

From:

Chronic Pain: Reflex Sympathetic Dystrophy Prevention and Management

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ELECTRICAL INJURIES

In our experience of over 100 patients suffering from electrical injuries referred to our clinic from different parts of the country, 42 patients had comprehensive extensive studies, both anatomical and physiological.

The rest of the patients had the majority of their studies done in other centers, and they are not included in this series of 42 patients being reported because there was not a complete battery of anatomical tests (MRI or CT), neuropsychometric tests (Halstead-Reitan or Luria-Nebraska), and neurophysiologic tests (evoke potentials and topographic brain mapping).

The following is a summary of the stereotypical clinical findings on electrical injuries. Thirty-three of 42 patients suffered from RSD. The treatment for the patients suffering from RSD secondary to causalgic pain of electrical contact was quite difficult and taxing to the clinician and to the patient.

As with other forms of RSD, the disease is classified into four stages.

Stage I: On the Scene of the Accident

1. No-let-go phenomenon. This was present in practically every patient except for the ones who had exposure to electricity to area that could not undergo gripping of the hand, such as contact with the foot or dorsum of the forearm (Figure 5a). No-let-go phenomenon means the electrical stimulation causes flexor spasm of the muscles, not allowing the victim to let go of the electrical source.
2. Ipsilateral extremity burn, eschar, and neurosensory damage. The same phenomenon to a lesser extent is present in the contralateral extremity - exit point of the electricity. The lesion specifically involves c fiber nerves and nerves around the arterioles (sympathetic nerve fibers) with ephapse in c fibers. As a result, the patient has severe pain in the involved areas, and the pain extends far beyond the eschar region.
3. The body is usually thrown away from the source of electricity in one massive myoclonic jerk. This results in falling from a ladder or other heights with secondary injuries.

4. Tonic and at times tonic clonic seizures of brief duration, followed by a brief loss of consciousness.
5. Cardiac arrhythmias and brief cardiac arrest with good response to resuscitation, followed by typical autonomic dysfunction in the form of abnormal cardiac rhythm, fluctuating blood pressure, and abdominal and chest pains. Attacks of apnea are quite frequent.
6. Blisters over the fingers, acute RSD of extremity, blisters and reddish discoloration over the contralateral exit point, as well as blisters and reddish discoloration over the anterior chest wall at T4 through T6 levels, which are the points of entrance of electricity through vascular and sympathetic fiber to the spinal cord (Figure 4d).

Stage II: Hospitalization

1. The patient is quite drowsy and at times confused and tired.
2. Labile vital signs in the first 24 hours, prolonged PQ, deep Q, irregular PQ interval, and arrhythmias on EKG. Orthostatic hypotension is quite common at this stage, resulting in syncopal attack when the patient tries to get up and walk.
3. Akinetic attacks. Usually myoclonic seizures are less frequent at the stage, and they are more likely to develop in Stages III of IV.
4. Vertigo and tinnitus, which can be quite intractable, lasting for months to years.
5. Painful extremities at the points of entrance and exit, eschars of different degrees, and RSD.
6. Sensory loss over the trunk distal to the T4 through T6 entrance of electricity to the spinal cord. This sensory loss is usually asymmetrical, and the patient usually develops a partial Brown-Sequard syndrome. This aspect of the examination should be checked on every patient. This is the most frequently overlooked and underdiagnosed sign of electrical injuries.

This sign of spinal cord injury explains the reason for the patient having myoclonic and akinetic seizures too deep to be recorded on EEG.

This is practically pathognomic and was present in every patient.

7. Frontal lobe dysfunction: tremor, positive snout reflex, masked fascies, irritability, and poor judgment were present in over 50% of the patients.

Stage III: First Few Weeks to Months after the Accident

1. Extremities pain, hyperpathia, and allodynia were present in 29 of 42 patients.
2. Akinetic or myoclonic seizures were present in 28 of 42 patients.
3. Anxiety, agitation, phobia, irritability.
4. Labile neurovascular symptoms and signs:
 - a. Cardiac arrhythmias
 - b. RSD
 - c. Labile blood pressure, orthostatic hypotension
 - d. Abdominal cramps
 - e. Diarrhea
 - f. Noncardiac origin chest wall pain usually due to sympathetic nerve injury (Figures 4d and 4e).
5. Poor recall, poor recent memory.
6. Depression and secondary insomnia were present in 28 of 42 patients.
7. Frontal lobe dysfunction, irritability, tremor, poor judgment, poor tolerance, and fatigue.

Stage IV: Over 6 Months

1. Loss of job (over 50% of the patients).
2. Loss of spouse, severe marital interpersonal strain (over 50% of patients).
3. Vertigo and tinnitus in one-third of patients.
4. Severe depression or schizoaffective withdrawal, anxiety, phobia in over three-fourths of the patients.
5. Akinetic and myoclonic seizures.
6. Poor recall and recent memory in over 60% of the patients.

7. Painful extremity (chronic pain) in over 30% of the patients.
8. RSD in 33 of 42 patients.
9. Impotence, neurogenic bladder, abdominal cramps, and chronic tremor.

DIAGNOSTIC TESTS FOR ELECTRICAL INJURIES

1. Anatomical tests, i.e., MRI or CT scans are normal.
 2. Physiological tests:
 - a. EEG usually is normal: 7 (14%) patients had sharp transients, and 4 (9%) had epileptiform discharges in the temporal frontal regions.
 - b. EKG abnormal in Stages I and II in over 50% of the patients. The EKG subsequently reverted to normal.
 - c. Thermography was abnormal in 33 of 42 patients, showing different degrees of RSD (Figures 4d, 4e, and 25).
 - d. Evoked potentials.
 - i. Visual evoked potentials are usually nondiagnostic.
 - ii. Baer showed abnormalities in interpeaks I-III in 30 of 42 patients and in 21 of 42 patients in stage IV.
 - iii. SSEP was normal in 39 of 42 patients in Stages I-III and 27 of 42 in Stage IV.
 - e. Topographic brain mapping was abnormal in 28 of 42 patients in Stages III and IV.
- None was done in Stages I and II.

The low percentage of EEG abnormality may be due to (1) involvement of deep structures, and (2) tests done in late (chronic) stages of the disease.

The abnormality on topographic brain mapping is usually in the form of frontal temporal asymmetry and suppression of background, which was bilateral in 22 of 28 brain mappings. In two patients, a dislocation was noted. This phenomenon refers to the fact that the afrequency power spectrum has shifted from the occipital lobes to the frontal lobes. These patients had suffered from prolonged coma after the electrical injury. They suffered from a marked frontal lobe dysfunction as mentioned above.

The only other condition that causes a dislocation on brain mapping is rare severe head injuries or cerebral anoxia, which shows "acoma" on the EEG as well.

The electrical injury causes CNS damage and follows the path of least resistance, which is the c fibers and the sympathetic nerve fibers surrounding the arteries to the thoracic spinal cord. The damage ascends up and down to the lower portion of the spinal cord in the final pathways of the nociceptive c fibers (Figures 4d-f, 25, and table 14).