#CLINICAL CORNER:

A Case of Electrical Injury:
Neuropsychological and Functional Imaging Considerations

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Introduction

The evaluation and treatment of individuals who are survivors of trauma is an important component of the clinical practice of many neuropsychologists, with traumatic brain injury (TBI) being the most common form of trauma. Yet, a growing number of health care providers are being asked to evaluate and treat patients who have sustained trauma secondary to electrical shock injury (EI). Indeed, EI accounts for about 5000 injuries annually in the US, accounts for 5% of all occupational fatalities and is the second leading cause of fatality in the construction industry, which is why about 90% of EIs occur in males age 20-34.

Electrical injuries are most commonly caused by mechanical contact with an electricity source. While mechanisms of injury to peripheral nervous system exposure have been well established (Lee, 1997), the impact and pathway of electrical exposure into the central nervous system remains a subject of intense investigation and speculation. When electricity enters the body it can disrupt the electrical rhythms of the heart leading to cardiac arrest and anoxia. Still other injuries can occur in falls secondary to an EI, such as if an individual was on a ladder at the time of the electrical exposure. Finally, if physical circumstances dictate (i.e., the individual completes a circuit that causes them to be in the most direct pathway to the ground), electricity can arc over to an individual without there being direct mechanical contact. These arc injuries can also be accompanied by explosions as the air between the individual and the power source can become very superheated, leading to the eruption of an acoustic/vibratory blast. By whatever means, as electric current passes through the body it follows the path of least resistance, and is hypothesized to preferentially affect nerve tissue and blood vessels more so than skin, which has a higher resistance. As such, it is possible to see EI with little to no evidence of thermal injury or what are commonly thought of as entrance/exit wounds.

Although the clinical occurrence of EI is much less common than TBI, many clinical neuropsychologists appear to judge EI by similar parameters as they would a traumatic brain injury. That is, if there is no loss of consciousness or post-traumatic amnesia the injury is not viewed as real. The same could be said for the lack of presence of entrance or exit wounds. In our experience, however, the differences between these types of trauma can by quite dramatic. Neuropsychological deficits can and do occur following EI and can be hypothesized to reflect the effects of electrical exposure on brain functioning. Indeed, neuropsychological studies, including case reports, have indicated that electrical injury survivors often experience a broad range of neuropsychological complaints, although until recently objective neuropsychological changes had not been verified via controlled investigation. In a recent study by Pliskin et al (2006) EI patients performed significantly worse on composite measures of attention, mental speed and

motor skills, which could not be explained by demographic differences, injury parameters, litigation status, or mood disturbance. Results from this and other recent studies suggest that cognitive changes can and do occur in patients suffering from electrical injury. Yet, important questions remain, such as where and how does electricity travel in the body? Moreover, to what extent can functional imaging of the brain provide further insights into the impact that electrical exposure has on central nervous system function?

The following case illustrates some of the challenges in the diagnosis and treatment of EI. The case comes from the annals of the Chicago Electrical Trauma program (ETP), a multidisciplinary group of investigators devoted to the evaluation and treatment of survivors of EI. The ETP is an interdisciplinary and multi-institutional program comprised by a team of clinicians, epidemiologists, biologists, and engineers working towards a better understanding, improved diagnosis, and treatment of electrical injuries.

Case Example

Mr. F is a 32 year old male who suffered an electrical injury in May 2003. He was digging a fence post using a one man auger in his backyard when he hit an underground electrical cable and short circuited a 7200 volt power line. Mr. F's recollection of the accident includes hearing a "pop" and being thrown 10-15 feet away. He landed on grass and cannot recall how he was thrown there. He is unsure if he lost consciousness.
When his wife returned home from work, she found him wandering around the yard in a confused state and shaking. His speech was incomprehensible and he was described as "talking gibberish". According to a neighbor, the power went out approx 20 minutes before his wife arrived home. Mr. F recalls have burning sensations in his hands and feet and was taken to the emergency room where blood studies were drawn demonstrating an elevated CPK and cardiac function was monitored. He was released the next day. He had not sustained entrance or exit wounds and his wife reported no evidence of thermal burns.

Since the accident, Mr. F noted a sharp decline in his stamina, being easily fatigued, increased sleep disturbance, persistent body soreness, and decreased upper body strength. Medical records indicated persistently elevated CPK (i.e., liver enzyme commonly associated with body trauma) levels (319 in June 2004, 193 in July 2004 and 182 in Aug 2004). Mr. F and his wife also noticed changes in his cognitive abilities, such as being forgetful (e.g., misplacing items), unclear thinking, concentration difficulty, stuttering, and not being able to complete sentences. Changes in personality and mood also occurred, including depression, mood lability, and having a "short-temper." Litigation was being pursued to cover medical expenses. Mr. and Mrs. F admitted to frustration about his medical care and a lack of understanding about his condition.

Mr. F was off work for 2 weeks as a technical supervisor for a cable company after the accident then returned to work full time and was able to maintain his employment. He supervises a crew of 14 people and noted difficulty organizing his work. He did not perform very physical work except very occasionally and noted that he is exhausted afterward. Although his performance has been affected, his company has been accommodating to his fatigue issues.

Mr. F's past medical history was unremarkable as he reported a fractured finger as a child, and was otherwise healthy. He completed his high school education and reported having a happy childhood with no specific difficulties in school. He is married and lives with his wife and 2 children. Mr. F used to participate in many sporting activities including volleyball and softball leagues. He still does participate but fatigues easily. He reported of having one alcoholic drink per week while use of illicit drugs and tobacco were denied.

Other Clinical Studies

Mr. F underwent a *high-resolution MRI* of the brain using a standard 3T MRI protocol that demonstrated an unremarkable examination with normal brain perfusion and intact white matter tracts. An *occupational therapy evaluation* found that functional strength and sensation was within normal limits throughout both upper extremities. However, *rehabilitation medicine* and *neurology* consultations detected periodic muscle twitching of the quadriceps muscles that were consistent with fasciculations. An *electromyography exam* was subsequently conducted and confirmed the abnormal fasciculations in the muscles of Mr. F's lower extremities. As a result, he underwent a muscle biopsy which showed some evidence of denervation (i.e., loss of nerve supply) in to the quadriceps muscles. Thus, despite the lack of any thermal injury, or entrance/exit wounds as part of his injury experience, Mr. F. had sustained nerve damage to his lower extremities. Finally, Mr. F. underwent a *neuropsychiatric evaluation* and was diagnosed with a mood disorder secondary to medical condition. Mr. F was treated with Wellbutrin, which both Mr. F and his wife agreed had helped to decrease his symptoms of depression and mood lability.

Neuropsychological Evaluation

As part of his Electrical Trauma Program evaluation workup, Mr. F was referred for an evaluation of current neuropsychological functioning due to his complaints of forgetfulness and thinking difficulty, depression, and emotional lability. His test results are presented in Table 1 below:

Table 1: Mr. F's Neuropsychological Test Scores

Domain	Score
Effort	**Raw**
TOMM Trial 1	49/50 Valid
TOMM Trial 2	50/50 Valid
VSVT # Easy	24/24 Valid
VSVT # Hard	23/24 Valid
Global Intellectual (WAIS-III)	**Standard Score**
Full Scale IQ	89
Verbal IQ	84
Performance IQ	97
Verbal Comprehension Index	80
Perceptual Operations Index	105
Working Memory Index	88
Processing Speed Index	103

Estimated Premorbid IQ	**Standard Score**
WTAR Reading	84
WRAT-III Reading	82
Academic Achievement	**Standard Score**
WRAT-III Arithmetic	81
Verbal Memory Immediate Recall	**Z Score**
CVLT-II Trial 1	-.5
CVLT-II Trial 5	.5
CVLT-II Total (trials 1-5)	52 (T score)
CVLT-II Free Recall Short Delay	.5
CVLT-II Cued Recall Short Delay	0
WMS-III Logical Memory I	9 (scaled score)
Verbal Memory Delayed Recall	**Z Score**
CVLT-II Free Delayed Recall	.5
CVLT-II Cued Delayed Recall	0
Yes/No Recognition True Positives	-1
Yes/No Recognition False Positives	-.5
Discrimination	0
WMS-III Logical Memory II	11
Visual Learning and Immediate Memory	**T Score**
BVMT-R Trial 1	41
BVMT-R Trial 3	46
BVMT-R Total (trials1-3)	44
WMS-III Visual Reproduction I	9 (scaled score)
Delayed Visual Memory	**T Score**
BVMT-R Delayed	49
WMS-III Visual Reproduction II	10
Attention and Mental Speed	**T Score**
Stroop Word	32
Stroop Color	39
Stroop Color-Word	39
Stroop Interference	50
Trails A	53
Trails B	35
CPT-II Omissions	41
CPT-II Commissions	38
Gordon Vigilance Hits	-1.34 (Z score)
Gordon Vigilance Commissions	.18 (Z score)
Gordon Vigilance RT	.54 (Z score)
Gordon Distractibility Hits	.56 (Z score)
Gordon Distractibility Commissions	-.68 (Z score)
Gordon Distractibility RT	-.32
Executive Functions	**T Score**
WCST % Errors	33
WCST % Preservative Responses	37
WCST % Preservative Errors	36
WCST % Conceptual Level Responses	32
TOL Moves	102
TOL Correct	96
TOL Rule Violations	104
TOL Total Time	102

Motor	T Score
Grip Strength Dominant Hand	36
Grip Strength Nondominant Hand	41
Grooved Pegboard Dominant Hand	33
Grooved Pegboard Nondominant Hand	41
Emotional/Personality	**T Score**
BDI-II	24 (raw) moderate depression
MMPI-II F	48
MMPI-II L	52
MMPI-II K	60
MMPI-II HS	84
MMPI-II D	89
MMPI-II HY	91
MMPI-II PD	69
MMPI-II MF	44
MMPI-II PA	42
MMPI-II PT	98
MMPI-II SC	82
MMPI-II MA	41
MMPI-II SI	75

Summary of Neuropsychological Test Findings

Test findings indicated that Mr. F is a man of low average premorbid intelligence, with stronger nonverbal abilities than verbal abilities, a likely longstanding pattern for him. He evidenced low average sight reading ability and mildly impaired arithmetic skills. Mr. F's verbal and visual learning and memory was average, though he experienced difficulty in the subsequent recognition of the learned visual information. Simple attention was intact while sustained attention was in the low average range. Divided attention, set shifting, and cognitive flexibility, however was mildly impaired. Higher-order problem solving and abstract reasoning abilities remained generally intact. A weakness was consistently demonstrated in Mr. F's dominant hand on motor tasks. Self-report measures and clinical interview indicated that Mr. F was experiencing emotional distress related to his electrical injury, including depression, mood lability, intense fatigue, health concerns and social isolation. He endorsed an avoidant coping style for dealing with his difficulties and other features suggestive of possible Post-Traumatic Stress Disorder.

Mr. F. demonstrated the types of cognitive changes observed in many EI patients. That is, he demonstrated subtle deficits in divided attention, mental slowing, shifting cognitive set and cognitive flexibility that could not be attributed to a lack of effort or interference from emotional disturbances or obvious fatigue given that other equally challenging tasks were completed successfully during the course of the single day evaluation. Unlike TBI, this EI patient, like many others, did not manifest a primary impairment in learning or memory. The problem remains, however, that neuropsychological testing alone has not proven to be informative in addressing the issue of underlying basis for these cognitive changes. Thus, the question remains how to best "image" the hypothesized changes in brain function that neuropsychological assessment appears to reflect. This has led us to develop a functional neuroimaging protocol that was applied to this patient and is described below.

Functional Neuroimaging Evaluation

In order to investigate the functional integrity of neural substrates supporting cognitive abilities, we administered two oculomotor cognitive activation tasks during a functional MRI scan. The tasks used were a memory-guided saccade task, tapping into the neurocircuitry underlying spatial working memory (Sweeney et al., 1996), and a predictive-saccades task, tapping into the neural network involved in implicit learning (Simo et al., 2005).

Cognitive paradigms using oculomotor tasks have been shown to robustly elicit brain activation in multiple brain regions simultaneously. As such, they provide useful, noninvasive tools to assess the functional integrity of widely distributed neural networks. In the spatial working memory task, subjects were asked to covertly attend to and learn the location of a briefly presented peripheral target stimulus, while maintaining their fixation onto a central cue. When the fixation cue was extinguished, subjects were asked to immediately direct their gaze to the remembered location of the peripheral target stimulus based on their memory of the target location prior to the delay period. Thus, this is a delayed response task in which subjects need to maintain a cued spatial location in working memory over time. In the implicit learning task, subjects were asked to direct their gaze onto a target that alternated between two locations in a fixed, predictable manner, but it was left up to the subject to learn that the stimulus presentation is predictable and utilize this implicitly learned information to guide their saccades more quickly to targets.

Using the subtraction method, patterns of activation on each task were compared to those obtained during a simple sensory processing task, in order to isolate the specific neural networks involved in spatial working memory and implicit learning. Mr. F's activation maps compared to a demographically matched control subject are presented in Figures 1-4 below.

[Insert Figures 1-4 about here]

The findings demonstrate different patterns of brain activation on both tasks between Mr. F and a demographically-matched control subject. Specifically, during the spatial working memory task, Mr. F exhibited increased activation in prefrontal cortices including bilateral middle frontal gyri, and left frontal eye fields, as well as increased activation in posterior cortices relevant to sensory processing including bilateral intra-parietal sulci (Figure 1). Conversely, during the spatial working memory task, the control subject exhibited a much more concise pattern of activation with a small cluster of increased activation in the right middle frontal gyrus (Figure 2). An opposite pattern of findings emerged from the implicit learning task. During this task, Mr. F demonstrated a small cluster of increased activation in the left cingulate motor cortex (Figure 3), while the control subject exhibited a widely distributed network of activation in multiple brain

regions including the left middle frontal gyrus, bilateral frontal eye fields, bilateral cingulated cortices and bilateral posterior cortices (Figure 4).

These findings suggest system-level, task-dependent differences in activation between Mr. F and the demographically matched control subject. These effects are similar to those we have observed in a larger sample of patients. The differences in activation during the spatial working memory task may reflect a "spill over" effect or compensatory mechanisms, in such that Mr. F requires more brain resources in order to perform the task. Indeed, many of our EI patients complain that this issue isn't that they cannot concentrate, but rather they must expend much more energy to do so. On the implicit learning task, the findings may indicate a failure to engage in or initiate neural networks involved in implicit learning of the predictable target movement sequence. The dissociation between the two tasks (i.e., overactivation on the working memory task and underactivation on the implicit learning task) effectively rule out the general effects of medication or psychiatric symptomatology as the primary bases for these findings.

Efforts to further elucidate the manner in which electrical injury alters brain functioning continue, and clearly these findings are those of one subject. The ETP is in the process of conducting a large scale fMRI investigation into the effects of EI on brain functioning in a large sample of electrical injury patients and a demographically-matched control group. We hope that the findings will contribute to existing literature by clarifying the neurocognitive sequelae of electrical injury and the extent to which they reflect alterations in central nervous system function.

Final Comments and Future Directions

The neuropathological mechanism mediating electrical exposure and the observed neuropsychological dysfunction is not yet clear. In order to improve current clinical care of EI patients and advance our knowledge regarding the effects of electrical exposure on brain integrity, it will be important to clarify the source of the observed neuropsychological dysfunction following EI. Neuropsychological abnormalities in the areas of attention and mental processing speed develop in some EI patients, and in the present case of Mr. F, we identified functional differences in his neural activation during spatial working memory and implicit learning oculomotor tasks compared to a matched, healthy control. However, at this point, it remains to be conclusively shown in a larger group of EI patients that observed neuropsychological dysfunction is the result of current-induced brain changes in neuronal substrates subserving cognitive abilities. Nevertheless, in the case of Mr. F., he had nerve damage to his legs with no obvious entrance/exit wounds. He experienced neuropsychiatric changes despite no prior psychiatric history, and his pre-accident medical history was unremarkable. He passed all symptom validity testing and evidenced neuropsychological changes without any obvious loss of consciousness. Cases such as Mr. F. highlight the fact that EI is not synonymous with TBI, and should not be judged clinically by the same standards.

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