining the descriptive statistics, visual inspection of histograms, as well as skewness and kurtosis values using SPSS. All variables in the model were found to reflect reasonably normal distributions. Skew and kurtosis values for all measured variables were acceptable with absolute values less than 2 and 7, respectively (Curran, West, & Finch, 1996). Data were also examined for extreme values or outliers, defined by scores more than 3 standard deviations beyond the mean. Two univariate outliers were detected within the Trails A variable (3.44 and 3.74 standard deviations above the mean), which were removed from subsequent analyses. In addition, of the 70 participants, there were two cases with missing estimates for duration of PTA. As this study used an archival dataset, recovering missing values was not feasible. Table 4 shows the descriptive statistics for the raw scores of all measured variables. For the purpose of computing descriptive statistics, missing data were addressed via pairwise deletion in SPSS.

Raw scores for all data (with the exception of gender, education, and PTA duration) were corrected for age by regressing out age and $(age)^2$ from all of the measured variables. The unstandardized residuals were retained and used in subsequent data analyses.

Table 4

Descriptive Statistics

Variable	Mean	Standard Deviation	Minimum	Maximum
PTA (days)	11.41	12.68	.00	49
Education (years)	13.61	2.66	8	20
Gender	1.20	.403	1	2
WAIS-IV: Coding	47.36	16.81	1	100
WAIS-IV: Symbol Search	21.10	8.05	0	45
WAIS-IV: DS Backward	7.11	2.39	1	13
WAIS-IV: DS Sequencing	7.01	2.16	1	11
Booklet Category Test	69.29	31.86	7	136
Trails A	41.89	18.44	18.85	107
Trails B	120.81	71.52	47	300
CVLT-II: List A Trials 1-5 Total	37.09	12.26	8	60
CVLT-II: List B Recall	4.49	1.717	0	8
CVLT-II: SD Free Recall	6.60	3.93	0	15
CVLT-II: SD Cued Recall	7.37	3.98	0	16
CVLT-II: LD Free Recall	6.09	4.17	0	15
CVLT-II: LD Cued Recall	7.27	4.09	0	16
CVLT-II: Total Recognition	12.09	3.55	2	16

Note. PTA=post-traumatic amnesia. Males in the sample were assigned a value of 1 and females a value of 2.

Model Estimation

The hypothesized model was drawn and analyzed using the structural equation modeling program, Amos 19 (Arbuckle, 2010). To set the scale of the latent variables, a path from each latent variable to one of the measured variables (indicators) was constrained to 1.0. Figure 2 shows the factor loadings for Coding, Digit Span Backward, and Total Recall A 1-5 were constrained to 1.0 to set the scale of their respective latent variables. On these measures, larger numbers indicated better performance. Age-

corrected raw scores were input into Amos and a covariance matrix was calculated using the full information maximum likelihood (FIML) method to account for missing data. All analyses were conducted using the FIML-derived covariance matrix in Amos (Appendix C). Intercorrelations among the measured variables were also estimated in Amos using FIML to deal with missing data and are presented in Table 5. Given that the normality assumption was met, the maximum likelihood estimation method was chosen to estimate the measurement and full latent variable models. The maximum likelihood technique is considered robust to moderate violations of the normality assumption and is one of the most frequently used estimation methods (Weston et al., 2008).

The general approach to model estimation involved the following steps, consistent with Anderson & Gerbing's (1988) two-step approach. First, the measurement portion of the model, defined by the paths from the latent constructs to the measured variables, was estimated. The measurement model (also termed confirmatory factor model) assessed the degree to which the indicators share enough variance to form the hypothesized latent constructs, and whether the measured variables in fact reflect the intended constructs. Theoretically acceptable respecifications were made to the measurement model to improve the fit of the model based on the modification indices and standardized residual covariances. The final measurement model was used as the basis for the subsequent full structural model. The structural portion of the model, which reflects a path analysis of the latent constructs, allows for estimating the presumed influence of one latent variable on another. This portion of the hypothesized model was just-identified. In the second phase, the measurement and structural portions of the model were estimated simultaneously (i.e.,

the full SEM model was tested). The full SEM model included injury severity, education

level, and gender as control variables in the model. Rather than testing competing

theoretical models, the magnitude and statistical significance of specific relevant paths of

interest were examined within the final full SEM model.

Table 5

FIML-derived Correlation Matrix of Measured Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. PTA (days)[a]	1.0																
2. Education	-.31	1.0															
3. Gender	-.09	.06	1.0														
4. Coding	-.13	-.06	.12	1.0													
5. Symbol Search	-.16	-.04	-.07	.70	1.0												
6. Trails A[a]	.30	.03	.15	-.51	-.57	1.0											
7. Category Test	-.03	-.06	.08	-.32	-.42	.45	1.0										
8. DS Backward	-.06	.08	.20	.45	.51	-.26	-.29	1.0									
9. DS Sequencing	-.09	-.05	.17	.45	.44	-.32	-.29	.65	1.0								
10. Trails B	.16	-.07	-.07	-.56	-.52	.60	.35	-.47	-.51	1.0							
11. Total Recall A1-5	-.10	-.09	.15	.38	.41	-.30	-.39	.39	.46	.39	1.0						
12. List B Recall	-.11	-.05	.02	.10	.06	-.15	-.10	.06	.23	-.07	.49	1.0					
13. Short-Delay Free	-.11	-.11	.09	.44	.47	-.34	-.42	.41	.51	-.39	.81	.41	1.0				
14. Short-Delay Cued	-.07	-.04	.11	.25	.28	-.29	-.40	.51	.43	-.33	.77	.34	.81	1.0			
15. Long-Delay Free	-.08	-.09	.13	.42	.45	-.35	-.46	.47	.51	-.42	.85	.35	.92	.84	1.0		
16. Long-Delay Cued	-.07	-.08	.11	.21	.25	-.27	-.39	.47	.43	-.32	.78	.38	.80	.94	.87	1.0	
17. Total Recognition	-.02	.05	-.03	.20	.30	-.14	-.30	.38	.20	-.23	.62	.35	.55	.58	.62	.63	1.0
M	11.22	13.61	1.20	0.00	0.00	0.07	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
SD	12.59	2.64	.40	15.77	7.68	17.46	29.62	2.37	2.14	70.88	12.12	1.68	3.90	3.94	4.13	4.04	3.46

[a] n = 68.

48

Evaluation of Model Fit

Various fit statistics are available to determine the degree to which a specified single model explains or "fits" the data. The fit of the measurement and full SEM models were evaluated by examining a combination of fit indices, including Root Mean Square Error of Approximation (RMSEA), Standardized Root Mean Square Residual (SRMR), Comparative Fit Index (CFI), and Tucker-Lewis Index (TLI).

The RMSEA index corrects for the complexity of a model and provides a measure of the approximate fit. RMSEA values of .06 and below are generally considered to represent a good fit (Hu & Bentler, 1999), and values of approximately .08 suggest a reasonable fit (Browne & Cudeck, 1993). Optimal RMSEA cutoff values may vary depending on sample size, with higher appropriate cutoff values for smaller samples and more stringent criteria applied for larger samples (Chen, Curran, Bollen, Kirby, & Paxton, 2008; Hu & Bentler, 1999; Weston et al., 2008). The SRMR is an index based on the covariance residuals and represents the average difference between the actual observed correlations and the model-implied correlations. SRMR values of .08 or lower suggest a good fit of the model to the data (Hu & Bentler, 1999), with values less than .10 considered acceptable (Bentler, 1995; Kline, 2005). The CFI and TLI are indices that compare the fit of the estimated model with that of the independence or "null" model, which assumes no relations among the observed variables. The CFI provides an estimate of the fit in the population, whereas the TLI is considered relatively independent of sample size and adjusts for parsimony (Keith, 2006; Tanaka, 1993). For both CFI and

TLI, values approaching 1.0 indicate a better fit, with values above .90 suggesting an adequate fit and values of .95 or greater suggesting a good fit (Hu & Bentler, 1999; Keith, 2006; Kline, 2005). For comparing nested models, in which one model can be derived from the other through adding or removing constraints, the χ^2 difference test was used. The Akaike Information Criterion (AIC) was also used to compare nested as well as non-nested models, with smaller AIC values indicating a better fit to the data (Keith, 2006).

<div align="center">Primary Analyses</div>

Measurement Model

The first phase in the SEM analyses required conducting a confirmatory factor analysis to evaluate whether the measurement model adequately fit the data. An initial test of this measurement model revealed only marginal support for the initial proposed model, as suggested by the majority of fit statistics which did not reach acceptable levels (i.e., SRMR = .067, RMSEA = .139, TLI = .841, CFI = .871). As a result, the modification indices and standardized residual covariances were examined to determine whether any modifications could be made that would lead to a better-fitting model. Model adjustments were limited to those that were deemed theoretically meaningful or consistent with past research.

The modification indices revealed two potential modifications to the hypothesized measurement model that made theoretical sense. First, results suggested that freeing (or estimating) the correlation (covariance) between the error variances for Short-Delay Cued Recall and Long-Delay Cued Recall would result in the largest improvement in model fit.

Allowing this revision suggests that these two variables share or measure something in common other than verbal learning and memory. This adjustment makes sense as the cued recall trials of the CVLT-II provide examinees with the categorical structure of the initial word list and requires them to retrieve the target words according to a language-based strategy by using semantic clustering (Delis et al., 2000). The second modification suggested was to estimate the correlation (covariance) between the error variances for Trails A and Trails B. This change makes sense in that it seems likely that part A and part B of the Trail Making Test share something in common other than their respective factors, such as visual scanning and sequencing. Relaxing this constraint was also supported by the highest positive standardized residual (1.694), suggesting that the proposed model may not have adequately accounted for the correlation between these two variables.

These two modifications were estimated one at a time to allow each adjustment to the model to be evaluated. These models were nested with the initial measurement model; therefore, the $\Delta\chi^2$ was used as the primary fit statistic for model comparisons. As shown in Table 6, allowing a correlated error between the short- and long-delay cued recall trials resulted in a statistically significant improvement in fit, suggesting that the two recall trials do measure something in common beyond verbal learning and memory. As a result, this model modification was retained in subsequent analyses. Allowing a second correlated error between Trails A and Trails B also resulted in a significant improvement in model fit. Thus, each of these modifications to the measurement model resulted in a statistically significant $\Delta\chi^2$, suggesting that the more parsimonious model (with higher

degrees of freedom) should be rejected in favor of the less parsimonious model (i.e.,

Modified Model 2). The smaller AIC value (183.44) and improvement in all other fit

indices also indicated that the second modified model allowing correlated errors between

the cued recall trials of the CVLT-II and between parts A and B of the Trail Making Test

provided a better fit to the data. Thus, both modifications did statistically significantly

improve the model and were both retained in subsequent analyses.

Table 6

Fit Statistics for Models with Chi-Square Difference Test for Nested Models

Model	χ^2 *(df)*	$\Delta\chi^2$ *(Δdf)*[a]	AIC	SRMR	RMSEA	TLI	CFI
Measurement Models							
Hypothesized Model	172.04 (74)		234.04	.067	.139	.841	.871
Modified Model 1 (SDCR ↔ LDCR)	126.23 (73)	47.81 (1)**	190.23	.066	.103	.913	.930
Modified Model 2 (Trails A ↔ B)	116.44 (72)	9.79 (1)**	182.44	.066	.095	.926	.941
Modified Model 3 (CT removed)	103.17 (60)		165.17	.056	.102	.925	.942
Final Full Latent Variable Model	133.06 (93)		219.06	.065	.079	.931	.946

[a] Compared to the previous model.

**$p < .001$.

Factor loadings for all of the indicators were statistically significant ($p < .001$).

However, two of the indicators, Category Test and List B, had relatively lower loadings

on their designated factors (-.48 and .40, respectively). Examination of the modification

indices and the pattern of relatively larger values in the standardized residual covariance

matrix revealed that the Category Test variable was a likely source of misfit in the model.

Specifically, results indicated that the Category Test variable, which was specified as an

indicator of the central executive component of working memory, was more related to

some indicators of the other latent factors (i.e., verbal learning and processing speed) than accounted for in the model. Thus, the nature of the Category Test seems to be factorially complex and it is possible that it may measure or load on more than one factor included in the model. As a result, the Category Test indicator was subsequently removed from the model altogether. Although the factor loading for List B on verbal learning was relatively lower (.40) compared to the other indicators on that factor, it was statistically significant at $p < .001$ and there was no substantial evidence of cross-loadings. Therefore, the List B indicator was retained in the final model. The results of the re-estimated model are shown in Table 6. Since this model is not nested with the initial hypothesized model, the AIC was used as the primary method for model comparisons. As shown in the table, the model with the Category Test indicator removed provided a better fit to the data than the previous model as indicated by the lower AIC value (165.17). The resulting measurement model achieved adequate levels of fit as suggested by the majority of fit indices, including SRMR (.056), TLI (.925), and CFI (.942), and was considered an acceptable measurement model to serve as the basis for testing the full latent variable model. The final modified measurement model with standardized estimates is presented in Figure 3. The correlations among the latent variables shown in the figure were all statistically significant ($p < .001$).
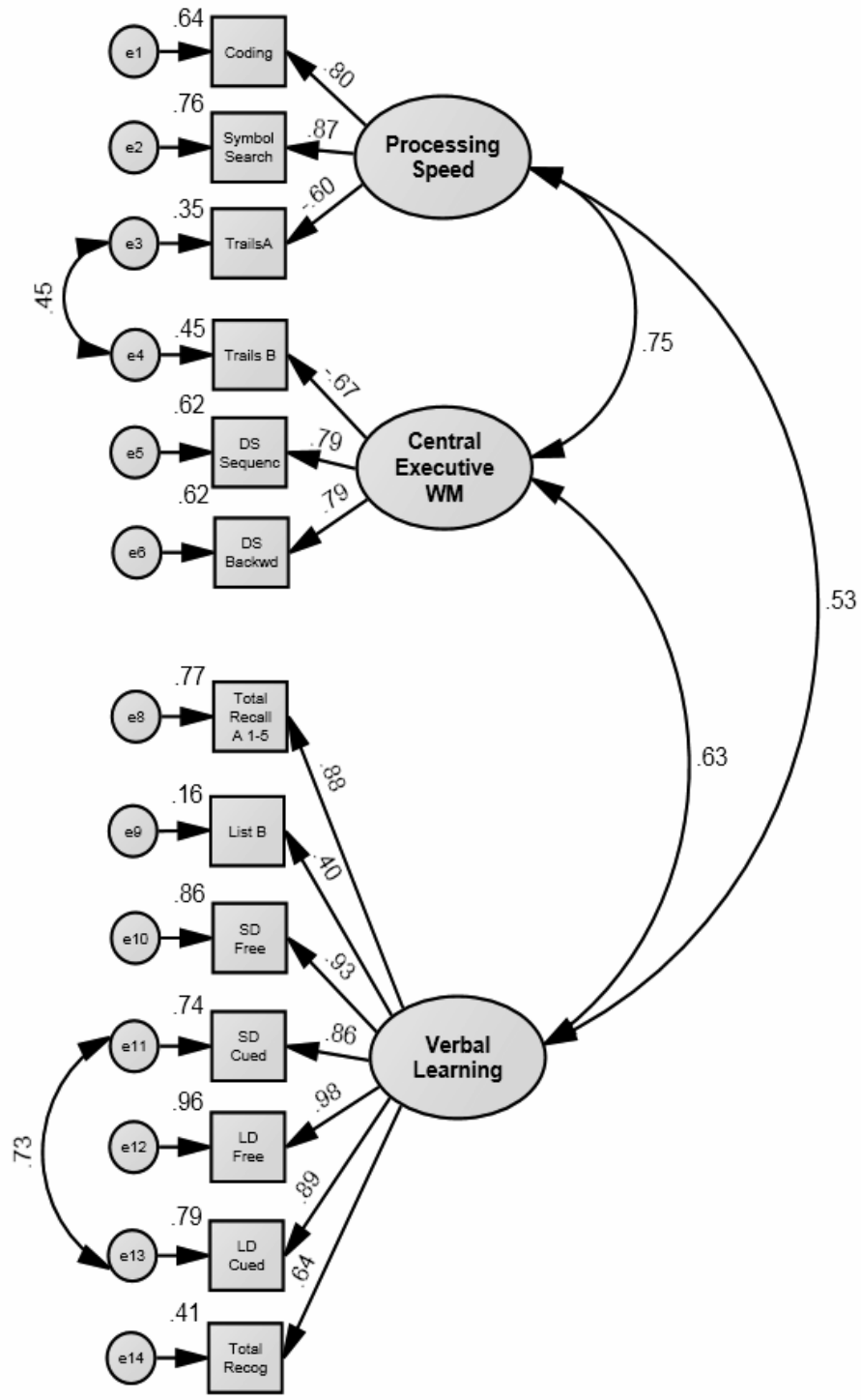
Figure 3. Measurement model with modifications.

Full Latent Variable Model

       Once an acceptable measurement model was developed, the full structural model was tested with the hypothesized relationships among the latent variables specified. In addition, level of education, gender, as well as duration of post-traumatic amnesia (PTA), an indicator of brain injury severity, were included in the model for the purpose of minimizing potential confounds and accounting for potential common causes.

       As summarized in Table 6, the results indicated that the full model fit the data reasonably well as suggested by the various stand-alone fit indices, including SRMR (.065), RMSEA (.079), CFI (.946), and TLI (.931). Standardized results for the final full SEM model are presented in Figure 4. With a sample size of 70 and 93 degrees of freedom, the estimated power for the final full model was .84. Since the full model adequately explained the data, the specific paths among the latent variables were investigated and interpreted in relation to the proposed research questions and hypotheses.
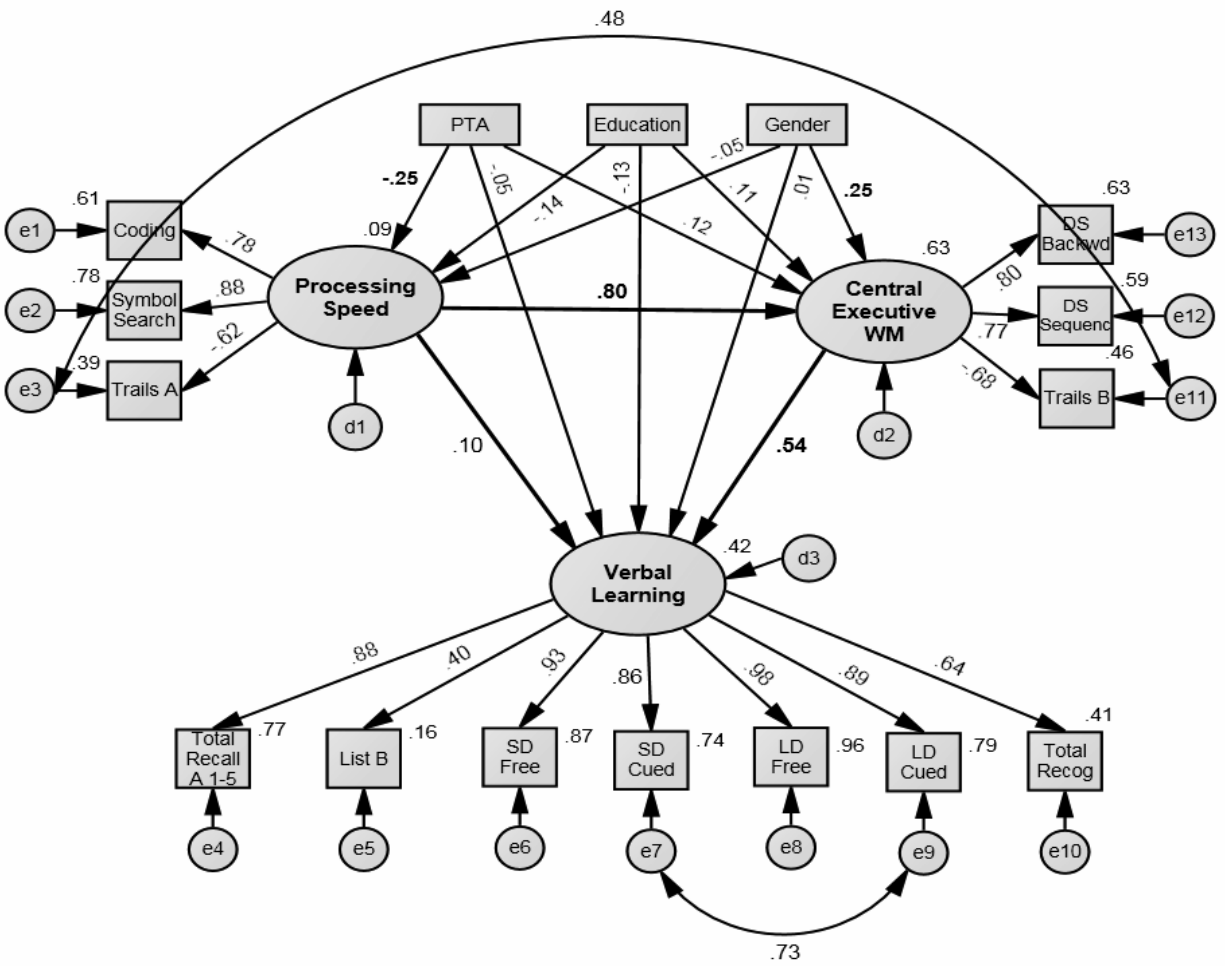
Figure 4. Standardized Estimates for the Final Full SEM Model.

Tests of Hypotheses

Hypothesis 1

Hypothesis 1 predicted that the central executive component of working memory would explain verbal learning ability after TBI to a statistically significant degree, even after controlling for processing speed. To determine the relative influences of working memory and processing speed on verbal learning and memory, the standardized direct effects from the full SEM model (Figure 4) were examined. The standardized direct, indirect, and total effects of the latent variables on verbal learning ability are summarized in Table 7. The standardized *total* effects of working memory and processing speed on verbal learning were both large (.54 and .53, respectively). After accounting for the effects of the other variables in the model, the standardized direct effect of processing speed on verbal learning was relatively small and statistically nonsignificant ($\beta = .10$, $p = .66$). In contrast, the standardized direct effect of central executive working memory on verbal learning was large and statistically significant ($\beta = .54$, $p < .05$), suggesting that for each standard deviation increase in the latent working memory variable, verbal learning and memory should increase by .54 of a standard deviation. In other words, individuals with greater working memory capacity after TBI tend to be able to learn and remember more target words. Taken together, after accounting for other relevant influences, the relative direct effect of working memory on verbal learning was stronger and more than five times the effect of processing speed on verbal learning (.54 and .10, respectively). As hypothesized, SEM analyses indicated that verbal learning and memory

57

ability after sustaining a TBI was explained primarily in terms of working memory, after controlling for effects of processing speed.

Table 7

Standardized Direct, Indirect, and Total Effects of Latent Variables on Verbal Learning

Latent Variable	Direct Effects	Indirect Effects	Total Effects
Central Executive WM	.54	—	.54
Processing Speed	.10	.43	.53

Hypothesis 2

Hypothesis 2 predicted that processing speed would primarily influence verbal learning indirectly through the central executive component of working memory. To determine how processing speed influences verbal learning ability, the standardized effects from the full SEM model were initially examined. As summarized in Table 7, the direct effect of processing speed on verbal learning was relatively small ($\beta = .10$) and was not statistically significant, but the indirect effect of processing speed on verbal learning was strong ($\beta = .43$). Processing speed had a large and statistically significant direct effect on working memory ($\beta = .80, p < .001$), suggesting that the faster TBI patients process information, the greater their working memory capacity. After accounting for the effects of the other variables in the model, the effects of processing speed on verbal learning were primarily indirect, through the central executive component of working memory (.80 x .54 = .43).

The statistical significance of the indirect effects was tested through a bootstrapping procedure in Amos. Bootstrapping is a resampling method that involves taking a large number of random samples, with replacement, from the original dataset. Bootstrapping has been recommended for testing indirect or mediated effects as it offers greater power and accuracy over conventional methods (Cheung & Lau, 2008; MacKinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2004; Preacher, Rucker, & Hayes, 2007; Shrout & Bolger, 2002; Williams & MacKinnon, 2008). The application of the bootstrap approach has been supported for use with even moderate or small sample sizes (Efron & Tibshirani, 1993; Shrout & Bolger, 2002). For the present study, a bootstrapping method was utilized, in which 2,000 bootstrap samples were drawn and used to estimate standard errors and bias-corrected confidence intervals for the estimated parameters. Table 8 shows the effects decomposition with bootstrap estimates of standard errors and 90% bias-corrected confidence intervals for the unstandardized coefficients.

The results from the bootstrapping procedure indicated that the indirect effect of processing speed on verbal learning through the central executive was in fact statistically significant ($p < .05$). As shown in the table, the 90% bias-corrected confidence interval for the indirect effect was between .10 and .88, with a p-value of .037 (two-tailed). In other words, the indirect effect of processing speed on verbal learning was significantly different from zero. Thus, these results suggest that processing speed primarily influences verbal learning indirectly, by influencing the central executive aspects of working memory. That is, on average, TBI patients with faster processing speed tend to have greater working memory capacity, which in turn improves verbal learning and memory.

59

Table 8

Effects Decomposition with Bootstrap Standard Errors and Bias-Corrected CIs

	Central Executive WM			Verbal Learning & Memory		
	b	*SE*[a]	90% BC CI[a]	*b*	*SE*[a]	90% BC CI[a]
Processing Speed						
Direct Effects	.12**	.03	.08 - .17	.09	.35	-.34 - .50
Indirect Effects	—	—	—	.37*	.33	.10 - .88
Total Effects	.12**	.03	.08 - .17	.46**	.14	.27 - .72
Central Executive WM						
Direct Effects				3.08*	2.70	.71 – 6.90
Indirect Effects				—	—	—
Total Effects				3.08*	2.70	.71 – 6.90

Note. Table is read from row to column. Unstandardized estimates are represented by *b*.

BC CI = bias-corrected confidence interval.

[a] Values are based on the unstandardized estimates.

p ≤ .05. **p* < .01.

CHAPTER 5

Discussion

Overview of Findings

The aim of the present study was to examine the roles of working memory and

processing speed in explaining verbal learning impairments in individuals with TBI.

Specifically, this study sought to clarify whether verbal learning and memory

impairments after TBI are primarily influenced by the central executive component of

working memory or by speed of processing. The way in which processing speed affects

verbal learning problems was also examined. A model was proposed and tested to

evaluate the nature of the relations among the central executive component of working

memory, processing speed, and verbal learning ability. The model was estimated using

latent variable structural equation modeling (SEM) with a sample of post-acute care TBI

patients. While several previous studies have examined the effects of TBI on memory

functioning, the present study is unique in that it applied a latent variable SEM approach

and simultaneously accounted for the relative contributions of both the central executive

component of working memory and processing speed.

As predicted, results from SEM analyses revealed that verbal learning and

memory difficulties following TBI were explained primarily in terms of the central

executive component of working memory, after controlling for the effects of processing

speed in the model. In addition, as hypothesized, the results indicated that processing

speed exerted its influence on verbal learning primarily indirectly, by influencing the

central executive component of working memory. In other words, the speed with which

TBI patients processed information influenced their working memory capacity, which in turn influenced how well they were able to learn and recall rote verbal information.

Roles of Working Memory and Processing Speed in Verbal Learning

In accordance with Baddeley (2000, 2003), the results of the present study confirm the importance of the capacity of working memory for verbal learning and memory performance after TBI. In particular, this study found that the central executive component of working memory had a strong and significant direct effect on verbal learning and memory ability in patients with TBI after accounting for the relative contributions of processing speed. This finding supports previous research showing that learning and memory impairments in TBI patients are attributable to deficits in the central executive or attentional control aspects of working memory (Mandalis et al., 2007; Mangels et al., 2002; Serino et al., 2006).

Of particular interest to this study was the role of processing speed and the way in which it influences verbal learning and memory in TBI patients. Results indicated that the direct effect of processing speed on verbal learning was not significant when working memory was taken into account in the model. Thus, present findings are consistent with an interpretation in which problems with learning and remembering verbal information following TBI can be directly attributed to reduced working memory capacity rather than slowed information processing. However, consistent with previous research (van Zomeren & Brouwer, 1994), results provided further support for the significant contribution of processing speed to aspects of working memory in TBI patients. Moreover, the present study found that processing speed primarily exerted an indirect,

rather than a direct, impact on verbal learning and memory ability following TBI. This suggests that TBI patients who process information at a faster rate tend to have greater capacity of the working memory system, which in turn aids verbal learning and memory ability.

The findings from the present study provide several contributions to the current TBI literature with regard to the cognitive factors that influence poor verbal learning and memory following TBI. First, results of this study help to clarify and expand upon the differing viewpoints concerning the primary cognitive mechanism (i.e., working memory versus processing speed) underlying impaired learning and memory subsequent to TBI. Second, this study improved on past investigations by using SEM to examine concurrently the relative effects of both processing speed and working memory on verbal learning ability after TBI. Using this methodological approach allowed for a more comprehensive understanding of the nature of the relations among these processes.

With regard to the differing viewpoints, the results of this study lend support for the working memory hypothesis, suggesting that verbal learning and memory problems after TBI can be primarily attributed to impaired executive or attentional control aspects of working memory. However, findings are also arguably consistent with the processing speed hypothesis to some extent, as processing speed was found to have a significant direct effect on working memory, and thereby exerting a significant indirect effect on verbal learning and memory. The current findings do not contradict either of the dominant hypotheses in explaining learning and memory problems after TBI, but rather, the results underscore the importance of *both* processing speed and working memory in

playing key roles in learning and memory processes. Specifically, the present findings revealed that processing speed operated on verbal learning indirectly through the central executive component of working memory. Thus, this study offers initial evidence to suggest that in TBI patients, processing speed works together with working memory to support performance on verbal learning and memory tasks, and both should be considered in any theoretical account of TBI-related learning and memory impairments. This pattern of findings is consistent with arguments and past findings within the literature on cognitive aging, which suggests that the influence of processing speed on memory is largely indirect and operates through working memory (Hedden, Lautenschlager, & Park, 2005; Park et al., 2002; Park et al., 1996; Salthouse, 1993).

Limitations and Future Directions

There are several limitations of the present study that should be recognized in light of the findings and implications. First, with regard to methodological considerations, a limitation of this study relates to its cross-sectional design and lack of a control group. Future studies should strive to include control participants from another patient group, such as orthopedic patients involved in a traumatic accident, as such patients are a preferable control group for individuals who have sustained TBI and favored over normal healthy control participants (Vakil, 2005). Second, the sample size of 70 was relatively small given the number of variables in the model and methods of SEM. However, research on power and the stability of factor solutions as a function of sample size, degrees of freedom, number of indicators per latent construct, and other factors, help to allay this particular concern (e.g., Guadagnoli & Velicer, 1988; MacCallum et al., 1996;

64

MacCallum et al., 1999; Muthen & Muthen, 2002). Nonetheless, replicating these findings with larger samples, as well as including a matched patient control group to explore whether the pattern of relations differs between groups, would be useful for future investigations in this area of research.

A third potential limitation concerns the indicator variables selected to represent the latent constructs of interest in this study (MacCallum & Austin, 2000). Although the indicator variables were selected to represent the latent constructs on the basis of theoretical considerations and previous research, there are many other tasks not included in this study that have been used as measures of processing speed, learning and memory, as well as the central executive component of working memory (e.g., dual-task paradigm, n-back task, PASAT). As such, it is possible that the use of alternative measured variables might have shifted the nature of the constructs and resulted in a different pattern of findings and interpretation. In a similar regard, although the present study offered a model that explained the data reasonably well, it should be recognized that there may well be other plausible models that fit the data equally well or better that were not considered in the current study (MacCallum & Austin, 2000). It would be useful for future studies in this area to specify and evaluate additional a priori models that may offer alternative meaningful explanations of the data.

At a broader level, as is the case with many traditional neuropsychological measures, it is important to acknowledge concerns related to the potential shortcomings of clinical learning and memory tests in predicting real-world functioning in everyday occupational, home, and social environments. In particular, there has been mixed

empirical support for the prognostic value of measures of memory functioning following TBI in predicting variables related to employment outcome (Ownsworth & McKenna, 2004) as well as everyday cognitive skills (see Chaytor & Schmitter-Edgecombe, 2003). As such, the degree to which the measures included in the present study translate into learning and memory skills necessary for everyday functioning remains unclear. However, there is evidence to support the psychometric properties of these measures, including their validity as indicators of the conceptual construct as modeled in this study (Delis et al., 2000, Strauss et al., 2006).

There are additional limitations of this study related to characteristics of the patient sample used. In particular, it is important to note that patients with TBI are a heterogeneous group, and there are a number of factors that likely contribute to the variability in performance among TBI patients and potentially lead to inconsistent findings reported in the literature (see Vakil, 2005). The present study was limited to participants who had suffered a brain injury of traumatic etiology and were between the ages of 16 and 65 years old. Consequently, findings from this study may lack generalizability to patient populations with neurological deficits of other etiologies as well as to ages outside of the included range. Further, it is possible that by including a wide age range of patients, as in the current study, may introduce potential confounds with the effects of age. Vakil (2005) suggested that studies restrict the patients' age range so as not to exceed a span of 15 years and that elderly participants be excluded, or otherwise included as a separate group. Additionally, identifying potential subgroups of TBI patients based on the different causes of injury (e.g., motor vehicle accident, falls,

struck by/against events, etc.) as well as the nature of the TBI would be particularly useful to examine whether the current pattern of findings hold or whether unique patterns exist across TBI subgroups. In this light, future work integrating neuroimaging data from functional MRI (fMRI), diffusion tensor imaging (DTI), and other functional brain imaging techniques will be especially important for characterizing differential patterns of brain activation and further delineating subgroups of TBI patients for investigation (Levin, 2003).

Finally, it is acknowledged that the present study did not include variables in the analyses that would implicate or account for the emotional or psychiatric state of the patients following TBI. It is important to recognize that there is a broad range of emotional and behavioral difficulties that may be experienced following TBI and the potential of these difficulties to affect cognitive performance. For instance, research has found an increased incidence of depression, anxiety, post-traumatic stress disorder, disinhibition, agitation, aggression, and loss of self-esteem after sustaining TBI (Hibbard, Uysal, Kepler, Bogdany, & Silver, 1998; Hiott & Labbate, 2002; Kim, 2002; Kreutzer, Sell, & Gourley, 2001; Olver, Ponsford, & Curran, 1996; Tyerman & Humphrey, 1984). Moreover, high levels of emotional distress have been shown to have a negative impact on memory functioning (Dalgleish & Cox, 2002; Gass & Apple, 1997). Hence, it is possible that the inclusion of additional, noncognitive factors in the SEM analyses may have yielded a different pattern of associations among the variables, and is worth further investigation. Analyzing the complex interplay among residual memory impairments and

the emotional and psychosocial consequences following TBI would be a useful topic to address in future research.

Conclusions and Implications

The current study has provided additional insights into the primary cognitive mechanism underlying verbal learning and memory difficulties following TBI, supporting the direct and significant impact of the central executive component of working memory in verbal learning processes. Additionally, findings provided further clarification regarding the nature of the relations between processing speed and verbal learning and memory difficulties in patients with TBI, suggesting the indirect role of processing speed through working memory.

This study has important implications for the rehabilitation of new learning and memory impairments following TBI. Results highlight both working memory and processing speed as fundamental cognitive processes of critical importance to verbal learning and memory after TBI. While working memory was found to have more of a direct role in verbal learning and memory performance, slowness of processing speed was found to be an underlying contributing factor, largely influencing learning and memory indirectly. In light of the present findings, it follows that post-injury cognitive rehabilitation efforts involving direct remediation or restorative interventions to specifically target the central executive component of working memory may be a useful means for improving verbal learning and memory capacity after TBI. There is emerging evidence in the literature to suggest that specific cognitive interventions may improve the central executive component of working memory in patients who sustained TBI

(Cicerone, 2002; Lundqvist, Grundstrom, Samuelsson, & Ronnberg, 2010; Serino et al., 2007; Vallat-Azouvi et al., 2009). Additional rehabilitation efforts aimed at teaching compensatory strategies that would minimize the functional impact of slowed processing speed and working memory problems could have significant value in enhancing learning and recall ability. To further address underlying impairments in processing speed post-TBI, adapting simple modifications within rehabilitation approaches, such as reducing the amount and rate at which information is presented thereby allowing patients more time to process the information, may prove useful (Chiaravalloti et al., 2003; Demaree, DeLuca, Gaudino, & Diamond, 1999). In sum, an integrative approach to rehabilitation, combining targeted remediation efforts designed to enhance an individual's working memory capacity and compensatory strategy training to help address slowed processing speed may be an effective method for facilitating new learning and memory capacity following TBI. Undoubtedly, ongoing research on the impact of cognitive rehabilitation and whether targeting these component processes post-TBI produces meaningful changes will be necessary.

Appendix A

Complete Neuropsychological Test Battery

Wechsler Adult Intelligence Scale – Fourth Edition (WAIS-IV)
    Core Subtests

Wide Range Achievement Test – Fourth Edition (WRAT-4)
    Word Reading
    Spelling
    Math Computation

Wechsler Memory Scale – Fourth Edition (WMS-IV)
    Logical Memory I
    Logical Memory II
    Visual Reproduction I
    Visual Reproduction II
    Verbal Paired Associates I
    Verbal Paired Associates II

California Verbal Learning Test – Second Edition (CVLT-II)

Halstead-Reitan Neuropsychological Test Battery
    Finger Tapping Test
    Grip Strength
    Tactile Finger Recognition Test
    Visual Double Simultaneous Stimulation
    Aphasia Screening Test
    Trail Making Test (Parts A and B)

Controlled Oral Word Association Test (COWAT)

Booklet Category Test

Minnesota Multiphasic Personality Inventory–2 (MMPI-2)

Beck Depression Inventory-Second Edition (BDI-II) [1]

Beck Anxiety Inventory (BAI) [1]

Neurobehavioral Functioning Inventory (NFI)

Preventive Resources Inventory (PRI)

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[1] *Administered if MMPI-2 is infeasible.*

Appendix B

Demographic Sheet

Patient: _____   **ID:** _____

Gender: 1 = Male   **Handedness:** 1 = Right
(circle) 2 = Female   2 = Left
   3 = Ambidextrous

Race: 1 = Native   **Date of birth:** _____
(circle) American/Alaskan
2=African American
3=Caucasian   **Age (yrs):** _____
4=Asian
5=Hawaiian Native/Pacific
Islander   **Education (yrs):** _____
6=Hispanic
7=Other

Occupation: 1=Unskilled   **Pre-morbid living:** 1=Alone
(circle) 2=Skilled   **(circle)** 2=With spouse or significant
3=Technical   other
4=Managerial   3=With parents
5=Professional   4=With roommate
6=Student   5=Military
7=Unemployed   6=Other _____

Group: 1=TBI   **Etiology:** 1=Motor vehicle
(circle) 2=Hypoxic   **(circle)** collision/accident
3=Vascular   (MVC/MVA)
4=Chemical dependency   2=Pedestrian Vehicle
   3=Motorcycle/ATV
   4=Cycling
TBI Type Subgroup: 1=Non-penetrating   5=Fall – Standing
(If you circled "1" above, 2=Penetrating   6=Fall > Standing
please further specify) 3=Blast   7=Fall – Stairs
   8=Assault
   9=Sports
   10=GSW
Date of Injury: _____   11=Struck by/against
   12=Other_____

Date of Admission: _____   **Days Post-injury:**
   (at testing) _____

Glasgow Coma Scale _____   **GSC – Location:** 1= Field
(GSC): (3-15)   **(circle)** 2=Emergency Dept

Rancho Los Amigos _____   **Posttraumatic** _____
Scale: (I-VIII)   **Amnesia (PTA):** (in minutes)
(at time of testing)

   PTA Russell Scale 1=<5 minutes (Very mild)
CRS-R (admission): _____   **(circle)** 2=5 minutes to 1 hour (Mild)
(0-23)   3=1-24 hours (Moderate)
Loss of Consciousness: 4=1 to 7 days (Severe)
(in minutes) _____   5=>7days (Very Severe)

FIML-derived Covariance Matrix of Observed Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1. Trails B	**5024.59**																
2. PTA (days)[a]	146.43	**158.56**															
3. Total Recognition	-57.26	-0.92	**11.98**														
4. Gender	-2.08	-0.46	-0.04	**0.16**													
5. Education	-12.90	-10.41	0.45	0.06	**6.95**												
6. Long-Delay Cued	-91.08	-3.48	8.75	0.17	-0.90	**16.34**											
7. Long-Delay Free	-122.91	-4.10	8.91	0.21	-0.99	14.60	**17.04**										
8. Short-Delay Cued	-91.61	-3.71	7.97	0.18	-0.46	14.90	13.61	**15.51**									
9. Short-Delay Free	-107.38	-5.41	7.35	0.14	-1.14	12.53	14.75	12.49	**15.19**								
10. List B Recall	-8.27	-2.40	2.03	0.02	-0.23	2.61	2.46	2.26	2.66	**2.82**							
11. Total Recall A1-5	-335.83	-14.54	26.01	0.75	-3.03	38.42	42.66	36.83	38.27	9.95	**146.99**						
12. DS Sequencing	-77.24	-2.39	1.45	0.15	-0.30	3.74	4.56	3.60	4.28	0.82	11.92	**4.59**					
13. DS Backward	-79.48	-1.89	3.09	0.19	0.48	4.53	4.58	4.76	3.79	0.23	11.32	3.31	**5.60**				
14. Category Test	735.62	-11.45	-30.66	0.94	-5.03	-46.91	-55.78	-46.60	-48.22	-4.93	-140.83	-18.40	-20.67	**877.60**			
15. Coding	-628.17	-24.85	11.12	0.74	-2.31	13.47	27.22	15.82	27.14	2.78	72.37	15.34	16.86	-151.39	**248.62**		
16. Symbol Search	-284.50	-15.66	8.00	-0.21	-0.88	7.70	14.12	8.34	14.04	0.76	38.22	7.16	9.31	-96.64	84.44	**59.03**	
17. Trail A[a]	743.99	65.29	-8.24	1.08	1.46	-19.28	-25.24	-19.86	-22.88	-4.34	-64.21	-11.97	-10.57	230.55	-139.10	-76.01	**304.70**
M	0.00	11.22	0.00	1.20	13.61	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.07

Note. Variances appear in bold along the diagonal.
[a] n = 68.

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