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Pragmatic Error Identification in Traumatic Brain Injury

by

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Thesis

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Abstract

Pragmatic Error Identification in Traumatic Brain Injury

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research are discussed.

Supervisor: Thomas Marquardt

Traumatic brain injury causes physical, neurobehavioral, and cognitive-linguistic deficits including problems related to pragmatic functioning and emotional processing. This study investigated the ability of 10 (9 male and 1 female) adults with traumatic brain injury and 10 neurotypical participants to identify errors in pragmatic behavior embedded in 25 videotaped interactions presented by computer. Statistical analysis revealed that the neurotypical participants identified significantly more violations of pragmatic rules than the participants with traumatic brain injury for two of the five deficit categories, excessive interruption and two deficits. Limitations of the study and directions for future

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INTRODUCTION

Each year in the United States, approximately 1.7 million people are diagnosed with traumatic brain injury. Of those injured, approximately 52,000 result in death and an additional 275,000 require some amount of hospitalization (Faul, Xu, & Ward, 2010). Many physical, cognitive-linguistic, and neurobehavioral sequelae result from a traumatic brain injury; deficient social pragmatic functioning and emotional processing deficits are acknowledged as two of the largest impediments to a high quality-of-life in the chronic stage of the injury (Dahlberg et al., 2007). The ability to effectively navigate social situations and understand emotional states of others is paramount to participation in society. Social functioning and emotional processing deficits resulting from brain injury have been linked to an inability to gain and keep employment (Temkin, Corrigan, Dikmen, Machmer, 2009), an increase in social isolation (Dahlberg et al., 2007), impaired academic abilities (Ashman, Gordon, Cantor, & Hibbard, 2006), and an increase in drug and alcohol abuse (Ommaya, Salazar, Dannenberg, Chervinsky, & Schwab, 1996). The decrease in quality of life caused by social deficits has been found to persist more than a decade after the initial injury (Thomsen, 1984). Remediation of these social deficits would allow affected individuals to participate more fully in the daily activities that allow for people to connect with each other.

Speech-language pathologists (SLPs) often are tasked with the rehabilitation of social pragmatic deficits following TBI. In order to provide effective intervention, SLPs

must know what level of awareness a patient has regarding their deficits. Impaired self-awareness has been shown to be common in individuals with TBI (Vanderploeg, Belanger, Duchick, & Curtiss, 2007). Video feedback has been used to increase awareness of and remediate deficits in individuals with TBI (Schmidt, Fleming, Ownsworth, & Lannin, 2012), but little research has examined whether individuals with TBI can identify deficient communication in others, which is an important skill for navigating common social interaction.

DEFINITION OF TRAUMATIC BRAIN INJURY

Traumatic brain injury (TBI) is the result of an external mechanical force or object acting on the skull that results in one or more pathologies, which may be temporary or permanent (Brookshire, 2007). A TBI is neither degenerative nor congenital. An individual may acquire a TBI even though the head has not struck nor been struck by another object, typically these injuries are caused by rapid acceleration-deceleration events like motor vehicle accidents or concussive blasts (Ashman et al., 2006; DePalma, Burress, Champion, & Hodgson, 2005). Individuals with TBI often present with a cluster of sequelae that include deficits in cognition, language, memory, behavior, and emotional processing (Langlois, Rutland-Brown, & Wald, 2006; Riggio, 2010).

EPIDEMIOLOGY

The leading causes of TBI among all age groups are, according to the Center for Disease Control (CDC), in descending frequency: falls, motor-vehicle accidents, being struck by or striking an object, and assaults. Cause of injury rates vary with age. Falls are the most common cause of TBI in adults over 75 years old and children younger than four years. In adults aged 20-24, motor vehicle accidents are the most common cause, followed by assaults (Faul, et al., 2010). Apart from age, the other significant risk factors for TBI are gender and low socioeconomic status. Men are approximately 1.5 times more likely than women to acquire a TBI. The risk for acquiring TBI for those without insurance is nearly twice as high as those with private insurance (Corrigan, Selassie, & Orman, 2010; Faul et al., 2010).

Mortality rates vary significantly with age and gender. Individuals over the age of 75 are most likely to incur a fatal TBI, with a mortality rate twice as high as the next nearest age group. A number of age-related factors may lead to higher mortality rates in the elderly including cognitive and motor impairments and increased cerebral inflammation post-injury (Roosen, Vandenbussche, & Depreitere, 2013). Men are more than three times as likely to acquire a fatal TBI (Faul et al., 2010). The significant difference between mortality rates for men and women may be due to men having a larger proclivity for high-risk behaviors (Corrigan et al., 2010).

BIOMECHANICS AND PATHOPHYSIOLOGY

Brookshire (2007) described the biomechanics and pathophysiology of traumatic brain injury. TBI can be classified as either an open head injury (OHI) or a closed head injury (CH). CHIs are more common that OHIs and are usually caused by falls, motor vehicle accidents, or a blunt object striking the head at a low velocity. CHIs can be further classified as either nonacceleration or acceleration-deceleration injuries.

Nonacceleration injuries are the result of a restrained head being stuck by a moving object. Trauma resulting from a nonacceleration injury is primarily a result of the deformation of the skull and the resultant focal damage to the meninges and cortical tissue below the point of impact. Nonacceleration injuries often result in less serious TBIs than do acceleration injuries.

Acceleration-deceleration injuries are the result of the head in a moveable, but resting, state being violently accelerated or by the head being rapidly stopped while already in motion. Acceleration-deceleration injuries can be further categorized based on the location of the force or object striking the head, into either angular acceleration or linear acceleration injuries. Angular acceleration injuries are caused by a force or object striking the head off center, which causes the head to rotate away from the point of impact. In an angular acceleration injury, the brain initially stays at rest while the skull rotates, then the brain begins to move, and then continues to rotate after the skull has come to a rest. Damage is caused both during the initial and final inertial mismatches between the skull and brain. The twisting and shearing forces induced by an angular

acceleration injury cause damage primarily at boundaries between grey and white matter, resulting in damage to major nerve tracts. Angular acceleration injuries typically produce more severe TBIs than linear acceleration injuries.

Linear acceleration injuries are caused by a force or object striking the head on a vector that passes through the central axis of the skull. Upon being stuck, the skull begins moving while the brain stays motionless as a result of inertia, compressing the brain against the skull. The brain then rebounds to match the movement of the skull. Once the skull has come to a stop, either as a result of striking another object or through physiological restraint, inertia causes the brain to strike the skull opposite from the initial point of impact. Such an injury is called a coup-contrecoup injury. Linear acceleration injuries typically cause focal injuries to the meninges and cortical tissue at the points of impact between the brain and skull.

SEVERITY

The severity of a TBI typically is classified as mild, moderate, or severe upon admission to a hospital or initial doctor's visit. There are several methods used for classifying the severity of a TBI, including the use of standardized scales and neuroimaging techniques. One of the most commonly used standardized scales is the Glascow Coma Scale (GCS; Teasdale & Jennet, 1974; Saatman et al., 2008). The most commonly used neuroimaging technique is computerized tomography (CT) (Shenton et al., 2012). Other methods of severity classification may be preferable, such as duration of

loss of consciousness or length of post-traumatic amnesia, depending on the specific setting and provider skill set (Corrigan et al., 2010).

The accuracy of using initial severity of injury as a predictor of the presence and magnitude of post-acute deficits is contested. Serino et al. (2006) indicated that severity is useful in predicting prognosis for recovery and course of treatment. Others, such as Riggio (2010) and Saatman et al. (2010), state that the use of a neurophysiologic diagnosis is unreliable in predicting chronic deficits due to the multitude of factors that contribute to recovery. Highly individual factors such as pre-morbid health, age, education, substance abuse and socioeconomic status have been found to have an effect on recovery from a TBI (Brookshire, 2007).

DIAGNOSIS

The Glascow Coma Scale uses a standardized set of criteria to assess the levels of consciousness and responsiveness after TBI. The GCS uses a numeric ranking of between 3 and 15 to broadly categorize a patient as having a mild, moderate, or severe injury. The GCS can be useful for managing TBI clinically, but unfortunately does not provide information about potential post-acute neurological and communicative deficits (Saatman et al., 2008).

Severe TBI, GCS scale of 3-8, includes the most significant loss of consciousness (LOC), lasting longer than 24 hours, and is operationally defined as a coma. Individuals who sustain a severe TBI may present with a number of secondary sequelae, such as: significant axonal injury throughout the brain and brainstem, hemorrhages, seizures,

microbleeds, cerebral edema, hypoxia, ischemia, compression of cerebral ventricles, and cell death (Andriessen et al. 2011; Brookshire, 2007; Papathanisou, Coppens, & Potegas, 2011). The quantity of axonal damage sustained from severe TBI often limits the amount of recovery, as the benefits of neuronal regeneration resulting from neuroplasticity are minimal in comparison (Brookshire, 2007).

Classified as a GCS of 9-13, moderate TBI also includes diffuse axonal injury in the brain and brainstem, but is less extensive than that observed in severe TBI. Focal lesions may be present in the inferior frontal and temporal lobes, in addition to hematomas (Brookshire, 2007). Many of the secondary sequelae in severe TBI can occur in moderate TBI, although both the severity and frequency of such sequelae are significantly decreased (Andriessen et al., 2011). Unlike severe TBI, individuals with moderate TBI will demonstrate a measure of physiological recovery as a result of axonal sprouting and dendritic proliferation resulting from neuroplasticity (Brookshire, 2007).

Mild TBI, also known as concussion, is classified as a GCS of 13-15 and accounts for approximately 75% of the total TBI cases in the United States (Bazarian et al., 2005). In addition to the requisite GCS score of 13-15, patients should have an LOC of less than 30 minutes and an altered mental state or post-traumatic amnesia no greater than 24 hours (Kay et al., 1993). The incidence rate of mild TBI may be significantly higher than current estimates because individuals who sustain a mild TBI are unlikely to go to an emergency room or be seen by a neurological specialist (Ashman et al, 2006; Flanagan, Hibbard, Riordan, & Gordon, 2006).

Computerized tomography is the most common imaging technique used for the diagnosis of moderate to severe TBI in the acute setting, as CT easily identifies intracranial hemorrhaging. Magnetic resonance imaging (MRI) has greater diagnostic sensitivity for non-hemorrhagic traumatic brain injuries such as cerebral contusions and diffuse axonal damage, although MRI is too time consuming for use as the initial diagnostic technique and is less likely to be available in emergency rooms (Kim & Gean, 2011, Shenton et al, 2012).

The diagnosis of mild TBI via neuroimaging is more difficult, as the brain can appear uninjured on both CT and MRI scans. Diffusion tensor imaging (DTI) can detect the diffuse axonal damage that is the most significant injury associated with mild TBI, but DTI is a relatively new technology and not widely available (Shenton et al., 2012). In lieu of definitive neuroimaging evidence of injury, the World Heath Organization (WHO) advocates the diagnosis of mild TBI based on the presence of one or more of the following four criteria: loss of consciousness for 30 minutes or less, post-traumatic amnesia lasting less than 24 hours, confusion or disorientation, and/or transient neurological signs such as seizures or intracranial lesions (Carroll, Cassidy, Holm, Kraus, & Coronado, 2004).

The diagnosis of neurobehavioral, cognitive, and communicative post-injury deficits can be difficult. Few TBI-specific standardized scales exist with which to classify patients' specific chronic deficits. Individuals who sustain mild TBI may not seek medical help and therefore the cognitive-linguistic, behavioral, and emotional deficits

that arise may be attributed to an etiology other than TBI (Ashman et al., 2006). Family members and heath care professionals may believe that the individual is exaggerating their impairments or classify the symptoms as psychogenic (Mayou, Black, & Bryant, 2000; McAllister & Arciniegas, 2002).

DEFICITS SPECIFIC TO COGNITION AND COMMUNICATION

The pattern of deficits resulting from TBI differs depending on the locations of damage, the type of damage (i.e. whether the injury was primarily focal or diffuse), and the amount of time that has passed since the injury. The positioning of the brain within the skull increases the likelihood of contusions on the inferior frontal lobes and the anterior temporal lobes (Flanagan et al., 2006) Damage to the frontal lobes results in executive dysfunction, including deficits in organization, self-regulation, task management, and planning (Douglas, 2010). Frontal lobe damage can also cause disinhibition of inappropriate behavior. Left temporal lobe damage can result in difficulties with auditory information processing and language processing, particularly with word memory and semantic comprehension. Prosopagnosia, i.e. difficulty with facial recognition, can result from right temporal lobe damage. Focal lesions have the potential to affect a limited number of cognitive functions, specific to the area damaged, although the severity of the resulting deficits can range from mild to severe. Diffuse axonal injury results in an unpredictable array of deficits due to the potential for disparate areas of both hemispheres to be damaged. Diffuse axonal injury is more likely to cause significant deficits than focal injuries, but the severity of deficits can also range from

mild to severe (Bear, Connors, & Paradiso, 2007). In the post-acute phase of TBI, approximately 6 months post-injury, persistent cognitive-linguistic deficits have been noted in the areas of executive functioning, concentration, attention, memory, fluency, and word-retrieval (Ashman et al., 2006; Dikmen et al., 2009).

Pragmatic Deficits

While no singular definition exists, researchers use similar language to define pragmatics. Turkstra, McDonald, and Kaufman (1996) defined pragmatics as "the skills underlying competence in contextually determined, functional language use" (p. 329). Martin and McDonald (2006) use a similar definition, calling pragmatics the "interpretation and use of language in context" (p. 202). As Coelho (2007) noted, "the common link in most definitions indicates that pragmatics is concerned with the social appropriateness of language" (p.123).

Multiple pathologies can cause an individual with TBI to exhibit pragmatic deficits, including: diffuse axonal injury, focal right hemisphere damage, and bilateral frontal lobe damage. The possibility of more than one of these pathologies occurring simultaneously in a TBI patient is high, especially in cases of moderate to severe TBI. These injuries can cause individuals to exhibit deficits in pragmatic understanding, awareness, and communication during discourse (Coelho et al., 2002; Cummings, 2007; Martin & McDonald, 2006).

Diffuse axonal damage, seen frequently in traumatic brain injuries, is not often constrained to a single hemisphere. The left hemisphere primarily processes lexical

information, while the right hemisphere is primarily responsible for the regulation of emotional processing. As a result, diffuse axonal injuries may result in deficits similar to focal right hemisphere damage; however, this is a simplified explanation and there are numerous cortical structures involved in both lexical and emotional processing. Multiple pragmatic deficits can arise as a result of right hemisphere damage, the majority of which center around the emotional content of language and the ability to draw inferences. Damage to the right hemisphere can produce deficits in the ability to express accurate prosody or facial expression or to perceive emotion conveyed through other individuals' speech or facial expressions (Dimoska, McDonald, Pell, Tate, & James, 2010; Green, Turner, & Thompson, 2004; McDonald, 1993; McDonald & Flanagan, 2004, Ross & Monnot, 2008, Ylvisaker, Szekeres, Henry, Sullivan, & Wheeler, 1987). Deficits in the ability to make inferences about speech include difficulties in understanding sarcasm and irony, difficulty recognizing social behavior violations, e.g. faux pas, and allowing others to assume incorrect assumptions (Johnson & Turkstra, 2012; Martin & McDonald, 2005; McDonald, 1999; Milders, Fuchs, & Crawford, 2003, Winner, Brownell, Happe, Blum, & Pincus, 1998).

Frontal lobe damage affects primarily the aspect of pragmatics related to executive functioning (Douglas, 2010). Pragmatic deficits resulting from frontal lobe damage include disinhibition of inappropriate responses, an inability to generate well-organized discourse, reductions in cognitive flexibility, difficulty with both topic maintenance and topic switching, difficulty maintaining attention, and difficulty in

initiating discourse (Burgess & Shallice, 1996; Busch, McBride, Curtis, & Vanderploeg, 2005; Coelho et al., 2002, Douglas, 2010). Frontal lobe damage can also affect overall cognitive processing speed and working memory, which can contribute in some measure to the presence of more specific pragmatic deficits (Busch et al. 2005; Mathias & Wheaton, 2007).

Theory of mind (TOM) deficits, or an inability to form representations of other people's mental states and then use those representations to predict and comprehend language and behavior, have been demonstrated in individuals with both RH and frontal lobe damage (Martin & McDonald, 2006; McDonald & Flanagan, 2004). Individuals with TOM deficits have been shown to have difficulty distinguishing between intentionally false statements, i.e. lies, and nonliteral ironic statements (Winner et al., 1998). TOM deficits can cause individuals to have increased difficulty in understanding stimuli that require the ascription of mental states over stimuli that require no inferencing of mental state (Happe, Brownell, & Winner, 1999).

Turkstra (2008) stated that the origin of pragmatic communication impairments is likely an as-of-yet unknown combination of non-social related cognitive deficits (e.g. executive function or inferencing deficits) and social cognition-specific impairments. The precise combination by which these specific impairments result in a single TBI patient's deficits is likely highly individualized and difficult to predict.

ASSESSMENT

Very few, if any, commercially available assessment tools exist which are specifically designed to gauge the broad range of pragmatic deficits in individuals with TBI. The Awareness of Social Inference Test (TASIT; McDonald, Flanagan, Rollings, & Kinch, 2003), is a commercially available video-based test created specifically for adults with TBI; however, the TASIT is limited to testing whether subjects can differentiate between sarcasm and sincere speech. Due to the lack of a TBI-specific instrument, pragmatic assessments designed for other populations are often employed to characterize the difficulty in communication that individuals with TBI experience. These assessments include measures designed for other non-TBI populations such as the Faux Pas Test (Baron-Cohen, O'Riordan, Stone, Jones, & Plaisted, 1999), designed for children with autism spectrum disorder (ASD) and the Hayling Test (Burgess & Shallice, 1996), designed for stoke patients. These measures also are limited in that the tests are administered in a manner that differ significantly from actual conversation and that they test a single type of pragmatic deficit. The Faux Pas Test (Baron-Cohen et al., 1999) measures only an individual's ability to use inferencing skills and the Hayling Test (Burgess & Shallice, 1996) measures inhibition exclusively. Turkstra (2008) noted that the Movie for the Assessment of Social Cognition (MASC; Dziobek et al., 2006), a 15minute video of 4 adults conversing, tests a wide range of pragmatic skills. Unfortunately, the MASC was designed for individuals who are developmentally unable

to learn appropriate pragmatic skills, not for those who had such skills and then acquired an impairment due to injury.

PURPOSE

The objective of this study is to investigate the ability of individuals with TBI to detect the occurrence of pragmatically deficient communication. Individuals with TBI frequently exhibit pragmatic language deficits including, but not limited to, poor turn-taking, poor topic maintenance, and an inability to infer the meaning of non-verbal behavior (Coelho, Liles, Duffy, & Clarkson, 1993; Coelho et al., 2002; McDonald & Flanagan, 2004). Due to an inability to recognize their behavioral deficits as a result of their brain injury individuals with pragmatic deficits are generally incapable of self-correcting (Vanderploeg et al., 2007). Speech-language pathologists are often employed to assess and remediate these impaired pragmatic skills. The ability of individuals with pragmatic deficits resulting from TBI to perceive their deficits or the deficits of others is not currently known. Should individuals with TBI-induced pragmatic deficits be unable to recognize deficits, SLPs would be tasked with teaching the individual to recognize pragmatic violations to increase communicative competence in various settings and modalities.

It is predicted that individuals with TBI will be unable to consistently differentiate between normal and deficient pragmatic communication and will have significantly more errors than the non-brain damaged (NBD) individuals. Additionally, it is predicted that individuals with TBI will be most successful at identifying deficient pragmatic

communication marked by a non-linguistic sound (e.g. laughter) and least successful at identifying deficient pragmatic communication consisting of multiple error forms.

METHOD

PARTICIPANTS

Ten individuals with TBI and ten non-brain injured individuals participated in this study. The experimental group was comprised of 9 males and 1 female brain injured individuals ranging between 19 and 46 years of age (mean age=31.7) (See Table 1). Participants were required to meet the following criteria: 1) a minimum of 6 months post closed-head traumatic brain injury, 2) demonstrate a pragmatic language deficit as determined by a certified speech-language pathologist, 3) no greater than moderate brain injury on the Scales for Cognitive Assessment of Traumatic Brain Injury (SCATBI; Adamovich & Henderson, 1992), 4) between the ages of 18 and 55, 5) native English speakers and 6) functional auditory and visual acuity sufficient for the experimental tasks as determined during a medical history interview. Patients with a history of concomitant receptive language deficits or visual neglect were excluded from the study. One participant was a bilingual Spanish-English speaker who had attended schools with English as the language of instruction from elementary school through high school. Table 3 contains subtest and total score results for the SCATBI (Adamovich & Henderson, 1992). for the TBI participant group. The **SCATBI**, (Adamovich & Henderson, 1992) is an assessment that tests the cognitive and linguistic abilities of individuals with closedhead brain injury aged 15 years and older. The test is comprised of five subtests: Perception/Discrimination, Orientation, Organization, Recall, and Reasoning. The TBI participants scored highest on the Organization subtest (X = 117.3, SD = 11.29), followed by the Orientation subtest (X = 114.6, SD = 9.28). The lowest scores were on the Recall subtest (X = 103.3, SD = 8.73) and Perception/Discrimination subtest (X = 103.4, SD = 7.01). The mean total severity score for the TBI participant group was in the mild range (X = 110.3, SD = 9.19). Two of the participants had a borderline normal total severity score, while five scored in the mild range, and one participant received a moderate severity score.

Participants with TBI were recruited from three locations: The Mary Lee
Foundation, a residential and vocational rehabilitation center located in Austin, Texas;
CORE Health Care, a post-acute and long-term care rehabilitation facility in Dripping
Springs, Texas; and Texas NeuroRehab, a medical and outpatient rehabilitation facility in
Austin, Texas.

Table 1. Age, time post injury, and gender for TBI participants (n = 10)

| Participant | Age (years) | Time Post Injury (years) | Gender |
|-------------|-------------|-----------------------------|--------|
| 1 | 46 | 19 | M |
| 2 | 29 | 3.8 | M |
| 3 | 41 | 10.2 | M |
| 4 | 39 | 10 | M |
| 5 | 27 | 3.5 | F |
| 6 | 40 | 1.1 | M |
| 7 | 29 | 2.3 | M |
| 8 | 25 | 0.5 | M |
| 9 | 19 | 0.35 | M |
| 10 | 22 | 0.5 | M |
| Mean | 31.7 | 5.1 | |
| Std. Dev. | 9.13 | 6.11 | |

Table 2. SCATBI subtest scores for TBI participants (n = 10)

Scales of Cognitive Ability for Traumatic Brain Injury (SCATBI)

| Scales of Cognitive Ability for Traumatic Brain Injury (SCATBI) | | | | | | | | | |
|---|--------|--------|--------|--------|--------|--------|----------------------|--|--|
| Participant | Perc* | Orie* | Org* | Rec* | Reas* | Total | Severity | | |
| 1 | 91 | 97 | 107 | 93 | 94 | 93 | Moderate | | |
| 2 | 113 | 119 | 129 | 98 | 102 | 114 | Mild | | |
| 3 | 104 | 119 | 129 | 98 | 103 | 112 | Mild | | |
| 4 | 95 | 97 | 115 | 105 | 121 | 106 | Mild | | |
| 5 | 104 | 119 | 129 | 101 | 102 | 112 | Mild | | |
| 6 | 113 | 119 | 107 | 110 | 125 | 120 | Borderline Normal | | |
| 7 | 101 | 119 | 115 | 95 | 97 | 104 | Mild | | |
| 8 | 104 | 119 | 98 | 103 | 103 | 104 | Mild | | |
| 9 | 101 | 119 | 129 | 123 | 117 | 126 | Borderline Normal | | |
| 10 | 108 | 119 | 115 | 107 | 117 | 112 | Mild | | |
| Mean | 103.40 | 114.60 | 117.30 | 103.30 | 108.10 | 110.30 | | | |
| Std. Dev. | 7.01 | 9.28 | 11.29 | 8.73 | 10.85 | 9.19 | | | |

Table 3. Age and gender of non-brain injured participants (n = 10)

| Participant | Age(years) | Gender |
|-------------|------------|--------|
| 1 | 24 | F |
| 2 | 37 | F |
| 3 | 39 | M |
| 4 | 47 | M |
| 5 | 21 | M |
| 6 | 28 | M |
| 7 | 35 | M |
| 8 | 23 | M |
| 9 | 24 | M |
| 10 | 29 | M |
| Mean | 30.7 | |
| Std. Dev. | 8.47 | |

The unimpaired group consisted of 8 male and 2 female non-brain injured individuals between the ages of 21 and 47 years of age (mean = 30.7) (See Table 3).

These individuals were native English speakers with functional visual and auditory acuity for the task as determined during an initial interview.

MATERIALS

In order to determine the severity of brain injury deficits for participant qualification, subjects were administered the *SCATBI* (Adamovich & Henderson, 1992). *SCATBI* scores for two participants were made available from a previous study conducted by the University of Texas at Austin; the test was not re-administered to these individuals. Once participants were qualified for the study, they were given a receptive pragmatic violation identification task and questionnaire designed by the principal investigator.

RePVIT. The Receptive Pragmatic Violation Identification Task is a novel video-based experimental task designed by the principal investigator to measure the ability to identify pragmatic communication deficits in conversation between two neurotypical speakers. RePVIT consists of 5 training video vignettes and 25 experimental video vignettes. The 5 training vignettes consist of one social conversation with no pragmatically erroneous behavior present and four social conversations demonstrating one of the following error behaviors: excessive topic switching, repeated interruption, inappropriate laughter, or inappropriate proxemics, i.e. standing too close. Each of the training videos corresponds to a potential answer on the response questionnaire. The

experimental vignettes consist of 5 sets of 5 videos each. The sets of videos are comprised of: 5 normal conversations, 5 conversations with excessive topic switching, 5 with repeated interruption, 5 with inappropriate laughter, and 5 videos of a conversation demonstrating two co-occurring deficits, excessive topic switching and excessive interrupting. No videos demonstrating inappropriate proxemics are included in the experimental set as this option on the response questionnaire is intended to act as a participant confound to identify individuals who have not understood the instructions or are not actively attempting to complete the experimental task. The content of the vignettes reflected general topics found in typical conversation such as: pet ownership, restaurants and dining with others, sporting events, weather conditions, family, entertainment (e.g. movies, music, books), and work.

The pragmatic error behaviors were selected to provide differentiation based on the cueing modality. Excessive topic switching is cued solely though semantic means, in that the participant must be cognizant that a speaker's preceding utterance has no broad semantic relation to the following utterance. Repeated interruption is indicated by both auditory and linguistic cues, with the sound of the two actors simultaneously producing utterances with semantic content. Inappropriate laughter is indicated by a non-linguistic auditory cue, i.e. the staccato, vowel-like, melodic nature of voiced laughter. The error proxemic pragmatic behavior chosen as a confound has an exclusively visual, non-linguistic, non-auditory cue, e.g. the observed distance between the two actors. Excessive topic switching and excessive interruption were chosen as the deficits for inclusion in the

Table 4. Error categories, cue modalities, and examples

| Error Category | Cue Modality | Cue Example |
|---------------------------|-------------------------|---|
| Excessive topic switching | Semantic | Subsequent utterances are unrelated |
| Excessive interruption | Semantic + Auditory | Two utterances occurring simultaneously |
| Inappropriate laughter | Non-linguistic auditory | Staccato, voiced laughter |
| Proxemic violation | Visual only | Distance between speakers |

set of videos with two error behaviors co-occurring because of the greater similarity between their cueing modalities (they both contain a semantic cueing component) than that of the inappropriate laughter, which lacks a semantic cueing component.

To account for potential attention deficits resulting from TBI, each vignette is brief (between 20 and 30 seconds) and has two speakers standing in front of a featureless background. Ecological and face validity are maintained in the stimulus videos; in accordance with the required age range of the experimental participants, the same two adult women actors appear in all of the vignettes. The volume of each video has been normalized in post-production using Adobe Premier Pro to ensure that stimuli are presented at similar volume levels. To prevent order effects, a random number generator was used to determine the order of vignette presentation for each participant in both the TBI and NBD subject groups.

The RePVIT response questionnaire is written in a large font to minimize unreported minor reading or visual deficits. The answer blocks are written in an identical

manner to reduce participant confusion and to minimize the amount of instruction necessary. Reading comprehension was not directly tested, but participants indicated that they had functional reading comprehension for the task by appropriately completing the response questionnaire. A sample page of the RePVIT response questionnaires is included in the Appendix.

PROCEDURES

The participants were tested at the living center where they resided or attended outpatient therapy. Five of the patients required two sessions to complete the requirements for the experiment; the initial 75-120 minute session consisted of a medical history interview and administration of the *SCATBI* (Adamovich & Henderson, 1992), the second 30-45 minute session consisted of the administration of the RePVIT. The two participants who had previously completed the *SCATBI* (Adamovich & Henderson, 1992) required a single 60-minute session, consisting of a medical history interview and the administration of the RePVIT.

The administration of the RePVIT began with an instructional segment. The investigator informed the participants that they would be watching a series of videos showing conversations between two women and that the participant was responsible for deciding whether "there was something wrong with the conversation." The investigator then presented the response questionnaire and informed the participant that if there was nothing wrong with the conversation they should check the "No" box, but if they felt something was wrong with the conversation they should check the "Yes" box. The

investigator then played the training video demonstrating a non-deficient conversation and asked if the participant thought anything was anything wrong. If the participant answered "no", the rest of the training vignettes were shown. If the participant answered "yes", the investigator replayed the video and explained that the vignette was an example of a normal conversation. The investigator then played the training videos demonstrating the deficient conversations, explaining prior to each vignette what type of deficit was demonstrated and pointing to the corresponding line of the first answer block on the response questionnaire. After each training vignette, the instructor asked the participant if they would like to re-watch the video. At the participant's request, the instructor repeated the training steps for that vignette. This process was repeated until all 5 training vignettes were viewed.

Once the instructional segment of the RePVIT was complete, the experimental procedures were initiated. The investigator explained that in the experimental vignettes, each video segment could have 0, 1, or 2 of the deficits shown in the training videos and that the participant should closely watch each vignette, and decide if a deficient conversation had taken place. Participants were instructed that if they were unsure of their decision after the initial viewing of an experimental vignette, a second viewing of the vignette was allowed, but no vignette would be played more than twice. The participants were then re-instructed on the method for filling out the response questionnaire. After verifying that the participant understood the instructions, the investigator played the 25 experimental vignettes. After the eighth and sixteenth

vignettes, the participants were reminded that each video could have 0, 1, or 2 deficits demonstrated by the actors.

Each participant viewed the RePVIT stimulus items while seated at a laptop computer. Stimuli were presented on a 11.5" wide by 6.5" high frame, approximately 15" away from the participant, in the center of the visual field on the computer display. Stimuli were presented at a listening level indicated as comfortable by the participant and headphones were made available at the participant's request. Construct validity was confirmed by two speech-language pathology graduate students and one certified speech-language pathologist. Interrater reliability was 100% as determined by the comparison of responses by three speech-language pathology graduate students.

RESULTS

The number of correct responses to the five experimental video vignettes for each behavior type was calculated for the NBD and TBI participants (See Table 6 and Table 7). Total mean scores reflected minimal error responses for the NBD participants (X =24.9, SD = 0.32) in contrast to increased error identification of behaviors for the TBI participants (X = 19, SD = 2.21). The lowest total correct score for the TBI subjects was 16 and the highest was 21. The pragmatic deficit category with the lowest mean correct in the TBI participant group was two deficits (X = 2.4, SD = 1.65), followed by excessive interruption (X = 3.3, SD = 1.16), excessive topic shifting (X = 4.1, SD = 0.88), and no deficits (X = 4.5, SD = 0.97). The inappropriate humor category had the highest mean correct score (X = 4.7, SD = 0.48) in the TBI subject group. In the NBD group, a single incorrect response, chosen by participant 7, occurred in the two deficits category. A oneway analysis of variance revealed a significant effect for groups (F = 13.85, p < .001). There was no interest in interactions between groups and conditions because of the ceiling effect in the NBD participants. Multiple t-tests with Bonferroni correction for multiple comparisons found significant differences between the mean errors of the TBI group and the NBD group for the two deficits and excessive interruption categories (p < .01), but not for the no deficits, excessive topic switching, or inappropriate humor pragmatic categories (p > .01).

To summarize, the TBI participants had significantly more error responses in total than the neurotypical participants. There were significant differences between the mean

correct responses for TBI and NBD participants in the two deficits and excessive interruption categories.

Table 5. Individual scores and mean correct by experimental item for TBI participants.

| Deficit and | d | | | | T | BI S | ubje | ect | | | | Total | | Std. |
|-----------------|-----------|---|---|---|---|------|------|-----|---|---|----|---------|------|------|
| Stimulus Nun | nber | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | correct | Mean | dev. |
| | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 9 | 0.9 | 0.3 |
| | 2 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 10 | 1.0 | 0.0 |
| No Deficit | 3 | 1 | 1 | 1 | 0 | 1 | 0 | 1 | 1 | 1 | 1 | 8 | 0.8 | 0.4 |
| | 4 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 10 | 1.0 | 0.0 |
| | 5 | 1 | 1 | 1 | 0 | 1 | 1 | 0 | 1 | 1 | 1 | 8 | 0.8 | 0.4 |
| | 6 | 0 | 1 | 0 | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 7 | 0.7 | 0.5 |
| Excessive | 7 | 1 | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 1 | 9 | 0.9 | 0.3 |
| Topic | 8 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 9 | 0.9 | 0.3 |
| Shifting | 9 | 1 | 1 | 1 | 1 | 1 | 0 | 0 | 1 | 1 | 1 | 8 | 0.8 | 0.4 |
| | 10 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 8 | 0.8 | 0.4 |
| | 11 | 1 | 0 | 1 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 8 | 0.8 | 0.4 |
| Excessive | 12 | 1 | 0 | 1 | 0 | 1 | 1 | 1 | 1 | 1 | 0 | 7 | 0.7 | 0.5 |
| Interruption | 13 | 1 | 0 | 0 | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 4 | 0.4 | 0.5 |
| interruption | 14 | 1 | 1 | 0 | 1 | 0 | 1 | 0 | 1 | 0 | 1 | 6 | 0.6 | 0.5 |
| | 15 | 1 | 1 | 0 | 1 | 1 | 1 | 0 | 1 | 1 | 1 | 8 | 0.8 | 0.4 |
| | 16 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 0 | 1 | 1 | 9 | 0.9 | 0.3 |
| Inonn | 17 | 0 | 1 | 1 | 0 | 1 | 1 | 1 | 1 | 1 | 1 | 8 | 0.8 | 0.4 |
| Inapp. Humor | 18 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 10 | 1.0 | 0.0 |
| Humor | 19 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 10 | 1.0 | 0.0 |
| | 20 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 1 | 10 | 1.0 | 0.0 |
| | 21 | 0 | 1 | 0 | 1 | 0 | 0 | 1 | 1 | 0 | 1 | 5 | 0.5 | 0.5 |
| Two | 22 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | 1 | 0 | 3 | 0.3 | 0.5 |
| Deficits 1 | 23 | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 1 | 1 | 5 | 0.5 | 0.5 |
| Delicity | 24 | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 1 | 1 | 1 | 6 | 0.6 | 0.5 |
| | 25 | 0 | 0 | 0 | 0 | 0 | 1 | 1 | 1 | 1 | 1 | 5 | 0.5 | 0.5 |

Table 6. Experimental task individual scores and means by category for TBI participants.

| | | Excessive | | | | |
|--------------------|---------|-----------|--------------|---------------|-----------------|-------|
| | No | Topic | Excessive | Inappropriate | Two | |
| Participant | Deficit | Shifting | Interruption | Laughter | Deficits | Total |
| 1 | 5 | 3 | 5 | 4 | 0 | 17 |
| 2 | 5 | 5 | 2 | 5 | 3 | 20 |
| 3 | 5 | 4 | 2 | 5 | 0 | 16 |
| 4 | 2 | 5 | 2 | 4 | 4 | 17 |
| 5 | 5 | 5 | 4 | 5 | 2 | 21 |
| 6 | 4 | 3 | 4 | 5 | 1 | 17 |
| 7 | 4 | 4 | 3 | 5 | 2 | 18 |
| 8 | 5 | 3 | 5 | 4 | 4 | 21 |
| 9 | 5 | 5 | 3 | 5 | 4 | 22 |
| 10 | 5 | 4 | 3 | 5 | 4 | 21 |
| Mean | 4.5 | 4.1 | 3.3 | 4.7 | 2.4 | 19 |
| Std. dev. | 1.0 | 0.9 | 1.2 | 0.5 | 1.6 | 2.2 |

Table 7. Experimental task participant scores and means for NBD participants.

| Participant | No Deficit | Excessive Topic Shifting | Excessive Interruption | Inappropriate Laughter | Two Deficits | Total |
|-------------|---------------|--------------------------------|---------------------------|---------------------------|-----------------|-------|
| 1 | 5 | 5 | 5 | 5 | 5 | 25 |
| 2 | 5 | 5 | 5 | 5 | 5 | 25 |
| 3 | 5 | 5 | 5 | 5 | 5 | 25 |
| 4 | 5 | 5 | 5 | 5 | 5 | 25 |
| 5 | 5 | 5 | 5 | 5 | 5 | 25 |
| 6 | 5 | 5 | 5 | 5 | 5 | 25 |
| 7 | 5 | 5 | 5 | 5 | 4 | 24 |
| 8 | 5 | 5 | 5 | 5 | 5 | 25 |
| 9 | 5 | 5 | 5 | 5 | 5 | 25 |
| 10 | 5 | 5 | 5 | 5 | 5 | 25 |
| Mean | 5 | 5 | 5 | 5 | 4.9 | 24.9 |
| Std. dev. | 0 | 0 | 0 | 0 | 0.3 | 0.3 |

DISCUSSION

Results from the study add to the body of information about the ability of individuals with traumatic brain injury (TBI) to identify pragmatically error behavior presented in a video format (Dziobek et al., 2006, McDonald et al., 2003; Turkstra, 2008). This study investigated if there was a significant difference between traumatic brain injury (TBI) and non-brain damaged (NBD) individuals in the ability to identify deficient pragmatic behavior with regard to total errors. Based on previous research by Coelho et al., 1993; Coelho et al., 2002; Dimoska et al., 2010; Douglas, 2010; Martin & McDonald, 2005; McDonald & Flanagan, 2004; McDonald et al., 2003; Milders et al. Turkstra, 2008; Turkstra et al., 2006; Vanderploeg et al., 2007; and Winner et al., 2008, it was predicted that individuals with TBI would make more total errors than individuals without brain damage. The results from the current study concluded that individuals with TBI had significantly lower mean total scores than did the neurotypical participant group. Findings from the current study reinforce previous research determining that individuals with specific types of brain injury have difficulty processing pragmatically based communication information. The observed reduction in performance on tasks of pragmatic error identification for individuals with TBI as compared to those without brain injury are potentially due to decreases in the ability to maintain appropriate attention for the task, a difficulty in perceiving emotion in the actors' speech, a decrease in cognitive flexibility, an inability to make inferences using theory of mind, and/or overall decreases in cognitive processing speed (Busch et al., 2005; Happe, Brownell, &

Winner, 1999; Douglas, 2010; Martin & McDonald, 2006; Mathias & Wheaton, 2007; McDonald, 1999). A decrease in overall cognitive processing speed or a reduction in the ability to attend to the vignette would interfere with the participant's ability to analyze linguistic information contained in the actors' speech. Therefore, the participant would have difficulty determining if semantic inconsistencies are present between sequential utterances (Douglas, 2010; Mathias & Wheaton, 2007). Perceiving the emotional content of speech is important for understanding the non-linguistic elements of pragmatic communication; a deficit in this skill could lead the participant to misinterpret the appropriateness of emotional cues (Green et al., 2004). Also, a decrease in cognitive flexibility could cause the participant to be unable to accurately identify a pragmatic deficit while simultaneously examining the vignette for the presence of other deficits.

The current study also examined if there was a significant difference in the recognition of pragmatically deficient behavior in individuals with regard to behavior category. Based on previous research by Douglas (2010) and Turkstra (2008), it was predicted that the TBI participants would be most successful at identifying the inappropriate laughter stimuli. Results confirmed that individuals with TBI were most successful at identifying a stimulus marked by a non-linguistic cue (e.g. laughter). As noted by Douglas (2010), individuals with TBI have difficulty in attending to the details of a conversation, in addition to difficulty attending to relevant information within a conversation. Turkstra (2008) noted that impairments in verbal working memory prevented individuals with TBI from accurately interpreting the lexical information in a

conversation. In contrast to the other stimuli contained in the RePVIT, the inappropriate laughter stimuli required no processing of lexical information to accurately identify the presence of a deficit, and therefore should have required the least use of the cognitive systems most affected by TBI. This theory is at least partially supported by the high success rate in identification of the inappropriate laughter stimuli. Significantly, individuals with TBI were more successful at identifying the inappropriate laughter stimuli than at identifying the stimuli with no deficits.

Finally, the experimental hypothesis predicted that individuals with TBI would be least successful at identifying the stimuli containing two deficits, based on research by Busch et al. (2005), Douglas (2010), and Mathias & Wheaton (2007). Results from the current study confirmed that individuals with TBI had the most difficulty in identifying stimuli containing two deficits; of the responses from the two deficit stimuli containing errors, all accurately identified one of the deficits present, but failed to identify the second. Mathias & Wheaton (2007) stated that TBI affects the ability to process even simple stimuli, the results of this study help to support this claim, as individuals with TBI had more difficulty correctly identifying stimuli demonstrating a single deficit, than did NDB individuals. Identifying multiple deficits co-occurring within a single stimulus could require greater cognitive flexibility, increased attention, increased inhibition, and increased verbal working memory over the identification of a single deficit; all of these systems are known to be compromised in individuals with TBI (Busch et. al, 2005; Douglas, 2010; & Mathias & Wheaton, 2007; Turkstra, 2008). The participants, once the

presence of a deficit was identified, had increased difficulty in examining the remaining portion of the vignette for cues of the occurrence of a second deficit due to reductions in cognition as a result of TBI.

LIMITATIONS AND FUTURE DIRECTIONS

The present study included a small sample size for the TBI experimental group (n = 10). A larger sample size would provide additional statistical power for detection of significant differences and would allow for consideration of factors such as sex of the participant, time post injury, and site of lesion. Related to the latter two factors, developing a more complete understanding of each participant's medical history would be helpful. The current study relied on self-reporting and some participants were unable to provide a specific date of injury and site(s) of lesion. Previous or, ideally, current neurological scans would allow for a more detailed consideration of the cortical structures involved in the perception of erroneous pragmatic behavior.

Although a certified speech-language pathologist made the determination of a pragmatic deficit for inclusion in the study, there was no standardized pragmatic measure used to determine severity, as the primary investigator was unaware of the existence of such a measure. Should such a measure become available, use in future studies is recommended for inclusion, allowing for examination of the correlation between severity of pragmatic deficits and performance on the RePVIT.

The success of the NBD group in correctly identifying nearly all stimuli results in low specificity for the RePVIT. Should revision of the RePVIT occur for future studies, consideration should be given to redesigning the stimuli to increase the specificity.

Additionally, five items in the RePVIT were answered correctly by all participants and four items were answered incorrectly by a single participant with TBI. In

future studies, these items should be reevaluated and possibly replaced with items that provide greater sensitivity, improving the utility of the test in differentiating between individuals with pragmatic deficits as a result of TBI and NDB individuals.

The inclusion of individuals with a greater range of deficits in future studies would provide greater information about the specificity of test. The current study tested primarily individuals with mild deficits. Were individuals with moderate or severe deficits tested, mean participant score would have likely been lower and errors would have been scored on all questions.

Future studies should also examine additional pragmatic deficits, expanding on the number of deficient behavior categories presented within the task, as well as the type of cue used to indicate the presence of deficient behavior. Additional behaviors in the experimental set could include higher-order pragmatic categories, such as sarcasm or irony, and proxemic deficit categories, which are lower-order pragmatic behaviors. Additional cueing modalities could include facial expression or prosodic changes as indicators of pragmatic error. Expansion of the task will allow for identification of deficit categories and cues that provide the greatest specificity and sensitivity in differentiating between NBD individuals and individuals with a larger range of deficit severity due to TBI.

CONCLUSION

The present study's findings are consistent with previous studies, suggesting that individuals with TBI have increased difficulty with the perception and identification of pragmatically incorrect behavior (Burgess & Shallice, 1996; Busch, McBride, Curtis, & Vanderploeg, 2005; Coelho et al., 2002, Douglas, 2010). Previous studies examining pragmatic identification using stimuli presented via video have employed longer or more complex stimuli and required expressive responses (Dziobek et al., 2006; McDonald et al., 2003; Turkstra, 2008). The current study attempted to differentiate between TBI and NDB participants using an exclusively receptive task employing brief, simple stimuli. Significant differences were found between the response accuracy of the TBI and NBD groups. Additional analyses indicated that the TBI participants were most successful in identifying stimuli that did not require perception of lexical information for cueing and least successful when perception of more than one cue was required. Clinically, conclusions from this study are useful in determining which deficient pragmatic behaviors are most difficult for patients with TBI to identify. These results can assist clinicians in deciding which behaviors to target for instruction during treatment. Further research is needed to develop a broader array of diagnostic tools for the detection and categorization of pragmatic deficits in individuals with TBI.

Appendix

Pragmatic Behavior Questionnaire (sample page)

| Date: | articipant Code: | |
|---|------------------|----|
| Select the choice or choices that best describe the pragmatic deficits displayed in each video clip. You will have two minutes after end of each clip to make your selections | | |
| 1) Is there something wrong with the conversation If the answer is YES, which of the following desc Inappropriate laughing | | NO |
| Interrupting | | |
| Changing topics too much | | |
| Standing too close to each other Would you like to explain further? | | |
| 2) Is there something wrong with the conversation If the answer is YES, which of the following descriptions. | | NO |
| Inappropriate laughing | | |
| Interrupting | | |
| Changing topics too much | | |
| Standing too close to each other Would you like to explain further? | | |
| 3) Is there something wrong with the conversation If the answer is YES, which of the following descriptions. | | NO |
| Inappropriate laughing | | |
| Interrupting | | |
| Changing topics too much | | |
| Standing too close to each other Would you like to explain further? | | |

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