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Delayed neurological sequelae of electrical injuries

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THIS REPORT reviews the neurological disorders that follow electrical injury to man to emphasize the long latent period that can intervene between electrical contact and the appearance of neurological dysfunction. The existence of such delayed effects received considerable mention in the past neurological literature,¹⁻⁵ but only a few sporadic cases have been reported in the past fifteen years.^{6,7} The significance of this problem for both clinical and medicolegal considerations can be inferred from the high mortality rate that exists at present from inadvertent electrical contact. Approximately 1,000 deaths occur annually in the United States and the number of injuries not resulting in death must be of a far greater magnitude. The fact that one-third of these fatal accidents resulted from contacts with household currents indicates that electrical injury is readily possible for most of the population.

CASE REPORT

A 67-year-old right-handed man was injured on June 1, 1964, by 18,000 volts (A.C., 60 Hz) from a high-tension line while at work as a pipefitter. He was standing on a rock guiding one end of a metal pipe with his right hand when the other end of the pipe hit the high-tension line. He remembers a loud "bang" and was told that he was thrown about 4 ft. from the rock. He was dazed for only a few seconds and resumed work immediately, even though he felt "shaky" for the remainder of the day.

The following day right arm and interscapular pain developed and he went to his physician who found an entrance burn on the right palm and an exit burn of the lateral aspect of the left little toe. These minor burns healed without complication. The patient received several injections of a local anesthetic intramuscularly around the right shoulder in the region of pain, with some relief over the next week.

During that same week, he noticed minimal

weakness of the left leg which did not interfere with his work and did not progress. He remained unchanged until June of 1966, two years after the electrical injury, when he experienced lightning-like pains starting in the low back, radiating around the left hip, and descending down the medial aspect of the left leg to the ankle. The pains occurred repeatedly during twenty-minute periods and then subsided, only to recur for five to six more periods each day. He also noted, for the first time, that this area of the leg was numb. He was seen by an orthopedist who noted the left thigh to be smaller than the right. Lumbar spine films revealed minimal osteoarthritis. The patient was placed in traction without relief. The leg weakness, which up to this time had been slight, gradually progressed so that he had difficulty in climbing stairs and had to resort to a cane for walking. In August of 1966, twenty-seven months after his injury, he noted the muscles in the left thigh were twitching. At no time, however, did the patient experience difficulties in voiding or defecation. A lumbar myelogram was performed in the same month and was normal. Spinal fluid was not examined at that time. The patient was then referred to the Stanford Medical Center in November of 1966 for further evaluation.

The patient had been healthy all of his life, except for diabetes mellitus, discovered while in the hospital in 1961 for a suprapubic prostatectomy for benign prostatic hypertrophy. The diabetes was well controlled by diet and phenformin therapy.

Physical examination: The blood pressure was 160/90, pulse was 86, and respirations were 16. The general physical examination was normal except for bilateral pterygia and a brawny discoloration of the skin over the tibiae. On neurological examination, the patient's mental status, cranial nerves, and deep tendon reflexes were within normal limits except for an absent left patellar reflex. The patient's gait was remarkable for his inability to completely extend the left knee on stepping

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forward with the left leg. The left thigh was atrophied and a few fasciculations were seen in the quadriceps. The right thigh measured 40 cm. and the left 36 cm., 10 cm. above the patella. Both calves measured 32 cm., 15 cm. below the patella. The following muscles in the left leg were weak (4, on a 0 to 5 scale):⁸ iliopsoas, sartorius, quadriceps, adductors, hamstrings, and posterior tibial.

The sensory examination was normal in the upper extremities. There was decreased sensation to pin, touch, temperature, and vibration in the distribution of the left second through fourth lumbar roots. In the area of the left tenth thoracic through first lumbar roots, diminution of the same modalities was inconsistently reported. Vibration and position senses were well preserved in the toes.

The laboratory studies revealed a fasting blood glucose of 220 mg. per 100 ml. A lumbar puncture showed an opening pressure of 140 mm. spinal fluid. The spinal fluid contained no cells, a protein of 32 mg. per 100 ml., and a glucose of 90 mg. per 100 ml. An electromyogram revealed no insertional activity, fibrillations, or fasciculations. About 50% of the motor units in the left adductor and quadriceps were polyphasic in form, 3 to 4 msec. in duration, with amplitudes of 1 to 1.5 millivolts. These same muscles showed a marked decrease in the number of motor units on maximal contraction. The right adductor and quadriceps muscles showed normal electromyographic patterns. Motor nerve conduction studies on the following nerves, tested bilaterally, were normal: common peroneals, posterior tibials, and ulnars. A muscle biopsy of the left quadriceps revealed groups of fibers that measured 15 to 20 μ in diameter among fibers of normal size. There were no inflammatory cells seen. This pattern was felt to be consistent with a neurogenic atrophy.

The patient's course has remained unchanged since hospitalization (as of May 1967). A diabetic amyotrophy or radiculopathy did not seem likely since the spinal fluid protein was normal and there was no evidence of slowed nerve conduction.

In summary, the patient is an elderly man

who experienced a high-tension electrical injury twenty-seven months prior to the study. The current passed from his right arm to his left foot. Twenty-four months after the injury, paresthesia, atrophy, weakness, and fasciculations of the left thigh became apparent. Our studies were consistent with neurogenic atrophy in this region.

DISCUSSION

We will discuss electrical injuries to the nervous system in terms of [1] the types of clinical disorders seen (Table 1) and [2] the possible mechanisms of such nervous system damage.

Cerebral syndromes. When the head is one of the contact points, the patient may suffer burns of the scalp and underlying bone and become unconscious. Those patients not rendered unconscious by the shock hear a loud sound or have tinnitus and may even become deaf. Headache and giddiness may be experienced for a period of time following the injury. Of more interest are the permanent neurological deficits which follow electric shock after a latent period.

Critchley⁹ reported a case of a 26-year-old man who was shocked by an unknown amount of current for about fifteen seconds. The patient suffered a burn of two fingers, paresis of the right arm, and an inability to speak. He recovered completely from the symptoms in a short time. Nine months later, the patient suddenly developed right hemiparesis with aphasia. Critchley proposed that the recurrence of symptoms resulted from a delayed vascular occlusion secondary to intimal damage from the original electric shock.

Langworthy³ described one of Foster Kennedy's patients, a man of 52, who was rendered unconscious by lightning while playing golf. Immediately, there was a partial paralysis of the right side of the face and a complete right hemiplegia. Movements returned within the next twenty-four hours. In the ensuing three weeks the patient's face moved normally on voluntary effort, but the right side of the face did not move during emotional expressions. The patient then developed a rhythmical tremor of the right upper extremity, a loss of associated movements of that arm, and some stiffness of the right leg while walking. It was

TABLE 1
CLASSIFICATION OF NEUROLOGIC DISORDERS
FROM ELECTRICAL SHOCK*

Cerebral syndromes:
Hemiplegia, with or without aphasia;
striatal syndromes; brainstem syndromes
Spinal syndromes:
Spinal atrophic palsies; hematomyelia,
spastic paraplegia
Peripheral nerve syndromes:
Isolated or multiple radiculopathies or
neuropathies

*Modified from Critchley, 1936

the author's opinion that the current from the lightning had damaged the basal ganglia on the left side of the brain.

Haase and Luhan⁷ reported a case of delayed thrombosis of the basilar artery following electrical injury. Their patient, a 45-year-old man, was shocked by a high-voltage transformer and experienced no symptoms. He continued to work for the next eighteen days. The patient's family noted some minor personality changes during this period. Then, on the nineteenth day, the patient suddenly collapsed to the floor with a right hemiplegia, became unconscious, and remained in a deep coma until his death a year later. An autopsy revealed occlusion of the basilar artery, resulting in encephalomalacia of the rostral end of the pons and thalamus. It was the contention of this group that the thrombosis was due to intimal damage of the basilar artery caused by the electric shock.

Spinal syndromes. Spinal cord damage is by far the most common of the permanent sequelae of electrical injury and occurs when the path of the current is either from arm to arm or from arm to the leg.

Panse⁵ collected 20 cases from the literature and reported 9 additional cases. The spinal forms may resemble either progressive muscular atrophy, amyotrophic lateral sclerosis, or a transverse myelitis. Sensory changes in the affected limb are also very common. Most of the time there is pain and paresthesia in the involved limb immediately, but this usually clears in a short time. Permanent symptoms occur with a latent period of days to months, are of gradual onset, and progress slowly.

Alexander's⁴ second case is a good example of a spinal syndrome. The patient was a 46-year-old man who was holding an overhead socket with his left hand while screwing in a light bulb with his right hand. Inadvertently, the pull-chain was pushed into the socket and the patient was shocked on the left hand, burning the middle finger. The current was estimated to be 25 milliamperes. The next morning he awoke with pain and weakness of the left arm. The patient was seen three weeks later and the left arm showed atrophy, fasciculations, and hyperesthesia in the distribution of the fifth through seventh cervical dermatomes. Over the next few months, the pain subsided

but there was progression of the atrophy. The lesion then stabilized and did not progress for the next two years when the patient was lost to follow-up. The author felt that the progression of neurological signs was due to a vasculitis of the spinal cord caused by electric current.

Delayed quadriplegia has recently been reported.¹⁰ The patient, a 10-year-old boy, was flying a kite with a stainless steel fishing line for a kite string when the wire came in contact with a transmission cable carrying 75,000 volts. The boy suffered severe burns to the body, with an entrance wound in the right hand and an exit wound in the left heel. Following the accident, the patient did well. Seven weeks after the injury, the patient was found confused and disoriented and, on the following day, developed a flaccid quadriplegia with a motor and sensory level at C-7. The patient died and histological examination of the spinal cord showed severe damage in the lower cervical and upper thoracic regions resembling those of recent anoxia of the spinal cord. There was edema, minimal perivascular infiltrate, intravascular thrombosis, and little evidence for an arteritis present in the involved areas. No evidence was found suggesting an infectious process.

Radicular and peripheral nerve syndromes. In most of the cases where peripheral and radicular nerves are involved, there are extensive burns to the limb. The peripheral nerves in the area may be directly burned or compressed by the resulting scar tissue, but a similar neurological picture may also develop in the absence of obvious burns.

Langworthy³ reported a 46-year-old switchboard operator who received a shock of unknown intensity from the switchboard. The patient suffered no burns or loss of consciousness but immediately experienced pain in the right shoulder and anesthesia of that same hand. The patient showed spasms of the right shoulder girdle muscles and loss of sensation over the right thumb and index and middle fingers, extending up the volar surface of the right arm to the shoulder. The biceps and triceps reflexes were depressed on that side. Laboratory studies were reported as normal.

Thus, almost any combination of neurological signs and symptoms may follow electrical

injury and a period of time, up to many months, may intervene between the injury and the development of the neurological dysfunction.

Mechanism of delayed injury. Experimental studies have defined some features of exposure to electrical currents that are significant for nervous system damage. The amount of current passed through the body appears to be the most significant factor in determining cellular damage. Alexander,⁴ in a study of the effects of a 60-cycle, 110-volt circuit, reported

that a current of 0.2 to 0.33 milliamperes applied to the skin felt like a "tap," 0.75 to 1 milliamperes felt like a "pinch," 1 to 4 milliamperes felt like a "grab," and 5 to 15 milliamperes caused muscle contraction. In experimental animals, tested with the same circuit, 25 milliamperes or more caused permanent damage to nerves and blood vessels and 70 or more milliamperes could be lethal immediately.

The skin provides protection from electric shock. It has been estimated that a dry, cal-

TABLE 2
REPORTED CASES OF DELAYED INJURY

Author and year	Voltage	Syndrome	Delay following shock
LeRoy de Mericourt, 1860 (cited by Panse)	Lightning	Atrophy, right hand	Some days
Colling, 1891 (cited by Panse)	1,000 v.	Atrophic paralysis of left deltoid and teres minor	Some days
Hoche, 1899 (cited by Panse)	1,000 v.	Atrophic paralysis of right deltoid	1 year
Eulenburg, 1905 (cited by Panse)	1,000 v.	Atrophic paralysis of right deltoid	1 month
Kurella, 1905 (cited by Panse)	500 v.	Atrophy of left shoulder muscles	3 months
Hoehl, 1906 (cited by Panse)	220 v.	Amyotrophic lateral sclerosis-like picture, starting right hand	15 months
Minot, 1908 (cited by Panse)	1,000 v.	Atrophy of right hand	5 weeks
Horn, 1916 (cited by Panse)	120 v.	Atrophic paralysis, right arm	4 months
Kramer, 1919 (cited by Panse)	120 v.	Diffuse atrophy of left arm	3 months
Jellinek, 1921 (cited by Panse)	5,000 v.	Atrophic paralysis of both arms	2 weeks
Stier, 1926 (cited by Panse)	550 v.	Atrophic paralysis of left foot and lower leg	8 weeks
Mendel, 1927 (cited by Panse)	500 v.	Atrophic paralysis of arm and left leg	8 days
Chartier, 1928 (cited by Panse)	500 v.	Atrophic paralysis of right underarm	4 months
Panse, 1931	200 v.	Amyotrophic lateral sclerosis-like picture, beginning left arm	10 months
Panse, 1931	380 v.	Atrophy, right thumb	3 days
Panse, 1931	500 v.	Atrophy, right shoulder girdle	Some days
Panse, 1931	550 v.	Atrophic paralysis of left leg	2 months
Panse, 1931	220 v.	Paresis of left arm	2 months
Critchley, 1932	Unknown	Right hemiparesis with aphasia	9 months
Alexander, 1936	110 v.	Spinal atrophic paralysis	3 weeks
Langworthy, 1936	Lightning	Left basal ganglion	3 weeks
Haase and Luhan, 1959	Unknown	Basilar thrombosis	3 weeks
Jackson and associates, 1965	75,000 v.	Transverse myelopathy	2 months
Present case	18,000 v.	Spinal atrophic paralysis	24 months

loused palm may have a resistance of up to 2 million ohms. Sweating decreases the resistance 12 times and immersion of the hand in water reduces the resistance 25 times. The duration of contact with the current is also important since continued contact reduces skin resistance significantly.³ As the resistance of the skin drops, current flow increases, in accordance with Ohm's law, in which the current in amperes equals the applied voltage divided by the resistance in ohms. It is easily seen that factors which reduce the resistance of the skin lead to the passage of increased currents and subsequent injury.

The mechanisms by which electrical currents applied to skin surfaces cause injury to nervous structures remote from the site of application are not clear. We assume that if the shock is to be damaging, current must pass through nervous tissue. Weeks and Alexander¹¹ measured the passage of currents in various tissues in animals subjected to a shock applied between the fore and hind limbs. They observed that the current traveled by the shortest route between the contact points and was of equivalent amplitude in blood vessels, spinal cord, and muscle.

The histopathological changes of acute electrical injury in experimental animals have been reported^{4,6,12-14} and consist of perivascular hemorrhage, demyelination with vacuolization, reactive gliosis, and neuronal death. The greater the number of electrical shocks or the longer the electrical contact, the more extensive were the pathological changes seen in the nervous system. Autopsies did not reveal any changes outside of the nervous system other than venous congestion.

The histopathological studies of the acute effects of electric shock on nervous tissue suggest that the current damages by directly heating the involved areas. Thus, the resultant vacuolization, nerve cell loss, perivascular hemorrhages, and demyelination are not unlike those seen following acute pyrexia of any cause. These acute studies do not clarify the mechanisms by which electric shock can result in the delayed appearance of neurological disorder. In man, the interval between the electrical contact and development of symptoms may be days or months (Table 2).

In the patient described in this report, the

slight weakness of the leg, experienced within the week after injury, only began to progress twenty-four months later. Critchley⁹ similarly reported an individual with a prolonged interval of nine months between electrical contact and the development of neurological symptoms.

The existence of a latent period between exposure and neurological disorder also occurs after irradiation of the nervous system: neurological symptoms can appear up to thirty-six months following such exposure. It is known that irradiation induces structural alterations in biological macromolecules such as deoxyribonucleic acid and enzyme systems that may be particularly lethal for cells undergoing division.^{15,16} It is likely that if such structural changes are not sufficient to cause cellular death, their presence may render the cell less able to cope with previously tolerated environmental changes (hypoglycemia, infection, electrolyte changes). Such a mechanism may explain why animals irradiated with nonlethal doses of X-rays suffer senility and premature death. We suggest that the mechanism of delayed effects following electrical injury may be similar. Biologically active proteins exposed to electrical fields undergo conformational change secondary to changes in weak chemical bonds.¹⁷ One of the consequences of passage of electrical currents through nervous tissue may be the production of similar types of structural alterations in biologically active proteins. This interpretation is compatible with the fact that blood vessels are most prominently affected both after irradiation and electrical injury, since, within the central nervous system, endothelial cells are perhaps the elements that most frequently divide.¹⁸ If, as a result of irradiation or electrically induced protein changes, these cells die or become manifestly abnormal following mitosis there would be a potential region for thrombosis and resultant alteration in blood flow. Though there is very little substantial evidence now at hand to support the proposed mechanism of delayed effects of electric current, the phenomenon is of sufficient biological and clinical significance to merit further investigation.

SUMMARY

A case of delayed neurological injury follow-

ing high-tension electric shock is reported. A classification of the various neurological syndromes and examples of these syndromes are reviewed. A mechanism of delayed damage secondary to changes in biologically active macromolecules is proposed. The increasing number of individuals undergoing accidental shocks requires awareness of the consequences and possible mechanisms of such injury.

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