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The Neurophysiological Aspects of Electrical Injuries

Hooshang Hooshmand, Farideh Radfar and Eleanor Beckner

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Vertigo

INTRODUCTION

Electrical injuries result in approximately 1,200 deaths annually in the United States.[1] This figure is similar to the 5 per 1,000,000 annual mortality rate in Switzerland, Canada, France and West Germany.[2-6] Males receive the majority of electrical injuries, usually in an occupational environment.[1-2] However, similar injuries may occur due to malfunctioning appliances. In addition, bathtub electrocution accounts for approximately 25 deaths a year in the United States.[7]

Electrical injuries related to lightning are quite variable, depending upon the circumstances at the time of the lightning. Currents of several thousand volts may be transmitted through the body to the ground[1] with variable degrees of injuries. Because of such variability, the lightning electrical injuries are excluded from our study.

MATERIAL AND METHOD

From 1976 through 1987 sixteen patients with early and late complications of alternate current electrical injuries were studied and followed for 5 to 9 years. Thirteen were male and 3 were female. The age range at the onset of the study was 21 to 47 years.

The place of exposure was an industrial and work environment for 12 and in the home for 4 patients. The main mode of industrial exposure consisted of contact with high voltage wires. This was in the form of crane extension of scaffolding touching overhead high voltage (13 kilovolts to 500 kilovolts) wires among 8 patients. The rest of the industrial accidents were in the form of exposure at work due to poorly insulated wires in a moist environment. The accidents at home consisted of exposure to poor wiring in the basement with washer and dryer appliances for 2 patients (240 volts) and exposure to poor wiring in other places in the house for 2 others (120 volts). Adverse working conditions such as exposure to electricity when sitting on top of a crane and exposure with a hand grasping a wire ("can't let go" phenomenon) was a common feature in the patients suffering from injuries to the upper extremities.[8-12]

RESULTS

The clinical complications were in early and late forms[2] as follows.

Early Complications

In all patients the early complications were severe enough to require a few days hospitalization, with loss of consciousness, pain in the extremities, and severe anxiety being the early clinical manifestations.

Hand-to-hand points of entrance and exit of the electrical current were noted in 2 patients, both of whom developed complications of post-traumatic depression, atonic seizures, and abnormal reflexes pointing to spinal cord injuries. The rest of the patients had hand-to-foot points of entrance and exit. Neurologic complications were not as drastic among these patients.

Neurologic examination invariably showed the presence of severe anxiety and depression in all 16 patients. Pathologic reflexes in the form of bilateral Babinski, unsustained clonus and hyperreflexia were present in 2 patients,

Requests for reprints should be addressed to Hooshang Hooshmand, M.D., Neurological Associates, Seizure Control Clinic, P.O. Box 7147, Vero Beach, Florida 32961.

Table 1

Psychological test results in 16 electrical injury patients.

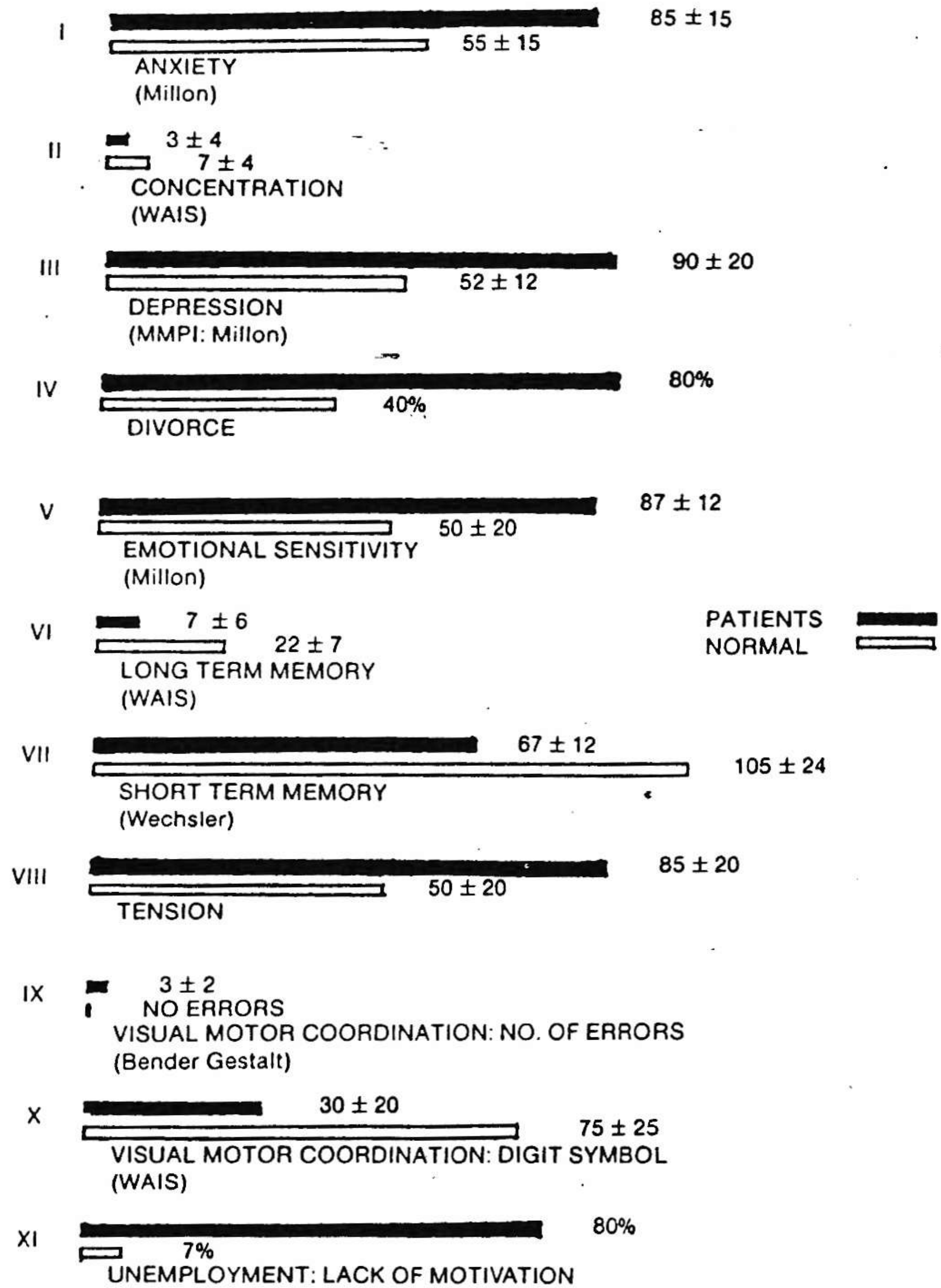

Table 2

Nocturnal plasma prolactin levels of three samples during non-REM sleep (half-hour intervals).

Seizure	No. of patients	Prolactin level
A. Nonepileptic (controls)	12	3 - 9.3 ng/ml
B. Pseudoseizures	12	3.5 - 9.2 ng/ml
C. Electrical injury with atonic, myoclonic, or major motor seizures	9	9.8 - 17 ng/ml

both victims of hand-to-hand points of entrance and exit of the electrical current. Two patients had positive snout reflex; all demonstrated poor recent memory, poor judgment, and poor concentration. In the extremities, the areas of eschar secondary to electrical burn were surrounded by moderate hyposthesia to all sensory modalities, as well as hyperalgesia.

Late Complications

The survivors of the initial electrical injuries developed late neurologic and psychological complications which consisted of neuropsychological complications, seizure disorder, tinnitus, dizziness, as well as 7th and 8th nerve injuries, cataract, visual disturbances, and progressive spinal atrophy (one patient).[2,4-24]

NEUROPSYCHOLOGICAL COMPLICATIONS

The 16 patients in the study were evaluated with Bender Gestalt Wechsler Intelligence, TAT, Shipley Elizure test and MMPI. In addition, 12 patients underwent the Halstead-Reitan test.

Within 3 months to 1 year all patients had disturbances of their daily life and demonstrated abnormal neuropsychological testing with poor scores regarding recent memory, concentration, judgment, verbal and non-verbal achievement. Fourteen of the 16 patients suffered severe depression. The depression was severe enough to prevent them from leading normal personal and professional lives in 11 out of the 16 patients (Table 1).

On the scale of emotion, the patients showed irritability and increased sensitivity. Invariably the patients lost their normal motivation and initiative capacity. They had problems activating their thoughts and long-term planning. The patients' judgment and insight were rated from fair to poor. Of the 11 married patients, all had poor interpersonal relationships which affected their marital status and caused problems with family relationships.

The patients had poor visual motor balance, decreased motor activity and poor attention and concentration spans which resulted in secondary side effects of poor recent memory (Table 1). After 5 years, 11 of the 13 employed patients had lost their jobs and 9 of the 11 married patients had been divorced.

SEIZURE DISORDER

Twelve patients suffered from one or more attacks of seizure. Three had an isolated seizure shortly after the accident, and nine suffered from repetitive seizures. Of the isolated seizure patients, one suffered from a major motor seizure immediately after the injury. The other two had violent myoclonic jerks for a few minutes after exposure to electricity.

Immediately after the accident, all the patients suffered loss of consciousness which lasted a few seconds to several minutes. The nine repetitive seizure patients were hospitalized and studied with 24-hour EEG recordings, in order to rule out the possibility of pseudoseizures.

The seizures, clinically observed in every patient, were not the type seen in psychogenic seizures, e.g., they did not have out-of-phase upper and lower extremity movement, pelvic thrusting, and side-to-side shaking movements of the head. The seizures were manifested in atonic, in-phase myoclonic, or major motor seizures. They could not be induced by the power of suggestion, while recording the EEG and injecting i.v. normal saline, in any of the patients.

The plasma level of prolactin hormone was measured in half-hour intervals during non-REM sleep on the nine patients. The results were compared to the same levels in twelve

Table 3

Brain stem auditory evoked potentials in electrical injuries.

	Wave Amplitude (uV)			Wave Latency (m.sec.)			Interpeak Latency (m.sec.)		
	I	III	V	I	III	V	I-III	I-V	III-V
CONTROLS	0.46	0.70	0.72	1.52	3.74	5.60	2.22	4.08	1.86
	±0.23	±0.32	±0.16	±0.16	±0.21	±0.20	±0.16	±0.12	±0.11
INITIAL VISIT	0.30	0.54	0.40	1.90	4.35	6.20	2.45	4.30	1.85
	±0.16	±0.17	±0.14	±0.12	±0.13	±0.18	±0.12	±0.11	±0.11
LAST VISIT*	0.38	0.57	0.46	1.92	4.34	6.20	2.42	4.28	1.86
	±0.12	±0.11	±0.12	±0.14	±0.12	±0.11	±0.11	±0.12	±0.12

Filter Settings 150 Hertz Low 3000 Hertz High
± Standard Deviation (SD)
*12-19 Month Intervals

non-epileptic control patients as well as twelve documented and diagnosed pseudoseizure patients. The plasma prolactin level has been demonstrated to become significantly elevated among patients suffering from seizure disorder.[13-15] The level range was 3 to 9.3 ng/ml among the twelve nonepileptic control patients, and the same level was measured at 3.5 to 9.2 ng/ml among the twelve pseudoseizure patients. The plasma prolactin level among the electrical injury patients with atonic, myoclonic, or major motor seizures showed a 9.8 to 17 ng/ml range (Table 2). This was significantly elevated when compared to the other two groups ($P < 0.02$).

The infrequent seizures were initially misdiagnosed as "syncopal" attacks. However, the seizure workup with 24-hour EEG-EKG recording ruled out syncopal attacks. The seizure disorder complications responded quite well to treatment with Clonazepam. Two patients had aggravation of depression with Clonazepam, and they were successfully treated with Valproate.

After a five to nine year followup, only two patients continued to have myoclonic seizures of an infrequent nature in spite of treatment. Both were noncompliant with low blood levels of anticonvulsants.

DIZZINESS

The complication of dizziness was present and persistent in ten of sixteen patients, in the form of true vertigo. With the passage of time, the dizziness became quite infrequent; after five years six patients continued to have infrequent attacks of vertigo. Only two patients complaining of vertigo had evidence of 8th nerve dysfunction on otological studies, with the remainder having CNS abnormalities on BAER.

OTHER COMPLICATIONS

In four patients, pain in the extremities persisted long after the eschar had healed. The

W.M. 52
1/29/84
SEN 10 µv/nm
HFF 70 Hz
LLF 1 Hz
Light Sleep
24 hr. EEG
3 previous EEG
Awake
Normal EEG

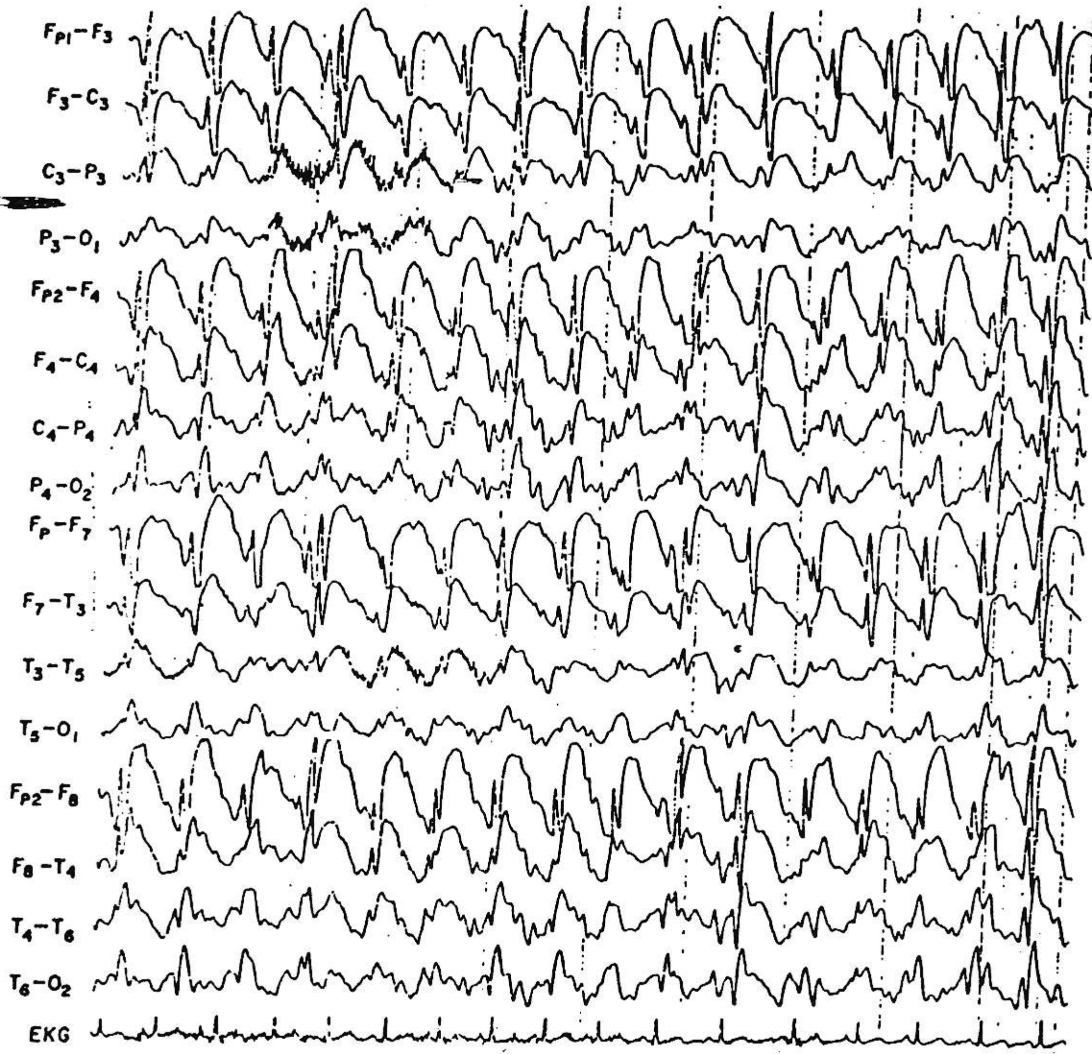

Figure 1. A single, prolonged (12½ second) spike and wave discharge during light sleep in a patient, age 52, with infrequent seizures after electrical injury. Waking activity was normal; 24 hour EEG recording.

Table 4A

Somatosensory evoked potentials in electrical injuries.

MEDIAN			
Initial Visit	16 Patients		224 Controls
Erb's Point	Left	Right	
P 9	11.88 ± 2.04	11.24 ± 2.76	8.50 ± 3.0
P 11	13.26 ± 2.96	12.96 ± 3.30	11.00 ± 1.96
P 12	14.98 ± 2.24	14.47 ± 2.68	12.50 ± 1.88
P 14	16.45 ± 1.74	17.04 ± 1.96	14.00 ± 2.08
N 20	22.46 ± 2.40	21.64 ± 2.40	20.00 ± 2.48
P 25	25.68 ± 2.68	26.04 ± 2.40	25.00 ± 2.36
N 30	absent	absent	26.28 ± 2.48
Last Visit*			
P 9	10.56 ± 2.74	11.76 ± 1.98	8.50 ± 3.00
P 11	14.04 ± 2.56	12.00 ± 2.24	11.00 ± 1.96
P 12	15.12 ± 2.34	14.74 ± 2.36	12.50 ± 1.88
P 14	17.64 ± 2.65	17.00 ± 2.48	14.00 ± 2.08
N 20	21.36 ± 1.76	22.00 ± 1.98	20.00 ± 2.48
P 25	25.00 ± 2.36	24.70 ± 2.16	25.00 ± 2.36
N 30	absent	absent	26.28 ± 2.48

Filter Setting Low 150 Hertz High 300 Hertz
± Standard Deviation (SD)
*12 - 19 Month Intervals

nerve conduction times were abnormal in the involved extremities of all four patients. Three of the four patients had evidence of moderate chronic sympathetic dystrophy on thermography. One patient suffered from compressed L3 lumbar vertebral body fracture due to major motor seizures.

Laboratory Tests

EEG abnormalities were not clear-cut. Mild generalized slowing was noted on the EEG's of nine patients. Five patients with atonic attacks

Table 4B

Somatosensory evoked potentials in electrical injuries.

ULNER	16 Patients		224 Controls
Initial Visit			
Erb's Point	Left	Right	
P 9	12.16 ± 1.98	13.00 ± 2.04	9.00 ± 2.75
P 11	12.04 ± 1.64	13.36 ± 1.76	11.90 ± 1.98
P 12	15.64 ± 1.98	15.24 ± 1.76	12.00 ± 1.98
P 14	18.76 ± 2.04	19.16 ± 2.16	14.50 ± 1.88
N 20	22.76 ± 2.16	23.16 ± 1.76	18.64 ± 2.00
P 25	26.84 ± 1.98	27.00 ± 1.76	26.64 ± 2.96
N 30	absent	absent	
Last Visit*			
P 9	12.00 ± 2.26	13.36 ± 2.20	9.00 ± 2.75
P 11	13.00 ± 1.98	14.16 ± 1.76	11.90 ± 1.96
P 12	15.54 ± 1.72	15.25 ± 1.90	12.00 ± 1.98
P 14	17.46 ± 1.98	18.00 ± 1.76	14.50 ± 1.88
N 20	22.56 ± 2.34	23.00 ± 2.20	18.64 ± 2.00
P 25	25.76 ± 1.88	26.00 ± 1.76	24.16 ± 1.92
N 30	absent	absent	26.64 ± 2.96

Filter Setting Low 150 Hertz High 300 Hertz
± Standard Deviation (SD)
*12 - 19 Month Intervals

and three patients with myoclonic seizures had generalized polyspike and wave ictal bursts recorded during the 24-hour EEG recordings (Figure 1).

Due to the high incidence of dizziness, tinnitus, and pain in the extremities, BAER and SEP were done on all 16 patients (Tables 3 and 4). The BAER abnormalities frequently pointed to lower brain stem dysfunction ($P<0.02$) The SEP abnormalities were more diffuse, showing involvement of the peripheral nerves in the burned extremity, as well as cervical spinal cord

Table 4C

Somatosensory evoked potentials in electrical injuries.

TIBIAL	16 Patients		224 Controls
Initial Visit	Left	Right	Left/Right
N 37	40.75 ± 7.70	42.10 ± 6.84	34.00 ± 5.55
L3-L5	absent	absent	9.00 ± 4.50
Last Visit*			
N 37	44.00 ± 2.10	42.76 ± 1.75	34.00 ± 5.58
L3-L5	absent	absent	9.00 ± 4.50
PERONEAL			
Initial Visit	Left	Right	Left/Right
N 25	38.00 ± 1.10	37.50 ± 2.00	25.00 ± 4.88
S 1 (iliac crest)	absent	absent	10.00 ± 3.75
Last Visit*			
N 25	35.38 ± 1.60	37.06 ± 1.84	25.00 ± 4.88
S 1 (iliac crest)	absent	absent	10.00 ± 3.75

Filter setting Low 5 Hertz High 250 Hertz
(±) Standard Deviation (SD)
*12 - 19 Month Intervals

dysfunction (Table 4A, B, and C). With the passage of time (12 to 19 months), there were no statistically significant changes in the evoked potential abnormalities, pointing to a tendency for permanent injuries.

The CT scan showed mild generalized cerebral atrophy in one of the 16 patients, the rest of the CT scans were normal.

DISCUSSION

Longterm follow-up of electrical injury patients revealed a stereotyped clinical picture.

This consisted of loss of consciousness at the time of injury, commonly followed by late complications of depression, seizures, dizziness, and tinnitus.

Adverse working conditions such as exposure to electricity when sitting on top of a crane and exposure to hand grasping a wire ("can't let go" phenomenon) can cause mortality and morbidity in patients exposed to alternate currents.[8-11] Another risk factor is the point of entrance and exit. There is a higher mortality when points of entrance and exit of the electrical current are hand-to-hand rather than hand-to-foot.[10,11]

The alternate current injuries result in cardiac arrest due to tetanic effect,[2,16] as well as generation of heat in the extremity which can cause more severe damage to nerves and arteries than to the other tissues.[12,17] The high incidence of post-traumatic depression may be related to the tendency for electrical injuries to result in multiple anatomical and chemical changes in the central nervous system in a diffuse fashion.[1,2,18-32]

In our study, the seizure disorder was quite common compared with other reports of infrequent seizure disorder secondary to electrical injuries.[19,25] One explanation may be that the seizure disorder is not typical, and is usually misdiagnosed as syncopal attacks. The atypical seizure disorder along with the paucity of epileptiform discharges on standard EEG recording may explain the difficulty in the diagnosis of the seizure complication. The abnormalities recorded on evoked potentials in the cervical spinal cord and brain stem region bring up the possibility that the seizure discharge is too deep to be easily recorded by standard surface EEG recordings.

The high incidence of psychosocial deterioration, unemployment and divorce, along with a high incidence of depression and seizure disorder, point to the moderate damaging effect of electrical injuries on the central nervous system. The emotional problems may mimic conversion reaction. The late development of such complications makes the correct diagnosis more difficult.[20] The same is true with spinal cord injuries. In a study of motor neuron diseases, the antecedent events were more likely to be electric shock and lightning accidents rather than other types of trauma.[21]

SUMMARY

Neurologic and psychologic studies were done on 16 victims of alternate current electrical injuries. The patients were followed for a period of over 5 years. The findings point to a stereotyped generalized cerebral dysfunction, resulting in depression, divorce, unemployment as well as a high incidence of atypical seizures (atonic and myoclonic seizures). The EEG and CT studies were nondiagnostic. Evoked potentials revealed abnormalities in the upper cervical spinal cord and lower brain stem regions, raising the possibility that the epileptogenic focus was too deep to be recorded by standard surface EEG recording. The fact that the electrical injury patients have a high incidence of severe emotional disturbance and post-traumatic depression along with atypical seizures, in the face of nondiagnostic EEG and CT studies, may result in improper management of such patients.

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