Neuropsychological, Psychological, and Injury Variables Associated with Post-Traumatic Stress Disorder in Individuals Who Suffered an Electrical Injury

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NEUROPSYCHOLOGICAL, PSYCHOLOGICAL, AND INJURY VARIABLES ASSOCIATED WITH POST-TRAUMATIC STRESS DISORDER IN INDIVIDUALS WHO SUFFERED AN ELECTRICAL INJURY

A DISSERTATION SUBMITTED TO THE FACULTY OF THE GRADUATE SCHOOL IN CANDIDACY FOR THE DEGREE OF DOCTOR OF PHILOSOPHY

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ABSTRACT

Electrical injury (EI) represents a major form of trauma that can greatly impact the individual cognitively, physically, and emotionally. EI can lead to a variety of cognitive impairments affecting attention, processing speed, motor skills, and memory. Furthermore, EI can lead to a variety of physical impairments from burns to cardiac injury. In addition to other psychiatric disorders, individuals who suffer an EI can eventually develop Post-Traumatic Stress Disorder (PTSD).

This study examined a clinical sample of 143 individuals (86.0% male, 85.3% Caucasian, 44.1% diagnosed with PTSD) who have experienced an EI to determine the factors associated with the development of PTSD after EI. By using a clinical sample, this study offered greater generalizability compared to previous research on EI which primarily used electricians. Also, this study applied a unique statistical approach that allows for the creation of subgroups within the context of the model. Classification tree analysis via Optimal Data Analysis determined the demographic and injury parameters, psychological, and neuropsychological factors associated with the development of PTSD in individuals post-EI. The strongest predictor of PTSD for the sample in this study was depressive symptoms. Mood symptoms may be utilized in clinical settings to determine individuals more likely to develop PTSD post-EI.
CHAPTER ONE
INTRODUCTION

The purpose of this study was to investigate neuropsychological, psychological, and demographic variables associated with the development of Post-Traumatic Stress Disorder (PTSD) in individuals who suffered an electrical injury (EI). Specifically, this study investigated the influence of demographic variables, injury parameters, neuropsychological functioning, and psychological functioning using an advanced statistical analysis, Optimal Data Analysis (ODA). Determining the factors linked to PTSD outcomes in EI patients allowed for a better understanding of PTSD and will help to improve treatment.

Clinical neuropsychological practice often involves the assessment and treatment of individuals who suffer a trauma. Traumatic brain injury is the most common form of trauma, and electrical shock injury represents a growing area in need of evaluation (Pliskin, Ramati, & Sweeney, 2007). In the United States, about 5,000 individuals per year suffer an electrical shock injury, and EI represents the leading cause of work-related trauma (U.S. Labor Departments’ Bureau of Labor Statistics, 2009). Clinical presentations of EI are extremely variable, ranging from minor to severe multisystem injury. Individuals who suffer an EI experience physical, cognitive, and emotional changes. Electrical shock enters the body disrupting the electrical rhythms of the heart leading to cardiac arrest and anoxia. Loss of consciousness (LOC) at the time of the injury has been associated with greater cardiac complications including cardiac standstill
and ventricular fibrillation (Arrowsmith, Usgoacar, & Dickson, 1998; Koumbourlis, 2002), although previous research has not found a link between LOC and neuropsychological performance (Pliskin, et al., 1998). Cardiac arrest induced in mice has demonstrated poorer learning for up to a week following the injury (Kofler et al., 2004). The cognitive outcomes following cardiac arrest for adults have been less clear given the variety of factors contributing to the development of cardiac arrest. It is not uncommon for individuals to suffer a fall secondary to the electrical shock which could lead to a variety of physiological, neuropsychological, and psychological sequelae.

While individuals may suffer a host of physical ailments after a traumatic injury, there are also psychiatric sequelae. For example, PTSD commonly occurs in individuals who experience an EI, and women may be especially vulnerable to the development of PTSD post-EI (Holbrook, Hoyt, Stein, & Seiber, 2002; Pietrzak et al., 2011). This study examined the demographic, neuropsychological, and psychological variables related to classification into groups based on the diagnosis of PTSD.

Research has noted changes in attention, mental speed, motor skills, and memory in individuals post-EI (Ammar et al., 2006; Duff & McCaffery, 2001; Pliskin et al., 2006). However, not all studies control for mood disturbance, particularly PTSD. Various psychiatric disorders, particularly PTSD, frequently develop after EI (Grossman & Tempereau, 1993; Kelley et al, 1994; Kelley et al., 1999; Mancusi-Ungaro et al., 1986; Ramati et al., 2009). Furthermore, PTSD itself is associated with impaired memory performance (Charney et al., 1993; Vasterling et al., 1998). One study to date has examined PTSD and memory performance in patients with EI, reporting that EI patients with PTSD performed more poorly on tasks of immediate and delayed recall as compared
with EI patients without PTSD (Ammar et al., 2006). However, these findings have not been teased apart from effort level during neuropsychological examination. Additionally, there have been mixed findings on whether litigation status may be a factor on their performance on neuropsychological measures. Therefore, a variety of factors may influence the development of PTSD post injury.

This study assessed cognitive functioning, emotional functioning, personality functioning, and effort. Additionally, other injury-related factors were taken into account in determining factors associated with PTSD in patients with EI. The current study aimed to use a multivariate classification tree analysis (Optimal Data Analysis; ODA; Yarnold & Soltysik, 2005) to explore the outcomes of individuals with EI. By using a range of variables in the exploratory analysis, this study allowed for unique interactions to emerge while maximizing classification accuracy.

Factors associated with the development of PTSD were examined in individuals who have suffered an EI. First, this literature review will cover the incidence, cost, and physiology of EI. The cognitive, physical, and emotional symptoms, especially PTSD, will also be discussed. Finally, the innovative statistical technique will be introduced, followed by a summary of the current study, and an explanation of the results.

Electrical Injury

In the United States, nearly 1,000 deaths are attributable to electric shock every year. Between 1992 and 1998, 2,287 U.S. workers died and 32,807 workers sustained days away from work due to EI (Cawley & Homce, 2003). In 2009, approximately 2,788 employees, their families, and their co-workers were affected by pricey on-the-job injuries and fatalities (Cawley & Brenner, 2012). More than 90% of these injuries occur
in males between the ages of 20 and 34 (Lee, 1997). Dangerous levels of electrical current passing through the body tissues causes injury by several distinct frequency-dependent pathophysiologic mechanisms. The pathophysiologic mechanisms will be discussed in greater detail later. Certain occupations experience greater exposure to electricity. According to a survey by Tkachenko and colleagues (1999), ninety-seven percent of electricians reported that they had suffered an electrical shock with 2.5% who indicated that they lost consciousness due to an electrical shock.

Medical care is not routinely sought following an electrical shock, as the survey respondents indicated that they sought medical care only if there was a loss of consciousness, severe burn, or fracture (Tkachenko et al., 1999). Outside of the workplace, most electrical injuries are due to lightning strikes or to domestic low voltage (<1000 V) electrical contact (National Safety Council, 1983). Since the mid 1970s, the clinical outcome for electrical injured individuals has improved with advances in triage, critical medical care, and tissue salvage procedures (Lee, 1997).

**Definition**

Research on electrical injury uses a variety of terms to refer to the individual, the shock, and the subsequent outcomes. High voltage electrical injury refers to contact voltage equal or greater to 1000V and low voltage electrical injury refers to contact voltage less than 1000 V. An injury is categorized as a “flash” if there are skin burns or exposed surfaces and no focal contact wounds, while “true” electrical injuries evidence focal contact wounds of a current path through the body (Lee, 1997). Lightning is the colloquial term for the phenomena of dielectric breakdown, or arcing, which occurs when
the voltage difference between clouds and other objects reach such a level that the electric field in the interposing air exceeds 2 million V/m.

**Cost**

One of the leading causes of work-related injury in the United States is electrical shock (U.S. Labor Departments’ Bureau of Labor Statistics, 2009). The economic burden of this type of injury is estimated to be over one billion dollars per year when taking hospital stay and lost wages into consideration (Lee, 1997; Occupational Safety & Health Administration, 1999). These estimates do not take into account individuals who do not seek medical treatment, yet experience EI-related difficulties.

**Physiology**

Electrical conduction refers to the results from electrical charge interactions, which is the force experienced by an electrically charged particle in the presence of a spatial gradient in electrical potential (Lee, 1997). Electricity’s effect on the human body depends on the strength and frequency of the electric field, the path of the current, and the histoarchitecture of the tissues (Lee, 1997). Electricity passes through the body from the entrance site to the exit site and for most injuries the point of contact serves as the entrance point. The majority of the current follows the path of least resistance and given that nerves and blood have lower resistance than either bone or fat, neurons and the brain may be especially vulnerable to EI (Bryan et al., 2006). Resistance is the amount of force that opposes the flow of electrical current.

Four mechanisms of cellular injury by electricity are presently acknowledged including: mechanical injury due to falls, the direct effects of the current, thermal burns, and electroporation (Spies & Trohman, 2006). Individuals who experience an EI may
have a loss of consciousness which may lead to a fall and mechanical blunt force trauma to the head. The combination of the EI and TBI makes differentiating the cognitive sequelae of the EI and the TBI nearly impossible.

Another mechanism of cellular injury is due to direct effects of the current. The passage of the current through tissue can cause muscle contractions. If pathways include the chest, they can induce systole and/or apnea and be life-threatening (Bryan et al., 2009). The body, composed of various tissues with different chemical properties, is a non-homogeneous electrically conducting material. When a current passes through the body, the electrical current distribution in the tissue depends on the relative electrical conductivity of various tissues and the frequency of the current. For low-frequency current delivered by contact with a small surface of the skin, the current density is greatest at the points of contact. While the body is discharging into the ground, the individual would experience a much smaller current.

The next mechanism of cellular injury refers to thermal burns. Following an electrical injury, an individual may experience epidermal breakdown (Lee, 1997). Complete epidermal destruction can occur at contact points when the voltage exceeds 200 V. The very brief period where the body surface potential can reach several thousand volts between the upper and lower body is the cause of the breakdown of the epidermis.

The final mechanism of cellular injury is electroporation. Electroporation refers to the process by which electricity directly causes pore formation in the lipid bilayers that form cell membranes. The cell will try to preserve its ionic gradients through great energy expenditures. The cell will eventually die if energy stores are depleted in trying to reseal pores. The larger the cell, the more likely it is to be injured by electroporation.
(Bryan et al., 2006). This process causes more rapid and diffuse necrosis in EI.

Disruption of cell membranes can wreak havoc on nerve and muscle tissue, although spontaneous sealing of membranes can occur (Lee, 1997). Even if the pores close, secondary injury processes such as the influx of calcium ions can be disruptive or fatal to the cell. The influx of calcium ions can cause the cytoskeleton of the cell to collapse, similar to what occurs in diffuse axonal injury (DAI). In conclusion, there are a variety of ways in which electricity injures cells.

**Cognitive Symptoms**

Electrical injury has been reported to lead to a variety of cognitive impairments. These impairments affect attention and concentration, memory, intelligence, and language. Research has shown that cognitive changes occur in EI survivors, even when the head was not in direct contact with the electrical power source (Pliskin et al., 1999, 2006). Some of the research on the cognitive symptoms associated with EI has simultaneously examined secondary medical or psychiatric complaints that may impact cognitive functioning while many studies have not.

**Memory**

Memory impairment is one of the deficits cited post-EI (Barres et al., 1994; Capelli-Schellpfeffer et al., 1994; Daniel, Haban, Hutcherson, Bolter, & Long, 1985; Hooshmand et al., 1989; Pliskin et al., 1994; Lee, 1997; Pliskin et al., 1998). Hooshmand and colleagues (1989) observed impairment on recent memory measures in 16 patients within three months to one year post-EI. Janus and Barrash (1996) observed selective deficits in verbal memory in 12 of 13 patients. The time elapsed between injury and observation varied widely. For example, one patient was seen one week post-injury while
another was seen 13 years post-injury. Although they found memory impairment in their patients across the range of time since the injury, a larger sample would have allowed for further analysis of different time periods (e.g., acute, short-term, long-term). Barrash and colleagues (1996) used the Rey Auditory Verbal Learning Test and the Benton Visual Retention Test to assess immediate and delayed verbal memory and non-verbal memory. They found verbal learning and verbal memory deficits in 18 EI patients relative to a group of TBI patients when compared acutely, in the short-term, and in the long-term. The EI literature suggests that memory impairment is a primary neuropsychological deficit in this population.

Executive Functions

Executive functions (EF) refer to higher ordered cognitive abilities such as judgment, decision making, social conduct, organizational skills, and planning (Delis, Kaplan, & Kramer, 2001a). Findings on EF deficits in EI patients have had mixed results. Some found that participants who suffer an EI have not been reported to have deficits in executive functions (Barrash, Kealey, & Janus, 1996; Pliskin et al., 2006). Despite attention and processing speed deficits, EI patients continue to perform within normal limits on the executive functioning tasks. Others have found deficits in EF post-EI (Duff & McCaffery, 2001).

Other Cognitive Functions

In addition to memory and executive function deficits, deficits in other cognitive functions have been observed. Deficits in attention and concentration after an EI have been reported (Crews et al., 1997). Pliskin and colleagues (2006) noted deficits on measures of attention and mental speed that were independent of secondary medical or
psychiatric complaints. Deficits in general intelligence have been reported in neuropsychological studies of EI patients (Martin et al., 2003; Pliskin et al., 2006). Impairments in language abilities post-EI have been reported (Hopewell, 1983). Aphasia is one of the less common neuropsychological complaints following electrical injury (Daniel, Haban, Hutcherson, Bolter, & Long, 1985). Pliskin and colleagues (2006) observed deficits on a measure of motor functioning (grooved pegboard) that were independent of secondary medical or psychiatric complaints.

Physical Symptoms

Electrical injury mechanisms can be direct or indirect and are influenced by field strength of the current, frequency, and duration of exposure. Individuals who survive an EI typically suffer severe thermal burns and may have damage to multiple organ systems (Barrash, Kealey, & Janus, 1996). When low-frequency electrical current passes through the chest, both cardiac and respiratory functions can be arrested (Lee, 1997). The individual may experience respiratory muscle spasm in response to tranthoracic currents (Dalziel & Lee, 1969; Sances, et al., 1979) and even a 20 milliamp will produce respiratory arrest (Dalziel & Lee, 1969). If the current possesses sufficient magnitude during the repolarization of cardiac cycle, atrial or ventricular fibrillation can occur (Lee, 1997).

There may be physical complaints following EI including skin injury, nerve injury, central neurologic effects, muscle injury, and cardiac injury. In electrical shock, there are always at least two points of skin contact. In ultrahigh voltage shocks (i.e., voltage ≤ 50 kV), several surface contact points are possible because current paths are established through multiple arcs (Lee, 1997). Electrical breakdown of the skin on
opposite sides of a joint is common and occurs most frequently around the underarm.

High-voltage electrical shock survivors frequently show physical signs of shock manifesting as a black metallic coating on the skin surface resulting from vaporization of the metal contacts (Lee, 1997). Clothing can become ignited if an arc initiates electrical contact. An individual can suffer burns and smoke inhalation. Fortunately, skin burns due to an arc are usually not that severe.

In addition to skin injury, an individual may experience nerve injury following an electrical shock. Peripheral nerves are sensitive to electric forces (Dalziel & Lee, 1969) and are commonly injured in victims of electric shock (Blom & Uglund, 1967; Grube & Heimbach, 1992). After minor electrical shock, temporary nerve dysfunction occurs, often persisting for only a few minutes or hours. The symptoms may include anesthesia, paresthesias, or dysesthesias (Grube & Heimbach, 1992). Anesthesia refers to the condition of having blocked sensation or sensation temporarily taken away. Paresthesia is a sensation of tingling, pricking, or numbness of a person’s skin with no long-term physical effect. Dysesthesia pertains to an unpleasant, abnormal sense of touch. Sometimes these symptoms are persistent, but in most cases the nerve injury is transient. Some case reports describe clinical impairment that worsens progressively (Kinnunen, Olaja, Taskinen, & Maitkenin, 1988).

Survivors of electrical injury frequently experience temporary autonomic nervous system disorders, more specifically, reflex sympathetic dystrophy (RSD) and hypertension (Cohen, 1995). The subsequent hypertension can be transient in nature and have a delayed onset. Following electrical trauma to the central nervous system, abnormalities in the regulation of cardiovascular function are observed. Research has
been unable to predict when this will occur and the extent of autonomic nervous system disorders varies. Also, cerebral anoxic injury can occur after cardiac and respiratory arrest. Cerebral injuries caused by falls may also take place in conjunction with electrical shock (Krob & Cram, 1983).

Immediately after the EI, there may be damage to skeletal muscle (Lee, 1997). The individual may show fixed, contracted extremity skeletal muscle in the rigor state. The loss of intracellular skeletal muscle can lead to concerns about the development of significant hyperkalemia, abnormally high levels of potassium in the blood (Lee, 1997).

In addition to damage to skeletal muscle, skeletal injury often occurs following an electrical injury. Common skeletal injuries include long bone fractures, joint dislocations, and cervical spinal fractures (Lee, 1997).

In addition to the previously mentioned physiological consequences to EI an individual may experience cardiac injury or central neurologic effects. An electrical current can be fatal through stimulation of lethal arrhythmias (Lee, 1997). Central neurologic effects most frequently entail spinal paralysis. Paralysis has been reported up to five years after shock without major intervening signs (Hooshmand, Radfar, & Beckner, 1989). Individuals who suffer an EI may subsequently endure a range of physical symptoms from skin injury to cardiac injury.

Emotional Symptoms

In addition to physical symptoms following EI, individuals may develop emotional symptoms and changes in personality. EI is associated with high psychiatric morbidity including major depressive disorder, anxiety disorders, and post-traumatic stress disorder (Duff & McCaffrey, 2001, Grossman et al., 1993; Kelley et al., 1994,
Changes in emotional symptoms have ranged from greater irritability to psychosis (Lee, 1997). Many EI survivors may receive initial medical treatment post-EI but be overlooked in terms of their psychiatric health. Survivors of EI are rarely referred for psychiatric evaluation until symptoms of serious psychiatric illness have developed (Kelley et al., 1999).

**Post-Traumatic Stress Disorder**

One of the most common psychiatric complaints following EI is the development of PTSD (Grossman & Tempereau, 1993; Kelley, Pliskin, Meyer, & Lee, 1994; Kelley et al., 1999; Mancusi-Ungaro, Tarbox, & Wainwright, 1986; Ramati et al., 2009). PTSD is characterized by the presence of symptoms that develop following exposure to an event that involves actual or threatened death or serious injury, or a threat of personal integrity to self or others. People differ in the degree of sensitivity to trauma, evidenced by the knowledge that not everyone who is exposed to a trauma develops PTSD. According to the Diagnostic and Statistical Manual-IV TR (American Psychiatric Association, 2000), symptoms of PTSD include persistent reexperiencing of the traumatic event, persistent avoidance of stimuli associated with the event, and persistent symptoms of increased arousal. The lifetime prevalence of PTSD is about 6% based on 34,653 adults in Wave 2 of the National Epidemiologic Survey on Alcohol and Related Conditions (Pietrzak et al., 2011).

The body manifests a hormonal response to stress in the environment in order to prepare the body. The hypothalamus responds to threat, simulating the pituitary gland, which subsequently releases adrenocorticotropic hormone (ACTH). The ACTH then travels through the bloodstream and stimulates the adrenal glands to release
catecholamines and cortisol, which energize the fight-or-flight response. The release of the catecholamines epinephrine and norepinephrine increase sympathetic nervous system activation. This increases heart rate, blood pressure, and respiration rate and decreases parasympathetic activation. The body has an emotional and physiological reaction to an emergency that increases readiness for action. Increased respiration and blood pressure makes more oxygen available to the muscles to invigorate an attack or to instigate escape. The adrenal glands also release cortisol, a hormone that increases the concentration of glucose in the blood to make fuel available to muscles.

Holbrook and colleagues (2002) examined rates of PTSD in men and women following traumatic events. PTSD was diagnosed in 35% of patients at a 6-month follow-up. Women were found to be at a higher risk for the development of PTSD after considering mechanism of injury and event-related factors. Pietrzak and colleagues (2011) also found that rates of PTSD were higher among women than men.

A few studies have examined predictors of PTSD in EI patients (Kelley, et al., 1999; Pliskin et al., 1998). Time since injury has been associated with the development of PTSD, such that patients seen beyond three months since the time of their injury present with more emotional complaints (Pliskin et al., 1998; Ramati et al., 2009). Litigation status was not associated with report of emotional symptoms (Pliskin et al., 1998). However, PTSD is associated with memory impairment (Charney et al., 1993; Vasterling et al., 1998) which makes it difficult to determine if memory impairment in EI patients was actually related to PTSD symptomatology or the EI itself.

In a retrospective study of 73 post-acute EI patients, injury parameters were examined as predictors in the development of PTSD. The injury parameters included: no-
let-go experience (involuntary muscle contraction response when sufficient current is present), being knocked away from the electrical source, loss of consciousness, and amnesia or altered states of consciousness. Patients who experienced a no-let-go phenomenon had an increased risk of PTSD compared to those who did not have such an experience. Patients who were knocked away from the electrical source did not have a statistically significant chance of developing PTSD compared to those who were not knocked away from the electrical source. Loss of consciousness as well as altered state of consciousness were associated with the development of PTSD. A subset of patients in their sample (n = 22) had some psychiatric history including substance abuse prior to EI, but the pre-injury status did not have a statistically significant association with PTSD development post-EI. In summary, greater time since injury, no-let-go phenomenon, and LOC or altered consciousness have all been associated with the development of PTSD post-EI.

Emotional Symptoms and Neuropsychological Performance

An EI can impact neuropsychological performance as well as emotional symptoms. However, individuals with an EI can experience neuropsychological deficits related to the emotional symptoms. Hooshimand and colleagues (1989) examined 14 patients who had severe anxiety and depression over a period of years following an electrical injury. Three quarters of the patients exhibited neuropsychological impairments on tests of immediate memory, concentration, judgment, and non-verbal achievement when compared with normative values.

Ammar and colleagues (2006) sought to examine the memory deficits seen in EI post-injury by comparing those with PTSD and those without PTSD. One hundred sixty-
five EI patients were administered the California Verbal Learning Test- Second Edition and psychiatric interviews determined PTSD status. EI patients with PTSD showed worse performance on tasks of immediate recall and delayed recall as compared to EI patients without PTSD. Also, EI patients with PTSD were more susceptible to retroactive interference. Therefore, the psychiatric status of the patient plays a role in regards to memory performance.

*Short-Term Effects*

The short-term effects following an EI persist for up to three months post-EI. The days between the time of injury and the neuropsychological testing has varied widely from study to study and even within studies. However, few studies have divided participants into groups based on time since injury. One study divided participants into groups based on the amount of time between injury and when they were examined (Barrush, Kealey, & Janus, 1996). The participants in the acute phase (less than 1 month since EI) exhibited mild impairments in immediate verbal learning in the context of otherwise intact cognitive abilities. They noted affective disturbances in half of the participants in the acute phase.

*Long-Term Effects*

In this study, the long-term effects following an EI pertain to symptoms that persist beyond three months after the injury. The research on the long-term effects of EI has several limitations. First, not all studies provide information on the time since the injury. Second, sometimes when the average length of time between injury and neuropsychological evaluation is provided, the range is so large (i.e., from immediately after injury to nearly 10 years) that the average does not provide useful information.
Third, studies that have included a long-term follow-up have utilized small sample sizes. Given the limitations of the research, the long-term outcomes of electrical injuries on cognitive, emotional, and behavioral functioning are unknown at this time.

Physical effects have been documented to continue for years past the time of the injury. Intolerance for cold may persist for 2-3 years (Lee, 1997). Some physical effects of electrical injury may not emerge for years after the injury. Neuromuscular problems, sensorimotor neuropathies, paresthesias, dysesthesias, and reflex sympathetic dystrophy may persist for years after the electrical injury (Lee, 1997). In 1-2% of victims of EI, cataracts have occurred (Lee, 1997). The various long-term effects of an EI can impact an individual’s daily functioning.

**Daily Living**

Individuals who experience an EI may experience disruptions in their daily living. Although the majority of people who experience an EI seem to recover, not all are able to return to work. Only 25-50% of electrical injury survivors are able to return to their previous employment while a third could not return to work at all (Noble, Gomez, & Fish, 2006; Therman et al., 2008). The ability to return to work can be impacted by neurological deficits, neuropsychological deficits, and psychological symptoms (Bryan et al., 2009). The exact contribution of each area in determining return to work is unknown. Hooshmand and colleagues (1989) followed EI patients for five to nine years. Patients reported severe depression (14/16 patients), job loss (11/13 patients), and divorce (9/11 patients).
Limitations of Electrical Injury Literature

There are several limitations to the previous work on neuropsychological and psychological associations with EI. As previously mentioned, time since injury has not always been provided and long-term studies have utilized small samples. Additionally, most research on electrical injury examined electricians who may not be generalizable to other populations. This study examined a clinical sample of electricians and non-electricians who have experienced an EI. Therefore, this study offers greater generalizability compared to previous research. Most studies describing neuropsychological deficits in EI participants did not control for the presence of mood disturbance in their samples.

Optimal Data Analysis

This statistical technique offers a method for maximizing predictive accuracy by using a multivariate classification tree (Yarnold & Soltysik, 2005). Other tree methodologies do not explicitly maximize classification accuracy as part of their computational algorithm. Independent of assumptions, ODA finds a decision rule for each predictor that maximizes the overall percent of classification accuracy for the sample. ODA offers advantages over other statistical techniques. For every problem analyzed with ODA there is one precise, optimal analysis (Yarnold & Soltysik, 2005). Also, every ODA analysis provides a goodness-of-fit index where 0 reflects the accuracy expected by chance for the sample, and 100 reflects perfect accuracy (Yarnold & Soltysik, 2005). No ODA analysis requires any simplifying assumptions, and $p$ is always valid and accurate (Yarnold & Soltysik, 2005). Other statistical techniques require assumptions to be met in order to interpret significant $p$ values. Specific hypothesis using
ODA will be discussed in further detail below. There have been no studies to date examining EI with ODA.

Summary and Current Study

Electrical injury represents a significant portion of trauma. EI can lead to a variety of cognitive impairments affecting attention, mental processing speed, motor skills, and memory. In addition to other psychiatric disorders, individuals who suffer an EI can eventually develop PTSD. These points emphasize the importance of this study.

Past research on EI has several limitations. Most research on electrical injury examined electricians, who may not be generalizable to other populations due to variation in exposure to minor electrical contact, and educational differences, among other differences. This study examined a clinical sample of electricians and non-electricians who have experienced an EI. Therefore, this study offers greater generalizability compared to previous research. By using a range of variables in the exploratory analysis, this study allowed for unique interactions to emerge while maximizing classification accuracy. Mounting evidence suggest that sequelae of EI are complicated to predict but are likely to occur in a significant minority of victims.

Hypotheses

Based on the previous literature with these clinical populations, a range of variables were hypothesized to predict outcome. However, it is important to note that the overwhelming majority of variables studied in the EI-PTSD literature have been main effects variables. The literature offers very little guidance on what will emerge from an exploratory statistical analysis designed specifically to reveal interactions—many ODA
studies unearth up to four or five total interactions. Therefore, the hypotheses below apply to the Univariate ODA analyses that were run and include a 2-way interaction.

The null hypothesis is that the class variable (e.g., PTSD) cannot be predicted as a linear cut-point on the continuous attribute (also referred to as an independent variable) and the alternate hypothesis is that the class variable can be predicted using this cut-point (Yarnold, 1996). Based on a review of the available literature and how the findings are best mapped onto neuropsychological, psychological, and demographic variables, the following factors are proposed to predict PTSD diagnosis outcomes for individuals who have suffered an EI:

1. Electrical Injury
   A. Main Effects
      i. Demographic Factors
         a. Longer time since injury will be associated with classification into the PTSD group.
         b. Greater loss of consciousness or altered state of consciousness will be associated with classification into the PTSD group.
         c. No-let-go experience will be associated with classification into the PTSD group.
      ii. Neuropsychological Variables
         a. Immediate and delayed verbal memory recall deficits are expected to be associated with classification into the PTSD group.
      iii. Psychological Variables
a. Higher scores on the BDI-II will be associated with the classification into the PTSD category.

B. Interaction Effects

i. There will be an interaction with gender such that females will be more likely to fall into the PTSD group than males, if they also have a longer time since injury.

Figure 1. An Example of Hypothesized Predictors of PTSD in Adults who have Suffered an Electrical Injury: Classification Tree Analysis via Optimal Data Analysis
CHAPTER TWO

METHODS

Participants

This study utilized archival data from 143 EI patients, derived from a convenience sample of treatment-seeking individuals who received comprehensive neurocognitive and psychiatric evaluations at an outpatient clinic in an urban, multicultural setting following EI. The original sample only included 38 patients but post-hoc power analyses demonstrated that a larger sample was needed to have adequate power to detect significant results. The details of the power analysis and sample size will be further explained in the results section. The EI group was further divided into EI participants who have PTSD (EI-PTSD: N = 63) and those without symptomatology (EI-No PTSD: N = 80) on the basis of a clinical interview, self-report measures of PTSD symptomatology, and clinical judgment. Clinical interviews were completed prior to further evaluation, and if trauma-related symptoms were suspected, patients completed self-report measures of trauma-related symptoms. The final diagnosis was determined by a Board-Certified Clinical Neuropsychologist based on clinical interviews and the patients’ responses on self-report measures of trauma-related symptoms. There was only one neuropsychologist determining the final diagnosis; therefore, inter-rater reliability for clinical interview and clinical judgment is not available. The source of electrical injury in this sample was limited to domestic and commercial power sources (i.e., patients with lightning injuries were excluded).
Procedure

Patients were administered a variety of neuropsychological tests that measure attention, verbal and non-verbal memory, visuospatial ability, motor ability, problem-solving, planning, and overall intellectual ability. They also completed a variety of psychological questionnaires assessing depressive symptoms, post-traumatic stress symptoms, and a general personality measure. Trained graduate neuropsychology externs and neuropsychology technicians tested participants using a semi-structured neuropsychological battery. Therefore, not all patients completed all of the measures used in this study. Release of information was obtained from the participant to collect relevant health professional data and review medical chart information.

Demographics and Injury Severity

Questionnaires were completed by individuals with the EI to obtain demographic information (e.g., age, gender, ethnicity/race, and socio-economic status). Medical chart reviews were utilized to assess the injury. Demographic information was gathered through significant others and family members. Efforts were made to gather information missing from the databases (e.g., injury characteristics) by examining medical records. Measures of PTSD were used in conjunction with clinical interview by a Board-Certified Clinical Neuropsychologist in determining the presence or absence of PTSD. In order to obtain board certification in neuropsychology, a licensed clinical psychologist must demonstrate knowledge of neuropsychology as well as psychopathology on a written and oral examination conducted by the American Board of Professional Psychology.

The following three measures assess PTSD symptomatology and were used in addition to a clinical interview in determining the presence of PTSD. None of the
participants completed all three measures and, in fact, only some of the participants completed one of the measures. The final diagnosis was made by a Board-Certified Clinical Neuropsychologist.

The Impact of Event Scale (IES)

This 20-item scale examines episodes of intrusion (9 items) and avoidance (11 items) (Horowitz et al., 1979). Items correspond directly to 14 of the 17 DSM-IV symptoms of PTSD. Respondents are asked to identify a specific stressful life event and then indicate how much they were distressed or bothered during the past seven days by each "difficulty" listed. Items are rated on a 5-point scale ranging from 0 ("not at all") to 4 ("extremely"). IES is a valid measure of post-traumatic stress symptoms but should not be used as a measure of PTSD. One reason is that the IES does not measure the hyperarousal symptoms included in the criteria for the diagnosis in the most recent version of the DSM.

Posttraumatic Diagnostic Scale (PDS)

The participants’ trauma-related symptoms were assessed using the PDS (Foa, 1996). The PDS is a 49-item self-report measure that proposes to assess severity of PTSD symptoms related to a single identified traumatic event. The PDS assesses all of the DSM-IV criteria for PTSD (i.e., Criteria A – F) and inquires about the past month (time frame can be adjusted for different uses). Thus, in addition to measuring the severity of PTSD symptoms (Criteria B, C, & D), it also inquires about the experience of a Criterion A traumatic events, about duration of symptoms (Criterion E), and the effects of symptoms on daily functioning (Criterion F).
The PDS has four sections. Part 1 is a trauma checklist. In Part 2, respondents are asked to describe their most upsetting traumatic event. Questions specifically ask about when the event happened, if anyone was injured, perceived life threat, and whether the event resulted in feelings of helplessness or terror. Part 3 assesses the 17 PTSD symptoms. Respondents are asked to rate the severity of the symptom from 0 ("not at all or only one time") to 3 ("5 or more times a week / almost always"). Part 4 assesses interference of the symptoms with the individual’s daily functioning.

A diagnosis of PTSD is made only when DSM IV criteria A to F are met. A categorical diagnosis of PTSD can be made with an algorithm that requires that the individual’s responses meet the following criteria: The traumatic event involves either injury or life threat; the person felt helpless or terrified during the event, endorsement (rating of 1 or higher) of at least one re-experiencing symptom, three avoidance symptoms, and two arousal symptoms; duration of at least one month; and impairment in at least one area of functioning. The PDS includes a symptoms severity score which ranges from 0 to 51 and this is obtained by adding up the individual's responses of selected items. The cut offs for symptom severity rating are 0 no rating, 1–10 mild, 11–20 moderate, 21–35 moderate to severe and >36 severe.

The PDS instrument was normed on 248 men and women between the ages of 18 and 65 who had experienced a traumatic event at least one month before they took the test. Individuals were recruited from women's shelters, PTSD treatment clinics, and Veterans Administration hospitals, in addition to staff of fire stations and ambulance workers.
The PDS has high face validity as items directly reflect the experience of PTSD with high internal consistency (coefficient alpha of 0.92). Test–retest reliability was also highly satisfactory for a diagnosis of PTSD over a 2- to 3-week period (kappa = 0.74). Test–retest using symptoms severity scores yielded a highly significant correlation (0.83). Analysis also revealed an 82% agreement between diagnosis using the PDS and the Structured Clinical Interview for DSM (Foa et al., 1997). The PDS does not incorporate any formal scales to detect faking or inconsistent responses.

_Trauma Symptom Inventory-Alternate (TSI-A)_

The TSI-A measure proposes to assess acute and chronic posttraumatic symptomatology, including the effects of rape, spouse abuse, physical assault, combat experiences, major accidents, and natural disasters, as well as the enduring effects of childhood abuse and other early traumatic events (Briere, 1995). The Alternate version of the TSI (the TSI-A) does not contain the Sexual Concerns or Dysfunctional Sexual behavior scales (only 5 sexual items remain), for use in circumstances where sexual item content is not desired. Each of the 86 symptom items is rated according to its frequency of occurrence over the prior six months, using a four point scale ranging from 0 ("never") to 3 ("often"). These items comprise 10 clinical scales: Anxious Arousal, Depression, Anger/Irritability, Intrusive Experiences, Defensive Avoidance, Dissociation, Impaired Self-Reference, and Tension Reduction Behavior.

In addition to the trauma-related measures that assisted in the diagnosis of PTSD, other measures were administered to gather neuropsychological and psychological information. Table 1 lists all of the measures that were included in this study.
Neuropsychological Measures

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

Memory was assessed using the California Verbal Learning Test- 2nd Edition (CVLT-II; Delis et al., 2000). This measure assesses the capacity to learn and retain a 16-item word list with five repeated exposure to the material (CVLT-II; Delis et al., 2000). Following the initial five repetitions of the word list, a second intervening word list is introduced to examine susceptibility to distraction and source memory confusion. Long and short delays assess free and cued recall ability. We utilized specific CVLT-II indices related to immediate (Short Delay Free Recall) and delayed (Long Delay Free Recall) recall and retention of information.

Test of Memory Malingering (TOMM)

The TOMM is a visual recognition test that was used to discriminate between malingered and true memory impairments (Tombaugh, 1996; Tombaugh, 1997). This measure assesses effort on memory tests and exaggeration or feigning of memory complaints in adults. The examinee is instructed that the examiner will assess their ability to remember 50 pictures of common objects and will then test how many of them the patient can remember. The measure was developed on a sample of 475 community-dwelling individuals ages 16-84 years, and on a sample of 161 patients referred for neuropsychological evaluations (Tombaugh, 1996).

Victoria Symptom Validity Test (VSVT)

The VSVT consists of a computer-administered and scored memory test designed to assess effort during test taking (Slick et. al., 1997). The measure uses a forced-choice
dichotomous memory test to assess potential exaggeration or feigning of cognitive impairment.

**Psychological Measure**

**Beck Depression Inventory- Second Edition (BDI-II)**

The BDI-II (Beck, Steer & Brown, 1996) assessed cognitive and behavioral features of depression. This 21-item self-report measure assesses the presence and severity of depressive symptoms reflecting areas such as worthlessness, concentration difficulty, hopelessness, and suicidal ideation. The total score is obtained by adding the responses from the 21 items. Each individual question is a list of four statements ranging from 0 to 3 with greater numbers representing a more pronounced depressive symptom, with a maximum score of 63. Individuals age 13-80 can be assessed using the BDI-II. Items on the BDI-II are intended to assess symptoms experienced in the two weeks prior to assessment.

The BDI-II was developed to have clinical sensitivity for assessing depression criteria reported in the DSM-IV. Psychometric characteristics of the BDI-II were established using four outpatient psychiatric samples and a college student sample. The BDI-II manual reported that the BDI-II demonstrated good internal consistency ($\alpha = .92$ for the outpatient samples and $\alpha = .93$ for the college student sample), test-retest stability ($\alpha = .93$ for a subset of outpatient samples) and good convergent and discriminant validity with respect to depression and anxiety respectively (Beck, Steer, & Brown, 1996). Cut score guidelines suggested in the manual are as follows: 0-13 (minimal range), 14-19 (mild range), 20-28 (moderate range), and 29-63 (severe range). However, the manual indicated that cut score thresholds may be raised or lowered to either reduce
or increase the number of false positives. For example, lowering the cut score will detect the maximum number of individuals presenting with depressive symptoms.

Table 1. Measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beck Depression Inventory- Second Edition*</td>
<td>Self Report</td>
</tr>
<tr>
<td>California Verbal Learning Test- Second Edition*</td>
<td>Objective</td>
</tr>
<tr>
<td>Immediate Recall</td>
<td></td>
</tr>
<tr>
<td>Delayed Recall</td>
<td></td>
</tr>
<tr>
<td>Impact of Event Scale</td>
<td>Self Report</td>
</tr>
<tr>
<td>Posttraumatic Diagnostic Scale</td>
<td>Self Report</td>
</tr>
<tr>
<td>Test of Memory Malingering</td>
<td>Objective</td>
</tr>
<tr>
<td>Trauma Symptom Inventory</td>
<td>Self Report</td>
</tr>
<tr>
<td>Victoria Symptom Validity Test</td>
<td>Objective</td>
</tr>
</tbody>
</table>

Note. * indicates measure used in statistical analyses.

Statistical Analyses

To create a prediction model for the development of PTSD in individuals who have suffered an EI, optimal data analysis (ODA; Soltysik & Yarnold, 1993; Yarnold & Soltysik, 2005) was conducted. This exploratory, non-parametric technique created a multivariate classification tree model for predicting PTSD symptoms. ODA finds a decision rule for each predictor (e.g., LOC) that maximizes the overall percentage of classification accuracy for the sample (Soltysik & Yarnold, 1993; Yarnold & Soltysik, 2005). Dissimilar to other statistical methods for constructing tree models (e.g., regression-based classification and regression tree or square based chi-squared automatic interaction director), ODA uses an exact permutation probability without basic distributional assumptions, evaluates the expected cross-sample generalizability of
classifications through a built-in jackknife resampling procedure, and finds main effects and non-linear interactions that optimally classify PTSD presentation.

Similar to other tests of interactions, ODA requires: (a) the distribution of scores on the independent variables have sufficient variability, (b) the range of values in the independent variables are not substantially restricted, (c) independent and dependent variables are measures with adequate reliability, (d) the dependent variable has sufficient numbers of observations to provide adequate statistical power, and (e) adequate total sample size to provide adequate statistical power to test for interactions. ODA offers a paramount statistical approach for incorporating various neuropsychological, psychological, and demographic variables in order to determine PTSD status among individuals with EI.

ODA’s method of statistical analysis was best suited for the current study. The approach to the testing of multivariate interactions used by ODA allows for numerous variables to be tested to fit into the optimal predictive model. Traditional analyses, such as ANOVA and regression, require the selection of specific predictors to be tested in a pre-described model. ODA permits the inclusion of many possible predictors without the specification of hypothesized interactions. Although some researchers argue that only those variables with supporting evidence in the literature should be included in the model of analysis, the techniques used by ODA are able to accommodate an unlimited number of variables without increasing the chance of a type I error (Yarnold & Soltysik, 2005). While this allows many variables to be examined, this makes ODA an exploratory technique. By not placing restrictions on those variables included in the model, ODA
allows variables not previously explored to be examined for involvement in PTSD outcomes for individuals that experience either an EI.

Additionally, ODA allows for the identification of subgroups of observations within the context of the model, rather than each variable needing to have a predictive effect for the entire group, as is the case in traditional models. For example, gender may moderate the effect of LOC on the development of PTSD post-EI. The methodology of ODA allows for the creation of a model that identifies the strongest predictors for each subgroup of the sample (Yarnold & Soltysik, 2005).

ODA techniques allow for the identification of both main effects and interactions. Main effects were tested using univariate ODA (UniODA; Yarnold & Soltysik, 2005). First, UniODA was performed for each predictor, revealing which variables significantly predict development of PTSD following EI and their effect size. After identifying those variables with a significant main effect, a Classification Tree Analysis (CTA) was created to provide information about other variables that interact with the variables with significant main effects in predicting PTSD symptomatology.

One classification tree model was created. The model examined PTSD outcomes in EI patients. The optimal predictor, the one variable with the greatest effect strength, was selected for the CTA model. ODA analyses provided a set of decision rules that divided the sample into subgroups. Once the sample was partitioned, ODA was again performed with all of the original variables, but this time only for those members of the particular subgroup. For example, if gender is determined to be the optimal predictor for development of PTSD for individuals who have suffered an EI, the second step in the CTA model selects one group, males or females, and determines the greatest main effect
for that subgroup, further dividing the original sample. This process continues, forming “branches” of the CTA, until the sample can no longer be subdivided (Yarnold & Soltysik, 2005). ODA was then conducted on each branch of the ODA tree until it could not be partitioned further. A minimum denominator of 13 was set as it represents roughly 10% of the sample. Significance was determined using the Sidak adjusted per-comparison $p$ values (Yarnold & Soltysik, 2004) for an experiment-wise alpha of 0.05. This procedure determines the adjusted Type I error rate according to the number of classifications conducted in the multivariate classification trees.

PTSD or no-PTSD was established as the class variable from which a decision-making “tree” was grown based on neuropsychological, psychological, and demographic factors. In creating the tree, two rules were utilized. First, we chose the attribute (and accompanying decision rule) with the strongest effect strength for sensitivity at each node in the classification tree model. Then, of those attributes, we chose those that provided the highest classification accuracy while remaining stable when subjected to a leave-one-out (LOO) jack-knife validity analysis (Lachenbruch, 1967; Yarnold & Soltysik, 2005). This jackknife analysis is a resampling procedure that leaves one observation out a time and reruns the analysis to determine that the model remains the same. The LOO analysis corrected for the possibility that the obtained results are sample-specific.

After the initial tree was constructed, two rules were used to prune the tree. First, we determined the statistical significance of each attribute in the final model by performing a non-directional Fisher’s exact probability test, with alpha = .05. Last, we used a sequentially rejective Bonferroni procedure to additionally prune the tree, to ensure an experiment-wise Type I error rate of $p< .05$. This Bonferroni adjustment
procedure corrected the generalized p-value for the number of predictors in the initial tree model.
CHAPTER THREE

RESULTS

A post-hoc power analysis was conducted for the CTA in predicting PTSD symptoms for the original sample of 38 participants. Because hypotheses and corresponding statistical analyses vary across multiple outcome measures and multiple types of measurement scales, statistical power was computed in assessing the overall classification accuracy of a hypothetical two-attribute tree model ($N = 38$) in predicting the presence (1) versus absence (0) of PTSD symptoms. The data revealed a distributional split of 47.37% ($n = 18$) reporting symptoms of PTSD and 52.63% ($n = 20$) not reporting symptoms of PTSD.

For the power analysis, an assumption was made that the overall model produced a total of 12 correctly classified cases (67% classification accuracy) in predicting the presence of PTSD symptoms, and 13 correctly classified cases (65% classification accuracy) in predicting the absence of PTSD symptoms. This assumed level of overall classification accuracy represents a Cohen’s $W$ effect size of .32, which is a medium-sized effect (Cohen, 1988). The power estimation of the two-predictor CTA model used a non-directional Fisher’s exact test with $p < .025$ to reflect Bonferroni criteria for experiment-wise statistical significance. This power analysis indicated that the original sample size of 38 provided only 35% power to classify participants accurately in the two-attribute CTA model.
Given 65% classification accuracy in predicting the absence of PTSD symptoms, the present sample size provided adequate (i.e., 80%; Cohen, 1988) power to detect at $p < .025$ classification accuracy for the presence of PTSD that is at least 83.29% (i.e., 15 of 18 actual observations, or Cohen’s effect size $W = 0.49$, which represents a “large” effect). Given the assumed “medium” ($W = .31$) overall effect size for the CTA model and assuming the same observed 47.37% base-rate of PTSD symptoms, current sample sizes would need to be increased to at least 48 respondents who reported the absence of PTSD and 43 respondents who reported the presence of PTSD, in order for the two-attribute CTA model to achieve 80% power. Variables previously used as exclusion criteria (symptom validity testing, previous EI, previous TBI, TBI secondary to EI, litigation, and evaluation number) were subsequently included in the analyses. Loosening the exclusion criteria allowed for these variables to be examined in the ODA analyses as predictors of PTSD. The final sample size, including the participants that had been excluded based on the original exclusion criteria, was 143 participants.

Descriptive Analyses

A summary of demographic characteristics can be found in Table 2 and the demographic breakdown by PTSD status is in Table 3. The sample of 143 patients was predominantly male (86.0%) and the average age was 39.4 years (S.D. = 10.0). Of this sample, 85.3% were European American, 7.7% were African-American, 3.5% were Hispanic, and 2.1% were another race. In regard to furthest educational attainment, 4.2% had fewer than nine years of education, 15.8% had some high school, 49.7% had a high school degree, 23.8% had some college, 4.2% had a college degree, and 2.8% had some education beyond college. Previous electrical injuries were reported by 6.3% with the
majority 85.3% denying previous electrical injury. Previous TBI was reported by 14.7% of participants with 74.1% denying a previous TBI. Only 28 participants engaged in symptom validity testing and 27 passed and one failed. At the time of data collection, 60.1% were involved in EI-related litigation and 31.5% denied involvement in EI-related litigation. For the majority of patients, the testing was their first neuropsychological evaluation (88.8%), and 9.1% it was their second evaluation, and 1.4% it was their third evaluation.

Multiple injury parameters were collected. The mean months since injury was 25.0 (s.d. =29.3). The mean loss of consciousness was 8.3 minutes (s.d. = 7.8). Twenty-one percent reported a no-let-go experience and 4.3% denied a no-let-go experience. Only 5.6% identified suffering a TBI secondary to the EI and 74.1% did not. Examples of the causes of electrical injury included domestic and commercial power sources.

In terms of emotional functioning, of the 143 participants in the study, 44.1% were diagnosed with PTSD and 55.9% were not diagnosed with PTSD. Mean depressive symptoms was 16.2 (s.d. = 10.3).

Table 4 shows a summary of the neuropsychological data across the group of patients with PTSD and the group of patients with no PTSD diagnosis. Of the 128 patients with immediate verbal recall data, the mean T-score was 41.6 (s.d. =14.2), a score in the low average range. For short delay verbal recall, there was data on 130 patients, the mean performance was $z = -0.7$ (s.d. = 1.4), a score in the low average range.
Table 2. Sample Demographics (N = 143)

<table>
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<th>Characteristics</th>
<th>N</th>
<th>%</th>
<th>M (S.D.)</th>
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</thead>
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</tr>
<tr>
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<tr>
<td>Female</td>
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<td>63</td>
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<tr>
<td>Loss of Consciousness</td>
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<tr>
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<tr>
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<td>9</td>
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<tr>
<td>No</td>
<td>122</td>
<td>85.3</td>
<td></td>
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<tr>
<td>Previous Head Injury</td>
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<tr>
<td>Yes</td>
<td>21</td>
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<td>74.1</td>
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<tr>
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<td>143</td>
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<tr>
<td>Pass</td>
<td>27</td>
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<td>Fail</td>
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<tr>
<td>Depressive Symptoms</td>
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<td>TBI Secondary to EI</td>
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<td>Yes</td>
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<td>Litigation</td>
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<td>127</td>
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</tr>
<tr>
<td>Race</td>
<td>Count</td>
<td>Percentage</td>
<td></td>
</tr>
<tr>
<td>--------------</td>
<td>-------</td>
<td>------------</td>
<td></td>
</tr>
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<td>Second</td>
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<td>9.1</td>
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</tr>
<tr>
<td>Third</td>
<td>2</td>
<td>1.4</td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>141</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>122</td>
<td>85.3</td>
<td></td>
</tr>
<tr>
<td>African-American</td>
<td>11</td>
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</tr>
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<td>Hispanic</td>
<td>5</td>
<td>3.5</td>
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</tr>
<tr>
<td>Other</td>
<td>3</td>
<td>2.1</td>
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Table 3. Means and Standard Deviations for Overall Sample (N = 143), Adults with Electrical Injury and PTSD (N = 63) and Adults with Electrical Injury and no PTSD (N = 80)

<table>
<thead>
<tr>
<th>Variables</th>
<th>PTSD</th>
<th>No-PTSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>93.7%</td>
<td>80.0%</td>
</tr>
<tr>
<td>Female</td>
<td>6.3%</td>
<td>20.0%</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>87.3%</td>
<td>83.8%</td>
</tr>
<tr>
<td>African-American</td>
<td>7.9%</td>
<td>7.5%</td>
</tr>
<tr>
<td>Hispanic</td>
<td>3.2%</td>
<td>3.8%</td>
</tr>
<tr>
<td>Other</td>
<td>1.6%</td>
<td>2.5%</td>
</tr>
<tr>
<td>Age</td>
<td>40.6 (9.5)</td>
<td>38.4 (10.2)</td>
</tr>
<tr>
<td>Months Since Injury</td>
<td>31.2 (36.4)</td>
<td>20.1 (21.0)</td>
</tr>
<tr>
<td>Previous Head Injury</td>
<td></td>
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<tr>
<td>Yes</td>
<td>11.1%</td>
<td>17.5%</td>
</tr>
<tr>
<td>No</td>
<td>77.8%</td>
<td>71.3%</td>
</tr>
<tr>
<td>Previous Electrical Injury</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1.6%</td>
<td>10.0%</td>
</tr>
<tr>
<td>No</td>
<td>90.5%</td>
<td>81.3%</td>
</tr>
<tr>
<td>Litigation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>68.3%</td>
<td>53.8%</td>
</tr>
<tr>
<td>No</td>
<td>27.0%</td>
<td>35.0%</td>
</tr>
<tr>
<td>TBI Secondary to EI</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3.2%</td>
<td>7.5%</td>
</tr>
<tr>
<td>No</td>
<td>73.0%</td>
<td>75.0%</td>
</tr>
<tr>
<td>Years of Education</td>
<td>12.2 (1.9)</td>
<td>12.5 (2.2)</td>
</tr>
<tr>
<td>Less Than High School</td>
<td>3.2%</td>
<td>5.0%</td>
</tr>
<tr>
<td>Some High School</td>
<td>22.2%</td>
<td>10.0%</td>
</tr>
<tr>
<td>High School</td>
<td>46.0%</td>
<td>52.5%</td>
</tr>
<tr>
<td>Some College</td>
<td>25.4%</td>
<td>22.5%</td>
</tr>
<tr>
<td>College Degree</td>
<td>1.6%</td>
<td>6.3%</td>
</tr>
<tr>
<td>Post-College</td>
<td>1.6%</td>
<td>3.8%</td>
</tr>
<tr>
<td>Loss of Consciousness</td>
<td>8.3 (4.1)</td>
<td>8.3(9.8)</td>
</tr>
<tr>
<td>No-Let-Go Experience</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>20.6%</td>
<td>21.3%</td>
</tr>
<tr>
<td>No</td>
<td>39.7%</td>
<td>55.0%</td>
</tr>
<tr>
<td>Evaluation Number</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First</td>
<td>85.7%</td>
<td>91.3%</td>
</tr>
<tr>
<td>Second</td>
<td>11.1%</td>
<td>7.5%</td>
</tr>
<tr>
<td>Third</td>
<td>3.2%</td>
<td>0.0%</td>
</tr>
<tr>
<td>Symptom Validity Testing</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------------------------</td>
<td>--------</td>
<td>--------</td>
</tr>
<tr>
<td>Pass</td>
<td>23.8%</td>
<td>0.0%</td>
</tr>
<tr>
<td>Fail</td>
<td>1.6%</td>
<td>15.0%</td>
</tr>
<tr>
<td>Immediate Verbal Memory (T)</td>
<td>41.0 (14.6)</td>
<td>42.4 (14.0)</td>
</tr>
<tr>
<td>Delayed Verbal Memory (Z)</td>
<td>-0.8 (1.4)</td>
<td>-0.7 (1.4)</td>
</tr>
<tr>
<td>Depressive Symptoms</td>
<td>20.0 (10.7)</td>
<td>13.3 (9.1)</td>
</tr>
</tbody>
</table>

*Note.* Dichotomous variables are represented as percentages. Interval variables are means followed by standard deviations in parentheses.
Table 4. Neuropsychological Variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>N</th>
<th>M (S.D.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediate Verbal Recall (T)</td>
<td>128</td>
<td>41.6 (14.2)</td>
</tr>
<tr>
<td>Short Delay Verbal Recall (z)</td>
<td>130</td>
<td>-0.7 (1.4)</td>
</tr>
</tbody>
</table>
Table 5. Univariate Associations of Theoretical and Demographic Attributes with PTSD 1) Versus No PTSD (0) for the Total Sample (N=143)

<table>
<thead>
<tr>
<th>Attribute</th>
<th>ODA Model</th>
<th>n</th>
<th>% PTSD</th>
<th>ESS</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>Female, predict 0</td>
<td>20</td>
<td>20.0</td>
<td>13.90*</td>
<td>0.025</td>
</tr>
<tr>
<td></td>
<td>Male, predict 1</td>
<td>123</td>
<td>48.36</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Months Since Injury</td>
<td>≤24.04, predict 0</td>
<td>77</td>
<td>36.36</td>
<td>19.67</td>
<td>0.120</td>
</tr>
<tr>
<td></td>
<td>&gt;24.04, predict 1</td>
<td>55</td>
<td>56.36</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous EI</td>
<td>No Previous EI, predict 0</td>
<td>9</td>
<td>11.11</td>
<td>9.39</td>
<td>0.044</td>
</tr>
<tr>
<td></td>
<td>Previous EI, predict 1</td>
<td>121</td>
<td>47.11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loss of Consciousness</td>
<td>≤4.0, predict 0</td>
<td>88</td>
<td>37.50</td>
<td>8.60</td>
<td>0.395</td>
</tr>
<tr>
<td></td>
<td>&gt;4.0, predict 1</td>
<td>10</td>
<td>60.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Previous TBI</td>
<td>No Previous TBI, predict 0</td>
<td>21</td>
<td>33.33</td>
<td>7.50</td>
<td>0.339</td>
</tr>
<tr>
<td></td>
<td>Previous TBI, predict 1</td>
<td>105</td>
<td>46.67</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Symptom Validity Testing</td>
<td>Pass, predict 0</td>
<td>28</td>
<td>53.57</td>
<td>6.25</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>Fail, predict 1</td>
<td>1</td>
<td>100.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressive Symptoms</td>
<td>≤11.5, predict 0</td>
<td>48</td>
<td>22.92</td>
<td>30.37*</td>
<td>0.002</td>
</tr>
<tr>
<td></td>
<td>&gt;11.5, predict 1</td>
<td>85</td>
<td>55.29</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No-let-go experience</td>
<td>No experience, predict 0</td>
<td>68</td>
<td>36.76</td>
<td>5.88</td>
<td>0.656</td>
</tr>
<tr>
<td></td>
<td>Experience, predict 1</td>
<td>30</td>
<td>43.33</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TBI secondary to EI</td>
<td>No TBI, predict 0</td>
<td>8</td>
<td>25.00</td>
<td>5.06</td>
<td>0.470</td>
</tr>
<tr>
<td></td>
<td>TBI, predict 1</td>
<td>105</td>
<td>43.81</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Litigation</td>
<td>No Litigation, predict 0</td>
<td>45</td>
<td>37.78</td>
<td>11.10</td>
<td>0.198</td>
</tr>
<tr>
<td></td>
<td>Litigation, predict 1</td>
<td>86</td>
<td>50.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Evaluation Number</td>
<td>First Evaluation, predict 0</td>
<td>126</td>
<td>42.86</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Second Evaluation, predict 1</td>
<td>15</td>
<td>60.00</td>
<td>6.59</td>
<td>0.288</td>
</tr>
<tr>
<td></td>
<td>Third Evaluation, predict 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Immediate Memory</td>
<td>≤38.5, predict 0</td>
<td>75</td>
<td>40.00</td>
<td>9.81</td>
<td>0.814</td>
</tr>
<tr>
<td></td>
<td>&gt;38.5, predict 1</td>
<td>52</td>
<td>50.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Delayed Memory</td>
<td>≤-0.25, predict 0</td>
<td>55</td>
<td>40.00</td>
<td>7.88</td>
<td>0.821</td>
</tr>
<tr>
<td></td>
<td>&gt;-0.25, predict 1</td>
<td>73</td>
<td>47.95</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: “ODA Model” indicates the cutoff point or decision rule by which ODA classified PTSD. Total sample sizes varied across attributes due to incomplete data. A sequentially-rejective Bonferroni adjustment was not employed for univariate analyses. Effect Size Strength (ESS) values followed by * were stable in jackknife (“leave-one-out) validity analysis, and are expected to show cross-sample generalizability.
Univariate Analyses

To describe simple relationships between PTSD and each attribute, we first conducted univariate analyses using ODA (Table 5). Consistent with previous findings, most demographic attributes were significantly related to PTSD, PTSD was significantly associated with sex, previous electrical injury, and depressive symptoms. Men were more likely to develop PTSD than women. Individuals who previously experienced an electrical injury were more likely to develop PTSD than those who did not experience a previous electrical injury. Individuals with greater depressive symptoms were more likely to develop PTSD than those with lower levels of depressive symptoms.

However, contrary to previous theory and research, attributes unrelated to PTSD included months since injury, loss of consciousness, previous traumatic brain injury, symptom validity testing, no-let-go experience, TBI secondary to EI, litigation, immediate verbal memory, delayed verbal memory, or evaluation number.

Classification Tree Analysis

Our primary interest was not to see simple relationships between each attribute and PTSD, but to see how multiple attributes combine to explain predictive profiles of individuals with and without PTSD following an electrical injury. Therefore, we used ODA to construct a hierarchically optimal CTA model. Following established procedures for constructing optimal CTA models, after applying a sequentially rejective Sidak-Bonferroni-type multiple comparisons procedure, only one node remained. The node was depressive symptoms measured by continuous scales ($p = 0.002$). Of the 143 participants in the study, 133 had completed the measure of depressive symptoms. Therefore, the model did not classify the 10 people that did not complete the measure of depressive
symptoms. The attribute of depressive symptoms was significant in the univariate analyses. Figure 2 shows the final hierarchically optimal CTA model for explaining PTSD in individuals who experienced an EI. In the figure, circles represent nodes, arrows indicate branches, and rectangles are prediction end-points. Numbers below each node indicate directional Fisher’s exact $p$ value for the node, and numbers in parentheses within each node indicate ESS for the node. Also, values next to each arrow indicate the value of the cutpoint for the node. The strongest predictor of PTSD for the total sample was depressive symptoms (ESS = 30.37%): the only node of the CTA model. The cutpoint for this attribute was 11.5.

Figure 2. CTA model for predicting PTSD versus no PTSD ($N=133$)

Circles represent nodes, arrows represent branches, and rectangles represent prediction endpoints. Numbers under each node indicate the exact $p$ value for each node. Numbers in parentheses within each circle indicate effect strength. Numbers beside arrows indicate the cutpoint for classifying observations into categories (PTSD versus no...
PTSD) for each node. Fractions below each prediction endpoint indicate the number of correct classifications at the endpoint (numerator) and the total number of observations classified as the endpoint (denominator).

Table 6 summarizes the overall classification performance of the CTA model, which correctly classified 84 (63.16%) of the total 133 adults who experienced an electrical injury and had completed the BDI-II. The ESS for this model was 30.37%, indicating that the model attained almost one third of the theoretically possible improvement in classification accuracy versus the performance expected by chance: this is considered to reflect a moderate effect.
Table 6. Predicted and Actual Class Status for CTA PTSD Model

<table>
<thead>
<tr>
<th>Actual Class Status</th>
<th>Predicted Class Status</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>No PTSD</td>
<td>No PTSD</td>
<td>37</td>
</tr>
<tr>
<td>PTSD</td>
<td>PTSD</td>
<td>38</td>
</tr>
</tbody>
</table>

Negative Predictive Value = 77.08%
Positive Predictive Value = 55.29%

*Additional Comments about Cutpoints*

Although the cutpoint for depressive symptoms was 11.5, what does this value indicate? Scores less than or equal to 11.5 were located within 18.25% on the absolute possible range, and the scores less than or equal to 11.5 reflects 18.25% of the absolute possible range in the amount of depressive symptoms. Descriptive statistics showed that the mean of depressive symptoms (range=0-46) was 16.2 with $SD=10.3$. Overall, 27.8% of respondents scored 11.5 or less, while 72.2% scored more than 11.5.
CHAPTER FOUR
DISCUSSION

The purpose of this study was to determine factors associated with the development of PTSD after an electrical injury. We predicted that longer time since injury, greater loss of consciousness, no-let-go experience, immediate and delayed verbal memory recall deficits and greater depressive symptoms would be associated with the classification into the PTSD group. However, in our study, only depressive symptoms predicted classification into the PTSD group. We analyzed the predictors of PTSD classification using classification tree analysis via Optimal Data Analysis. After applying a sequentially rejective Sidak-Bonferroni-type multiple comparisons procedure, only one node remained-- depressive symptoms. The strongest predictor of PTSD for the total sample was depressive symptoms and the cutpoint for this attribute was 11.5. Individuals scoring less than or equal to 11.5 on the Beck Depression Inventory-Second Edition were correctly classified as not having PTSD about 77% of the time. Individuals scoring greater than 11.5 on the Beck Depression Inventory were correctly classified as having PTSD about 55% of the time. The following section will discuss the outcomes of each of the hypotheses we proposed to predict PTSD diagnosis outcomes for individuals who suffered an EI.

Hypotheses on Demographic Factors

With regard to injury parameter variables, longer time since injury, greater loss of consciousness, and no-let-go experience were hypothesized to be associated with
classification into the PTSD group. However, the current study did not support these hypotheses. Although other studies documented a relationship between time since injury and the development of PTSD, such that patients seen more than three months after injury had higher rates of PTSD (Pliskin et al., 1998; Ramati et al., 2009), we did not replicate those findings. Such discrepancies across studies may have been due to how the samples were recruited. For example, some past studies collected random samples of trauma victims which would be expected to yield a lower rate of PTSD than was found in the current study, based on the work of Holbrook and colleagues (2002) who diagnosed PTSD in 35% of patients at a 6-month follow-up. The current study included patients that self-selected for a neuropsychological evaluation because they were often times experiencing mood related symptoms and about 44% of patients in this study carried a PTSD diagnosis (which is greater than 35%).

Although we hypothesized that greater loss of consciousness would predict PTSD, our analyses did not support this hypothesis. One possible explanation is that while we associate greater loss of consciousness with a more traumatic event there may be a counteracting force. Greater loss of consciousness could also have a direct relationship with post-traumatic amnesia, a symptom that is believed to reduce the prevalence of PTSD in patients that experience it. Thus, greater loss of consciousness as a single construct might not have a causal relationship with PTSD and studies that control for symptoms of post-traumatic amnesia might be more likely to find a significant result.

Prior electrical injury research found a relationship between no-let-go experience and the development of PTSD. Our hypothesis was that patients who experience no-let-go during an EI would have a more vivid memory of the trauma, thus making them more
likely to experience PTSD symptoms. On the other hand, a variety of environmental factors contribute to the intensity of a person’s trauma memories. No-let-go is one such factor but there are also likely to be others such as the magnitude/length of pain sensations, as well as other sensory experiences (i.e., smell). Collecting accurate data on these other factors can be challenging; however, it would be interesting to control for all other factors and isolate the effect of no-let-go.

Also, based on the literature, we hypothesized that litigation status would not be associated with PTSD post-EI. Congruent with a previous study (Pliskin et al., 1998), litigation status was not associated with PTSD in the current study. Litigation aims to compensate the victim for their medical expenses, loss of wages, as well as emotional suffering. For electrical injury victims that are seeking treatment, we did not expect that the status of their litigation would be strong enough on its own to increase the likelihood of PTSD.

Hypotheses on Neuropsychological Factors

With regard to neuropsychological variables, immediate and delayed verbal memory recall deficits were expected to be associated with classification into the PTSD group. The analyses did not support this hypothesis. A plethora of factors may complicate an individual’s cognitive presentation, including motivation, stress, effort, pain, pain medications, emotional state, and personality traits. This study examined effort and emotional state in conjunction with neuropsychological factors, but other factors may also play a role in cognitive presentation. Therefore, an association between cognitive variables and PTSD may be reported in one study but not another.
Hypothesis on Interaction Effects of Gender and Time Since Injury

We hypothesized an interaction with gender such that females would be more likely to fall into the PTSD group than males, if they also had a longer time since injury. Our results did not support this hypothesis. One reason for this finding may be that there were only 20 females in this study and only four women had a diagnosis of PTSD. With a larger sample, the interaction between gender and time since injury may have yielded a different result. Another reason for the lack of a significant effect may be differences in symptom manifestation. The majority of EI research examined predominantly male samples, an indication of employee demographics in professions where workers are at risk to experience an electrical injury. In one sample of 34 females and 59 males who experienced low voltage injury, males more commonly reported unexplained moodiness, short-term memory loss, and dizziness, while females more often reported a chronic pain syndrome (Morse & Morse, 2005). An individual’s subjective pain experience was not a variable examined in this study.

Hypothesis on Psychological Factors

In terms of psychological variables, higher scores on a measure of depression were hypothesized to be associated with classification into the PTSD group. The current study supported this hypothesis. About 30-50% of patients with PTSD have comorbid depression (Blanchard & Hickling, 1996; Blanchard et al., 1998; Boudreaux et al., 1998; Nixon, Risick, & Nishith, 2004; Ginzburg et al., 2009; McLean et al., 2011; Pietrzak, et al., 2011). Breslau and colleagues (1997) noted that 83% of the women in their study with PTSD met criteria for at least one other psychiatric disorder, compared with only 44% of those without PTSD. While PTSD frequently occurs simultaneously with other
psychiatric disorders, depressive disorders appear most often with PTSD (Brady et al., 2000).

Several causal pathways have been theorized to explain the association between PTSD and depression following exposure to a traumatic event. Evidence suggests that the presence of PTSD may increase the risk for first onset of major depression (Breslau et al., 1997; Kessler et al., 1995) and, conversely, preexisting major depression heightens one’s risk for developing PTSD post-trauma (Breslau et al., 1997; Bromet, Sonnega & Kessler, 1998). Given that PTSD and depression increase susceptibility for each other, O’Donnell, Creamer, & Pattison (2004) suggested a shared vulnerability for both disorders. Shared risk factors such as childhood abuse, event severity, and female gender support their hypothesis. They concluded that depressive symptoms are often integral to PTSD and attempting to distinguish depression as a separate disorder when comorbid with PTSD is challenging and perhaps impossible.

While the study above examined PTSD and depression following trauma in general, a recent study examined predictors of psychiatric symptoms, including PTSD following a specific trauma- electrical injury. Shin and colleagues (2010) reviewed 709 electrically injured patients’ medical records to examine risk factors for psychiatric complications. Patients were admitted to Hanil General Hospital in South Korea from 2002-2007. The DSM-IV criteria were used to diagnose depression, acute stress disorder, and PTSD. Incidence of psychiatric complications was 27.5% in total (depression: 15.8%, acute stress disorder or PTSD: 17.6%). Factors associated with increased psychiatric morbidity included high voltage injury, burns, and amputations. Patients with high voltage injuries had psychiatric complications 2.38 times higher than low voltage
injured patients. The total burn surface area was computed for each individual and classified into the following categories: 0-5%, 6-10%, 11-20%, and 21-30%. The total burn surface area was associated with the incidence of psychiatric complications; 1.83 times in 6-10% of burn surface area, 2.01 times in 11-20%, and 2.41 times in 21-30% higher than in 0-5% of burn surface area. The psychiatric morbidities occurred 1.96 times more when the site of the burn included their face. Amputations were also a risk factor for the development of psychiatric symptoms with minor amputation demonstrating 2.39 times incidence and major amputation demonstrating 7.70 times incidence for depression, acute stress disorder, or PTSD morbidities. They recommended that earlier psychiatric consultation may help manage psychiatric complications of electrical injury in patients with the aforementioned risk factors. The current study did not examine voltage, burns, or amputations as risk factors for the development of PTSD following an electrical injury.

Limitations of the Study

There are several limitations to this study that need to be acknowledged. The current study relied on self-report for some measures. As such, the associations reported among self-report measures may be inflated due to common method variance particularly for relations between depressive symptoms and PTSD. Univariate analyses showed that PTSD was significantly associated with depressive symptoms and individuals with greater depressive symptoms were more likely to develop PTSD than those with lower levels of depressive symptoms. Information on depressive symptoms was gathered subjectively on a self-report measure and PTSD symptoms were examined with self-report measures of PTSD as well as a clinical interview. Other variables examined in this study were objectively measured by the patient’s performance (immediate and delayed
memory performance) or were collected based on their medical chart and objective self-report (i.e., months since injury, loss of consciousness, experience of traumatic brain injury). Excluding depressive and trauma-related symptoms, the information collected based on the patient’s self-report was objective.

Another limitation is the lack of a non-electrically injured comparison group which limits our ability to attribute the depressive symptoms to the classification into the PTSD group. EI symptom patterns have been compared to patterns observed in diffuse cerebral injury, damage to the limbic system of hypothalamic-pituitary axis, and in electroconvulsive therapy (Pliskin et al., 1998; Pliskin et al., 1999). Perhaps a comparison group comprised of individuals with the aforementioned experiences would elucidate some of the unique experiences of electrical injury.

Another limitation is the lack of diagnostic inter-rater reliability. Only one neuropsychologist diagnosed PTSD; thus, inter-rater reliability could not be examined. An additional limitation is the homogeneity of the sample (78.9% Caucasian and 86.0% male). This sample is representative of individuals who experience an electrical injury, although it does not generalize to the United States population. Also, a limitation of the current study is the possibility of a non-response bias, as each variable was not available for all 143 participants. The lack of longitudinal data limits our ability to determine causality. The final limitation is that the use of self-report items to assess depressive symptoms and injury parameters may not have accurately reflected actual levels because of social desirability, memory limitations, and motivation to recall.
Directions for Future Research

Future research should explore the nature of the EI in more detail (e.g., voltage, burns, or amputations as risk factors for the development of PTSD) and the etiology (i.e., shared vulnerability) of psychological sequelae post-EI. The percentage of one’s body burned in an electrical injury was shown to be related to psychiatric morbidity (Shin et al., 2010); however, it was not assessed in the current study. Additionally, the voltage of the electric shock and amputation status are also important factors to explore related to the development of PTSD post-electrical injury. This study used depressive symptoms endorsed at the same time point as trauma-related symptoms were assessed. Examining mental health problems prior to the injury may also elucidate the subset of patients who experience an electrical injury who eventually develop PTSD. Ramati and colleagues (2009) revealed that in a large group of EI patients studied through an electrical trauma program, 76.0% had been diagnosed with a psychiatric condition, though merely 10.0% of EI victims had any history of mental health problems prior to their injury. Psychological symptoms can be examined prospectively by examining data collected on individuals prior to an electrical injury. In terms of shared vulnerability, the work of Yehuda and colleagues (2010) examined the impact of early life stress as a risk factor for the development of PTSD, as well as depression, suggesting a genetic predisposition in certain individuals.

Conclusion and Implications

This study demonstrated that depressive symptoms were associated with PTSD symptoms post-EI. Health care providers can improve the care provided to their patients who experience an electrical injury by being aware of the development of trauma-related
symptoms and making appropriate referrals to mental health providers. This research is relevant for those in health care, but the implications are widespread. PTSD as well as other mental health disorders have a tremendous financial impact. For example, exposure to traumatic stress is linked to reduced labor market outcomes based on a review of the labor force participation research for individuals who have experienced a traumatic exposure (Fairbank, Ebert, & Zarkin, 1999). Although specific to military personnel, the 2008 report from the Rand Corporation put the economic impact of PTSD including medical care, loss of productivity, and suicides at $4 to $6 billion over a two year time period. While this number reflects a larger group of individuals with PTSD, it still demonstrates the profound economic burden of PTSD. The experience of an electrical injury and the subsequent development of PTSD has a profound effect on the individual as well as society. Based on the findings of the current study, depressive symptoms should be evaluated in clinical settings to determine which individuals are more likely to develop PTSD post-EI.
REFERENCES


VITA

Jana Wingo was raised in Rolling Meadows, Illinois. Before attending Loyola University Chicago, she attended the University of Notre Dame, where she earned a Bachelor of Arts in Psychology, graduating magna cum laude in 2007. She earned her Master’s degree in Psychology in May 2009 from Loyola University Chicago.

As a student at Loyola, Jana has served on the Information Committee and as the Vice-President and President of the Clinical Student’s Association. Currently, Jana is working as a graduate student intern in the Department of Psychiatry and Behavioral Science at the University of Chicago. She lives in Mt. Prospect, IL with her husband.