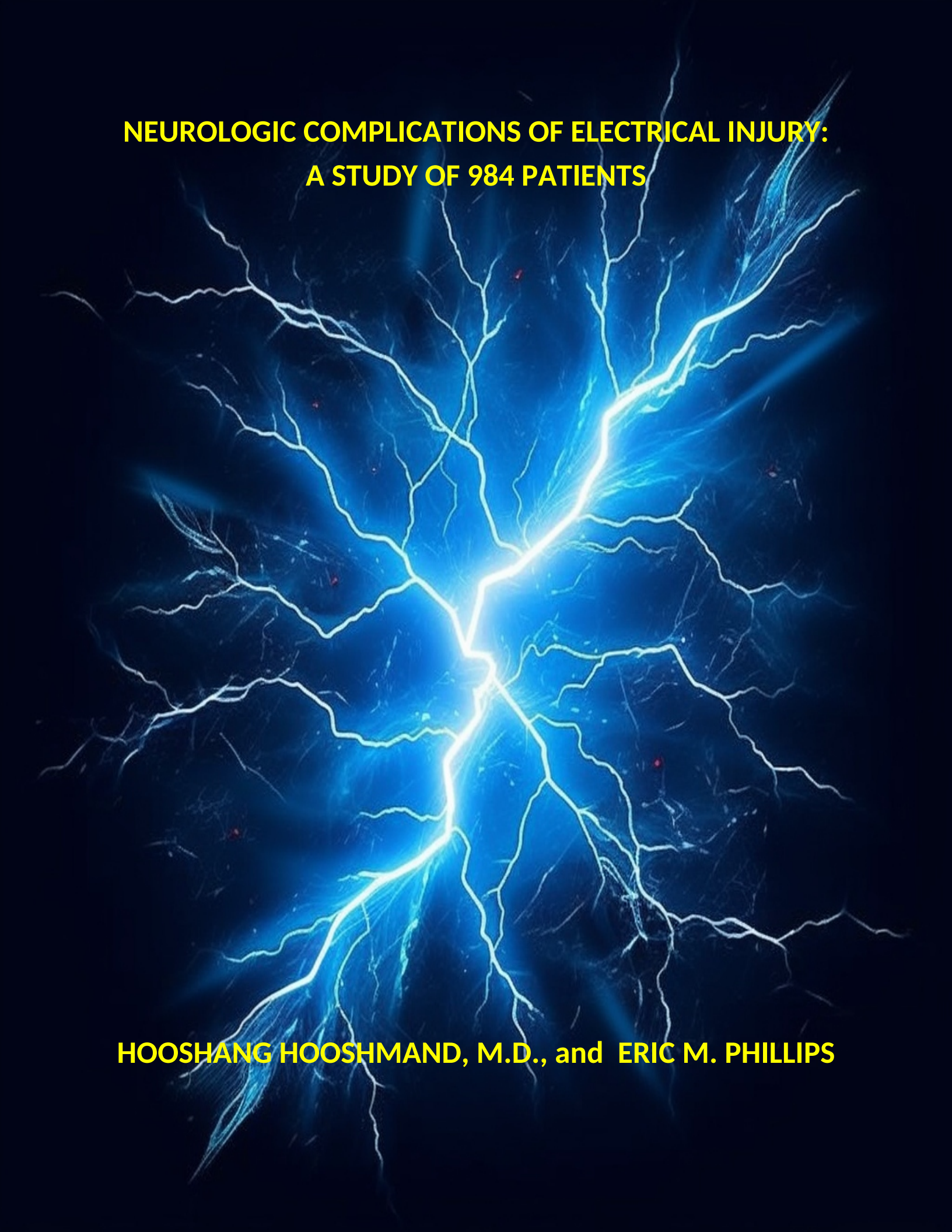
NEUROLOGIC COMPLICATIONS OF ELECTRICAL INJURY: A STUDY OF 984 PATIENTS



HOOSHANG HOOSHMAND, M.D., and ERIC M. PHILLIPS

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Other books published by the authors:

Doctor Hooshang Hooshmand:

Chronic Pain-Reflex Sympathetic Dystrophy: Prevention and Management (1993).

Don't Diet: Change Your Eating Habits - Proper Eating for Good Health (2020).

Eric M. Phillips:

Complex Regional Pain Syndrome (CRPS): Patients' Perspective of Living in Chronic Pain: Volume 1 (2020).

Complex Regional Pain Syndrome (CRPS): Patients' Perspective of Living in Chronic Pain: Volume 1-Picture eBook (2020).

Don't Diet: Change Your Eating Habits - Proper Eating for Good Health (2020).

Complex Regional Pain Syndrome (CRPS): Patients' Perspective of Living in Chronic Pain: Volume II (2020).

Complex Regional Pain Syndrome (CRPS): Patients' Picture eBook Guide: Volume II (2020).

What is CRPS? A Helpful Guide to Teach Children About Complex Regional Pain Syndrome (CRPS): Volume III (2020).

Complex Regional Pain Syndrome (CRPS) and Amputation: A Patients' Picture eBook Guide: Volume III (2021).

Complex Regional Pain Syndrome (CRPS) and Amputation: A Difficult Decision to Make: Volume IV (2021).

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Complex Regional Pain Syndrome (CRPS): Patients' Perspective of Living in Chronic Pain: Volume V (2021).

Complex Regional Pain Syndrome (CRPS): Patients' Perspective of Living in Chronic Pain: Volume VI (2022).

Complex Regional Pain Syndrome (CRPS): Patients' Perspective of Living in Chronic Pain: Volume VII (2023).

Complex Regional Pain Syndrome (CRPS): Learning About The Different Aspects of a Painful Syndrome: Volume I (2022).

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Complex Regional Pain Syndrome (CRPS): Learning About The Different Aspects of a Painful Syndrome: Volume III (2023).

Complex Regional Pain Syndrome (CRPS): Learning About The Different Aspects of a Painful Syndrome: Volume IV (2023).

Complex Regional Pain Syndrome (CRPS): Learning About The Different Aspects of a Painful Syndrome: Volume V (2024)

Complex Regional Pain Syndrome (CRPS): Learning About The Different Aspects of a Painful Syndrome: Volume VI (2024).

Complex Regional Pain Syndrome (CRPS): Learning About The Different Aspects of a Painful Syndrome: Volume VII (2024).

Complex Regional Pain Syndrome (CRPS): Learning About The Different Aspects of a Painful Syndrome: Volume VIII (2025).

IN MEMORIAM

Doctor Hooshang Hooshmand (1934-2019)

Doctor Hooshang Hooshmand, dedicated his life to medicine (Neurology), teaching, and caring for his patients for over 40-years.

He had a special interest in the management of electrical injuries, lightning strikes, reflex sympathetic dystrophy (RSD), and other neurological conditions such as multiple sclerosis (MS), and epilepsy.

Doctor Hooshmand had a great philosophy and understanding of medicine. He was ahead of his time and always looked outside of the box with his approach to treating such conditions as electrical injuries, lightning strikes, and RSD.

His philosophy on treating electrical injuries, and lightning strike injuries was to approach it in a multidisciplinary fashion with the use of proper non-addictive medications, proper nerve blocks, proper physical therapy, having a proper diet, and avoiding unnecessary surgeries.

He was a great advocate for his patients. He truly loved helping his patients to receive the proper treatment and care they needed. He always tried giving them a better quality of life from the pain they were suffering. He always treated his patients with compassion and with the utmost respect.

I consider myself incredibly fortunate to have had the opportunity to know and collaborate with Doctor Hooshmand for more than 25 years. He has been an exceptional mentor and a true friend, providing guidance that is hard to find. My gratitude for his willingness to share his vast knowledge with me and others is immeasurable.

He has now left us for his next journey in life, but he has left a lasting impression on so many of us that we can never repay him back. His life's work through his research and his writings will help many people for generations to come. I am saddened that Doctor Hoosh (as I would call him) is unable to see the completion of this book, but I know he would be proud that it is finally finished.

Eric M. Phillips

QUOTE BY: DOCTOR HOOSHANG HOOSHMAND

“WE DOCTORS think we are god. We blame the patient if we can’t find anything wrong with them. We tell patients with electrical injuries it’s all in their heads. Well, it is in their heads-their brain cells are cooked.”

-Hooshang Hooshmand, M.D.

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PREFACE

Electrical injuries are not uncommon, and they occur more frequently than one might expect, highlighting the importance of awareness and safety measures in environments where electrical hazards are present. These injuries can range from minor electric shocks to severe electric burns, lightning strikes, and even fatalities, depending on the voltage and duration of exposure.

Individuals working in construction, maintenance, or any field involving electrical systems are particularly at risk, as they may encounter live wires or faulty equipment. Also, there are electrical injuries seen in the home environment as well as.

Lightning causes more deaths than any other weather phenomenon (1). The fatal incidence of electrical injuries is figured at five per one million or 1200 to 1300 deaths a year in the United States of America (2-8). According to the National Oceanic and Atmospheric Administration (NOAA), millions of lightnings striking the mainland USA result in approximately 600 lightning deaths (approximately 100 in Florida alone) each year. The clinical manifestations are different in early and late stages. The late development of neurologic complications makes the correct diagnosis more difficult (7-21).

Electricity is a tool utilized to perform work. Like all other tools, it is both a beneficial instrument and a potential hazard. The beneficial effect of the electricity is channeling its power, which is also called electromotive force (EMF) (electromotive force) or voltage (V). It is the method by which the power of electricity is transformed into work that makes it a potential hazard to the human organism.

INTRODUCTION

From January 1990 through January 1998 a total of 984 home bound, industrial, and lightning strike electrical injury patients were evaluated at our clinic in Vero Beach, Florida. Among the 984 patients studied, 459 patients were monitored over a period ranging from three to eight years.

The age at onset varied significantly, occurring in individuals as young as two years of age and as old as 68 years. Among the patients, 74% were male, while 26% were female.

The majority of industrial injury victims were male, while there was no significant gender preference observed for household or lightning-related injuries. Among the 98 cases of lightning strike injuries, all occurred in workplace settings. Additionally, there was an overlap of 11 patients who were classified under both industrial and electrical injuries.

Electrical injuries result in both acute stage as well as chronic clinical manifestations. In the acute stage the nervous system, blood vessels, visceral organs, heart, skin, and muscles were at high risk. In chronic stages, the nervous system involvement was in the form of electroporation (gradual deterioration of defective cell membranes), resulting in latent neurologic complications.

The alternate current (AC) was three times more damaging to the heart (due to tetanic contraction)and direct current (DC) of lightning strike.

The critical factors determining mortality and morbidity are low skin resistance (wet skin), the length of exposure, and the resultant high amperage affecting the body. The voltage is less important factor.

The high voltage meeting a dry, resistant skin with a short temporal exposure, may cause a significant skin burn. but the majority of the electrical thermal energy is consumed at the expense of the skin burn. As a result, less current (amperes) shields the skin.

The length of exposure and wet skin allow more current (amperage) to pass through the heart and central nervous system (CNS).

The low voltage currents (110-240v) are usually more likely to cause mortality and morbidity than the high voltage current (over 1,000v). This is due to the temporal factor of "no-let-go-phenomenon." Whereas high voltages over 1,000v, such as seen in lightning strike and electric and telephone line workers, a have a tendency to arc and jump over the dry skin in search of a point of lower resistance to penetrate, the low voltage currents (110-240v) are commonly seen in house or place of work injuries. The low voltage currents are more likely to cause mortality and morbidity than the high voltage current due to the lengthy exposure through the "no-let-go-phenomenon", and due to the improper use of ground fault interrupters (GFI). The reflex contraction of the muscles of the hand causing "no-let-go-phenomenon" allows the passage of current (amperes) through the skin during longer period of time, and causing more damage to the heart and brain.

The wet skin causing low resistance allows more current to enter the body. The combination of wet skin, length of exposure, and the critical points of entrance and exit are the risk factors overshadowing the electrical potential (voltage) factor.

Whereas the lightning strike usually enters and exits the body vertically, causing more damage through the CNS, guts, and the lower extremities (feet), the entrance and exit of the AC current are usually hand to hand or hand to foot. The hand to hand exposure is a high risk factor due to passage of electricity across the body through the heart.

The electrical injury usually causes a typical clinical picture which cannot usually be mistaken for other diseases.

However, the electricity entering the body through moist skin can cause very little if no scar while it is causing maximum damage. For this reason, the diagnostic tools for measurement of the electrical injury damage and dysfunction should be mainly physiological tests such as careful neurologic examination, somatosensory evoked potential (SSEP), infrared thermal imaging (ITI) (thermography), positron emission tomography (PET), and objective neuropsychometric testing such as Halstead–Reitan neuropsychological test battery (HRNB). Anatomical tests such as CT scan and MRI, as well as standard electroencephalogram (EEG) which mainly studies the cerebral hemispheres, is not sensitive enough for proper diagnosis of long term neurologic complications of electrical injuries.

The enforcement of electrical safety at the work place (proper use of breakers, GFI, and proper protective hats), and the use of mobile telephones are a few of the life-saving precautionary measures.

Treatment with anticonvulsants, proper antidepressants, avoidance of surgery, detoxification from habituating drugs, and (in advances stages), the use of infusion pump are helpful in the management of electrical injuries.

CHAPTER 1
CLINICAL MANIFESTATIONS

The electrical injuries (EI) result in acute and chronic clinical manifestations. In the acute stage, the critical vital target is the heart. Exposure to the electricity of 60 milliamperes or higher has the potential of causing cardiac arrest and death (1). The cardiac arrest among the electrical injury patients is different than that seen in other types of cardiac pathology. Electrical injury patients have an excellent chance of survival with resuscitation (22). Successful resuscitation after several minutes (up to 20 minutes or longer) is not uncommon(10). This is due to the fact that the electrical injury causes an electric shock on the cardiac plexus which has excellent potential for being reversible(1,10,22). The asystole is practically instantaneous in severe cases, and can be reversed with persistent resuscitation and electrical stimulation (1). This is in contrast with other arrests (such as coronary artery disease). The death is not necessarily due to myocardial infarction, but due to the prolonged cardiac arrest. Extensive work-up in the form of stress test usually does not show coronary dysfunction (23,24).

The other acute complications of electrical injury consist of extensive burns with secondary complications of infection and shock; damage to gastrointestinal, liver, kidney, muscle (myoglobinuria), peripheral nerves, and central nervous system (CNS).

In 1934 Doctor MacDonald Critchley, reported that "Doctors have advised the efforts of resuscitation to be abandoned: the workmen, however, have continued the efforts after the doctor's departure and the victim's life thereby saved"(10).

The neurologic manifestations of acute and chronic electrical injury (EI) are summarized into four categories (Table 1).

The long-term neurological side effects of the EI are quite stereotyped in nature (Figure 1). In mild to severe cases of chronic EI, similar selective neurologic deficits are noted in different degrees. The stereotypical clinical findings as outlined below, are quite consistent (Figure 1)(3):

- Too stereotypical and complex.

- Too selective and discriminative in organs involved (e.g., spinal cords, brain stem, and limbic system).

- Too consistent and predictable among different types of EI victims to be hap-hazardous, or "functional" due to any tendency by the patients to concoct such complex neurologic deficits.

TABLE 1 THE CATAGORIES OF NEUROLOGICAL MANIFESTATIONS OF ACUTE AND CHRONIC ELECTRICAL INJURIES (EI)
High voltage alternate current (A.C.)
Low voltage alternate current (A.C.)
Direct current (D.C.) (the use of an instrument, e.g., paint spray gun)
Lightning injuries

THE EFFECTS OF ELECTRICITY ON BODY TISSUES

Electricity can affect all the three main physiologic functions of electrical, magnetic, and chemical balances of the body. It can cause electrothermal damage, electromagnetic dysfunction, and cell membrane electrical and chemical dysfunctions. The electromotive force, (electromotive force, (EMF, often denoted as E) is utilized from work. Like all other tools, it is potentially both beneficial and hazardous. The beneficial effects of the electricity is channeling its power for electrical communication. The electric force (E, or V) and the current intensity (I), in the path are hampered by resistance (R). The resistance converts and wastes the electricity to heat (watt or W).

- $E\ (V) = RxI$
- W (watt)

Electric power for residential use is 120-240v. Factories require 500-660v. Transferring electricity to remote areas is hampered by the distance which wastes voltage energy. To compensate for this waste, the electricity has to be transferred in high voltage (7,000-56,000 volts or higher).

Following the principle of the above Joule's Law, $P=RI^2$, it becomes clear that if the skin resistance is low (moist skin), then the same constant power of electricity, or voltage, causes high current to enter the skin and vice versa. The critical vital factor in regard to mortality and morbidity is not the power of electricity (voltage-V), but the intensity of the current (I, or amperes). It is the current or amperes (I) that causes the death of myocardium or nerve cells.

The power entering the body through wet skin meets very low resistance. The normal dry skin resistance is 100,000 to 300,000 Ohms. On the other hand, wet skin has no more than a few thousand Ohms. The most descriptive example is a woman in the bathtub using an electric hair curling iron with an electric short in it. The electric short charges the entire water in the bathtub, causing instantaneous death without a single scar anywhere over the patient's body. The ultimate humidity opens the gate for the current (I), to enter the deep structures.

Paradoxically, a dry skin causes a high resistance (R) resulting in generation of electrothermal heat and microwave affect. The dryer the skin, the more resistance against the electricity and more severe skin burn.

The electrothermal waste of energy due to high resistance allows less current (I) to enter the deep structures with less damage to the nerves, blood vessels, and brain. The dry human skin is the best natural protective shield against the damaging effects of electric power on the heart and nervous system.

The skin serves as a remarkably effective protective barrier against the wide range of potentially noxious chemical, physical, biological, and electrical factors. The outer layers of the cells contain keratin, a sulfur-containing fibrous protein, renders the skin relatively inert. The skin is electro-resistant and thermo-resistant. The key to protecting the inner organs from medical shock is to prevent the initial excitation of the electron. Under simple conditions, this can be accomplished easily with gloves, rubber soles shoes, grounding wrist straps, and various other insulators. The resistance to electron flow increases when no positive pole is provided at attack it. Standing on a wooden surface with rubber soled shoes, limits the body from assuming a positive ground potential. This prevents the flow of the electrons and disrupts the circuit.

AII the above mentioned insulators are relative in their insulating action, and can be overcome by intensity of the electric power. Due to the extreme electromotive force (E), the very high voltage electricity is quite likely to find the easiest path to ground. This phenomenon is referred to as arcing, which plays a major role in the presentation of otherwise resistant skin. In rare individuals who have been struck by electricity three or more times, the infrared thermography shows preexisting patches of very low resistance in the skin identified on the infrared imaging as heat leakage. These multiple patches of very low resistance allow the arcing to take place and the lightning to enter the otherwise dry and highly resistant skin. Arcing provides the heat that burns the skin through its microwave affect. This is due to the physiology of the subdermal layers of the skin controlled by the sympathetic system.

The microwave effect of the electricity entering through the lower resistant areas of the skin causes bombarding of water and blood molecules within the skin with electromagnetic radiation, exciting the subatomic particle and thus creating heat. This gives rise to the popular notion that microwaves cook from the "inside out." This is the reason the microwaves are unable to "brown" the outside of a roast.

The lack of browning and toasting effect of the microwave on the skin by the lightning strike phenomenon of arcing, causes minimal if any discoloration of the skin in the face of the damage to subdermal structures which is usually deeper than it meets the eyes during the acute phase of the electrothermal burn.

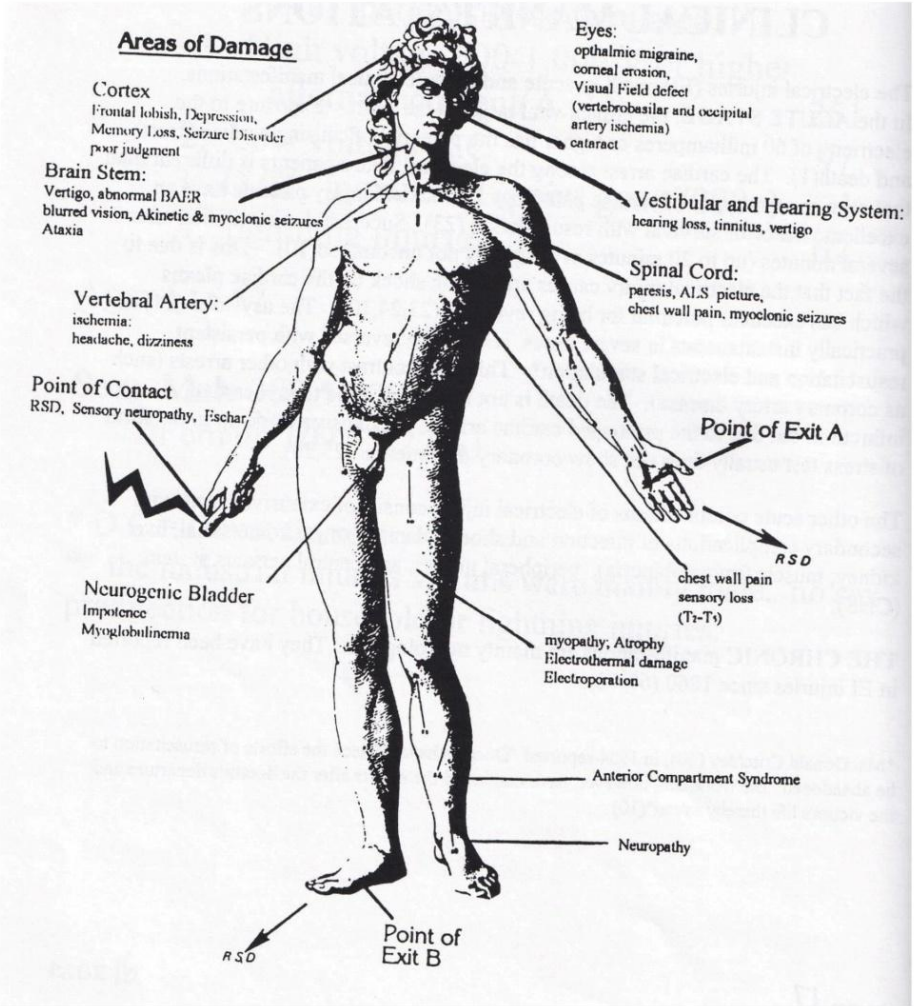
DIELECTRIC EFFECT

In AC electrical injuries, the skin acts as a capacitor. The skin separates the negatively charged conductor from the positively charged non-conductive membrane (dielectric). The current will tend to build up on each side of the dielectric until it can no longer be contained. Then the current jumps the dielectric barrier. This results in an immediate rise in the current (I) and penetrance through the skin. What may limit the capacitance effect in the skin penetration is the small

exposure area. The more focused the area of the entrance of electricity, the more easily it breaks down the capacitance effect of the skin. This is usually noted as an area of the skin that is left with less keratin and more locally thinly damaged and scared layers of the skin.

These areas of electric penetration are best identified on infrared thermal imaging test. The damaged area shows a "button-hole" The center of the button-hole is the area of the least capacitance allowing heat leakage hyperthermia. This area is surrounded by a reactive hypothermic circle as a defense mechanism by the sympathetic vasoconstriction to reduce heat leakage and waste (Figure 1).

FIGURE 1. NEUROPSYCHOLOGICAL COMPLICATIONS OF ELECTRICAL INJURIES (EI) (28)
H. Hooshmand, M.D.



CHAPTER 2
THE FOUR STAGES OF ELECTRICAL INJURIES

The clinical manifestations have been divided into four stages (1,8). The neurologic manifestations in acute (stage 1) and subacute (stage 2) stages are usually temporary in nature. However, latent complications may appear months, or years after the accident.

STAGE 1: ON THE SCENE OF THE ACCIDENT(28)

1. No-let-go-phenomenon (22-27). This was present in every patient with exposure to electricity to the palm of the hand. However, even the trunk of the body or lateral aspects of arm or leg can become attached to the electric source. The "no-let-go-phenomenon" means the electrical stimulation causes flexor spasm and tetanic contraction of the muscles not allowing the victim to let go of the electrical source.

This phenomenon does not apply to lightning strike victims hit directly by the thunderbolt, pointing to better prognosis in regard to exposure time to lightning (which is usually quite brief, measured in a few milliseconds).

2. Ipsilateral extremity burn, eschar, and neurosensory damage. The same phenomenon to a lesser extent is present in the exit points of the electricity. The lesion specifically involves c fiber nerve: e.g., nerves around the arterioles (sympathetic nerve fibers). As a result, the patient has severe pain which extends beyond the eschar region.

3. The body is usually thrown away from the source of electricity in a massive myoclonic jerk of the paraspinal muscles. This results in disengagement of the victim from the electric source at the expense of falling from a ladder or other heights with secondary injuries (e.g., fracture of spine or skull).

4. Tonic, and at times, tonic-clonic seizures of brief duration, at times followed by a brief loss of consciousness.

5. Cardiac arrhythmias and cardiac arrest with good potential response to resuscitation (10,22); followed by typical autonomic dysfunction in the form of abnormal cardiac rhythm, fluctuating blood pressure, abdominal and chest pain. Attacks of apnea are not infrequent.

In lightning strike victims, the electricity traverses vertically (air to ground or ground to air) with tendency for GI damage (throat, esophagus, and guts).

6. Skin burns. Blisters over the fingers, acute sympathetic dysfunction of extremity, reddish discoloration over the entrance and exit points, as well as reddish discolorations over the anterior chest wall at T2 through T5 levels, which are the points of entrance of electricity through vascular and sympathetic fibers to the spinal cord. The burns in the first one to two days may appear limited to extent and depth, only to show more extensive damage in the following week.

7. Amnesia or loss of consciousness (LOC). In our series, amnesia was seen six times as common as LOC. LOC is usually a sign of temporary or prolonged brain stem dysfunction. The memory loss implies limbic system (especially temporal lobes) dysfunctions, and is more apt to result in long-term cerebral dysfunction and disability (8).

STAGE 2: HOSPITALIZATION(28)

1. The patient is quite drowsy, tired, and may be confused.

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. In lightning strike victims, severe abdominal pain and diarrhea starts at this stage.

3. Akinetic and myoclonic seizures (infrequent).

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.

6. Sensory loss over the trunk distal to the T-2 through T-5 entrance of electricity to the spinal cord. The sensory loss is usually asymmetrical, and the patient occasionally develops a partial Brown-Sequard syndrome. This aspect of the examination should be checked on every patient. This is a frequently overlooked and under diagnosed sign of electrical injury (Tables 2 and 3). Only in severe electrical exposures the patient suffers from overt spinal cord Keraunoparalysis (lightning paralysis). However, the majority of patients (72%), show chest wall sensory loss, and abnormal SSEP test (63%) (Table 3). This sign of spinal cord dysfunction explains the reason for the patient having neurogenic bladder, impotence, incontinence, as well as myoclonic and akinetic seizures too deep to be recorded on EEG.

7. Frontal lobe dysfunction (FLD): tremor, positive snout reflex, masked facies, irritability, and poor judgment (Table 2 and 3). In acute hospitalization stage the patients may manifest tendency for indifference, emotional withdrawal, and flat affect. This has been referred to as a "frontal lobotomy" picture by the relatives and nurses. The same patients subsequently may develop irritability, agitation, and bouts of violent behavior-eventually leading to divorce or unemployment.

STAGE 3: FIRST FEW WEEKS TO MONTHS AFTER THE ACCIDENT(28)

1. Pain in extremities, hyperpathia and allodynia (Table 2).

2. Akinetic or myoclonic seizures.

3. Anxiety, agitation, phobia, and irritability.

4. Labile neurovascular symptoms and signs:

- Cardiac arrhythmias
- Sympathetically mediated pain (SMP) and, less frequently, Reflex sympathetic dystrophy (RSD) (10,28-30).
- Labile blood pressure, orthostatic hypotension.
- Abdominal cramps.
- Diarrhea.
- Noncardiac origin chest wall pain usually in the distribution of T-2 through T-5 nerve roots due to spinal cord dysfunction.

5. Poor recall, poor recent memory.

6. Depression and secondary insomnia.

7. Frontal lobe dysfunction: irritability, anxiety, agitation, phobia, tremor, poor judgment, poor physical tolerance, and fatigue.

STAGE 4: OVER SIX MONTHS (28)

1. Loss of job (over 50% of the patients) (Tables 2 and 3)(8).

2. Loss of spouse, severe marital interpersonal strain (Tables 2 and 3) (8).

3. Vertigo and tinnitus in one-third of patients(Tables 2 and 3).

4. Depression or schizoaffective withdrawal, anxiety, phobia, organic brain syndrome. No definite generic PTSD was seen in our series.

5. Akinetic and myoclonic seizures.

6. Poor recall and recent memory.

7. Painful extremity (chronic pain).

8. Reflex sympathetic dystrophy (RSD) (38%) or sympathetic mediated pain (72%)(Tables 2 and 3)(10, 28-30).

9. Impotence, neurogenic bladder, abdominal cramps.

10. Progressive muscle atrophy and spasticity (a picture of Lou Gehrig Disease (ALS)); tremor (Tables 2 and 3) (14,31).

11. Progressive paralysis (Kerauno-paralysis) (10).

12. Disturbance of immune system (18%).

13. Cataract (7%).

14. Severe fatigue (Tables 2 and 3).

15. Neurodermatitis (8%).

The neurologic manifestations in electrical injuries are different from those of traumatic, anoxic or vascular cerebral injuries. In head injuries, the patients go through a critical stage of approximately two years during which they usually become more stable. Even a few years after the trauma the patient's neurologic status may partially improve. In the electrical injury patients, a biphasic "double hump" course of aggravation of the neurologic deficits is noted. Whereas the patient's neurological state improves in the first 18 to 24 months, in 36% of patients a latent deterioration emerges in the form of progressively deteriorating memory loss, poor judgment, seizure disorder, disturbance of interpersonal relationship (loss of spouse, loss of job, etc.), development of cataract, atrophy of the muscles almost resembling amyotrophic lateral sclerosis (ALS) (Lou Gehrig Disease), peripheral neuropathy, or the development of autonomic nervous

system dysfunction in the form of weight loss, orthostatic hypotension, and sympathetically mediated pain (SMP)(12,14,28,29,31).

This biphasic deterioration of neurologic complications seems to be due to the principle of "electroporation" which is outlined below under the risk factors (32-35). The electroporation is a major physical phenomenon in electrical injuries leading to a latent deterioration in neurologic function secondary to the direct electrical damage on the cell membranes. The electricity causes the formation of holes (pores) in the wall of the nerve fibres-hence called electroporation. another 7% shows mixed electrothermal and direct electroporation damage to nerve cell membranes with resultant gradual leakage and loss of biogenic amines and other vital chemicals (32-35).

In our series of 984 patients, 36% of the patients who were followed for three years or longer showed the above "double hump" biphasic deterioration. These were usually more severe in lightning strike or industrial injuries manifesting themselves acutely with very little skin burn due to the low resistant moist skin (which allowed more current to enter the body).

For further detail see the "risk factors" outlined in chapter 8.

TABLE 2. A COMPARISON OF THE SYMPTOMS OF NEUROLOGICAL ASSOCIATES PATIENTS WITH L.S.V. SYMPOMS VERSUS THE SYMPTOMS OF ENGELSTATTER AND ASSOCIATES		
SYMPTOMS	**NEURO ASSOCIATES**	**ENGELSTATTER ASSOCIATES**
Depression	88%	63%
Memory loss	78%	71%
Sleep disturbance (insomnia: 2 cases of malignant insomnia)	76%	74%
Fatigue (chronic)	72%	48%
Easy fatiguability	37%	0
Chest pain	68%	16%
Vertigo	66%	48%
Pain (excluding chest)	62%	65%
Epileptic seizures	32%	24%
Pseudo seizures	1.25%	Not specified
Tinnitus	49%	48%
Loss of job	68%	57%
Divorce	56%	5%
Frontal lobe (poor judgment, irritability, distractibility, adult ADHA, and Wizelsucht)	62%	60%
Muscle spasms	64%	63%
Paraesthesia	66%	63%
External burns (sometimes extremely small in the form of punctate burns)	72%	54%
Disturbance of libido	60%	55%
Neurogenic bladder	36%	35%
Akathesis	48%	45%
Personality change	42%	29%
Latent neurologic deterioration (electroporation)	36%	Not specified
Movement disorder (tremor, rigidity, etc.)	38%	63%

TABLE 3. NEUROPSYCHOLOGICAL FINDINGS (SIGNS)		
SYMPTOMS	**NEUROLOGICAL ASSOCIATES**	**ENGELSTATTER ASSOCIATES**
Depression	88%	32%
Memory loss	78%	71%
Attention and concentration	46%	48%
Irritability and agitation	44%	42%
Sensory loss and allodynia in extremities	74%	Not specified
Anterior chest wall sensory loss	72%	Not specified
Mild hearing loss (left ear 46%, right ear 24%)	70%	25%
Ataxia	56%	40%
GI symptoms and findings: diarrhea, weight loss, persistent vomiting, gastric irritation, (mostly lightning strike patients)	26%	Not specified
Dry eyes (Sjogren's syndrome) corneal irritation	16%	11%
Hypertension	49%	48%
Speech and communication problems.	28%	32%
Apraxia (clumsiness and disturbance of skillful function)	26%	Not specified
Attacks of confusion, distractibility and easily becoming frustrated	32%	34%
Somatosensory evoked potential (SSE) pointing to thoracic spinal cord dysfunction	63%	Not specified
Brain stem evoked potential (BAER) slowing of conduction mainly in the lower brain stem.	48%	Not specified
Positive snout reflex	22%	Not specified
Perineal "onion skin" sensory loss	27%	Not specified
Trigeminal sensory loss	26%	28%
Sympathetic maintained pain (SMP)	72%	28%
A full blown RSD (80) along with tremor, etc.	38%	Not specified
Totally disabled from work	52%	54%
Totally dependent on others	12%	19%
Suicidal	15%	17%
The neuropsychological changes were mainly the manifestations of limbic dysfunction.		

CHAPTER 3
SYMPTOMS

The symptoms of the electrical injuries are relatively stereotyped in nature (Figure 1). To understand the symptoms and signs, one has to understand how the electricity affects the human body.

Electricity follows the path of the least resistance. The electricity does not cause neurologic complications unless the resistance of the skin is broken down and the current penetrates the body. The patient may suffer from extensive skin burn, yet may be neurologically intact-and vice versa.

Usually, in low voltage AC (110-240) injuries, the severity of the neurologic manifestations has an inverse relation with the degree of the skin burn. The dryer the skin, the more skin burn, the more electricity is absorbed and dispersed at the surface and the less electricity enters the body (36). According to the studies by Gourbiere et al, at Electricite de France (EDF), 78% of electrical burns are arc burns: similar to non-electrical, or thermal burn; with damage being limited to skin (36). Only 15% of the electrical burns were electrothermal, allowing the passage of electricity through the body (36). This passage of electricity results in damage to the deep structures-including electroporation. Another 7% shows mixed electrothermal and arc burns (36).

After the electricity penetrates the protective shield of the skin, the current follows the path of the least resistance. This path is the oxygenated, rapidly moving, warm, and ionized arterial blood. As the electricity follows the conductive arteries, and arterioles it causes burn and damage at the wall of the blood vessels with resultant injury to the small c-fiber nerves in the wall of the blood vessels (the thermoreceptor afferent nerves of the sympathetic reflex arch).

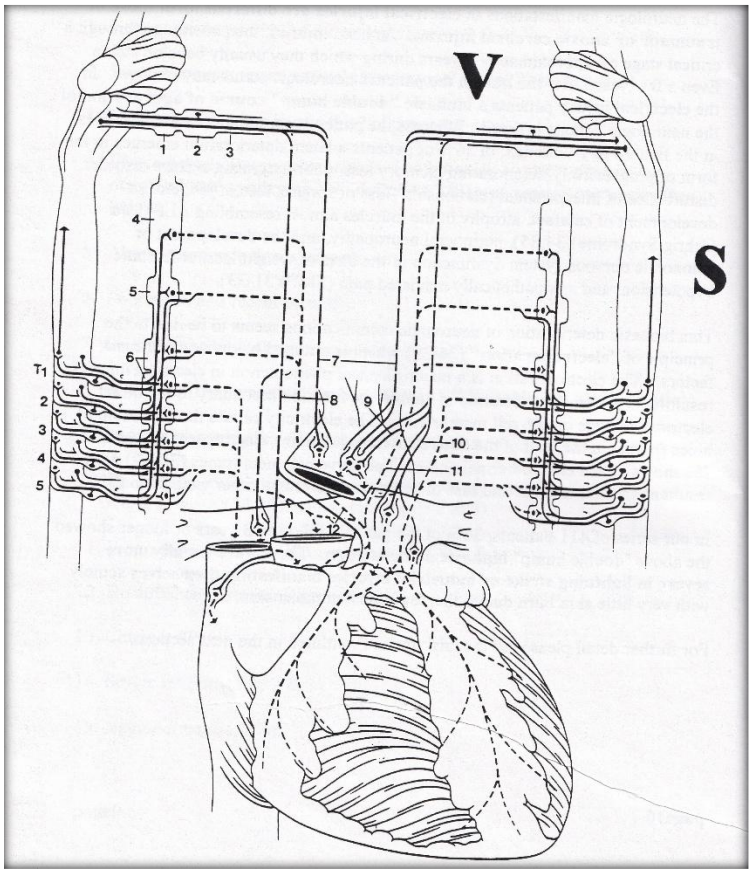
As the electricity follows the arteries to the heart, it affects the precordial sympathetic plexus. This results in the development of chest pain, fibrillation, and cardiac arrest.

Simultaneously, the electricity follows the sympathetic nerves as they enter the spinal cord at the upper thoracic cord level (at T2 to T4 levels) (Figures 2 and 3) (16,37-40). From there the electricity follows the sensory nerves to the spinal cord, it causes dysfunction of the spinal cord in the form of moderate sensory loss over the anterior chest wall, spasticity especially in the lower extremities, paralysis and weakness in the extremities, urinary urgency, frequency, or incontinence and impotence.

Somatosensory evoked potential (SSEP) is abnormal in 63% of electrical injury patients (8,41). The abnormality is limited to thoracic spinal cord conduction in 92% of these patients. The sparing of cervical spinal cord is another fact corroborative of the passage of electricity through the afferent sympathetic fibres to the thoracic spinal cord.

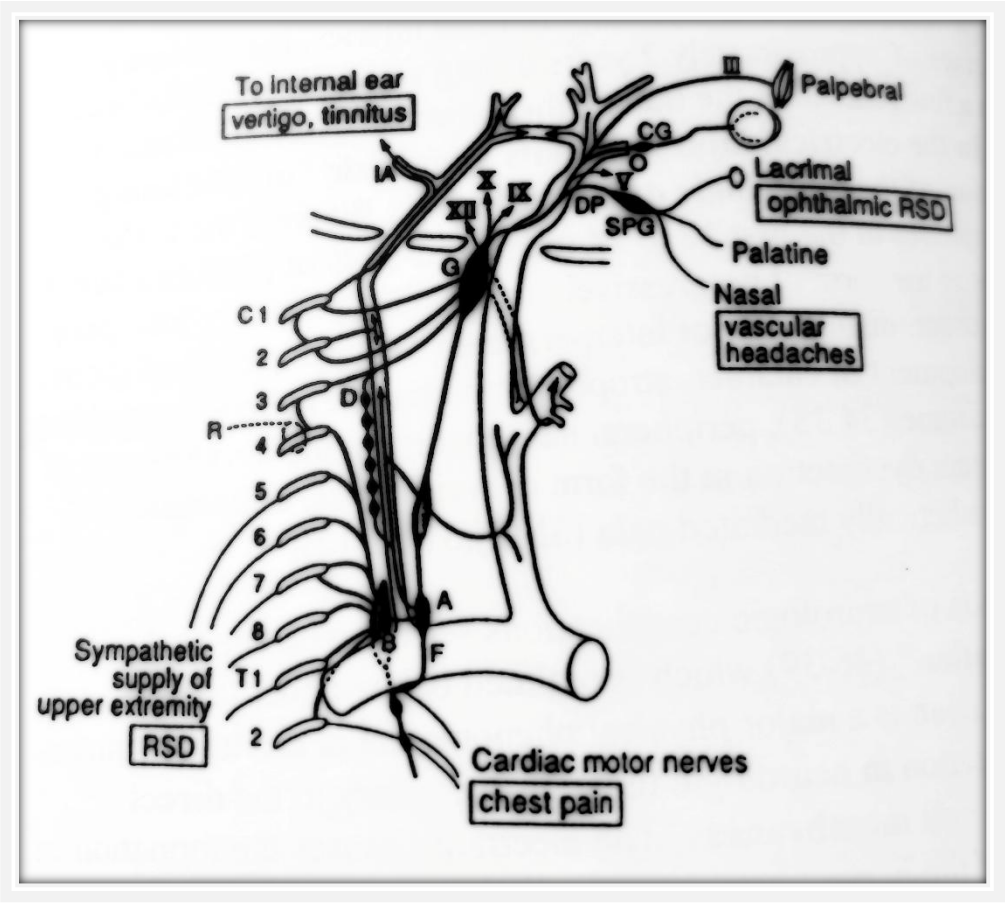
Figure 2. The sympathetic sensory nerves enter the spinal cord from T1-T5 levels. This may explain the patient's chest wall pain and numbness in Electrical injuries.

Vagal (V) and sympathetic (S) relation to cardiac plexus and the spinal cord.



(Reproduced with Permission)

Figure 3. Sympathetic system around blood vessels of head and neck. Electrical injury affects this system causing severe headaches, dizziness, tinnitus and chest pain (28).



As the electricity ascends through the least resistant and the least myelinated (spinoreticular) fibers, it causes damage to the nerves in the brain stem with resultant unilateral hearing loss, dizziness, tinnitus, and temporary loss of consciousness (the hearing loss is twice as common on left side regardless of points of entrance and exit (Table 3)(8,42). Simultaneously, the electrical current stimulates the nerves around the vertebral arteries in the cervical spine resulting in vasoconstriction and secondary dizziness, syncopal attacks, loss of consciousness, and vascular headaches. Further damage to spinal cord and brain stem causes myoclonic and akinetic seizures (8). Following the ascending thalamocortical fibers, the electricity ascends to the thalamic nuclei and from there it follows the path of the nerves to the frontal polar and temporal regions (limbic system)(8,43).

The limbic system is the system responsible for memory, judgment, and mood. As the electricity causes dysfunction of the limbic system, the patient becomes irritable, edgy, short fused, depressed, insomniac, uses poor judgment, and is handicapped by significant recall and recent memory disturbance.

Halstead Reitan psychological evaluation was compared with computerized mapping in 316 of 984 electrical injury patients: There was over 90% overlap of localization between the two tests (8). Over 80% of abnormal Halstead Reitan test involved anterior head regions. This was true even among the cases with evidence of multifocal abnormalities in anterior, as well as posterior (parieto-occipital) head regions.

On the basis of the above background of electrical dysfunction, the symptoms of our 984 cases were compared with the symptoms outlined in a study by Doctor G.H. Engelstatter, Ph.D. (Table 2).

The fact that two independent groups of investigators have come up with such close statistical data, points to a stereotyped pattern of symptoms and signs.

The symptoms as well as the signs discussed below are representative of the path of the electricity through the body. As noted on, neuropsychological symptoms are quite frequent (Tables 2 and 3). This does not imply that the patient is imagining them or that the symptoms are not on "organic basis." The fact that we did not encounter a generic PTSD (as defined by American psychiatric Association) in 984 patients underlines the organic basis of neuropsychologic symptoms and signs (44).

CHAPTER 4
SIGNS

The neurological and neuropsychological findings (signs) are dependent on how carefully and extensively the neurologic examination was done. For example, one common sign of the electrical injury victims is anterior chest wall sensory loss (Figure 3). This was present in 72% of our cases. Every patient, to the last person, said that prior to our examination this aspect of sensory examination was not checked. The other signs commonly not checked were snout reflex and corneal reflex which was positive successively in 22% and 20% of our cases. Finally, in patients with neurogenic bladder or impotence, a perineal "onion peeling" sensory loss was found on one side of the perianal regions, pointing to unilateral damage to the spinal cord in 8% of the patients. Again, no patient nor the relatives could recall if such a test were previously carried out.

The neurological residuals follow the path of the least resistance of electricity: arterial circulation, small efferent c-fibers in the wall of the arteries causing pain and sensory loss in the thermatomal and brachial plexus distribution nerves, cardiac plexus involvement, sensory loss over T2 through T5 nerve roots distribution, thoracic spinal cord dysfunction (myoclonic seizures, spasticity, impotence), brain stem dysfunction (vertigo, ataxia, hearing loss), and limbic system dysfunction (memory loss, depression, and insomnia) (Figures 1 and 3) (37).

One significant observation was absence of post-traumatic stress disorder (PTSD) as defined by the American Psychiatric Association (44). According to the APA definition, PTSD is a result of an experience "outside the range of usual human experience and that would be markedly distressing to almost anyone. The traumatic event is persistently experienced, and persistent avoidance of stimuli associated with the trauma or numbing of generalized responsiveness occurs. Persistent symptoms of increased arousal are present." In electrical injury patients, the patients usually suffer from amnesia; hence, they are not capable of "persistently experienced" memory rumination as seen in PTSD. Simple symptoms of nightmares, phobia, or anxiety are not enough for the diagnosis of PTSD.

The frontal lobe personality disorder in the form of poor judgment, irritability, agitation, being short fused, having a problem with interpersonal relationship was seen in more than half the patients (62%).

On the other hand, conversion disorder was seen in rare cases. Twenty patients (1.25%) suffered from conversion reaction (44). Four of these patients (20%) manifested pseudo seizures. In one patient both seizure and pseudo seizure were recorded.

The majority of the attacks of seizures were in the form of myoclonic or akinetic seizures (17%), complex partial seizures (11%), and only 5% suffered from major motor seizures.

One difficulty in the diagnosis of seizure disorder among electrical injury patients is the fact that in the akinetic and myoclonic seizure patients, 60% do not have surface EEG abnormalities, but demonstrate significant abnormality on SSEP test (8,41). In such patients, treatment with anticonvulsants such as Depakote or Klonopin in therapeutic doses completely stopped the seizures. Poor compliance (low drug level of blood) or discontinuation of the anticonvulsant, resulted in recurrence of the seizures. This finding is suggestive of the source of seizure disorder originating in the spinal cord or brain stem which would make it impractical to record the epileptiform discharge on surface scalp EEG.

CHAPTER 5
NEUROPSYCHOLOGICAL DYSFUNCTION IN ELECTRICAL INJURIES

Besides the Halstead Reitan Test showing significant cerebral dysfunction in our patients, similar to Daniel and colleagues, Minnesota Multiphasic Personality Inventory (MMPI) test showed a nonspecific tendency for somatic preoccupation, depression, and anxiety (8,45). Both in our previous study and in the experience of Daniel, et al, a high percentage of such patients have become disabled from work (8,45). In our previous study, 11 of 13 employed patients lost their jobs and 9 of 11 married patients were divorced during the late stages of electrical injury (8).

The high incidence of post traumatic depression among electrical injury victims points to the organic cerebral dysfunction due to the electrical injury rather than simple emotional effect of the victim's experience (e.g., post-traumatic stress disorder-PTSD) (2,8,45,46). The depression in electrical injury is not at all similar to the effect of electro-convulsive therapy (ECT) which has been used to successfully treat intractable major depression. According to Sackeim, et al, the epileptogenic effect of ECT does not seem to be the factor in improving a major depression (54). The condition seems to be quite complex, and some other effect of electricity may play a role in the paradoxical effect of ECT-versus accidental electrical injuries-on depression. Whereas electrical injuries definitely result in a significant degree of depression, ECT improves the acute episode of major depression (even though the patient continues to need to take antidepressants after the ECT (47).

The frontal lobes are the critical cerebral structures in ECT patients and in EI victims. However, the clinical outcome shows a marked contrast.

In the case of ECT, the electricity acutely damages the larger excitatory, pyramidal cells. This results in amelioration of agitation. On the other hand, in EI patients, the electroporation phenomenon affects the smaller inhibitory granular cells more severely. This may be due to the vulnerability of thinner cell membranes of the small inhibitory granular cells. As a result, lack of inhibition of the large pyramidal cells causes agitation, irritability, and distractibility (Adult attention deficit and hyperactive disorder - ADHD).

NEUROPSYCHOMETRIC TESTING (HALSTEAD REITAN)

The neuropsychological tests show abnormal cerebral function of multifocal nature (1,16,19-28). The Halstead Reitan neuropsychometric test was the most sensitive tool in our studies of evaluation of head injuries, and electrical injuries (8,48). The above studies have demonstrated the high yield and accuracy of Halstead Reitan test. In mild head injuries the CT scan and MRI had the lowest (11 of 135) and Halstead Reitan the highest diagnostic yield (over 80%) in assessment of the cerebral dysfunction among the electrical injury patients with 82% yield (48).

This test mainly pointed to anterior head regions, especially frontal lobes dysfunction in over 80% of the abnormal Halstead Reitan test group. This was in sharp contrast with abnormal neuropsychometric tests other than Halstead Reitan which were noted in only 58% of the electrical injury patients (8,47,48).

The main abnormalities on neuropsychological test results among the electrical injury patients were poor judgment, apraxia, and poor recall.

AREAS OF CEREBRAL HEMISPHERIC INVOLVEMENT

It is obvious that MRI or CT scan cannot localize the "software" abnormalities of the high cerebral functions in EI patients. Our previous studies demonstrated the sensitivity of Halstead Reitan test in localization of the cerebral dysfunction (8,48).

Other neuropsychometric tests such as MMPI have pointed to abnormalities in a high percentage of EI patients (45). However, the abnormalities have been nonspecific termed as depression, anxiety, and somatic pre-occupation. More recent studies have shown that the most widely used MMPI tests (MMPI-1, MMPI-2), even though illuminating, fail to address a more fundamental set of problems with personality assessment-hence inherently constraining and limiting their utility (49-52). To quote Gamsa, "the accumulated evidence fails to provide support for the central tenet of psychoanalytic explanations: that emotional conflicts give rise to bodily pain (50). Although such a formulation undoubtedly explains some causes of pain, the view that psychological causes generally explain otherwise undiagnosed pain is not supported.

Similarly, research has failed to identify a typical personality profile in patients with chronic pain" (50). It is interesting that in the first half of the 20th century the psychoanalytic school even created a personality profile of multiple sclerosis patients (called "MS personality"). In this regard, it is interesting to note that of all stressors aggravating and bringing on a new attack of MS, only electrical injuries have proven to do so with statistical certainty (53). It is a well-known fact that long term pain affects the normal psychological state of the mind; it can cause anxiety, depression and insomnia in the strongest character and personality type of patient. Whereas the diagnosis of PTSD has been assigned as an explanation to EI burns, subsequent authors have emphasized that the diagnosis of PTSD had not been clarified and had been used in a loose and generalized fashion (54,55).

It is true that the EI patients demonstrate high levels of distress, depressive symptoms, and a high rate of unemployment and divorce (8,32-35). The delayed manifestation of these neuropsychological symptoms is similar to the delayed neurologic complications such as neuropathies, ALS picture, muscle atrophy, and late onset of seizure disorder. The phenomenon of electroporation may explain the latent development of the above symptoms and signs (see electroporation in Chapter 8) (32-35). This phenomenon has been suspected since 1977 (16,39,40). The delayed manifestations of this phenomenon has been reported as early as 1934

(10). It would be illogical to think that the electroporation limits itself to spinal cord and peripheral nerves and spares the higher cerebral hemispheres. Daniel et al have reported deterioration of Halstead Reitan test after showing improvement in the first few months post electrical injury (also please see the PTSD versus depression discussed above; and APA definition of PTSD discussed in (Table 3) (47).

AVERSIVE CONDITIONING: Aversive conditioning has been suggested as an explanation of the PTSD among the electrical injury patients (54). On the other hand, the same visual or electrical trauma that may result in aversive conditioning can as easily be due to the direct effect of electrical injury on cerebral hemispheres.

It is well known that without the protection of surge-suppressors; the software of any computer is affected by lightning. The same susceptibility obviously is instrumental in the sensitive and complex computer of cerebral cortex.

The subject of conversion reaction (and pseudoseizure) is discussed in later chapters. The incidence of conversion reaction in our series was 1.25% (20 patients) four of these (25%) manifested pseudoseizures.

The fact that the psychological disturbances are associated in different degrees with neurological deficits which are quite stereotyped and the fact that the neurologic and neuropsychological disturbances occur whether or not the head is a point of direct contact for the electrical current makes it difficult to separate the neuropsychological effects as "functional" and yet to accept the neurologic deficits such as aphasia, seizure disorder, tinnitus, disturbance of memory, muscle atrophy, weakness, etc., as "organic" (Figure 1) (8,55,56)

Both among our 984 patients and in the literature it has been noted that the EI patients in acute stage were diagnosed as "essentially normal neurological examination" even though such patients suffered from disorientation and memory disturbance during and after hospitalization (55,57). The fact that at this stage the detailed psychological tests by a psychologist is not carried out, dilutes and sacrifices importance of neuropsychological cerebral dysfunction in such patients. Even in late (chronic) stages of the disease many of the neurologic findings are of "soft neurological sign" type-such as positive snout reflex (frontal lobe dysfunction), sensory loss in T3-T5 nerve roots distribution, mild neurosensory hearing loss, mild suppression of corneal reflex, and abnormal evoked potentials (Figure 1). Not looking for such subtle neurologic abnormalities does not prove their absence. Not performing neuropsychometric tests in early stages of the disease does not prove normalcy.

Few temporal serial follow up studies have been rarely done (8,58). Troster and Ruff studied three EI patients, one of which was serially assessed (58). That one patient showed the same disturbance of verbal learning and memory, arithmetic, logical, and abstract thinking as well as selective attention disorder as was noted in the 16 patients previously studied by us (8). One

handicap in the neuropsychological study of the EI patients has been lack of standardized tests which makes it difficult to compare different groups of patients. The Information Subset of the Wexler Memory Scale and the Memory Quotient Tests, which is a measure of immediate memory capacity has been found deficient in EI patients (55). However, tests such as the Luria-Nebraska Neuropsychological Battery (LNNB) or Halstead Reitan have not been routinely applied to EI patients.

As is the case with all the other manifestations of electrical injury, the degree of neuropsychological deficits is quite variable depending on the length of exposure time and the severity of the cerebral injury. However, as in the other aspects of neurologic deficits, the EI burn patients may have a limited or a temporary neuropsychological deficit (59,60). In regard to neurological and neuropsychological deficits, four different grades of neurological dysfunction are noted.

GRADE 1: In such patients both the neurological and neuropsychological findings are minimal, mild, and are referred to as "soft neurological and neuropsychological findings." The best example of this group is a brief exposure to lightning, or a high voltage electrical burn of low risk such as hand to the same arm or foot to the same leg electrical current. In such patients the burn may be extensive because of the dry skin, but the neurologic and neuropsychological complications are quite mild. Another good example of Grade 1 patients is when the alternate current does not cause "no-let-go-phenomenon": e.g., electrical contact to the dorsum of the forearm or to the anterior aspect of the thigh with brief electrical contact.

GRADE 2: Moderate electrical injuries. In this condition there are different degrees of electrical burn, but due to the length of exposure, moisture of the skin, or "no-let-go-phenomenon", the neurological and neuropsychological deficits are more evident. The more neurologic abnormalities, the more depression, memory loss, insomnia, disturbance of judgment, and frontal lobe behavior (attention deficit, irritability, poor judgment, and disturbance of interpersonal relationship).

GRADE 3: Severe electrical injuries. In these cases, chest pain, cardiac arrhythmias, and other cardiac abnormalities are quite common. The patient usually suffers from hearing loss mainly on the same side of the electrical injury. The neuropsychological tests show significant organic brain syndrome (OBS), and Halstead Reitan test shows more disturbance of function in multiple areas of cerebral hemispheres.

In this group, the lightning strike patients are more likely to demonstrate severe GI complications such as intestinal perforation, persistent severe abdominal pain and diarrhea, and weight loss. The high voltage alternate current patients are more likely to show sympathetic maintained pain (SMP) or RSD as well as headache, dizziness, tinnitus, and marked disturbance of memory.

Among the Grade 2 and Grade 3 patients, a biphasic phenomenon of frontal lobe dysfunction is

noted. Among 11 of the lightning strike and 34 of the DC patients, in the acute stage, a clinical picture of withdrawal, inactivity, indifference, and a tendency for being quiet is noted. The relatives and the psychologists resemble such a patient to a person who has undergone frontal lobotomy. The same patients after one to two years usually develop a clinical picture of agitation, irritability, being short fused, having problems with interpersonal relationships, and adult attention deficit hyperactive disorder (ADHD).

GRADE 4: In this group the neurological and neuropsychological deficits are severe and persistent. There is a history of prolonged cardiac arrest which may last up to 20 minutes. In this group the neurologic deficit is the combination of direct effect of electricity, electroporation, as well as hypoxia. Obviously, when the patient can be resuscitated after up to 20 to 25 minutes, there is some degree of damage (10). However, the fact that such survival after prolonged cardiac arrest is unique to the electrical injuries, points to some protective effect of the electric shock in acute stage to enable the patient to survive the prolonged cardiac arrest.

The majority of such survivors after prolonged resuscitation are the victims of lightning strike (six of nine in our series of patients).

The skin burn in the Grade 4 patients is usually minimal. The exposure is usually in a humid environment with resultant low resistance of the skin. The Grade 4 neurologic deficit patients have evidence of diffuse, generalized peripheral and CNS damage.

Whereas neuropsychometric tests in Grades 3 and 4 patients point to a diffuse cerebral hemispheric dysfunction, in the Grades 1 and 2 patients there seems to be a predominance of frontal lobe dysfunction.

Once the electricity passes the threshold of the skin resistance and enters the internal environment, it is spread instantaneously and diffusely in all the tissues. However, different tissues show different resistance (Tables 4 and 5)(16). Even in regard to the central nervous system (CNS), the small c-fibers and their termination over the limbic system (frontal polar and temporal lobes) are more likely to show a more severe damage than the large, myelinated nerve fibers in the neocortex versus the paleocortex (limbic system) (Table 5) (Figure 4) (43). As a result, in milder (Grades 1 and 2) electrical injuries, the frontal lobe dysfunction becomes more obvious on neuropsychometric testing. Even in severe EI cases, the Halstead Reitan test shows more severe frontal lobe neuropsychometric deficit than the rest of the brain (Figure 5)(61).

The main contribution of Halstead Reitan test was a clear lateralization, localization, and objective documentation of the cerebral dysfunction which was mainly over the frontal regions in milder cases, and diffuse in nature in more severe injuries.

The Halstead Reitan test clarified the issue of "organicity" versus "functional", and in 82% of patients pointed to organic cerebral dysfunction.

It is imperative to accurately diagnose the neuropsychological disturbances of electrical injuries and to differentiate them from other benign nonspecific and minor psychological disturbances such as post-traumatic stress disorder (PTSD) (see above). Even though anxiety, phobia, and nightmares are seen in adults and children (in combination with cardiac involvement), these symptoms are not enough to arrive at the diagnosis of PTSD (2,44).

The selective involvement of the frontal polar regions in electrical injury patients (in different degrees of severity) as well as the hyperpathic allodynic pain (burning pain aggravated by even simple touch) seen in over two-third of such patients, is compatible with selective dysfunction caused by the passage of electrical current through the arteries, sympathetic nerve fibers surrounding them, and eventual termination of the small afferent nerve fibers in the frontal polar regions (Figure 4).

According to Benarroch, the afferent autonomic sympathetic nerve fibers do not end up in the parietal somatosensory neocortex of the brain (43). Instead, they end up through the reticular system in the thalamus and the limbic system. This results in dysfunction of the limbic system in the form of poor memory, poor concentration, agitation, irritability, depression, as well as even attacks of seizure disorder (complex partial seizures).

This distinction and clarification is not just an academic exercise. It helps the clinician to understand the nature of the injury, and to apply a more sensible medical treatment.

TABLE 4. TISSUE RESISTANCE IN OHM	
Mucosa	100
Wet skin	100-40,000
Dry skin	100,000-200,000
Callous sole of foot	1-2 million

TABLE 5. PATHS OF THE LEAST RESISTANCE (22)	
Small c-fiber nerves	R
Arterial blood	RR
Myelinated nerves	RRR
Muscle	RRRR
Skin	RRRRR
Tendon	RRRRRR
Fat	RRRRRRRR
Bone	RRRRRRRR
Dry skin 100,000-200,000 Ohm	RRRRRRRRR
Callous dry skin around 1 million Ohm	RRRRRRRRRR
R= Resistance	

Figure 4. Neuropathic pain (sympathetic mediated pain) ends up in the limbic or emotional part of the brain causing insomnia and depression.

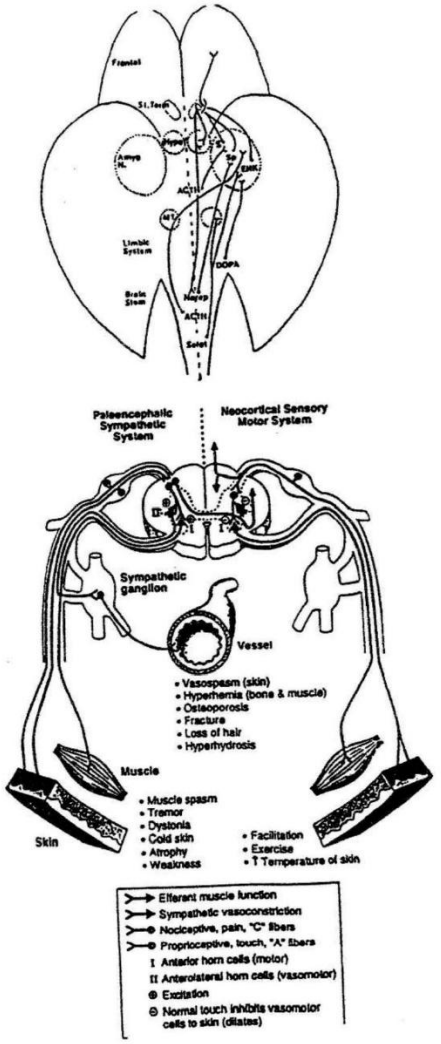
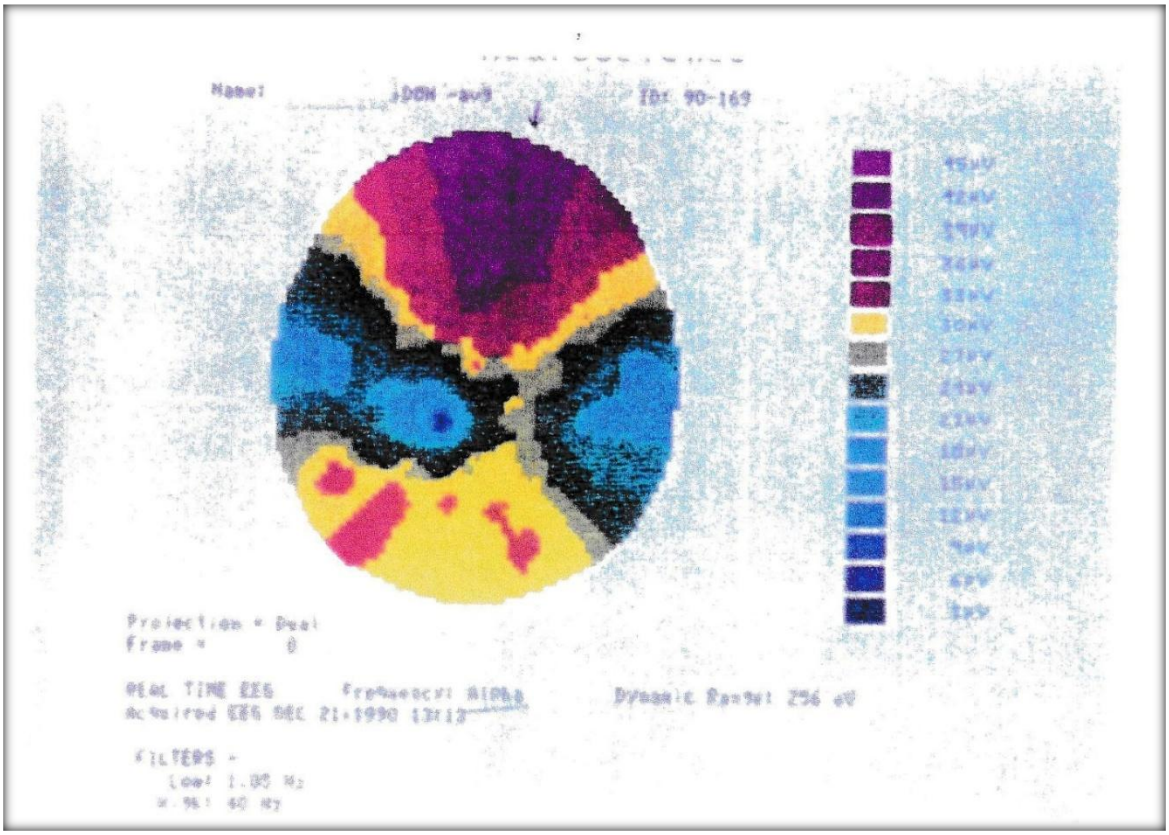


Figure 5. The alpha power spectrum is normally in occipital regions. In severe electrical injuries (EI) it can be displaced to frontal regions.



CHAPTER 6
LABORATORY FINDINGS

The diagnosis of electrical injury is hampered by practically normal results on anatomical tests (CT, MRI) mistaking the disease for malingering, somatization, multiple sclerosis, etc. (Table 6).

- X-rays: Fracture of skull in three patients. Compression fracture of the thoracic spine in four patients. Compression fracture of L1 or L2 in four patients.

- CT scan: Two patients showed nonspecific changes.

- MRI: Four patients had abnormal MRI (less than 1%). One patient had a small lower brain stem infarct (age 22 years) and another 42 year old patient had a hypodense lesion at the pontocerebellar junction on the right side with thrombosed vein leading to the center of the hypodense lesion. Both these patients were lightning strike victims. In one patient, suffering from malignant insomnia after a bath tub electrical injury, a normal MRI post-accident changed to a moderately abnormal MRI (eight years later) showing cortical and subcortical loss of nerves and encephalomalacia: Most likely due to electroporation (32-35). Patel and Lo, have observed cerebral vein thrombosis in electrical injury (62). Three patients in our series suffering from AC injuries showed venous thrombosis in the white matter of right frontal lobe, (one case) and in the white matter of cerebellum (two cases).

- Three patients showed demyelination in spinal cord MRI-mistaken for M.S. (Table 6). Altogether, of 984 patients only six patients showed the above-mentioned MRI abnormal findings.

- EKG irregularities: Four percent in our group, 6% in Engelstatter 's group (Table 2) (8). Abnormal EKG and CPK-MB enzymes do not usually point to myocardial infarction, but a temporary myocardial distress (18,63,64).

- EEG: Epileptiform discharges in 18%. Otherwise, EEG findings were nonspecific.

- Abnormal SSEP: Pointing to thoracic spinal cord dysfunction in 63% of the patients (8,41). The sparing of cervical spinal cord may be explained by the entrance of afferent sympathetic fibres of head and neck to the upper thoracic spinal cord (T2-T5 levels)(Figure 2).

- Abnormal BER: Pointing to mild lower brain stem dysfunction in 48% of the patients.

- Low voltage response to nerve stimulation, and painful neuropathy (6%) (65,66).

- Abnormal computerized EEG/mapping: In 72% the main computerized EEG/mapping abnormality was bitemporal and occasional bitemporal frontal suppression of faster frequencies. In three patients "Alpha dislocation" was noted in the form of higher voltage potential of alpha frequency in the frontal regions rather than occipital regions (Figure 5). The patients with this form of alpha dislocation abnormality had a history of prolonged loss of consciousness (over 12 hours), and showed marked neuropsychological deficit on Halstead Reitan Testing, or standard EEG testing "alpha coma" which has a grave prognosis, as has been previously reported in electrical injury patients (61).

- Sympathetic function tests: Neuropathic pain is not unusual in electrical injury patients (Table 3). This may be in the form of a simple hyperpathic pain, or a full scale RSD (10,28-30). Critchley, as early as 1934, pointed to the fact the "many of secondary effects of lightning and electricity are trophic in character...such phenomena as the common trophic in character...such phenomena as the common "electrical edema"; cyanosis; peripheral arterial spasm; and Horner's Syndrome"(10).

The high incidence of abnormal evoked potentials, and the low incidence of surface EEG epileptiform discharges bring up the possibility that the seizure discharges are too deep in the spinal cord to be recorded on the surface (8).

Sympathetic maintained pain (SMP) was seen in 72% of our group of patients. The SMP refers to patients who did not show the full-blown picture of RSD, but showed pain relief after sympathetic block.

The symptoms of RSD in the form of the following:

- Allodynic (sensitive) burning pain.

- Tremor or muscle spasm.

- Swelling (edema).

- Insomnia as well as depression; was noted in 38% of our patients (Table 3).

Accurate diagnosis is essential in order to provide proper treatment (see Chapter 12).

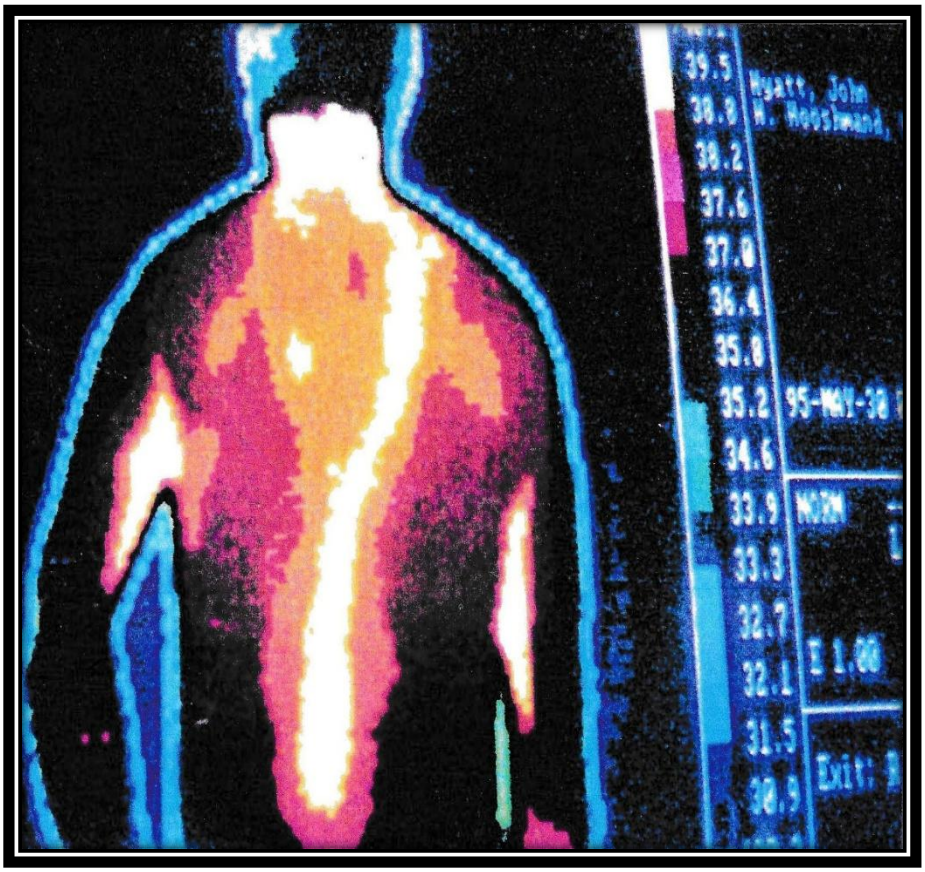
RSD is a clinical diagnosis made by confirmation of the above four criteria. Bone scan in 65% of patients, and infrared thermography 85% of cases may be diagnostic (Figure 6) (28). RSD is not a diagnosis by exclusion, but by confirmation of the above criteria (28).

Pseudoseizures: Were diagnosed by the following:

- Normal levels of serum prolactin in spite of clinical "seizures."
- Recording of patient's behavior and simultaneous normal EEG in the face of thrusting, side-to-side shaking movement of the head, patient stayed responsive during the attack.
- IV injection of normal saline and power of suggestion would bring on a "seizure" and would stop it as well(8,67-69).

TABLE 6. **ELECTRICAL INJURIES MISDIAGNOSED AS:** **(In order of frequency)**
1. Fibromyalgia
2. Somatization
3. Malingering
4. Post-traumatic stress disorder (PTSD)
5. Multiple Sclerosis
6. Amyotrophic lateral sclerosis (ALS) (29,30)
7. Münchhausen Syndrome
8. Idiopathic Epilepsy
9. Idiopathic malignant insomnia

Figure 6. Lightning strike causing damage to the sympathetic system, and heat emission over the patient's back.



CHAPTER 7
INDUSTRIAL VS. LIGHTNING STRIKE INJURIES

The symptoms and signs were slightly different among the lightning strike victims direct current (DC) as compared to the alternate current (AC) household or work place injury victims.

The lightning strike victims showed a pattern of usually descending (or ascending) vertical path of the electricity going through the body. The point of entrance was usually over the vertex of the head, face, neck, upper extremities, or upper part of the torso, and the points of exit were usually in the lower extremities especially the sole of the feet, bursting open the sole of the shoes. The lightning strike would burn and blow out the shoe to go through the body and to ground. As a result, the majority of GI symptoms and signs were seen in the lightning strike patients.

The very brief (a few milliseconds) exposure of the thunderbolt may explain the reason for survival of lightning strike victims.

The lightning strike injury showed small areas of skin burn over the scalp, behind the shoulder, or through the posterior cervical spine region-as well as the soles of the feet. The scar over the scalp or skin of the neck or shoulder was usually small in size with permanent brownish or reddish discoloration. The path of the electricity went through the throat the GI tract and down to the leg or foot (upward, or downward).

Whereas in both DC (lightning strike) and AC (industrial and household) currents, the autopsy findings show predominantly severe damage to the arterial vascular structure with charred necrosis of the blood vessels, the GI (gastrointestinal) findings are quite common. In the fatal cases, the GI findings are in the form of necrosis, perforation, and microwave effect in guts. In milder cases it is mainly in the form of burn scars over the glossopharyngeal region, esophagus, gastric mucosa, and small bowel regions. Intestinal perforation may develop secondary to electrical burn (70).

In the AC electrical injuries, the GI findings are minimal, and the main abnormalities are noted in the distribution of the arteries in the path of the entrance and exit of the electricity. In lightning strike, the path of electricity is vertical (cephalad or caudal), and the AC has a tendency to go through a horizontal pathway to find an exit. This vertical path is more likely to cause GI damage.

The exit point may be the opposite extremity, as well as the sweaty creases of skin (underarm, below the breast, or on volar aspect of the elbow).

The degree of damage from the electricity through the body depends on the risk factors operational at the time of the electrical injury.

CHAPTER 8
RISK FACTORS

No two electrical injuries are similar. Due to the circumstances of the accident, there is a wide variation in risk factors.

Not every electrical injury causes severe morbidity or mortality. Only 2.5-3% of electrical injuries are fatal (1).

The high risks in order of importance are:

- Alternate current (AC) versus direct current (DC).
- Low skin resistance.
- Length of exposure time.
- Strategic points of entrance and exit of electricity.
- The intensity (I, or amperes) of the current.
- The voltage which is a constant factor influenced by the above listed variables.
- Professional risks.

RISK FACTORS IN ELECTRICAL INJURIES (EI)

In our series of 984 patients, the accidental industrial or household injuries were at least three to four times more frequent in occurrence, than lightning strike. The majority of the victims were affected by alternate current (AC) exposure (Table 1). The direct current (DC) patients were usually lightning strike victims. In one case (1 of 984) an industrial DC injury was seen due to exposure to a high voltage DC generator (a high voltage paint spray gun used in automobile industry) (Table 1).

Simple exposure to electricity does not necessarily cause serious injuries. Electrical injuries usually are deflected by the dry human skin which is a strong shield against penetration of electricity (36). The dry human skin has 100,000 to 200,000 Ohm resistance. At the expense of causing extensive heat in its process of resisting electricity, it protects the deeper structures. In the process, the patient may develop grade II or III skin burns.

However, if the skin is wet, or other risk factors outlined below cause the penetration of the electricity through the skin, then the electrical injury causes damage to multiple organs (Figure 1)(Tables 2 and 3).

RISK FACTOR NUMBER 1
ALTERNATE CURRENT (AC) VERSUS DIRECT CURRENT (DC)

The household or industrial AC currents are three times as dangerous and potentially fatal as lightning DC currents of the same voltage (Table 7) (71,72). The AC currents of low (household) voltage (60HZ) are more likely to cause tetanic spasm of the muscles (no-let-go-phenomenon) which is potentially fatal (3,72,73).

TABLE 7.
DIRECT CURRENT (DC) VS. ALTERNATE CURRENT (AC) (121)

DIRECT CURRENT (DC)	ALTERNATE CURRENT (AC)	.
0-75 Milliamps	0-24 Milliamps	Brief apnea, and no cardiac damage
80-30 Milliamps	25-65 Milliamps	Cardiac arrest, high blood pressure, and apnea. Beyond 30 seconds exposure: ventricular fibrillation (V-fib)
300 Milliamps to 3 Amps*	800 Milliamps to 3 Amps	DC fatal thru certain paths going through heat. AC can only survive with less than 0.3 second exposure: otherwise, irreversible ventricular fibrillation (V-fib)
***DC is less fatal if the current bypasses the heart.**		

RISK FACTOR NUMBER 2
LOW SKIN RESISTANCE

Contrary to its superficial appearance, the extent of the skin burn has a reverse relation to visceral and neurological damages of electrical injuries.

The normal dry skin is one of the most resistant of body tissues (12,30,40). Victim shows no points of entrance and exit, no scar on the skin, a dry skin undergoes extensive electrothermal injury resulting in extensive second and third degree skin burn. This high resistance (100,000 to several thousand Ohms) of the dry skin absorbs the brunt of the electrical current (amperes) and shields the deeper structures (Table 5). The best example of this principle is the use of a hair dryer with defective wiring while the victim is immersed in the bath tub (74). The hair dryer charge is low voltage (110 volts) yet the defective wiring charging the water results in a generalized entrance of the electricity into the body-due to the fact that the wet skin has no resistance to current-and

allows the high amperage enter the body. As a result, the victim shows no points of entrance and exit, no scar on the skin, and in a matter of a few seconds develops cardiac arrest and death.

In contrast, the entrance of electricity over the dorsum of the dry skin of the wrist, and its exit through the arm may result in extensive skin burn, but no serious CNS and visceral damage. Another example is a lightning bolt of thousands of volts of short duration flashing over (arcing) the dry skin, leaving minimal or no damage to the skin and finding a point of low resistance on an object close to the person who was hit by the lightning. What saved this person was the high resistance of the skin as compared to the surrounding wet environment.

Applying Ohm's Law, V=R x I, the V (voltage) is a constant depending on the source of the electricity. It may be low voltage (110-220 volts) or high voltage (over 600 or 1,000 volts). If the resistance is high, the I (intensity of current or amperes) is going to be low, and less electricity enters the body. If the resistance is low due to humidity, the same 110 volts will generate a larger electrical current entering the body.

Application of the same Ohm's Law interprets the electrical current as I=V/R. Exposure of 110 volts electricity to the dry skin results in 110/100,000 Ohm (R)= 0.11 milliamperes. This current is too low to cause a significant damage. On the other hand, if the skin is soaking wet, then the resistance of the skin drops from 100,000 Ohms to 100 Ohms. Then the equation changes to 110 V/100 Ohm (R)=11 amperes which is over the 60 milliamperes threshold of cardiac arrest and death.

Fortunately, usually the skin of a victim is not so soaked with water. The skin resistance plays a role in the following phenomena:

- Arc burns.
- Electrothermal burns.
- Combination of the above.
- Electroporation.

ARC BURN

Arcing is the passage of electricity through the space with no need for direct contact. The path of the electricity can jump through a vacuum (or air) and can keep arcing-jumping around until it finds a lower point of resistance to pass through the next object. Arcing can occur during lightning (DC current), or in the vicinity of the electromagnetic field of a high voltage utility power line (AC).

The arcing is facilitated by a humid environment. One example is a worker not using his insulated protective helmet while working within inches of the highly charged wire. The humidity of air and scalp facilitate the arc burn and electrocution occurs through the scalp without a direct contact. Of the 984 patients, only nine demonstrated such high voltage arcing.

This phenomenon is more frequently seen among the lightning injury patients. The extremely high voltage electricity facilitates the arcing. In the case of lightning strike, the principle of flash over explains the reason for the lightning jumping over the high resistance skin without causing any penetration, if it can find an adjacent area of lower resistance such as a nearby wet tree or metallic object. The arcing usually does not result in cell membrane damage. This explains the survival with no damage in some lightning strike patients.

If a small part of the skin is humid (due to sweating over abdomen, underarm, groin, or at the crease of elbow), then arc burns cause more extensive bodily damage. Usually, if the skin is dry, the arc burn is a thermal skin burn without penetration of the electricity through the skin to the viscera and nerves. In one study reported by Electricite' de France, 78% of the electrical injuries were simple arc burns with good prognosis (14).

The small areas of humid skin (over the axilla, the elbows, the groin, or the fat pad of the abdomen) may allow multiple points of exit of electricity causing confusion regarding identification of point of exit (3,12,30,40).

ELECTROTHERMAL BURNS

When the skin is not as dry, electrothermal burns cause deep thickness skin burn and more damage to the deep structures. In the study by Electricite' de France, these burns consisted of 15% of the of patient population (36). The electrothermal burn refers to a relatively low skin resistance, allowing a high voltage penetrating the skin, and following the path of the least resistance in the body. This is in contrast with arc burns which cause a simple thermal burn, usually without penetrating the skin shield (36).

MIXED DAMAGES

The mixed damages refer to a combination of the arc burn and electrothermal damage to the small areas of electrothermal injury such as through the wet areas of axilla, groin, or abdominal crease where the skin is wet and sweaty with significant visceral and neurologic damages.

RISK FACTOR NUMBER 3
TIME FACTOR
"NO-LET-GO-PHENOMENON"

The third risk factor is the length of exposure to the electric source. The time factor is quite vital in lightning strikes. The lightning strikes (AC) are usually in the range of tens of thousands to millions of volts (6). However, lightning usually has a time duration measured in a few milliseconds. Such brief temporary exposure to the electricity is not enough to cause cardiac arrest. Between the protective effect of high skin resistance and the short duration of electrical exposure, the fatal outcome in lightning and other electrical injuries is rare. Less than 3% of electrical injury victims end up with a fatal outcome (2.5-2.7% of cases)(75).

With brief duration of the lightning bolt, arcing and flash over phenomena occur which in many cases may leave no serious damage. The AC currents (household and industrial injuries) are three times as likely to be fatal than the DC (lightning) currents(22).

In the AC electrical injuries, the so-called "no-let-go-phenomenon" (Grip Tetanus) results in prolonged damage and eventually fatal exposure to the electricity (10,22-27). This phenomenon refers to the fact that if a source of electrical current touches the palm of the hand, it causes an instant reflex contraction (tetanic contraction) of the hand with resultant constant grabbing of the source of the electricity, i.e., the electric wire or the electrical device, resulting in the no let go phenomenon. The prolonged exposure of the electricity results in the cumulative effect of the current entering the body (amperes), cardiac arrhythmia, ventricular fibrillation, cardiac arrest, and death.

The lower voltage (110 or 220 volts) AC current are more likely to be fatal by causing the "no-let-go" phenomenon than the high voltage AC currents. The high voltage currents pass through the points of entrance and exit so fast that the muscle does not have time to react with tetanic contraction (101,105). This results in less chance of long exposure to electricity, and explains the remote effects of high voltage electricity in other parts of the body (62,76).

The no-let-go-phenomenon (Grip Tetanus) is a common phenomenon in industrial and household injuries. Whereas in 86% of the no let go phenomenon victims in our series the point of entrance was through the hand, in 14% the tetanic contraction was due to the contact over the forearm, wrist, gastrocnemius, or quadriceps regions. In such cases, the no let go phenomenon manifests itself in the form of muscle contraction pushing the patient against the source of the electricity. The victim usually resembles the phenomenon to "a magnet" attracting the extremity to the electric source. This is especially the case when a witness attempts to rescue the electrocution victim.

A low voltage electrical source can become fatal due to the "no-let-go-phenomenon" (22-27). In the example of Ohm's Law (V=RxI), 110 volt electrical exposure to 100,000 Ohm normal skin

resistance results in 0.11 milliamperes current. This is not enough to cause cardiac arrest. On the other hand, if the time of exposure is prolonged due to the no-let-go-phenomenon, then there is an accumulation of current enough to cause cardiac arrest. In addition, the no-let-go-phenomenon usually causes enough electrothermal damage of the skin to make the skin less resistant to the source of electricity.

The time factor is summarized in the following abbreviation of Ohm's Law:

- V=RxI
- V(t)=RxI(t)
- WATT(W)=IxV
- WxT=JOULE

Where R=resistance, I=intensity or amount of the current passing through the organs especially the heart, T=the length of time of exposure and V=voltage. The other two formulas, WATT and JOULE, emphasize the importance of time and intensity of the current passing through the body.

In addition to the no-let-go-phenomenon, two other mechanisms of electrical injury are dependent on the time factor:

- Electroporation.

- G.F.I. safety feature.

ELECTROPORATION

Electrical exposure results in acute electrothermal damage which is simply a heat generation damage to the cells in the form of heating, coagulation, and microwave effect.

In addition, there is another form of electrical damage which affects the subcutaneous and deep tissues with long-lasting deficits. In survivors of electrical injuries, the exposure time is usually too brief to explain the extensive damage in multiple organs. Frequently, the body is thrown away from the electric source by a massive tonic extension of the paraspinal muscles. This reflex massive spasm occurs in a matter of a fraction of a second after exposure. This leaves little time for the thermal effect to cause the extensive pathologic changes. An example is the fatal electrical damage in a victim immersed in water. In this situation, no skin burn is seen, and no significant heat is generated, but cell death occurs. This death of the cells has been blamed at electroporation referring to the break-down of the cell membrane (nerve, muscle, etc.) and formation of pores due to the direct effect of the electricity on the cell membrane (electroporation) (32-35).

The stabilizing forces in the cell membrane are chemical (e.g., sodium-potassium pump), anatomical (e.g., blood brain barrier or BBB), and electrical and electromagnetic: the membrane resting potential and thickness form an electrical field at cell membrane level. The electrical injury obviously affects this field (Figure 6).

The delayed electrical injury manifestations have been reported since 1860 (38,77-79).

The electroporation was first suspected by Panse in 1977, Christensen in 1980, and Starr in 1986 (15,16,39). These authors reported protein changes in the cells due to electrical injury.

The electroporation has a tendency to result in subacute and chronic death of the cells due to the leaky cell membrane. The pores gradually drain the protective shield of the cell membrane and result in a slow, gradual death of the cell. This explains the chronic neurologic deterioration in 36% of the 984 electrical injury patients followed for three years or longer (Tables 2 and 3).

In acute stage, electroporation results in breakdown of vascular endothelium and muscle membrane resulting in the second degree electrothermal burn to become further complicated by cell membrane damage to deep internal tissues, cell membrane leakage, and secondary complications of edema and infections (such as clostridia) . The leakage of the cell membranes results in myopathy, myoglobinuria, and renal failure, and anterior compartment syndrome (38,73).

In chronic stage, this phenomenon causes a biphasic clinical manifestation in electrical injuries in contrast with other forms of trauma. The two years rule of thumb for achieving stabilization and recovery-usually applied in head injuries-does not hold true in electrical injuries: aggravation of memory loss, development of cataract usually longer than two years after the injury, latent development of myelopathy resembling ALS may be some of the latent manifestations of electroporation (Table 3).

The electroporation is the logical explanation in atrophic and dystrophic phenomena such as reported by Blom and Ugland of patients developing muscular atrophy and weakness in the hands up to six years after an arc burn (38). Delayed causalgia and RSD also been reported in electrical injury patients (Table 3) (28-30).

Atrophy of the muscles as well as development of kerauno-paresis (paraplegia due to spinal cord damage by electrical injury may develop several months or even years after an electrical injury (5,80,81). This phenomenon, rather than being an exception to the rule-is not uncommon in electrical injury patients. It is most prominently noted in spinal cord dysfunction due to electrical injury (80,81).

GROUND FAULT INTERRUPTER (GFI)

The time of exposure becomes prolonged if the safety feature of ground fault interrupter (GFI) is not functioning properly. Normally, the breaker box disconnects the short-changed circuit due to electric short (or grounding), through human body, or other conductors. As the electricity is by-passed from the main circuit through the victim's body, the GFI does not receive the standard current and automatically disconnects the circuit.

The GFI is usually designed for 110 volt electricity. However, a lot of washers, dryers and other utility electrical devices function on 220 volt electricity. The standard 110 volts GFI does not interrupt the 220 volt current. The electric short may take away even a 100 volts from 220 volts current. Even then, the GFI designed for 110 volts does not interrupt the shorted circuit. As a result, the length of the electrical exposure becomes prolonged due to lack of the protection of proper GFI. This prolongation of electrical exposure can cause fatal damage to the heart even though the electricity is only 220 volts in contrast to tens of thousands of volts of lightning strike of very short duration.

Both the 110 and 220 are classified as low voltage current (since high voltage is considered as any current higher than 600-1,000 volts) but they are more likely to cause mortality and morbidity. In absence of proper 220 volt safety feature, the victim is exposed to 220 volt current for a lengthy period of time. In this situation, the victim's body becomes grounded for several seconds. Light bulbs in the house start popping, burning and bursting open (pointing to the failure of the breaker system to cut off the current) while the patient is in the path of the electrical current for several seconds with resultant potentially fatal passage of current through the heart.

RISK FACTOR NUMBER 4
POINTS OF ENTRANCE AND EXIT

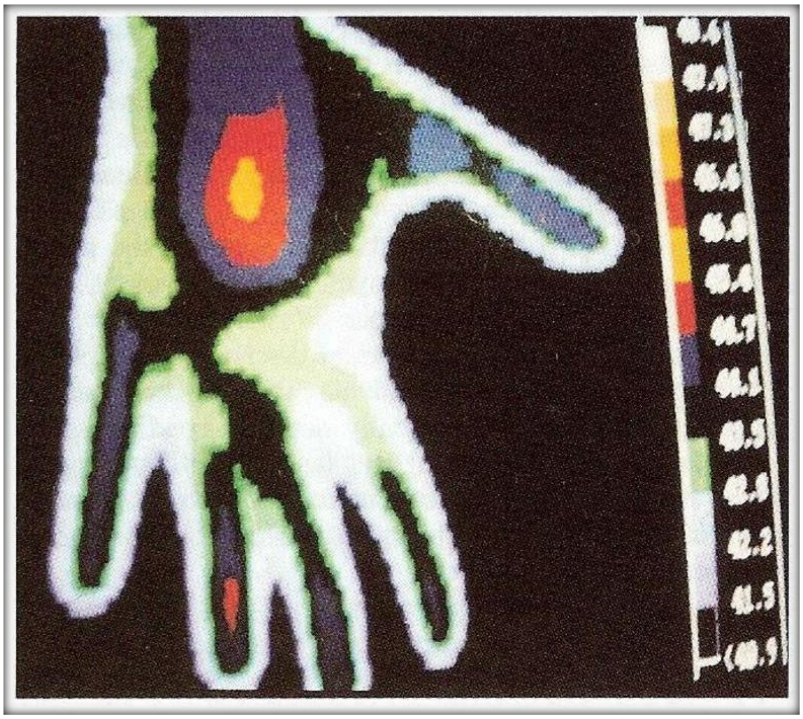
The fourth risk factor (in order of frequency of morbidity) is the points of entrance and exit. The strategic location of the points of entrance and exit plays a major role in the outcome of the complications of electrocution. In the case of a body immersed in a pool of water charged with electricity, there is no need for points of entrance and exit. If the patient is exposed to electricity the point of entrance can be identified but the point of exit is too diffuse in the wet environment to be recognized.

Infrared thermography shows the frequent occurrence of one point of entrance, and several

points of exit-usually in sweaty crease of elbow, groin, or fat pads of anterior abdominal wall (Figure 9).

The points of entrance or exit are in upper or lower extremities in 82% of patients of AC injuries with the order of least resistance being nerves, arteries, muscle, skin, tendon, fat, and bone (Table 5) (3,16,73).

Figure 9. Reflex sympathetic dystrophy (RSD) due to electrical injury. Point of entrance in palm of the hand. Three concentric thermal changes typical of sympathetic nerve damage and dysfunction. The central vortex of the damage area has lost its thermal vasoconstrictor function, resulting in heat leakage. The second circular margin shows less hyperthermia. The third and fourth circular margins are hypothermic (intact sympathetic function).



SAME EXTREMITY

In the case of exposure to electricity in a dry environment, if the current enters the body through the hand and exits through the elbow or shoulder of the same extremity, most of the current bypasses the heart and only affects that one isolated portion of the extremity. The skin burn is usually extensive requiring skin graft. This form of electrical exposure causes the least amount of current going through the heart and points to a better prognosis. The same applies to the lower extremity.

SAME SIDE

On the other hand, if the electricity enters and exits the upper and lower extremities on the same side, more current passes through the rest of the body but still the path of the electricity does not necessarily directly involve the cardiac muscle and cardiac sympathetic plexus. This form of entrance and exit is the second less damaging (Figure 9).

OPPOSITE SIDES

If the entrance and exit are through upper and the lower extremity on the opposite sides, then more current passes through the heart with a higher mortality and morbidity.

HAND TO HAND

The most serious type of electrical damage, short of immersion in the water, is electricity entering one hand and exiting the other (73). This puts the heart in direct path of the electrical current with rapid and severe damage to the nerves to the myocardium, myocardial contraction, and death. The incidence of fatal heart attack in this type of exposure has been reported to be over 60% of the fatal cases(73).

HEAD AND FACE

The second most serious current is entrance of electricity is through head and face down to extremities. This usually happens in lightning injuries.

VERTICAL VS. HORIZONTAL CURRENT

In AC electrical injuries, the current usually is transmitted horizontally (hand to hand) even though the current will go through any direction to ground itself (16,73). In lightning strikes the current ascends or descends vertically. Commonly, in lightning victims, the entrance is above, and the exit is below the waist. The vertical lightning current commonly causes both cardiac and gastrointestinal damages (diarrhea, attacks of severe abdominal pain, and weight loss). The autopsy of lightning strike victims shows necrosis, burning of tissues, and moderate to severe damage to the arteries, brain, pharynx, esophagus, and the guts. The vertical currents (hand to opposite foot AC current, or lightning DC current) are more likely to cause cardiac pathology (22,79).

RISK FACTOR NUMBER 5
THE AMOUNT OR THE INTENSITY OF THE ELECTRICAL CURRENT (AMPERES OR I)

The final determinant of the damage from the electricity is the intensity (the amount of electricity, measured in amperes). The increase in amperes entering the body results in complications from pain to death (Tables 8 and 9).

The critical fatal risk in electrical injury victims is the number of amperes entering the body. The voltage is a predetermined constant factor. The electrical or electromotive force (effective voltage) entering the body is controlled by humidity, length of exposure, etc., outlined above. However, the fatal factor is the amount (intensity) of electricity going through the cardiac plexus and muscle (over 60 milliamperes is fatal).

TABLE 8. ELECTRICAL INTESITY (AMPERES) AFFECTING THE ORGANS	
0.001 to 0.01 Amperes	Painful tingling
0.01 to 0.02 Amperes	Muscle contraction (no-let-go-phenomenon)
.02 to 0.05 Amperes	Respiratory arrest
0.05 to 0.10 Amperes (usually 0.06)	Ventricular fibrillation (V-fib) and cardiac arrest

TABLE 9. AMPERES CAUSING NO-LET-GO-PHENOMENON	
Children	0.004
Women	0.007
Men	0.009 to 0.01

RISK FACTOR NUMBER 6
VOLTAGE

The body may be exposed to 1 volt to 10 kilovolts (KV) of electricity (Table 10) (82). In any given case, the voltage is a constant factor influenced by the AC current versus DC current (AC is three times more fatal); skin resistance, and other risk factors outlined above (71-73).

The higher DC voltage has more tendency for arcing. The higher DC voltage is less likely to cause tetanic muscle contraction, and more likely to take a direct path to ground (17,38,71).

The exposure to electricity is also influenced by the electromagnetic changes in the environment. The high voltage electrical injuries (over 600-1,000 volts) have a tendency to cause electromagnetic changes in the adjacent environment.

This phenomenon is at times noted when a metallic crane accidentally touches a high voltage wire and conducts the current to the body of heavy equipment.

In lightning injuries, in rare cases (3 of 984), the extremely high voltage arcing into the room or

stable or the garage, can cause pipes and wires inside the walls to spring out of the walls due to the electromagnetic field generation.

Capacitors add more voltage to the 110 household or office currents. In this regard, the computer operating on 110 volts exerts much higher voltage (over 500v, or higher) exposure on the victim.

TABLE 10. VOLTAGE RANGE IN ELECTRICAL INJURIES (EI)		
Low voltage	Telephones (land-lines) Household utilities	24v 60-65v 110-120v 220-240v
High voltage	Industrial utilities Lightning strikes	600-100v and higher 1000v to hundreds of thousands of volts

RISK FACTOR NNUMBER 7
PROFESSIONAL RISKS

In our study of 984 electrical injury patients, the highest risk factor was exposure to a high voltage wire among the utility and telephone company workers.

- Professions such as electricians, phone repairmen, power line maintenance works, heavy equipment operators, roofers, lifeguards, hotel maids, and housewives are high risk.

- Whereas in two cases the contact with high voltage wire (voltage over 1,000 volts) was due to the accidental contact of metallic objects and poles in the back yard by children, and in four patients the exposure was due to the accidental contact of a crane against the electrical wire, the rest of the group (52 patients) belonged to the power and light utility or the telephone company workers who came into close contact with the high voltage wire. The improper usage of helmets was a major factor in arcing and electrical contact.

- The next group of high risk workers were air conditioning, roofing, and television repair workers coming into contact with power generators or improper wiring of the units (13 patients).

- The fourth high risk group of workers were the electricians working in the factories or high rises coming in contact with electricity due to another worker turning the breaker on or due to the use of scaffolding coming to contact with a hot wire (7 patients.)

- The next group of high risk workers were the individuals coming into contact with improperly grounded ice box and other utility units in walk-in freezer areas (6 patients).

- The housewives are exposed to a relatively high risk of electrical injury. This is specifically true in regard to the old houses with lack of proper GFI wiring. The old washer and dryer in a wet basement is a high risk source. Other sources consist of the use of old washing machines with improper wiring, multiple plug connectors and wire extensions.

Of all the high risks in the house, the most frequent and most fatal electrical injury is the bathtub electric short due to the use of a hair dryer or other electrical devices while the person is immersed in the bathtub.

RISK FACTOR NUMBER 8
TELEPHONIC TRANSMISSION

The next example of the risk of exposure to high intensity current is the telephonic transmission (18,63,64,83-90).

Of the 984 patients in our study, eight patients were due to telephonic lightning injury while using outdoor telephone booths (three patients from Hawaii were in shell-type out door telephone booths). Another three patients suffered from this type of lightning arcing of the electricity while using the telephone indoor or standing by a gate or open space or window. The low incidence indoor versus outdoor injuries may be due to better construction and better grounding in newer houses (83).

The above group of patients usually described the same phenomenon as in outdoor lightning victims in the form of a blue or white light arcing; (even a blue light "ball" approaching at high speed and passing through the body of one victim resulting in instantaneous death). Similar phenomena have already been recorded in the literature (1).

Lightning injury and death is not limited to outdoors. The indoor victims become involved due to the phenomenon of “side flash.” The side flash phenomenon is usually through the telephone wiring. However, water pipes, electrical appliances, computers, and hair dryers are some other instruments that can facilitate the occurrence of side flash. The best prevention is the use of portable (cord-less) phones.

In the telephonic side flash, a voltage difference is generated between the person and the telephonic equipment (83). The telephone wires may be struck directly by the 48lightning outside of the house with a transfer of the current to the victim's body. On the other hand, the person may be grounded by leaning against a metallic object or standing barefoot on a moist floor. The lightning results in a voltage difference between the headset and the body. This in turn causes a spark between the headset and the ear. This usually causes electrical burn involving the

external ear, rupture of the ear drum, and damage to the inner ear (18,63,64,83-90). The complications can on rare occasion be fatal (83).

In such an arcing, a spark from the telephone to the ear and the head can occur in typical phones when the potential of the phone relative to the ear exceeds above 10 kilovolts (83).

CHAPTER 9
PATH OF THE LEAST RESISTANCE

The body as a volume conductor, allows the electricity to pass through simultaneously. The resultant damage is influenced by the resistance of the tissues (Table 5). Kouwenhoven, has shown that as electricity penetrates through the skin, the internal environment of the body becomes evenly charged with electricity which is constant at any cross section (6,33,91). However, obviously the less resistant tissues suffer from more damage during a short exposure to electricity (obviously with longer exposure the tissue become necrotized and there is no chance of survival).

- Nerves, especially the small c-fibers of the sympathetic nerves surrounding the walls of the arteries and arterioles.
- Arterial blood, which is warm, ionized, oxygenated, and fast moving.
- Larger, myelinated nerve fibers.
- Muscles and viscera such as liver and kidney which result in acute complications of myoglobinuria, renal insufficiency, nephrosis, and liver damage mistaken for "hepatitis C" among electrical injury patients.
- Wet skin.
- The GI tract. In lightning strike victims and in rare AC injuries-the current entering the head, face, and soft tissues of digestive system- the oral pharynx, esophagus, gastrointestinal mucosa and nerves-become damages with long-term complications of chronic diarrhea, autonomic dysfunction, severe epigastric pain , and weight loss.
- Fascia and tendons.
- Cortex of the bones.
- Dry skin.
- Callous skin.

This variation in the tissue resistance against electricity explains the high incidence of neurologic and vascular latent complications of electrocution. Structures rich in blood vessels and nerves are more likely to sustain damage; so, the long term side effects usually involve the peripheral and central nervous systems, the sympathetic nerves, the vascular structures, followed by liver, kidney, and GI complications.

Among the 984 patients we studied, a high percentage suffered from sympathetic mediated pain (SMP) due to the fact that the sympathetic nerve fibers are quite small in diameter, and usually follow the walls of the arteries (Table 3) (28-30). This may explain the high incidence of SMP in such patients.

LIMITS OF ELECTROTHERMAL PHENOMENON

The electrothermal damage extends beyond the surface of the skin and affects the deeper structures. Once the voltage exceeds 200 volts, and the exposure is prolonged, the skin resistance becomes negligible as the damage goes through the entire thickness of the skin, and the current enters the internal environment (Kouwenhoven Principle)(33,91).

According to Faraday's Law, the current traveling from a portion of the extremity throughout the rest of the body, is distributed across the extremity. The electrical power is practically constant in any cross section perpendicular to the axis of the extremity (33). The tissues with the least resistance (nerves, blood vessels, liver, heart, guts, and muscle) undergo a more rapid destruction than the adjacent tissues (fascia, ligament, and bone). Usually, the survivor is jolted violently and reflexly from the source-saving his life by shortening the time of exposure. With longer exposure time, the entire internal environment becomes saturated with electricity, and in severe cases, death equalizes the damages in all tissues. Fortunately, we study the survivors.

The thermal effect achieves the maximum damage to tissues with lower threshold, resulting in maximal denaturation, dehydration, and damage to the cell membranes with the end result of necrotic death of parts of the organs (91). As the susceptible tissue becomes necrotic, it loses its rate of electrical conduction transport (91). Electric short (coupling) affecting the skeletal muscles lead to a high voltage discharge, forcing the victim away from the source of electricity. The physiological excitation contraction coupling time for muscles in the extremities is only 10-30ms with resultant jolting and disengagement of the body from the source to a significant distance resulting in compression fracture of the spine and other injuries.

However, this short length of exposure (up to 30ms) is not long enough to explain the long term effects of the electricity (33). The phenomenon of electroporation seems to be the logical explanation for delayed manifestations of electrical injury.

ACUTE STAGES VERSUS CHRONIC STAGES

In acute stages, cardiac arrhythmia and chest pain, muscle pain and necrosis, renal damage, abnormal liver tests, myoglobinuria, skin burn, skin infection and septicemia, abdominal pain, vomiting, and diarrhea are the main features (Figure 1).

In chronic stages, allodynia and hyperpathia, as well as tremor and flexion spasm of the extremity are the main features of the sympathetic nervous system dysfunction (28-30). The dysfunction of the cortical representation of the sympathetic system (small c-fibers)(Figure 4) ending through the reticulo-thalamic and thalamocortical pathways in the limbic system, result in insomnia, depression, agitation, poor memory and judgment, and mood swings(43).

The central nervous system (CNS) involvement of the electrical injury results in myoclonic jerks, other forms of seizures, as well as pseudoseizures (Tables 2 and 3). Other manifestations are in the form of neurogenic bladder, vertigo, mild hearing loss, and neuropsychiatric dysfunctions (Tables 2 and 3)(Figure 3).

CHAPTER 10
PATHOLOGIC DAMAGES DUE TO ELECTRICAL INJURY (EI)

The pathologic damages consist of coagulation necrosis, thermal damage arc burn, electrothermal damage (penetration of electricity through the skin), edema, crush injury, and electroporation (cell membrane defect) which may become irreversible.

The pathological effects of electrical injuries have been documented for the last two centuries. Among the significant contributions to this field is a remarkable medical report by the renowned neurologist Doctor MacDonald Critchley from England. This pivotal work was published on January 13, 1934, in the journal Lancet (10).

Even in this original early report, Doctor Critchley divided the clinical features to immediate, secondary, and remote effects. The secondary and remote effects just means that it was considered a delayed complications of the electrical injuries. He discussed kerauno-paralysis (paralysis due to the electrical injury) in detail, and emphasized that usually this kind of paralysis is of temporary nature even though it does leave significant residual among the patients. He emphasized the fact that the neurologic sequelae of the electrical injuries do not show "morbid appearances," and the normal appearance of the patient masks the serious complications of electrical injury (10). In 1909 Doctor Jellinek first reported vasculopathy in electrical injuries (92).

In 1931, Panse highlighted the occurrence of significant spinal cord injuries and subsequent atrophy in patients who had suffered electrical injuries. (93).

In 1938, Leo Alexander published a comprehensive and influential report on the pathological injuries observed in patients who suffered electrical injuries (21).

In the above mentioned reports the pathologic findings have consisted of selective damage to the arterial circulation, as well as to the gastrointestinal system, liver, kidney, and nerves. The above authors have emphasized the fact that the delayed sequelae of electrical injuries caused muscle and nerve damage in the brain, spinal cord, and extremities by "vascular disease of a chronic progressive vasopathy" (21,38). This "vasopathy" is clinically identical to sympathetically mediated pain (SMP) which is seen commonly in EI patients (Table 3)(28). The incidence of SMP is around 28% to 72% (Table 3)(28).

The pathologic findings and the consensus of the literature of the turn of the century to the present time is that the blood vessels (arteries) and the surrounding nerves are the main targets of electrical damage. As a result, the patient develops "vascular atrophy" or "vasopathy" as termed by Panse and Alexander (21,93).

In 1967, Ugland, along with Jellinek who reported similar findings as early as 1909, documented instances of brain edema and spinal cord injuries in individuals who suffered electrical injuries. (65,92).

In the past 10 years, the phenomenon of electroporation has been the key mechanism of the latent development of the neurological and neuropsychological complications (32-35).

The thermal effect of the EI is directly related to the resistance and intensity of the current (heat=amps 2xR). As the skin resistance drops it results in formation of blister, followed by breakdown of the skin insulation. This breakdown is followed by colorization (thermal necrosis) and death of the tissues in different degrees according to their resistive susceptibility.

The burns due to EI may be the result of direct thermal effect, remote flash, arc burn, or combination of the above thermal effects.

The ionic and enzymatic leakage of the cell membrane is due to the electroporation or disruption of the cell membrane seal of protection, can be reversible or irreversible depending on the severity and the duration of the electrical exposure. When an excitable membrane is depolarized of the resting potential, sudden change in the membranes conductivity takes place leading to a propagation of action potential into the cell. Any increase in cellular potential would correspondingly increase the already enormous field across the membrane. The resulting molecular forces have important cellular consequences including electroporation. In this regard, electroporation can be defined as penetration of the electricity across the critical threshold of depolarization of the cell membrane.

As the electroporation is mainly an ionic and enzymatic exchange disturbance, it can best be detected by its long term side effect of disturbance of the protective shield of the cell membrane and blood brain barrier (BBB).

EKG and muscle enzyme abnormalities do not usually point to myocardial infarction (79,94,95).

Gastrointestinal (GI) injuries are quite common, and even a low-voltage injury can lead to serious complications such as necrosis and ulcers in the intestines (70,96). The GI damages are more commonly seen among lightning strike patients. This is probably due to vertical-usually head or shoulder to feet-direction of the electric current. This path is more likely to cause perforating ulcers and necrosis of the guts than a hand to hand or foot to foot AC injury.

Chronic intractable diarrhea (16 patients) and abdominal pain (12 patients) respond positively to celiac, epidural, and paravertebral nerve blocks (21 of 28 cure).

In rare cases (2 patients) hiatal hernia developed in absence of any past history or any high risk factors (obesity, etc.).

SPINAL CORD INJURIES

Spinal cord injury is usually subtle, but can be quite disability. According to Varghese et al, "spinal cord injury is perhaps the most significant permanent sequala of the electrical injury to the nervous system (37). Such injuries are more frequently encountered than is generally recognized" (37). This may be due to the compact low resistance nature of the cord and its rich vascular structure (38). The pathology may be acute, or more commonly, delayed (electroporation) (38,39).

As the electricity follows the path of the least resistance of arteries and small c-fiber nerves, it becomes obvious that it finds its way through the sympathetic system surrounding the arteries towards the cardiac plexus and towards the entrance and exit of the sympathetic nerve fibers at upper thoracic spinal cord level (T3 through T5 levels). The spinal cord is no more in danger of being affected by the electricity as the rest of the CNS (central nervous system). However, being a compact structure, it manifests the damages blatantly in the form of acute and chronic neurologic changes. The acute damages are usually transient (10,77,78,97). However, the delayed development of electrical injuries is not at all unusual and has been reported as early as 1931 by Panse (93).

The pathologic findings on autopsy of spinal cord as reported by Alexander in 1938, are not limited to demyelinating changes (21). As early as 1909 Jellinek reported vasculopathy in the spinal cord in electrical injury patients (79). The vasculopathy is mainly in the form of vasospastic changes; as well as in more severe cases, secondary punctate lesions may be seen in the spinal cord (21,79). This phenomenon may explain the reason for absence of demyelinating changes on MRI of the spinal cord myelopathy (41,97). Even the typical demyelinating diseases such as multiple sclerosis involving the spinal cord are less likely to manifest pathologic changes on standard MRI-let alone electrical injuries involving the spinal cord (41,97-101).

However, both neurologic examination and neurophysiological tests in the form of somatosensory evoked potential (SSEP) as well as central motor conduction abnormalities have demonstrated definite delay of sensory and motor conduction of the spinal cord (8,41,102).

The SSEP done among our patients showed a high incidence of delay in sensory conduction in 63% of 241 patients tested. The SSEP was done in majority of cases in absence of kerauno paresis (post electrocution paralysis), but due to the presence of weakness and spasm in the lower extremities, pathologic reflexes, and sensory loss in the distribution of T2 through T5 levels over the anterior chest wall as well as a high incidence of urgency and frequency of urination, impotence, and occasional incontinence (Figure 2) (Table 2). The central sensory delay among our patient was similar to the findings of Davidson and Deck (102).

The prominent abnormal pattern in the SSEP test was the fact that stimulation of the sensory nerves in the upper extremities showed statistically nonsignificant central delay whereas

stimulation of the sensory nerve in the lower extremities showed a significant nerve conduction delay, especially involving the thoracic spinal cord. Specifically, the N13-N20 central conduction time delay was noted as has been previously reported (41,102).

Among the patients with delayed development of kerauno paresis, the clinical picture prior to the proper diagnosis has been complex enough that in three cases, the patients have been diagnosed as suffering from ALS. This confusion has already been reported in the literature (29,30).

An accurate differential diagnosis separating the kerauno paresis from the true ALS is not an academic exercise. The kerauno paresis patients have better prognosis and live longer than ALS patients. At the present time we are carrying out a project of treating kerauno paresis patients with ACTH. The final results are not yet available. The ACTH, which is a polypeptide and not a corticosteroid, has been shown to be quite different in its therapeutic effects than corticosteroids (e.g., Lennox-Gastaut Syndrome (LGS)) enhance and detoxification (28,103). Even without any treatment the kerauno paresis patients does not show any shorter life expectancy than other EI patients regardless of how closely the clinical picture resembles ALS.

SYMPATHETICALLY MAINTAINED PAIN (SMP)

Electrical injury (EI) is an ideal model of eliciting pathologic response from the sympathetic nervous system (SNS). The multiple factors of the conduction of electricity through the path of the least resistance causing damage to small arterioles and the sympathetic nerves surrounding them, and causing damage and dysfunction of the spinal cord results in a high incidence of sympathetically maintained pain (SMP) among EI patients. As noted in (Tables 2 and 3), the symptoms and signs of neuropathic pain are quite common among the EI patients. However, a full blown RSD is seen in only one third of such patients. The difference between the SMP and the full blown picture of RSD is SMP, as well as weakness and tremor of the extremity, insomnia, agitation, and depression, and trophic changes of edema as well as skin trophic changes is only seen in one third of EI patients. The SMP, is practically twice as common manifesting itself by the allodynic hyperpathic pain, which is successfully blocked by sympathetic nerve block (SNB), but is not accompanied by the rest of the clinical picture of full blown RSD, is seen in 72% (Table 3) of EI patients.

The spinal cord dysfunction makes the patient more susceptible to develop SMP. The spinal cord dysfunction results in susceptibility to the development of SMP and RSD (8,10,41,92). The extensive studies by Wallin and his associates have demonstrated that spinal cord injury results in partial paralysis of cutaneous sympathetic reflex (104-106). The cutaneous sympathetic responses are not proportional to the visceral vascular changes. The spinal cord injury deprives the spinal cord from the higher inhibitory influences on the anterior lateral horn cells and the motor vasoconstrictive function of the spinal cord. The same studies showed that the decentralized spinal cord does not respond normally to vascular or visceral changes (104-106). The resultant dysautonomia is not only due to increased pressor response to norepinephrine, but also due to the lack of inhibition of the higher CNS centers on the autonomous spinal cord. Secondary systemic hypertension is independent of renal vascular changes, and more dependent on the abnormal reflexes in response to visceral changes such as distention of the bladder or inflammation (106).

The SMP (see "sympathetic function tests" under "Laboratory function) or RSD due to the electrical injury should be treated like any other type of RSD. This is mainly in the form of detoxification, the use of alpha one blockers (such as Clonidine Patch, Hytrin, or Dibenzyline), extensive physical therapy, moist heat, exercise, avoidance of ice, and inactivity.

It is imperative to treat the condition early and to prevent the change of SMP to a full blown picture of RSD.

CHAPTER 11
PREVENTION

The following is the result of our observation and study of the nature of the electrical injuries among 984 patients. The electrical injuries are mostly preventable. The hazards are countless. Strict enforcement of the safety regulations is not routinely practiced.

The following is the breakdown of the preventable factors in industrial, household, and lightning injuries.

INDUSTRIAL

The industrial accidents were due to poor enforcement of the existing electrical safety codes.

- The number one cause of industrial electrical injury in this study was the liberal, out of control use of the breaker box. One worker turns off the box while another worker is not in the area for a coffee break. While the first worker is handling the wires under the safety of the current being already turned off, the next worker returns back to work and unknowingly turns the breaker box on. Proper warning signs and on the job training should prevent this fatal risk (36).

- Stricter use of insulator hats with proper chin strap (while working around high voltage power lines). The hat is accidentally thrown off by the wind exposing the already warm, wet, and sweaty scalp of the worker. This results in the disastrous arcing of the electricity from the high voltage power line to the worker's head. A simple addition of a strap to the safety hats can prevent the fatal accident.

- Stricter quality control, and the use of 220 volt GFI, as well as 110V, in the breaker box. The use of 110 GFI is standard. However, most utility equipment work on 220 volts. If the proper 220 volts GFI is not provided for such a circuit, then the 110 volt breaker box does not recognize the drop of voltage from 220 to 120 volts (still above the 110 volt threshold) while the victim's body is the conduit for the electric short. The 110 or 220 volts current are already higher risk factors because of the fact that they are low voltage currents (below 600 volts) and are more likely to cause tetanic contraction (no-let-go-phenomenon). The improper GFI makes it more dangerous.

- In this study the construction site was a high risk zone. A contractor may provide proper safety measures for the job site and a subcontractor may try to make improvisations and cut corners with resultant substandard safety features.

- The use of cranes and the use of rods by workers or by children in the vicinity of a megavoltage power lines results in fatal accidents. This can be prevented by allocating wider safety zone space between the power lines and the neighborhood.

- The steel radial tires are conductor of electricity. The spare wheel or tire laying on top of a heavy equipment acts as a source of deadly shrapnel when the body of the equipment is charged with electricity. An example is when the crane touches a high voltage power line resulting in the charging of the body of the equipment.

- As is the case with other industrial countries such as Japan, the use of metallic devices (screw drivers, etc.) should be replaced with plastic, nonconductive materials.

HOUSEHOLD PREVENTION

- The use of wireless phones should be encouraged.

- If on a clear day the user of the telephone hears thunder and hears crackling sounds on the wire, she should automatically stop the conversation (1).

- No electrical utilities (radio, hair dryer, hair curlers) should be allowed to be used around the bathtub or shower.

- Proper enforcement and usage of 110 as well as 220 volts GFI. Next to bathtub deaths, lack of 220 volts GFI is the common cause of residential electrical injuries. In absence of a 220 GFI safety device, prolongation of electrical current through the victim's body is fatal (see industrial prevention).

- No improvisation in grounding of swimming pool or jacuzzi lights.

- Avoidance of the use of electrical utilities (telephone, computer, hair dryer, etc.) close to the window during the storm.

- Avoidance of the use of washer, dryer, or ice box while standing on a moist or wet floor.

LIGHTNING

Our experience with lightning strike victims points to the following conclusions regarding risk aversion and precaution.

- Education to run for safety before the lightning catches up. Most of the damage happens on a clear day with lightning as a forewarning of an approaching storm. .

- Taking shelter under a tree or next to a vehicle is high risk. The tree or the car act as lightning rod. The deep roots of trees, such as oak, act as an excellent ground for the lightning.

- Golf cart or motorcycle are not safe (50). However, it makes sense to use the vehicle to run away from the storm.

- The risk is much higher if the body is wet or sweaty such as playing strenuously, swimming or surfing.

- The lightning injuries may be prevented by the use of high-voltage coaxial down conductors (82).

- Some geographic areas are high risks and require more strict precautions. In the state of Florida one example is the "lightning alley" of the Tampa, Bradenton, Sarasota megapolis.

In this study the telephonic injuries were more likely to occur in Hawaii. These incidents are usually due to the use of "shell-type" outdoor phones. This is in contrast with the very low incidence of telephonic injuries in the standard, closed-in, telephone booths.

Of all the risk factors, the one area that needs the most desperate attention and correction is the enforcement of safety features at the place of work, especially insuring the breaker box being off while a worker is handling a bare wire. A simple correction of the safety hats with the addition of a proper chin strap, and enforcement of avoidance of working too close to high voltage power lines are the most glaring risk factors that need to be corrected.

CHAPTER 12
TREATMENT

In dealing with a patient with complex chronic pain, high incidence of depression, seizure disorder, chronic fatigue, insomnia, chest pain, dizziness, vascular headaches, visual disturbance, and urgency and frequency of urination, the patient requires multidisciplinary treatments.

Adding to this complex clinical picture is the high incidence of iatrogenic dependence on addicting opioids and benzodiazepines.

A complex chronic pain which is many causes is in the form of neuropathic pain is going to require the analgesic of choice for chronic pain is to use antidepressants.

ANTIDEPRESSANTS

Chronic pain is a common symptom of EI. Antidepressants are the analgesic of choice for chronic pain. Whereas tricyclics-especially Desipramine and Amitriptyline are effective analgesics for treatment of chronic pain, they have the side effects of weight gain, lassitude, and a tendency in some cases for aggravation of fatigue (80). The tricyclic antidepressants have been referred to as "dirty" drugs because of their side effects listed on the next page.

In the past quarter century, tricyclics have not been the only antidepressants used for management of pain. The non-tricyclics (Trazodone) and selective serotonin re-uptake inhibitors (Prozac or Fluoxetine; Zoloft; or Sertraline; and Paxil or Paroxetine) as well as the newer group of antidepressants (Effexor or Venlafaxine) which is not an SSRI inhibitor, but inhibits the re-uptake of both serotonin and norepinephrine similar to Amitriptyline, have been tried in management of complex chronic pain.

The non-tricyclic antidepressant Trazodone (Desyrel) is not only analgesic of choice for chronic pain and an enhancer of REM sleep (81,107,108). In this regard, it helps the patient to be detoxified from addicting benzodiazepines. It has been observed by King et al that chronic pain patients develop affinity to the intake of benzodiazepines to counteract insomnia (108). Trazodone, provides a more natural REM sleep and helps the patient to be detoxified from the addictive benzodiazepines (75,81,107,108).

Prozac (Fluoxetine) has been found to be effective in treatment of headache as well as other forms of pain (109-113).

Paroxetine (Paxil) has been shown to be an effective antifatigue and analgesic without the autonomic side effects noted with the use of tricyclics (114,115).

By the time the patients were seen for their chronic complications of electrical injury, they were usually under treatment with tricyclic antidepressants especially Amitriptyline or Imipramine.

These time honored antidepressants which have been applied for treatment of pain in the past quarter of the century, have side effects as follows.

SIDE-EFFECTS OF CHRONIC USE OF TRICYCLICS

Chronic use of tricyclic medications can lead to various side effects that may impact a patient's overall well-being. These side-effects can include the following:

- Cholinergic effect.
- Muscarinic effect.
- Obesity.
- Aggravation of chronic fatigue.

Another problem with the use of tricyclic antidepressants is the fact that the blood level of antidepressant is rarely used as a guideline for the dosage of the medication, so the dosage is not tailored to the patient's body weight or individual tolerance of the medication.

The problems of hypotension, fatigue, drowsiness, and inactivity (lack of motivation) result in weight gain, and persistent aggravation of fatigue.

In the past 13 years, we have practically routinely switched the patients from tricyclic antidepressants to Trazodone, and more recently to SSRI antidepressants.

The Trazodone provides an increase in the REM sleep, and helps the patient with control of chronic pain without the above mentioned side effects.

The dosage is adjusted under the basis of two guidelines. One is the degree of drowsiness. If the patient takes enough Trazodone, providing relief of pain during the night and return of sleep pattern back to seven to eight hours a night without drowsiness the next day, then the dosage would be optimal. On the other hand, if the patient still wakes up with pain during the night, then the dosage is gradually increased.

The second guideline is the maximal level of Trazodone which helps in adjusting the dosage within therapeutic range.

In addition, Trazodone has beneficial side effect of helping the patient have better erectile function which is helpful in patients with poor erection.

SSRI ANTIDEPRESSANTS

In the past five years, the complex chronic pain and depression of electrical injury patients have been treated with the help of SSRI antidepressants. Fluoxetine (Prozac) has been quite helpful from the standpoint of being an analgesic, antidepressant, and counteracting fatigue. However, because of its long half-life of almost six days it can have a tendency for cumulative accumulation of the medication in the central nervous system with resultant side effects of aggravation of agitation, insomnia, and poor appetite. In therapeutic doses Prozac helps counteract the obsessive over eating and helps the patient lose weight. On a long-term basis, it can cause too much weight loss and too much agitation.

Taking into account the side-effects of poor erection and poor sexual desire as well as agitation with SSRIs. Trazodone should be the first choice for EI patients.

Zoloft in dosages of 50-200mg a day has been quite effective in treatment of depression, and especially in management of chronic fatigue. In approximately half the patients that have been treated with Zoloft, we have had to prescribe the medication to be taken in the morning and because of persistence of insomnia the patient is instructed to take Trazodone at bedtime.

Treatment with SSRI or Trazodone as well as non-addicting BZs e.g., Klonopin facilitates the detoxification of the patients. This is specifically true in regard to taking the patient off addicting benzodiazepines used for anxiety or insomnia.

In patients suspected of seizure disorder, Wellbutrin (bupropion) Clozaril, and Prozac should not be prescribed.

If the patient cannot tolerate antidepressant due to excessive drowsiness, a combination of small dose of Paxil (1/2 tablet) or Prozac (10mg) in the morning, and small dose of a tricyclic such as Desipramine or Amitriptyline (25mg) in evening may work very well. The treatment by Prozac and its inhibitory function of enzyme P450 blocks the break-down of the tricyclic and provides good sleep.

DETOXIFICATION

The long term use of addicting benzodiazepines (Valium, Halcion, Ativan, Tranxene, Restoril, etc.) and addicting opioids (Codeine, Percodan, Lortab, Demerol, and similar medications) is quite a serious problem in management of complex chronic pain of the problem of chronic pain of the electrical injury (EI) patients.

The above mentioned benzodiazepine (BZ) and opioids result in suppression and arrest of formation of natural endo benzodiazepines (endo BZ) and endorphins. As the result, the patient develops tolerance and dependence towards these addicting drugs, and the pain recurs every

four to five hours when the drug level of opioids drops in the plasma. The same applies to the use of addicting benzodiazepines and endo BZ.

With the help of proper use of Trazodone and/or SSRI antidepressants, non-addicting muscle relaxants, and non-addicting opioids (Stadol and Tramadol or Ultram), the patients have been detoxified from the use of addicting opioids. With the temporary use of Clonazepam (Klonopin) and Buspar or Thioridazine, the patient is tapered off benzodiazepines. The use of Trazodone has been quite helpful in counteracting insomnia and detoxifying the patients from benzodiazepine sedatives such as Halcion and Restoril. Amitriptyline and Effexor have a dual function of blocking the reabsorption of serotonin and norepinephrine. Blocking the reabsorption of norepinephrine may aggravate the neuropathic pain (SMP) that many of the electrical injury patients suffer from (Table 3). The Amitriptyline and Effexor treatment should be discontinued in such patients and should be replaced with SSRI antidepressants or Trazodone.

NON-ADDICTING OPIOIDS

In addition to switching the patients to antidepressants of choice for chronic pain (as the analgesic of choice), the patient should be detoxified from strong addicting opioids. Medications such as Nubain, Talacen, Tramadol, and butorphanol tartrate (Stadol) achieve such a goal-as long as the patient is taken off the addicting narcotics abruptly ("cold Turkey)-so that the combination of morphine antagonists (e.g., Stadol or Nubain) does not cause nausea, vomiting, and excessive drowsiness.

MUSCLE RELAXANTS

Due to spinal cord dysfunction, the patient may need muscle relaxants. The most effective muscle relaxant for this condition is baclofen. The use of carisoprodol (soma) should be avoided due to the fact that this medication is metabolized in vivo to Meprobamate which is a strong tranquilizer with a strong tendency for abuse.

Flexeril has a tendency to aggravate depression. Robaxin has a short half-life and requires repetitive dosage of medication. This can cause the side effect of drowsiness and depression.

In rare and severe cases of spasticity and keraunoparalysis, the patient may need treatment with such large doses of Baclofen that it may cause excessive drowsiness. In such cases, the medication can be infused in small amounts directly in the spinal fluid through an infusion pump (see the next paragraphs regarding infusion pump).

ANTICONVULSANTS

The old fashioned Phenytoin combined with Phenobarbital treatment has no place in the treatment of EI patients. The Phenobarbital has to be detoxified and discontinued. A patient who

already has a problem with disturbance of the function of limbic system in the form of poor memory, poor concentration, irritability, agitation, cannot afford taking Phenobarbital. This strong sedative causes more handicap and disturbance of function of the limbic system by causing drowsiness, withdrawal agitation, strong dependence (addiction), and affecting and aggravating the patient's irritability, agitation, and disturbance of judgment.

Unfortunately, Phenobarbital is ubiquitous in most medications given for pain and headache in the form of combination of aspirin or Acetaminophen and Phenobarbital. If at all possible, such medications should to be discontinued to achieve a better function of an already defective limbic system. This can be achieved by switching the patient to non-addicting analgesics outlined above. For the treatment of seizure disorder, the newer anticonvulsants are more effective.

Recently, there has been a tendency for the use of Ativan, Diazepam (valium), and even Xanax in treatment of recurrent attacks of seizure disorder. These addicting benzodiazepines may help control the seizure disorder temporarily at the expense of the strong side effects of dependence (addiction) excessive sedation, and withdrawal agitation. In this regard, they are no better than Phenobarbital and maybe even worse (Figure 10) (Table 11). In such cases because it takes several weeks or months to detoxify the patients off the addicting benzodiazepines, the above mentioned BZs can be replaced with Clonazepam which has the least competitive binding with the endo benzodiazepine receptors and the least addicting risk of all BZs (Table11).

Figure 10. Combination of addicting drugs and unnecessary surgery causes rapid deterioration of the disease (28).

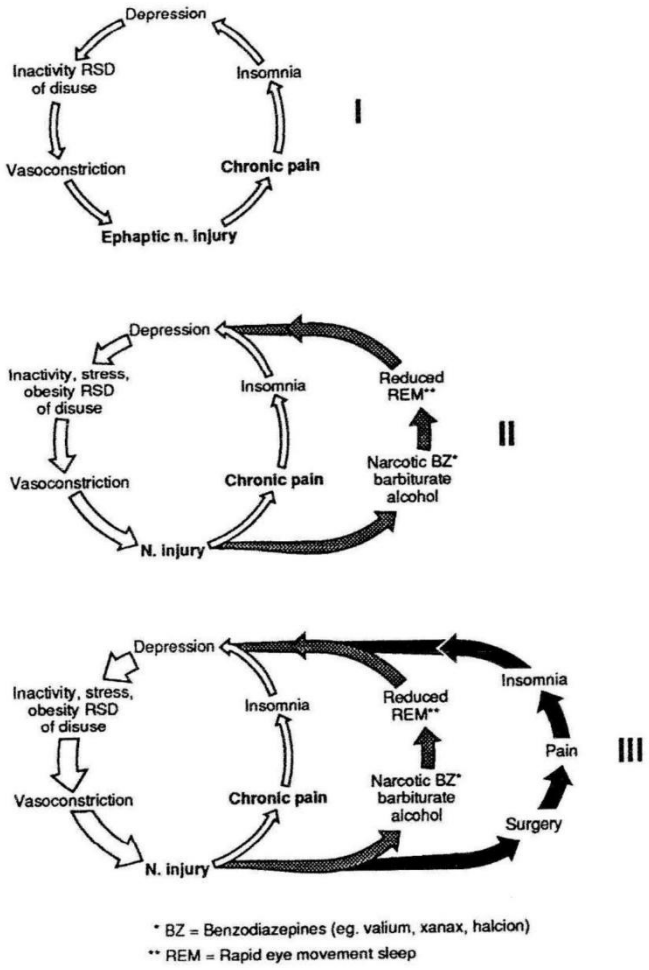


TABLE 11.

BENZODIAZEPINES

ADDICTING AND NON ADDICTING (28)

BZ Hypnotics

1. Flurazepam (Dalmane): half-life, 8 hours; N, desalkyl, Flurazepam (NDF) — its metabolite — half-life 50-300 hours causes depression, poor memory, daytime sedation
2. Temazepam (Restoril): half-life, 8 hours: side effects — poor memory, depression
3. Triazolam (Halcion™): a dangerous hypnotic: half-life 5 hours; longer metabolite binding effect
   a. Strong receptor binding to brain stem (BZ1): strongly addictive
   b. Strong receptor binding to hippocampus (BZ2): confusion, poor memory, depression, anxiety
   c. Withdrawal: daytime anxiety even with occasional use

Insomnia

1. Acute anxiety type: insomnia at onset of sleep
   Rx: L-tryptophane in dairy products; avoidance of ETOH; Rx with chloral hydrate
2. Chronic, intractable anxiety (CIA) with resultant depression: disrputed sleep with frequent awakening
   Rx: antidepressants, e.g., Trazodone (50–100 mg); avoidance of ETOH
3. Cerebral atrophy: early morning awakening
   Aging: usually no Rx needed
   Alcoholism: abstinence, chloral hydrate
   Advanced atrophy: Thioridazine (Mellaril)
   Haloperidol

Endogenous Benzodiazepines (ENDO-BZ)

1. Endo-diazepam: 3 pmol/cc in CSF
   a. In ETOH hepatic encephalopathy the CSF endo-diazepam is 30 pmol/cc (10x normal)
   b. Flumozemil, diazepam antagonist, rapidly reverses hepatic encepahlopthy
   c. Nonhepatic encephalopathic patients have normal CSF endo-diazepam (J. Rothstein, et al., *Ann. Neurol.*, 1990)
2. N-desmethyldiazepam, and 4 other diazepam metabolites, potentiate GABA activity of cerebral cortex

Tendency for Benzodiazepine Binding in Diencephalic — Limbic System[a]

Most binding[b] (dependence)	Average days (detox)	Medium binding	Average days	Least binding[c]	Average days
Triazolam	68	Alprazolam	8	Clonazepam	2
Lorazepam	27			Oxazepam	3
Diazepam	25				
Clorzepate	23				
Chlordiazepoxide	12				

[a] A study of 316 patients detoxified at Neurological Associates, Vero Beach and New Horizons, Ft. Pierce, FL.
[b] The most binding group has the highest tendency to dependence, and a few weeks of detoxification is not enough.
[c] The least binding group (Clonazepam and Oxazepam) did not need detoxification. They were additional drugs among prescription narcotics group that required admission.

INFUSION PUMP

Infusion pump (in the form of Morphine, Dilaudid, Marcaine, Fentanyl, Sufentanil, or Baclofen) works on the principle of the application of a very small dose of medication infused in a continuous fashion ("drip irrigation") providing relief as the medication bypasses other organs such as liver, kidney, etc., and has direct access to the spinal cord and brain. It should be applied to the patients who have documented objective cause of chronic pain that has failed every other form of treatment.

The daily dosage of the treatment is 1-6mg Morphine Sulfate is given in a continuous drip irrigation. This dosage is on the average 1/20 the dose of the same pain medication given in other forms (by mouth, MS Contin skin patch, IM, or IV).

There is an optimal dosage for control of the pain which usually is somewhere around 3-9mg per day. Once the optimal analgesic dose is established, then there is no need to increase the dosage. This is because of the fact that the continuous drip irrigation prevents withdrawal effect, and the amount of analgesic is so small that it does not cause tolerance to the medication.

Interestingly, if the patient tries to additionally use narcotic pills, then the pain becomes worse. Once the optimal dosage is increased by additional narcotic pain medication, the patient experiences more severe pain, and excessive drowsiness.

The fact that the pain becomes worse if the optimal dose is exceeded is suggestive of the large dose of narcotic blocking the formation of natural endorphins.

Addiction has four main principles.

- Tampering with the brain's natural essential hormones and biogenic amines (in the case of pain, blocking the formation of endorphins).

- Withdrawal effect. Even a normal person using narcotics for anxiety or temporary tension headache develops severe pain four to five hours after repetitive use of a strong narcotic.

- Tolerance. As seen in the case of addiction with heroin, alcohol, or cocaine. In such addictions the brain requires more and more dosage of the drug to achieve the same pleasure.

- Destructive effect of the drug on nerve cells producing biogenic amines and hormone to control pain or depression (examples: alcohol, LSD).

The infusion pump bypasses all the three principles and pillars of addiction.

- Infusion pump provides such a small dose of medication that theoretically it does not effectively block the brain formation of endorphins. The best proof of it is when too much of analgesic is applied through the infusion pump, then the pain becomes worse rather than better suggesting the blockade of the cerebral endorphins.

- The medication is continuously administered so there is no withdrawal.

- The infusion pump in optimal dose provides pain relief without the patient realizing that he or she needs to take any medication. The patient cannot self-inflict tolerance because the patient has no control over the adjustment of the dosage of the infusate (the medicine given in the pump).

In our experience of nine years of application of infusion pump in late advanced stages of RSD, electrical injury, and failed back syndrome (over 80 patients), the success rate of two years and longer has been 88%. The other 12% failure rates have been due to the following factors.

- In advanced (stage IV) stages of RSD, or electrical injury, the immune system is so dysfunctional that the patient has a tendency to develop infection around the pump.

- In rare cases, the patient cannot tolerate any type of infusate applied directly into the spinal fluid. In such patients addition of small doses of clonidine (75 to 125 micrograms) enhances pain control with minimal use of analgesics in the pump.

- The patient's poor judgment and addiction to alcohol or other drugs is apt to mess up the function of the infusion pump. The alcoholism is incompatible with any form of treatment of chronic pain including infusion pump treatment. If the patient has any tendency for alcohol abuse, then the pump should not be considered-for that matter, no treatment will do any good.

The process of determining whether a patient can effectively tolerate the infusion pump typically spans several weeks, allowing both the surgeon and anesthesiologist to monitor the patient's response. During this trial period, which usually lasts a few weeks, either the surgeon or anesthesiologist can assess how well the patient adapts to the infusion pump. This evaluation is crucial, as it helps identify and eliminate those patients who may not be suitable candidates for this treatment option. By the end of the trial, most individuals who do not respond positively can be ruled out, ensuring that only those who are likely to benefit from the pump continue with the infusion pump treatment.

One such example is the patient who has hysterical pain. Unbeknownst to the patient, the patient gets relief from pain with even normal saline or sugar and water inserted into the spinal fluid. This also sorts out the patients who are not the proper candidates. However, this specific placebo test should be done on every infusion pump patient as long as one takes into consideration that if the patient has been given narcotic in the infusion pump, it cannot be immediately discontinued and placebo tried because of the fact that the narcotic effect in the spinal fluid can last up to four to five days after infusion of the medication.

CHAPTER 13
OTHER COMPLICATIONS CAUSED BY ELECTRICAL INJURIES (EI)

PSEUDOSEIZURE

According to Critchley in 1934, "one of the earliest records of injury due to electricity is that of a case of hysteria described in the journal Philosophical Transactions in 1754 by Doctor Cheney Hart" (10, 116).

A pseudoseizure gone undiagnosed delays the proper care of the patient. When approached tactfully, by educating the patient and the relatives about the nature of the pseudoseizure (and the fact that the pseudoseizure is different from malingering), the patient accepts the diagnosis quite nicely and considers it a relief. In addition, at times the pseudoseizure and the true epileptic seizure disorder co-exist. Just proving that the patient has pseudoseizure is not the end of the diagnosis and treatment. Even among the pseudoseizure patients every attempt should be made to diagnose any co-existing pathology.

Pseudoseizures were diagnosed by:

- Normal levels of serum prolactin in spite of clinical "seizures" (8,67-69).

- Recording of patient's behavior and simultaneous normal EEG in the face of thrusting, side-to-side shaking movement of the head, while the patient is responsive during the attack.

- IV injection of normal saline and power of suggestion would bring on a "seizure" and would stop it as well (8).

Our protocol has been to first of all document and ideally observe the seizure disorder. Secondly, objectively make the diagnosis of conversion reaction by observing the patient's type of seizure during an attack-as well as the patient's response to placebo versus to anticonvulsant.

Realizing that in myoclonic seizures (seizures originating from the spinal cord or lower brain stem in the form of myoclonic or akinetic attacks) the surface EEG shows abnormality in about one-fourth of such patients, we have had to rely on the clinical picture, the type of seizure, and the requirement of an objective abnormality on evoked potentials (SSEP and BAER).

Even after all the above documentation has been done and the placebo trial has been made, there is an additional safeguard to avoid improper treatment, in the form of anticonvulsant level of blood (compliance).

Especially in of myoclonic seizures, non-compliance has a direct relationship with the recurrence of seizure. In the case of conversion reaction patients, there is no relationship to blood level of anticonvulsant; and the seizure is more likely instigated by emotional crisis.

The treatment of choice for myoclonic seizures in our group of patients has been Valproic Acid. In 28 such patients, Valproic Acid did not control the seizures, and such patients were tried on Klonopin with good results (89). In occasional intractable seizure patients, we have had to resort to other therapies such as treatment with a combination of standard anticonvulsant and Neurontin.

HEADACHE

Headache is quite a common symptom among the EI patients. Usually, the patients are treated with a combination of Inderal (Propranolol), analgesics that contain barbiturates, and injections of Imitrex. This combination does not work for EI patients. Propranolol aggravates the chronic fatigue, and results in weight gain and depression. Barbiturates (such as Fiorinal) aggravate the depression and cause a strong tendency for dependence (addiction). The Imitrex did not help a single case of vascular headaches due to EI. The EI victims quite frequently suffer from a combination of headache, dizziness, and blurring of vision. The vertigo, headache, and visual problems are due to trigeminal vascular dysfunction accompanied by vertebral artery constriction with resultant vertigo and blurring of vision. In occasional cases, the vertebral artery vasoconstriction can result in altitudinal visual field defects as well-which are usually transient.

Such patients suffer from other manifestations of electrical injury that contribute to this type of headache. These consist of severe cervical paraspinal spasm and marked paravertebral nerve irritations in the zygapophysial paravertebral nerves (Figures 11 and 12). Paravertebral as well as epidural nerve blocks in these patients are quite successful in treatment of the severe headaches. On the same basis, occipital nerve blocks are quite effective as well. Combining the nerve blocks with proper muscle relaxants (Baclofen, Skelaxin, or Norflex), physical therapy, and application of alpha blockers such as clonidine patch are quite helpful in control of the vascular headaches, blurred vision, and vertigo. Discontinuation of the Propranolol and replacement with alpha blockers is quite helpful. A strict migraine vascular diet is also very helpful in management of the headaches (see section about diet below).

FATIGUE

Chronic fatigue is usually the result of multifocal central nervous system (CNS) EI injuries. Electroporation aggravates the chronic fatigue.

The combination of proper antidepressants that provide normal REM sleep (such as Trazodone), proper diet (see below), and application of proper muscle relaxants such as Baclofen or Skelaxin are quite helpful in the management of chronic fatigue. Paxil (10-60mg/day) or Zoloft in the doses of 50-200mg a day also are quite helpful in the management of the chronic fatigue. Chronic fatigue aggravated by severe chronic anxiety responds nicely to the treatment with Buspar.

Alternating proper exercise and rest also helps the problem of chronic fatigue.

In more intractable cases, treatment with Symmetrel may help.

Figure 11. Electrical Injury(EI) causes referred pain and headache that can be helped with nerve blocks (28).

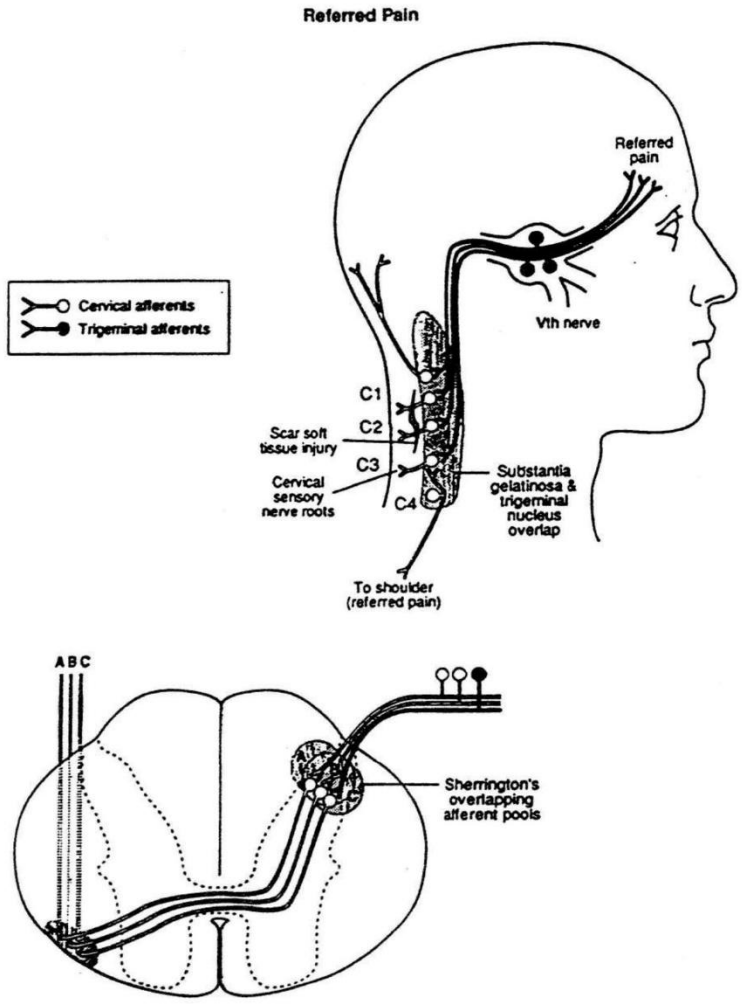
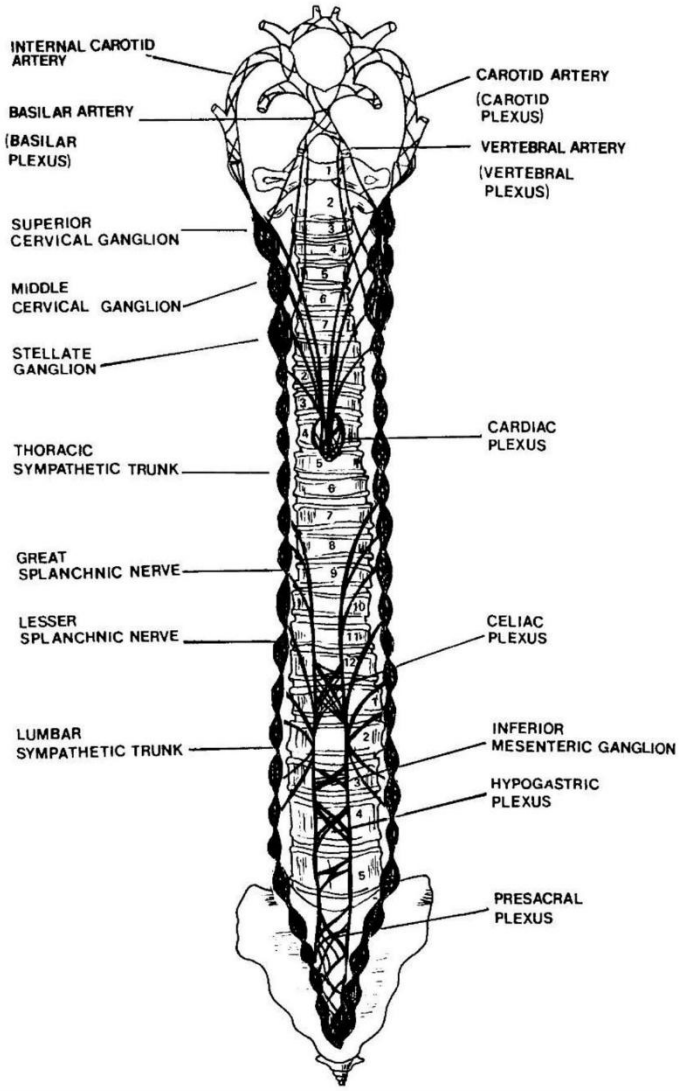


Figure 12. The sympathetic system is interconnected across the midline and vertically resulting in the spread of pain, as well as headache and dizziness, to other parts of the body.



NEUROPATHIC PAIN

Neuropathic pain (sympathetically maintained pain-SMP) treatment mainly consists of nerve blocks, alpha one blockers, discontinuation of addicting benzodiazepines and narcotics, extensive physical therapy, heat and exercise, application of Epsom salt and hot water, and specifically avoidance of ice which aggravates the vasoconstrictive tendency due to the SMP (28-30).

Avoidance of wheelchair, assistive devices, braces, and inactivity is essential in treatment of SMP.

Use of Trazodone and, if needed, SSRI antidepressants (e.g., Prozac or Paxil) should be considered as the number one line of defense against chronic pain. Alpha blockers such as Clonidine, Hytrin, Dibenzyline, and Yohimbine are effective in treatment of SMP, or its less frequent form reflex sympathetic dystrophy (RSD).

However, sympathectomy should not be considered because it is apt to fail in late stage of the disease due to the fact that the pain is not just an SMP pain, but also sympathetically independent pain (SIP). In addition, sympathectomy does not prevent the spread of the sympathetic dysfunction proximally or distally in the chain of the sympathetic ganglia with the spread of the disease from upper extremity to lower extremity and vice versa.

GASTROINTESTINAL COMPLICATIONS

The GI complications can be quite intractable, especially in the case of lightning strike patients. These consist of bouts of diarrhea, severe abdominal pain, weight loss, and paralysis of gag reflex, esophagitis with severe chest and epigastric pain, dysphagia, and chronic gastritis.

Treatment with Prilosec, Zantac, and Liquid Aloe are quite effective in treating the above mentioned complications. In the more severe cases, treatment with Tincture of Belladonna or Elixir of Paregoric may be helpful.

The epigastric stabbing and causalgic type of pain may respond to Coeliac ganglion block in combination with treatment with anticonvulsant Tegretol. In more severe cases, addition of Neurontin in therapeutic doses (1800-3600mg) may control the condition.

In more severe cases when everything else fails, then the last resort is infusion pump (see below).

ELECTROPORATION

Treatment with calcium channel blockers, (e.g., Verapamil) ACTH, Sodium channel blockers (e.g., carbamazepine, phenytoin, IV lidocaine, and Mexiletine) are helpful. More effective treatment is desperately needed.

SURGERY

Quite frequently the electrical injury patients are told that they need surgery for the problem of cervical or lumbar disc bulging or herniation.

The electrothermal effect of the EI frequently causes disc desiccation.

The intense force of electricity can propel a person away from its source, sometimes up to six or seven meters, leading to fractures in the vertebrae, particularly in the thoracolumbar junction area.

Disc bulging and disc herniation are not at all uncommon among the EI patients. The disc bulging is by far more common than the disc herniation.

Even in patients who have not suffered from electrical injury, disc herniation should not be treated surgically. Over 95% of the disc herniations are successfully treated conservatively with physical therapy, traction, moist heat, etc.

Disc bulging should practically never be operated on. Disc bulging is not a sign of impingement of the nerve roots.

In late stages of electrical injury, any kind of operation has the potential to aggravate the sympathetically mediated pain (SMP). A patient who has had a simple SMP quickly changes to a full blown reflex sympathetic dystrophy after an unnecessary operation for disc herniation, disc bulging, or "carpal tunnel syndrome." The nerve conduction studies in EI patients usually show borderline slowing of conduction time and low amplitude response to electrical stimulation. Mistaking such conditions for carpal tunnel syndrome may have disastrous results

DIET

Correction of bad eating habits is not just necessary for the patients suffering from GI symptoms, but is also essential for patients suffering from pain, depression, chronic fatigue, and other complications of electrical injuries.

Certain foods aggravate the pain, depression, and GI symptoms of electrical injuries. Any food that contains large amounts of tyrosine is a stimulant and specifically aggravates the SMP (sympathetically maintained pain). The best examples are sausage, kielbasa, cold cuts, and liver.

Chocolate is another stimulator of the sympathetic system due to the fact that it contains Phenylethylamine which is a precursor of Norepinephrine and Dopamine. Phenyletholamine is from the cocoa tree and its effects are similar to cocaine.

Hot dogs, containing nitrates, also aggravates the chronic pain as well as disturbing the immune system.

In patients suffering from severe GI symptoms due to the electrical injury, treatment with yogurt, buttermilk, and potable aloe vera can be quite helpful. The yogurt and buttermilk help balance the bacterial content of the guts, and aloe vera helps soothe the ulcerative lesions. The autopsy of electrical injury victims shows necrosis, ulcers, and marked inflammation of the mucosa of the guts.

The patients should routinely consume regular three meals and a snack at bedtime. Fasting from after dinnertime (usually 6PM) all the way up to 7AM the next morning results in a drop of the blood sugar in the early hours of the morning, with resultant mobilization of the glucose from muscle and liver due to the stimulation of Epinephrine. This aggravates the insomnia, and disturbs the natural REM sleep with secondary tendency for depression and fatigue.

CHAPTER 14
THE EFFECTS OF ELECTRICITY ON BODY ORGANS

SKIN

In vertebrates the skin consists of two major layers: the richly cellular relatively thin outer layer, or epidermis, and a thicker and tougher inner part composed of fibrous connective tissue, called the dermis or corium.

The human skin varies in thickness from less than one millimeter on the eyelids to three millimeters or more on the palms of the hands and the soles of the feet. The average thickness of the epidermis is only about 1mm, although on the palms and soles it may be 10 to 15 times thicker, largely due to the greater thickness of the outermost cornified layer.

The epidermis consists of the cornified outer layer, the stratum lucidum, the stratum granulosum, the stratum mucosum and the stratum germinativum.

The stratum lucidum presents a major barrier against the passage of water and salts through the skin. Since there are no blood vessels in the epidermis, its cells must receive their nutrition from intercellular tissue fluids in the stratum mucosum, thus creating a moist environment just below the outer barriers.

The cerium intrudes with finger-like projections into the stratum mucosum; these fingers bring blood and nerve fibres near the skin surface. As already described, these are ion-rich viscous containers, or simply conductors.

SWEAT

Sweating, an important factor in cooling and stabilizing the body temperature and the body salts, also causes a sharp, precipitous drop in the skin resistance. This drop reduces the heat and electrical burn, but results in more current (amperes) entering the body resulting in more damage to nerves, muscles, blood vessels, and the nerves in the deep structures of the body.

Various physiological factors control the sweat mechanism, but the most prominent one is the sympathetic nervous system. The main purpose of the sympathetic nervous system is control of the vital signs, control of the immune system, and the control of the body temperature. The control of the body temperature is mainly achieved by preserving the heat by constricting the blood vessels to the skin, or emanating and wasting the un-needed heat through the function of sweating. The main purpose of sweating is controlling body temperature (evaporation-the same principle used in air conditioning). However, it also maintains normal hydration of the outer layer of the thickness of the skin.

ELECTRICAL PENETRATION

The skin serves as a remarkably effective protective barrier against a wide range of potentially noxious chemical, physical, biological, and electrical factors.

The outer layers of the cells contain keratin (referring to the Greek word for horn, a sulfur-containing fibrous protein, which renders the skin relatively inert. This undoubtedly accounts for the electrical resistance of the epidermis.

The key to protecting the inner organism from electrical shock is to prevent the initial excitation of the electrons. Under simple conditions, this can be accomplished easily with gloves, rubber soled shoes, grounding wrist-straps and various other obvious mechanisms. The dry skin is quite resistant to electricity (100,00 to 300,000 ohms). The wet skin has 100's to 1,000's of volts less resistance.

The resistance to electron flow increases when no positive node is provided to attract it. Standing on a wooden surface with rubber soled shoes limits the body from assuming a positive ground potential. This prevents the flow of the electrons and disrupts the circuit.

ARCING AND SKIN RESISTANCE

The antithesis to this neat logic of electron resistance is arcing.

The higher the voltage, the more likely it is to continue flowing. Due to the extreme electromotive force (E), the very high voltage *is* quite likely to find the easiest path to ground.

Arcing plays a role in the penetration of otherwise resistant skin even when it may not appear to be the case. The physiology of the subdermal layers argues for it. Arcing provides the heat that burns the skin through the microwave effect.

THE MICROWAVE EFFECT

A microwave "oven" cooks by bombarding water molecules within food with electromagnetic radiation, exciting the subatomic particle and thus creating heat. This gives rise to the popular notion that microwaves cook from the "inside out." That is the reason the microwaves are unable to "brown" the outside of a roast.

In arcing, and other forms of electrical burns, the damage is usually deeper than it seems to the eyes during the acute phase of the burn.

CAPACITANCE EFFECT

The skin resistance against the alternate current (AC) is best explained by the capacitance effect. When a negatively-charged conductor is separated from a positively-charged one by a fine non--conductive membrane (a dielectric) the current will tend to build up on each side of the dielectric

until it can no longer be contained. Then the current jumps the dielectric barrier. This obviously results in an immediate rise in the current, with a concomitant immediate fall in resistance.

The voltage attempting to penetrate the skin keratin to get at the conductive inner tissues may set up a capacitor, which unloads forcefully and causes a breakdown to resistance by increasing voltage. The increased voltage would produce conductive heat, which would release moisture from the corium layer of the skin, thus producing a cascade effect.

What may limit the capacitor effect in skin penetration is the small exposure area. The more focused the area of the entrance of electricity, the more easily it breaks down the capacitance effect of the skin. This is usually noted as an area of the skin that is left with less keratin and more focally damaged and scarred layers of the skin.

THE BODY AS CONDUCTOR

The next consideration of conductors involves the skin effect. Here, the skin does not refer to human skin, but the surface of the conductive material.

The resistance of a conductor is not the same for alternating current as it is for direct current. With alternating current there are internal effects that tend to force the current to flow mostly in the outer parts of the conductor. This decreases the effective cross-sectional area of the conductor, with the result that the resistance increases.

The moist skin results in a decrease of skin resistance resulting in more deep organs and neurological damage.

The higher skin resistance and skin burn, the less visceral and brain damage (and vice versa).

It may be that invasive current tends to flow in specific channels, such as blood vessels and nerve fibres, with their high water content and high concentration of ions. It cannot be overlooked, however, that current flows somewhere, and that somewhere will have a positive potential compared to the current source.

Furthermore, it can be demonstrated that the current following bone and tendons (highly resistant compared to blood vessels) will arc across a joint.

NERVE CONDUCTION

The higher animals receive information about their environment, or about their bodies, along sensory nerves (afferent) to a central nervous system (CNS), or central processing unit. The processing unit then may send a response through motor nerves (efferent).

The sensory afferent fibres are subdivided into the somatic - those concerned with perception of external factors or the movements of muscles and joints; and the visceral - those concerned with the internal organs and economy of the body temperature and vital signs.

Likewise, the efferent fibres send responses to the voluntary muscles through the somatic, and to the internal organs and glands through the visceral. Generally, the visceral efferent fibres constitute the autonomic system, which is further divided into the sympathetic and the parasympathetic systems.

The autonomic electrical pathway is vaguely similar to the transformer described above in that the nerve pulse arises in the brain or spinal cord (CNS), but ends in a ganglionic interface; these ganglionic fibres in turn stimulate adjacent ganglionic fibres that cause a second pathway to carry the signal to the organ innervated.

While nerve fibres conduct the necessary impulses, the cells that serve them are merely supportive; they are located close to or within the spinal cord.

The bulk of human nerve fibres consist of a protoplasmic core (axon) surrounded by a sheath of fatty substance known as myelin; this sheath is interrupted at regular intervals by a short gap. Some nerve fibres are not myelinated; they consist of simply the protoplasmic core encased in a membrane.

While commercially available electrical currents are transmitted electrostatically, organic electrical transmission is propagated electrochemically.

The protoplasmic core of the nerve fiber is aqueous (watery) and therefore a reasonably good conductor of electricity. This liquid medium contains a high concentration of potassium ions, and a relatively low concentration of sodium and chloride ions. In extracellular space the opposite is true.

In contrast to the protoplasm of the nerve fiber, the enveloping membrane is insulative, resisting any electrical potential across its structure. When an electrical change creates an impulse, it is known as an action potential.

Action potentials last about 1/1000 of a second, and travel at speeds up to more than 200 MPH. The diameter of the fiber, the velocity, and myelinated fibers conduct faster unmyelinated ones.

Significantly, in any given fiber the magnitude and velocity of the action potential is constant, and is not altered changing the strength or quality of the stimulus. The reason is the nature of the electrochemical process involved.

This process is totally unlike the method of propagation of an electrical signal in a metallic conductor. Electrostatic conduction pushes electrons free by repelling them with a negative

charge. Electrochemical conduction is achieved by creating a positive/negative potential – i.e., a voltage potential - across a membrane through a chemical change.

The resting membrane is impermeable to the sodium ions in the external solution, but is moderately permeable to the potassium ions in the protoplasmic core. Thus, a resting potential can be measured of usually about 0.07 volts negative relative to the external solution.

As conductivity begins, the membrane apparently becomes less resistant to the sodium ion in the external solution. The change in permeability generates the action potential and the movement of sodium ions provides the inward current on which conduction of impulses depends. The inside medium swings momentarily positive; at the peak of the action potential the inside of the fiber is about 0.04 V positive to the outside solution.

Shortly after a stimulus has caused a response, the nerve experiences a refractory period, during which no stimulus, no matter how strong, can evoke a second response. This period is usually about 1/1000th of a second; there is a shoulder period following the refractory period during which a second impulse can be evoked, but only by a stimulus stronger than normal.

The restoration of the normal potential difference is brought about by an outward movement of potassium ions and is accelerated by a rise in potassium permeability, which takes place subsequent to the initial rise in sodium permeability.

CHAPTER 15
SAFETY DEVICES

Electrical safety devices originally relied solely upon the heat principles already mentioned. The word fuse means to reduce to a liquid fusion by means of heat. This led early to the invention of a circuit interrupter employing a carefully engineered conductor designed to burn across when the desired safe current threshold was reached. Due to flash the fusible conductor was enclosed in a safe container - usually glass tube, allowing the consumer to see whether the fusible material had separated or not.

One of the drawbacks to this system was its insensitivity to slowly increasing heat. Nominally, with our formula (P=IE) in mind, it is easy to see how a direct short between the conductors would cause a rapid rise of heat within the circuit. Since the current is relatively constant throughout the circuit, it follows that the heat is relatively uniform throughout the circuit as well. These dynamics would lead to the blowing of the fuse on the principle of the weakest link.

However, resistance can vary greatly throughout the circuit; obviously, resistance is a primary source of heat. As a consumer utilizes more and more appliances at any given time on the same circuit, the wattage would naturally rise in steps. These dynamics do not always result in the burning open of the fuse, even though it does usually accelerate the oxidation of the conductor.

Another source of increasing wattage within the circuit is a poor connection. This commonly occurs when a relatively high current device is connected to the circuit, and the connection is not technically perfect-for example, a degraded wall receptacle. Heat builds up across the conductive connection, and is slowly conveyed along the wires, again causing oxidation of the conductors.

Oxidation of conductive materials, commonly copper in our example, causes more resistance to build in the circuit, which naturally produces more heat.

The purpose of the fusible safety device is not to protect the consumer directly; it is to protect the conductors!

When conductors, which are usually fastened to wood in the residential setting, become hot enough, they will ignite adjacent materials. Under the conditions of slowly increasing current already mentioned, the local heat in the conductors would exceed the heat in the fusible link and can cause a fire.

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ACRONYMS

Adult Attention Deficit Hyperactive Disorder (ADHD)

Amyotrophic Lateral Sclerosis (ALS) (Lou Gehrig Disease)

Alternate Current (AC)

Basic Electrical Rhythm (BER)

Blood Brain Barrier (BBB)

Central Nervous System (CNS)

Computed Tomography (CT) Scan

Current Intensity (I)

Direct Current (DC)

Electric Force (E)

Electrical Injuries (EI)

Electrocardiogram (EKG)

Electro-Convulsive Therapy (ECT)

Electromotive Force (EMF)

Frontal Lobe Dysfunction (FLD)

Gastrointestinal (GI)

Ground Fault Interrupter (GFI)

Halstead–Reitan Neuropsychological Test Battery (HRNB).

Infrared Thermal Imaging (ITI) (Thermography)

Kilovolts (KV)

Lennox-Gastaut Syndrome (LGS)

Luria-Nebraska Neuropsychological Battery (LNNB)

Magnetic Resonance Imaging (MRI)

Minnesota Multiphasic Personality Inventory (MMPI)

Multiple Sclerosis (MS)

Ohm's Law (V=RxI)

Positron Emission Tomography (PET)

Post-Traumatic Stress Disorder (PTSD)

Rapid Eye Movement (REM)

Reflex Sympathetic Dystrophy (RSD)

Resistance (R)

Somatosensory Evoked Potential (SSEP)

Sympathetic Nerve Block (SNB)

Sympathetic Nervous System (SNS)

Sympathetically Maintained Pain (SMP)

Voltage (V)

Watt (W)

Doctor Hooshang Hooshmand, dedicated his life to medicine (Neurology), teaching, and caring for his patients for over 40-years. He had a special interest in the management of Reflex Sympathetic Dystrophy (RSD), and other neurological conditions such as Multiple Sclerosis (MS), Epilepsy, and Electrical Injuries. He had written and published more than one hundred medical articles and abstracts on the subject of Reflex Sympathetic Dystrophy (RSD). He also had written the first Textbook on Reflex Sympathetic Dystrophy (RSD) (1993).

He had a great passion for writing on many medical topics such as Electrical and lightning strikes Injuries, RSD, Cerebrospinal Fluid Pressure, Topographic Brain Mapping, Thermography, and including this book on the neurological complications of electrical injury. He had made many landmark contributions to medicine over his 40-year career.

Eric M. Phillips is the President and Founder of the International RSD Foundation in Massachusetts.

Eric has worked in the RSD-CRPS Community for over 35 years. He has authored and co-authored over 20 medical articles and 15 textbooks on the subject of Complex Regional Pain Syndrome (CRPS).