Detection and diagnosis of malingering in electrical injury

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Abstract

This paper sought to demonstrate that diagnosable malingering does occur in Electrical Injury (EI) and examine the relationship of malingering to potential indicators of the presence and severity of neurological injury. Eleven consecutive EI patients seen for neuropsychological evaluation were presented. Over half the patients met the Slick et al. (1999) criteria for at least Probable MND. Most of the MND patients lacked evidence of a biologically meaningful exposure to electrical current. These findings highlight the importance of considering biological markers of neurological injury and of non-neurological factors, including effort/malingering, in the study of the neurocognitive consequences of EI.

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Electrical injury (EI) is a potential source of injury and death and most EI (excluding lightning strike) occur in the workplace (Duff & McCaffrey, 2001; Goubiere, Corbut, & Bazin, 1994; Heilbronn & Pliskin, 1993). A number of studies have demonstrated cognitive performance deficits in EI patients and the observed patterns of cognitive symptoms and neurobehavioral deficits are very similar to those seen in traumatic brain injury (TBI; see Duff & McCaffrey, 2001, for an excellent review). However, in contrast to the recovery pattern in TBI, there has been speculation that EI may result in persistent, progressive, and/or delayed-onset neurocognitive and emotional effects (e.g., Farrell & Starr,
Many EI patients reporting neurocognitive impairment have suffered clearly demonstrable neurological injuries that one would expect to produce significant neurocognitive sequelae (e.g., coma, focal neurological signs; Hopewell, 1983). However, the neuropathology in other cases is more ambiguous. Therefore, when attempting to attribute cognitive deficits to the direct neurological effects of an electrical exposure, it would be helpful to have indicators that the body had actually absorbed a sufficient amount of electrical energy to produce injury.

Entry and exit (EE) wounds are a certain indication that the body has conducted a significant amount of electrical current because they result from the conversion of the electrical energy into thermal energy due to the resistance of pathway tissues (e.g., skin, hair, bone; Solem, Fischer, & Strate, 1977). In the only study of the relationship of EE wounds to cognition, Grossman, Tempereau, Brones, Kulber, & Pembrook (1993) found a higher incidence of neurobehavioral problems in patients with EE wounds versus those without. While EE wounds are an indication that the body has absorbed sufficient electricity to cause injury, their presence does not necessarily indicate that the electrical exposure has caused nervous system injury. At the same time, the absence of EE wounds does not necessarily mean that EI has not occurred. Therefore, other indicators of acute central nervous system dysfunction such as those used to grade TBI severity (e.g., Glasgow Coma Scale score, length of coma and/or post-traumatic amnesia, structural damage to the nervous system) may also be helpful. Unfortunately, these have not been studied systematically in EI.

The acute presence of burns, altered consciousness, focal neurological signs, and/or structural pathology would arguably indicate that exposure to electrical current has led to an internal dose sufficient to alter physiological function and damage neurological systems. However, like many mild TBI patients, EI patients reporting neurocognitive sequelae often have little or no objective evidence of acute injury (i.e., they have more ambiguous injuries). In the absence of such evidence, it is reasonable to be very cautious in attributing observed or reported cognitive deficits to neurological damage caused by electrical current, because, just like in TBI (Binder, 1997; Binder & Rohling, 1993), non-neurological factors including personality traits, stress, motivation, effort, and financial incentive likely influence the report of cognitive deficits in EI. In fact, a high proportion of electrical injuries do occur in a compensable context (Cherington, 1995; Duff & McCaffrey, 2001; Heilbronner & Pliskin, 1993). Mittenberg, Patton, Canyock, and Condit (2002) suggest that the base-rate of malingering is as high as 40 percent in compensation seeking samples in general and 25 percent in EI in particular. However, published research on the cognitive and emotional effects of EI has not assessed, much less controlled for, exaggeration/malingering nor have the implications of potentially including malingerers in research samples been addressed.

This paper presents a series of EI patients who have been carefully examined for malingering and in whom we have applied the criteria of Slick, Sherman, and Iverson (1999) for Malingering Neurocognitive Dysfunction (MND). The purpose of this paper is twofold: (1) demonstrate that malingering does occur in EI and can be diagnosed with the Slick et al. criteria; and, (2) examine the relationship of malingering to
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