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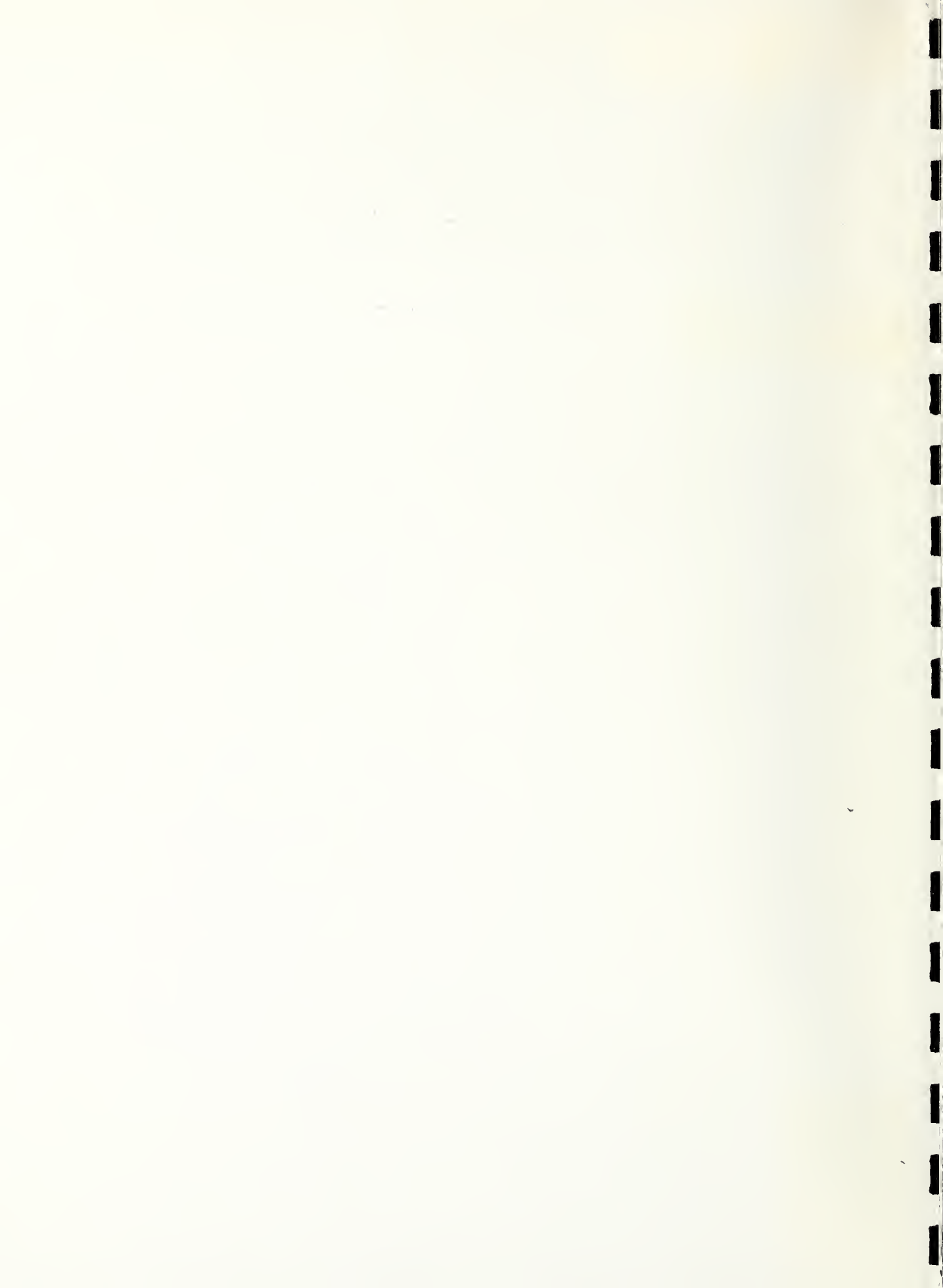
Electrical Injuries - A Literature Review

Yung-Chi Wu

Product Safety Technology Division
Center for Consumer Product Technology

February 1979

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Report to:
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Interagency report NBSIR 79-1710

U.S. DEPARTMENT OF COMMERCE, Juanita M. Kreps, Secretary

Jordan J. Baruch, Assistant Secretary for Science and Technology

NATIONAL BUREAU OF STANDARDS, Ernest Ambler, Director

INTRODUCTION

Ever since electricity was first put into commercial use, electrical injuries have occurred. During the hundred years since the first recorded accident in 1879 (1), both the number and variety of electrical injuries have increased with the advancement of technology and the utilization of electricity. In the first quarter of this century, the number of fatal electrical accidents in the U.S. averaged about 750 annually (1), and currently are only slightly more frequent, around 1110 per year (2,3). Today "there are more than 4000 extension and appliance cord injuries (in 1974 alone) serious enough to require hospital emergency room treatment." Of these, approximately 20% involved burns to the mouths of children (4). Electrical injuries have become a significant concern for consumer safety, hence this study was initiated and sponsored by the Consumer Product Safety Commission.

Humans frequently sustain traumatic injuries from the interaction with electricity. It is generally believed that the effect is due to the magnitude of electric current flowing through the body (1,2,5-8). However, for a given condition, the electric current is a function of electromotive force (E.M.F.), or voltage. Moreover, after an accident, the current cannot be reconstructively determined: only the voltage can be assessed. For this reason, electrical injuries are generally classified in terms of high or low voltage, depending upon the voltage involved in the accident. "Low voltage" has been conventionally defined as a voltage of 1000 volts or less. Unless otherwise indicated, this definition is used throughout this report. With respect to consumer products and their safety, the low voltage injuries are of particular interest and will be discussed here. However, because electrical injuries are not delineated by specific categories, it is important to attempt to assess the relative frequency and severity of low voltage and high voltage injuries.

The basic principles of electricity are applicable to both high and low voltage electrical injuries, whereas the physiological reactions are different and manifest different injurious phenomena. The causation and severity of an injury depend in part upon the physical parameters of the electrical source, and in part on the physiological parameters of the human body as it reacts to the electricity supplied by the source. These parameters will be reviewed and discussed in conjunction with the mechanism of injuries.

Since that first reported electrical accident in 1879, a voluminous body of literature has been produced by both life and physical scientists. The older literature has been thoroughly reviewed and critically analyzed by Jellinek (9) in the first part of this century; by Jaffe (1) in the twenties, and by Alexander (5) in the thirties. Over the past forty years, the increased frequency and alarming effects of injury have led to more studies of electrical injuries and more reports in the literature. Many of these are case studies dealing with clinical treatments, and only a few have provided a different approach to the problem. Among the latter, reports of

Dale (10), Lewis (11), Artz (12), Ugland (13), Skoog (14), DiVincenti (15), Hunt (16) and their coworkers are noteworthy.

The so called "mouth burn" type of electrical injury was not specifically addressed until about forty years ago, after which the number of injuries reported annually has been increasing. This problem was not discussed in the earlier literature and has been reviewed only recently (4,17-23).

In the vast body of literature there are some consensus interpretations of the causation of the injurious phenomena. However, the exact nature of how electricity affects the physiological entity remains unresolved. It is the purpose of this study to review the current state-of-the-art knowledge of the phenomenon, especially the non-fatal (but serious) low voltage electrical burns.

The term "electrical burn" is not clearly defined in the literature. In a narrow sense, the term may be interpreted to mean an injury resembling a burn, that is caused by electricity; in a broader interpretation, the term simply refers to an injury caused by electricity. Thus, Artz (12) observed that "many physicians refer to the injury caused by electricity as electrical burn" and that "It is not uncommon to see an electrical injury of an extremity resemble a burn and be so treated, only to find that clostridial myositis* has developed (deep within the injured part, author's note) in two or three days." Dale (10) noted that "All workers are agreed that the electrical injury, as Jellinek names it, is unique, and that whether it is caused by heat coagulation or by specific electrical effects, its clinical appearance and characteristics are the same." In the literature, it is apparent that the terms "electrical burn" and "electrical injury" are often used in referring to the same injury. In this report, therefore, no attempt is made to define or interdistinguish the terminologies, and the terms "electrical burn" and "electrical injury" are loosely employed in referring to injuries caused by electricity.

*For this and similar terms see Glossary at the end of this report.

STATISTICS OF HIGH AND LOW VOLTAGE ELECTRICAL BURN INJURIES

Statistics on the severity of electrical trauma (which may range from a non-injurious, tingling shock to fatality), the frequency of occurrence of electrical accidents, and their origins appear to be only partially reported in the literature. In 1958, Lewis (11) observed that "In most cases these data (frequency of non-fatal electrical burns) are included in the statistics relative to burns per se, and are not classified under initial causes." Only recently, the National Center for Health Statistics (3) has compiled the annual statistics for hospitalized electrical injuries in which both high and low voltage injuries are lumped together and no initial causes are given. These statistics show the annual rate for electrical burns in the United States is about 4% of the total number of burns (thermal, chemical and electrical inclusive). This figure compares favorably with estimations reported in the literature for different periods and for different nations (3, 14, 15, 24). All these data are summarized in table 1. There is no further detail given in these statistics.

In 1977, Butler (25) reported on a group of electrical injuries, summarized in table 2. This table provides somewhat more detailed information than is evident in table 1; however, the causes of the 2% domestic injuries (low voltage) are not specified. In the interest of consumer safety it is necessary to know the frequency of low voltage electrical injuries related to consumer products. Since this type of statistic does not seem to be directly available, an effort was made to obtain such data through a few randomly selected hospitals. The results, listed in table 3, only confirm the general lack of detailed information. This is probably due to the infrequent incidence of electrical burn injuries in comparison with other hospitalized injuries or diseases (3), so that relative little effort has been exercised in keeping detailed records. However, special effort has been made by the "Project Burn Prevention" in Boston, Massachusetts (26), to collect data from 18 hospitals in the Boston and Springfield areas. A total of 171 electrical injuries from 1974 to 1976 were reported. Half of these were treated in the emergency room and released; the other half required hospitalization. About 41% of the released cases and 53% of the in-patient cases involved indoor, low voltage electrical sources. The National Electronic Injury Surveillance System data show a total of 2644 extension and appliance cord cases treated in emergency rooms in 1977, of which 548 were mouth injuries*. From the data cited above, it is difficult to reach any definite conclusions. However, the limited information appears to indicate that low voltage injuries, especially those that are related to consumer products, are relatively small in number. Among these, the number of mouth burns is relatively high.

*These data are product-oriented and do not provide detailed information on physical causation. The numbers cited here may not all be electrical injuries.

THE NATURE OF ELECTRICAL INJURY

Two fundamental factors directly govern the electrical injury. One is the physical parameters of the electrical source which transmits electricity to the human body when the latter comes into contact with it. The other is the set of biophysical parameters of the physiological entities (such as organs, tissue, nerve fibers, and bio-fluids) which react to the electricity. These parameters are listed as follows:

Electrical Source

1. . Electromotive force, or voltage
2. Direct or alternating current
3. Frequency of alternating current
4. Physical properties of the contact surfaces (entrance and exit)

Physiological Entity

1. Contact surface conditions (wet or dry)
2. Contact area
3. Contact duration
4. Resistivity (more generally, electrical characteristics) of organs, tissue, nerve fibers and fluids (see tables 4 and 5)

It should be noted that the data given in table 4 were measured in vitro; they may be very different in vivo, hence, they can be used only as reference values. Thus, Kouwenhoven (8) showed that the resistance of the skin is not constant, but varies with the amount of moisture content, the temperature and the applied voltage (76). Jaffe (1) suggested that dry skin could have a resistance of 50,000 to 1M Ω (megohm), whereas wet skin may have a resistance of only 2500 Ω . Further, during the passage of current, the resistance can decrease from 260K Ω to 380 Ω . Kouwenhoven measured the skin resistance of cadavers at 60 Hz and gave the following results: at 50 V, 5000-18,000 ohms; 500 V, 800-1800 ohms; and 1000 V, 800-1800 ohms. He noted further that, in vivo, "dry epidermis has a high resistance which may reach 1,000,000 Ω /cm² (ohms per square centimeter)."

Since the epidermis is the surface most likely to contact an electrical source in an electrical accident, the condition and area of contacting skin will affect the distribution and density of current flow, hence the severity of injury. Furthermore, the basic principles of electricity reveal that the current flow, for a given circuit, is a function of applied voltage, and takes the least resistant path. According to table 4, the bio-fluids, nerve fibers and blood have the least resistances among the

physiological entities. It may be inferred, then, that the bio-fluids, nerve fibers and blood stream conduct most of the current which flows through the body. Various current and current density thresholds are shown in table 6.

High frequency alternating current (A.C.) is relatively harmless (1, 32). Thus, D'Arsonval (33) and others (34,35) found that, for a short duration, currents up to 3 amperes at frequencies from 400 kHz to 1 MHz have no effect on the human body. Kennelly (36) observed that "An alternating current of sufficient strength to light up an ordinary 0.5 amp incandescent lamp can be tolerated at high frequency when the human body is in the circuit," and maintained that "the skin effect in the human body is practically negligible and that up to the highest frequency (100 kHz) here considered the current diffuses throughout the cross-sections of the body in substantially the same manner as a continuous current." He further suggested that "The nerve stimulation of one alternation does not have time to register an effect before it is annulled by the next succeeding alternation." Kouwenhoven, et. al. (37) demonstrated that the current required for fibrillation at 1200 Hz is about 12 times that required at 60 Hz. The domestic current most commonly used today is 50-60 Hz, which is in the most dangerous range of frequencies.

In general, 50-60 Hz alternating current is more dangerous to life than direct current (1, 5, 10, 38). Dale (10) reasoned that this is "because the rapid electrical impulses produced by the alternations throw the muscles into tetanic spasm, causing the victim to close a vise-like grip on the electrodes, from which he can be removed only with difficulty. Direct current, while perhaps less lethal, is more liable to cause severe local injuries."

THE PHENOMENA OF ELECTRICAL BURN INJURIES

There are two types of electrical burn injuries. One is caused by flame, flash, or spark of external electrical energy, in situations where the body is not directly involved in the electrical network. This type of injury is a pure thermal burn and will not be examined in this discussion. The other type of injury is caused by contact with, or arcing over of, electricity, in which the body is a part of the electrical network. The phenomena of this type of injury may be listed as follows:

1. Mild shock
2. Involuntary muscle contraction
3. Neuro-system damage
4. Ventricular fibrillation
5. Respiratory paralysis
6. Tissue damage
7. Instantaneous death

The extremes in this listing, mild shock and fatality, are outside the scope of this investigation, which reviews only non-fatal electrical injuries. Specific cases will be discussed after a review of general observations appearing in the literature.

General

As discussed above, electric current flows through the human body when it contacts an electric source and becomes part of the circuit. If the current is above the threshold values listed in table 6, injurious events occur. A person who suffers a tingling shock or mild involuntary muscle contraction is likely to recover almost immediately if the contact duration is short. However, if the contact duration is long or the current is sufficiently high (see table 6), damage to the nervous system and other tissue may result. A current on the order of 100 mA (1, 5, 10-14) flowing from hand to feet will cause heart fibrillation, respiratory paralysis and, usually, unconsciousness. Often, resuscitation efforts must be continued for as long as 12-18 hours in order to save the victim (1, 5). It should be noted that the quantity of current and the duration of time involved in an electrical injury can never be reconstructively determined. At best these can only be described qualitatively as above.

If the current penetrates muscles (12), follows the path of blood vessels leading to the head, and/or follows the nerve fibers leading to the nerve centers controlling vital functions, the resulting injury is usually severe. The higher the current, the more severe the injury. If only the

peripheral blood vessels and nerve fibers are involved, the accident may cause thrombosis and sensory disturbance at the site of, or even some distance from, the original injury (1,5,10-14,24,25). This led Brinn (39) to believe that "there may be progressive central nervous system or peripheral nerve damage leading to paralysis, sensory disturbances or vasomotor phenomena. There may also be vascular disturbances with progressive vessel thrombosis as well as weakness and fragility of blood vessel walls. Tissue repair is often poor." In general, a patient surviving an electrical injury will usually have more tissue damage than might be expected on initial examination (1,5,10-15,24,25).

Arc burns are very destructive (1, 5, 10-15). Jaffe (1) described that "when permanent damage occurs, it is usually due to the mutilating effect of the electric arc which necessitates the amputation of extremities." Dale (10) observed that when the local lesion of electrical injury is caused by arcing, "the effect on the tissue is immediate and profound. . . Small electrical injury presents as a clear circumscribed area of yellowish grey skin surrounded by a sharp line of hyperemia. In the center there may be charring, in which case it is probable that an electrical arc has occurred. The lesion is bloodless, cold and painless. The destruction of tissue is deep and often involves muscles, tendons and bones."

Local injuries caused by electric current from contact or arcing are apparently due to "prolonged vasoconstriction, followed by vasoparalysis leading to formation of parietal and occluding thrombi and to necroses of vessel walls which are followed by immediate, insidious or delayed necrosis of tissue, usually preceded by edema" (5). Thus, "The separation of dead tissues is extremely slow. After the slough has eventually separated, a process which may take several weeks, a slow healing ulcer is left" (10).

Because of vascular alterations, which are initiated by the passage of current through the blood vessel walls, especially the intima (5), it may be assumed that the circulatory disturbance has produced numerous perivascular necroses (1, 5, 10). This may lead to massive gangrene (10) and delayed occurrence of nervous system damage (5,10,11,13).

High Voltage Injuries

According to the statistics cited earlier, high voltage injuries account for 2/3 of the total electrical injuries, or more than 2500 annually. The severity of burns may be classified into three groups, according to Barnes (40):

Severity Group	Body Surface Burn (%)	
	1st-2nd degree	3rd degree
I	< 15%	< 5%
II	15-30%	5-15%
III	> 30%	> 15%

This led Uglund to classify his observations of 64 cases of high voltage** burn as follows:

	Severity Group		
	I	II	III
High voltage burn	40	16	7

The statistics given by DiVincenti, et. al. (15), for a set of 65 cases, show that "The total area of burn ranged from 1% to 87% of the body, with an average of 20%. . . The hospital days for electrical injury averaged 85 days, which was 20 days longer than for non-electrical burns, thus indicating a more severe injury." Skoog (14) summarizes that "thrombi are apt to form within the damaged vessels and ischemic necrosis is common in high voltage burns." The involvement is deep and extensive. Amputation of extremities is sometimes required (11, 13). Cases reported are numerous (5, 10-15, 24-25, 39, 41-56). No further detail will be given here. Additional information may be found in the references cited above.

Low Voltage Injuries

For low voltage injuries (5, 11, 13-14, 38, 55, 56, 58-63) other than mouth burns, Muir (62) has described that "the majority of burns involved the hands. . . They are generally small in size and of relatively minor severity. . . Skin is dead white and surrounded by a bright red margin. . . The limit of injury shortly after electrical burns is clearly defined. . . Later this skin becomes black and mummified for many days and weeks without changes occurring." However, in some cases, "tissue previously thought to be viable sloughs, and tendons, joints, or bones thought to be out of danger are exposed and at hazard." Skoog has summarized that "low tension burns were almost exclusively of the contact type; but burning did not occur where one contact point was large," and asserted that "the small lesion of the so-called current mark heals spontaneously, leaving insignificant scar." (11,13,14,39).

O'Flannagan (63) reported the following case of 240 V, A.C. injury. "The victim was unplugging an electrical drill from an extension cord and apparently came in contact with the live terminals. He didn't fall or lose consciousness. After a 10-15 sec delay his wife disconnected the extension lead. He had no apparent burn marks on his hand. . . He was tender over the upper half of the humerus. Movement of shoulder was severely restricted. . . X-rays showed an incomplete fracture of the surgical neck below the greater tuberosity and crack fracture of the greater tuberosity."

For low voltage direct current injury, Alexander (5) reported an incident involving 115 V, 25 mA⁺ that occurred to a healthy male of 46 who was changing a light bulb in a hanging lamp with a pullchain. He suddenly felt an electric shock in his left hand with which he held the socket. He was able to remove his left hand immediately from the socket and noticed

**High voltage, in this case, is defined by the author as the voltage of the electric source at or above 380V.

⁺This value was given by the author without mention of how it was determined.

that he had received a burn on the end of the fourth finger of his left hand; otherwise everything was normal. However, the next morning he noticed pain and weakness, almost paralysis, of his left arm. Three weeks later the pain and weakness became definitely worse and quivering of his left upper arm was also noticed. Six weeks later, examination revealed muscular atrophy. The circumference of the upper left arm measured 4 cm smaller than that of the right arm. Panse (64) has collected and described a number of similar cases.

Mouth Burns

When a child's mouth or lips come in contact, by chewing or sucking, with exposed live elements of an extension cord, a mouth burn injury usually results. The saliva and the wet tissues of the mouth provide a highly conductive environment, and current flows through the mucosa, taking a path along the peripheral blood vessels and nerve fibers. In most cases, the wound is found to be much deeper than it initially appears. Usually, variable expanses of both lips, laterally, plus the corners of the mouth will be found coagulated or charred. Detailed descriptions of the phenomena have been given by Pitts (17) and others (4,17-23,65), and also reviewed by Oglesby of NBS (66). Specific case histories can be found in those reports.

THE RELATIVE MAGNITUDE AND SEVERITY
OF LOW VOLTAGE ELECTRICAL BURNS

In view of the preceding discussions, it appears that the annual rate of non-fatal, low-voltage injuries requiring hospitalization (mouth burns are included) is about one third of the total of 4000 electrically injured patients (25). This annual rate is about 1.5% of all burns and is about 0.04% of all injuries (3.5 million in 1975) requiring hospitalization (3). This amounts to about one low-voltage injury in every 2500 injured patients. The rank is relatively low. If mouth burns are excluded, the severity of low voltage electrical injuries is relatively minor compared to those caused by high voltage.

ELECTRICAL BURN MECHANISM

Since electrical injury results from human interaction with electricity when the body is involved in an electrical network, both the electrical and physiological parameters listed in the preceding section require examination. As described above, electric current flows through the body, taking the least resistant paths. The resulting physiological reaction to the electricity is the essential cause of injury. The quantity of current through those paths depends upon the EMF of the source, the contact area of the conducting surface, the resistance of each path, and the physiological distribution and structure of the paths. Furthermore, the distribution and structure of physiological entities differ at the micro level among individuals and within an individual body. Therefore, it is very difficult, if not impossible, to quantitatively evaluate the current distribution for any typical path. In addition, individual responses differ and there are no identical electrical injuries (11, 12). The physiological reaction to electricity is current dependent, and may result in only a tingling sensation from low current, but almost instantaneous death from high current flow. For nonfatal electrical injury, the apparent results of physiological reaction to electric current are disturbance or destruction of the natural functions of the entities in the current paths.

Since knowledge of the distribution and structure of physiological entities in the electrical network involved in an electrical accident is limited, there can be no quantitative interpretation and assessment of the injurious phenomena and mechanisms. Qualitatively, however, there are two contemporary theories of interpretation and assessment, viz., the traumatic effect is due to (1) the Joulean heat effect I^2R , and (2) the specific electrical effect.

The theory of heat effect derives from the generation of heat as a current flows through a resistive path, with the amount of heat proportional to the square of the current and to the amount of resistance (I^2R). This theory was initiated by Jaffe and his coworkers (67) in a controlled experiment where current was passed through a section of the femoral artery of a dog, in situ, for a duration of 2 minutes. During the experiment, the flow of blood through the artery could be stopped or allowed to continue normally. When the flow was stopped, damage to the vessel wall occurred; during normal flow, no change in the arterial wall was observed. In another experiment, the same artery was touched with a heated 3 mm steel wire. In the latter experiment, damage to the vessel wall closely resembled that caused by the electric current. They concluded that for the electrical experiment "it was the heat liberated in the obstructed vessel that was detrimental to the tissue of the vascular wall." Hunt and his coworkers (16), in 1976, reported on a series of experiments with animals. Two electrodes were taped to the distal end of one fore and one hind paw of rats. The animals were shocked with constant sources of 125, 250, 500, and 1000 volts, A.C. until arcing occurred, at which time "the amperage falls to zero." The durations of the shock (before arcing occurred) were found to be inversely proportional to the applied voltage, and ranged from 20 seconds at

125 V to 2.5 seconds at 1000 V. Currents from 1 to 8 amperes were recorded. The tissue temperature was measured proximal and distal to the contact sites. Adjacent to the contact sites, tissue temperatures of from 60-95°C were observed during current flow. This was associated with significant damage to deep muscles and other tissues. They concluded that "an electrical burn is simply a thermal injury." Many investigators adopt this theory to interpret the phenomena of electrical injuries (13-15,21,24,25,41,43,44,46,51-53,55-61). Even though some authors (13,14,24,25,45,68,69) have noted clinical distinctions between the electrical burn and thermal burn, they believe that Joulean heat is generated during the passage of current and that this heat destroys cells.

Ugland (13) has reviewed 122 clinical cases and much of the available literature up to 1967. He concludes that "the transformation of electrical energy into heat is therefore maximal in the skin at the contact site and in the adjacent tissues. . . The voltage drop and the resultant release of heat along a pathway of varying resistance is directly proportional to the latter at any one point." He provided a simplified circuit diagram of current pathways through the human body, and suggested that "the most severe local lesions would be expected with current flow through the pathway of greatest resistance." However, as regards peripheral nerves, he distinguished between thermal and pure electrical effects (70-72). He performed a series of elaborate experiments on the sciatic nerve of rabbits, using various electrical shocks from 250 to 1700 mA, at 200 to 500 V, for 5 sec. He observed that "following electrical injury there was a decrease in conduction velocity. . . It seems feasible that derangement of the myelin (70) should be associated with changes in conduction velocity." He concluded that "Electrical current shocks of strength sufficient to cause serious injury in man, produce changes in the function of peripheral nerves in acute test animals. . . These changes are most likely caused by direct neuronal damage induced by the current itself. Heat plays a minor role, if any."

The theory of specific electrical effect was first suggested by Jellinek (73), recapitulated by Alexander (5,70) and followed by some of their contemporaries (71,72) in the twenties and thirties. This theory, in a broad sense, suggests that electric current interacts with a physiological entity to alter its natural function, hence injury results. Dale (10) and Buris (74) observed histological differences between thermal and electrical burns in that (10) elastic fibers remain in the tissue after electrical burns, but are not seen after thermal burns. This led Dale (10) to believe that cells are destroyed not by heat, but by electrical action which causes an imbalance of ionic exchange. In explaining the syndrome of electrical injury, Spitzka (75), as early as 1912, postulated that the process of electrolysis occurs, and Mills (51) has recounted recently that "The phenomenon of electrolysis undoubtedly occurs, and it has been postulated that the syndrome of electric shock may be partially explained by electrolysis and changes in cellular composition." Chasmar (45) has suggested an alteration in chemical balance, and Brinn (39) has described electrical injury to bones and joints (non-mechanical) as due to decalcification. These effects are quite distinct from thermal effects.

The validity of the Joulean heat and specific electrical effect theories may be examined by comparison with the interpretations of injurious phenomena as described in the preceding section. The better theory should be able to explain, at least qualitatively, all types of electrical injuries from minor shock to fatality.

Physical Aspects

When the human body completes an electrical circuit, a current flows from higher to lower potential through the contact areas and the body. The size of the entrance and exit sites on the body affects the current density and depends on the effective contact area and the amount of moisture on the body surface. Variations in these factors lead to different injurious phenomena.

When the contact surface of the human body is wet, its surface resistance is substantially reduced, from approximately 100,000 to 300 ohms. In this case, the flow of current through the contact area is similar to that through an electrolyte solution (76). The solution, in turn, connects to the tissue fluids and the ends of blood vessels and nerve fibers through capillary diffusion. These ends are membranes, with a resistance of about 1,000 Ω (28,77). While the membranes of blood vessels are resistive, the membranes of nerve fibers permit free ion mobility, hence a conductive path is formed. The current will, therefore, have a greater tendency to perfuse through the tissue fluids and flow along the nerve fibers (77), giving rise to high current density in these specific pathways of the network flow.

On the other hand, if the surface is dry, the high resistivity of the epidermal layer of skin will act as a dielectric. Immediately on contact, but before the start of current flow, an electric charge will begin to accumulate at the contact point and the dipoles will line up along the field. The higher the voltage, the higher the charge concentration becomes, until the phenomenon of arcing occurs (78). The arcing may involve an area of pinpoint size, a small circle, or a treelike branching which may spread over an extensive area. The epidermal layer of that area will be destroyed, and the underlying tissue will be exposed. The current will pass through the exposed surface, taking the least resistant path, and flow through the fluids in the perivascular spaces and the vascular channels (79), the nerve fibers and the cerebrospinal fluid (80), and the blood stream (11, 79).

The exit sites (through which the current leaves the body to complete the circuit) also depend on the contact surface condition and the potential difference. The phenomena at these sites are similar to those described for entrance sites.

Jaffe (1) found that electric current spreads radially from the point of entrance and is collected again at the exit point of the body involved. This led Lewis (11) to believe that the greatest density of current is to be found at the sites of entrance and exit.

In the human body, the tissue fluids, the cerebrospinal fluid and the blood stream are electrolyte solutions in equilibrium with different kinds of proteins and biomolecules. The nerve fibers are also filled internally with an electrolyte solution, and are bathed in a similar electrolyte solution separated by a membrane, across which a delicate balance of ionic concentration is maintained (see table 7) (81-82). The membrane permits ion movement under the influence of an action potential (83-86). Thus, the nerve fibers may be considered as electrolyte solutions for conducting current.

According to the theory of heat effect, Joulean heat develops and causes cell damage when an electric current traverses body paths. On the other hand, the theory of specific electrical effect provides no detailed explanation of injury mechanisms, hence requires further examination.

When current passes through electrolyte solutions, the ions carry the charges from higher to lower potential under the applied voltage. If it is direct current, an electrolysis phenomenon results wherein the processes of charging and discharging, ionic association and dissociation, and the forming and reforming of molecules continue until the circuit is broken (87). If the current is alternating, the processes of electrolysis are the same except that they periodically reverse direction because of the applied alternating EMF. At 50 Hz, the direction of current flow and the processes reverse every 1/100 of a second. However, this period of 1/100 sec provides sufficient time for ions to move a long distance in terms of molecular dimension. For example, the mobility of a sodium ion (Na^+) is 5.2×10^{-4} cm/sec (88); thus in 1/100 sec it will traverse 520×10^{-10} m. The thickness of the membrane of a nerve fiber is in the order of 100×10^{-10} m (77) and the crystal radius of Na^+ is 0.95×10^{-10} m (89). It can be seen that the distance the Na^+ ion traverses in 1/100 sec. is about 250 times its own diameter and five times the thickness of a nerve fiber membrane. The other ions commonly contained in physiological fluids are potassium (K^+), calcium (Ca^{++}), and chloride (Cl^-). There are also hydrogen (H^+) and hydroxyl (OH^-) ions from the dissociation of water, in addition to many minute quantities of other inorganic and organic ions. When a positive ion meets a negative ion, a neutral molecule may be formed and this molecule may or may not redissociate when the polarity of the applied EMF reverses. This process of molecular formation and reformation depends upon the chemical affinity and bonding energy of the involved ions, and is beyond the scope of this study. It is not known whether or not the processes which take place in a given half cycle of current flow are reversible during the following half cycle. However, judging from the injurious phenomena some of those processes are irreversible. If this is the case, the constituents of the electrolyte solutions will be altered and their natural functions will be disturbed, changed, or even destroyed.

Physiological Aspects

In the human body, every physiological entity has a specific function. If the entity is changed constitutionally by an external agent, such as

Joule heat or specific electrical effect, its natural function will be disturbed, changed or destroyed, and injury results.

Joule heat is strongly time dependent, whereas electric current flow is essentially instantaneous. In an electrical accident it is commonly observed (10, 11) that a strong muscular contraction may fling the body away from the contact and break the current, or the victim may be unable to release the conductor until the current is broken. Lewis (11) has observed that when electric current traverses the body, strong tetanic contractions of the entire musculature are produced. These could lead to extreme opisthotonos (i.e., extreme arching of the back), with torn muscles, dislocated joints and fractured bones (11, 90). This muscular reaction may take place instantly as a result of a severe disturbance to the nervous system. Joule heat could not induce such a spasmodic effect, but specific electrical action could. Since the nerve system is controlled under a delicate ionic balance (81, 82), the disturbance must be caused by unnatural ion transfer. This could only be induced by current, not by heat.

The ionic unbalance in the nerve system induced by electricity (77, 83-86) could also explain other phenomena of shock, such as ventricular fibrillation and respiratory paralysis, for the latter are also controlled by the nervous system. Urquhart (91) demonstrated that the heart beat was inhibited with the passage of electric current through the vagus nerve centers of animals. When the vagus nerve centers were controlled by the administration of atropine, the heart beat was not affected by passage of current. Obviously, the minute amount of atropine could not diminish the I²R effect, were it to occur, but the chemical might readily alter the ionic activity in the nerve fiber. It therefore appears that ventricular fibrillation is caused by the specific electrical effect, rather than from the heat effect.

In the case of non-arcing contact injury, the theory of specific electrical effect again appears to offer a better explanation. An experiment conducted by Hooker, et. al. (92) serves as evidence. In this experiment, the diagonally opposite fore and hind leg of a dog were shaved and immersed in separate containers of a 1% saline solution. Through electrodes in the solutions, a current of 5 amperes from a 2000 V, 60 Hz source was passed for 5 seconds through the body of the dog. Breathing and heart action both stopped during current flow, but recovered normally soon after the circuit was broken. No tissue or organ damage was immediately apparent, nor was observed for the following six years. This experiment appears to contradict the I²R effect since the current was high and time duration was long. Under these conditions, the Joule heat effect would have been deemed to be sufficient to cause damage comparable with many other low voltage electrical injuries. This experiment also yields further evidence to support the observations of Skoog (14) that for large and wet surface contacts, no burn occurs. Bathtub type electrocution accidents which result in heart fibrillation or respiratory paralysis are comparable to the experiment cited above.

If arcing occurs at the contact site, temperatures on the order of 2000-3000°C could be developed. The arcing phenomenon generally occurs in high voltage accidents. Because of the high current density at the entrance and exit sites, electrical energy transformed into heat is very high and local damages are severe. Delayed effects of tissue damage often occur. The theory of heat effect could explain the local damages but the delayed effect does not conform to the phenomena usually observed with thermal burns (10-12). However, when the epidermal layer is destroyed by intense electrical energy, the dermis and adjacent tissues will be exposed. The current will flow through the tissue fluids, blood stream and nerves. The process of electrolysis could take place; electrolytes could shift and the natural functions of the entities involved in the process could be disturbed. Hence, the delayed effect of tissue damage could result. The theory of specific electrical effect seems to provide a better explanation of observed phenomena.

For low voltage injury and mouth burn, commonly encountered with 110-220 V, 60 Hz A.C., current marks (9) generally appear at the site of contact. The lesion is bloodless, cold and painless (10), and delayed tissue necrosis commonly occurs (1, 5, 10, 11). For mouth burns, the injury is usually localized, for the current enters and exits in the same general vicinity. In the general assessment, the current involved is small (5, 10, 11, 17), on the order of 100 ma (5). At low current levels the heat developed should be very small.

In view of the foregoing arguments, the theory of specific electrical effect appears to better explain the mechanism of low voltage electrical injuries than does the theory of Joulean heat. Both theories are qualitative. A quantitative description of the mechanism requires further experimental determinations of the functional relationship of the physiological interaction with electricity.

CONCLUSIONS AND RECOMMENDATIONS

The non-fatal electrical injury is a complex phenomenon, both clinically and physiologically. It may range from a minor lesion, resulting in an insignificant scar, to severe damage requiring amputation. Clinically these injuries may be treated in emergency rooms, by plastic surgery or in special burn units, hence hospital records for electrical injuries may be scattered over several departments. It is a difficult, if not impossible, task to obtain detailed national statistics for electrical injury. The statistical data presented in the preceding sections are cited for reference only, and are not necessarily to be considered representative. The discussion of the mechanism of electrical injury is only a qualitative assessment of the current state-of-the-art, and it leaves many open questions. The major problem is that our knowledge of the structure, distribution and properties of the physiological entities in the current pathway is limited. The electrical characteristics of their biochemical constituents are far from quantitatively determined, hence neither the I²R effect nor the specific electrical effect can be quantitatively assessed at this time.

In order to understand this type of trauma more fully, a systematic experimental examination of both the physical and physiological parameters discussed in the preceding sections is necessary. This will require a carefully planned, long-range program involving cooperative investigations by physicians, physiologists, physical scientists and other experts using sophisticated, state-of-the-art techniques, equipment, and facilities. Specific recommendations on the approach and nature of these investigations is beyond the scope of this report and should be developed by a multidisciplinary team of experts familiar with the many aspects of the problem.

TABLE 1. Annual Rate of Non-Fatal Electrical Injuries

State	Period	Total # of Burns	Total # of E.I.	Annual Rate of E.I. Burns	Source
U.S.A.	1974-1976	100,000/yr	4,000/yr	4%	National Center for Health Stat.
U.S. Army Hospital, Texas	1951-1967	2,477	65	3%	DiVincenti (14)
Birmingham, England	1951-1955	1,893	70	4%	Davis (24)
Sweden	1955-1970	2,000	141	7%	Skoog (13)

TABLE 2. Electrical Injuries (25)

182 cases in 20 years

3 female
 5 deaths
 95% utility worker
 3% industrial
 2% domestic

of which

67% high tension
 33% low tension

TABLE 3. Burn Cases Treated in Selected Hospitals

Hospital/years	Total Burns All Types	Total Electric Burns (all)	High Voltage	Low Voltage	Mouth Burn
U. of Virginia Hospital Charlottesville, VA 73-6/78	461	30	17	-	13
San Francisco General Hospital, San Francisco, CA 70-77	1,182	28	25	-	3
Shriners Children Hospital, Cincinnati OH 70-6/78	784	56	18	-	38
Massachusetts General Hospital, Boston, MA 74-76	-	11	8	3	-
Shriners Burn Institute Boston, MA 74-76	-	20	-	12	8
King's County Hospital Center, Brooklyn, NY 70-78	1,690	96	-	-	-

TABLE 4. Resistivities of the Human Body (27)

Body Segment	freq.(Hz)	temp.(°C)	Resistivity(ohm-cm)	Remarks
Blood	D.C.	40	150	Normal subjects
	20-5k	40	155	
	1k	37	165 avg.	
Body fluid	1k-30k	24.5	64.6	
Physiological solution 1% saline	-	38	50	
Cardiac muscle	1M	room	132	2-3 hours after death
Skeletal muscle	D.C.	37	245	longitudinal
	100-1k	37	240	longitudinal
	100-1k	37	675	transverse
Lung	200M-900M	27	161 avg.	autopsy
Kidney	1M	room	126	2-3 hours after death
Liver	1M	room	298	2-3 hours after death
Spleen	1M	room	256	2-3 hours after death
Fat	1M	room-body	2,180	freshly excised
Bone	low	-	16,000	freshly excised
	1M	room-body	1,800	
Skin	1M	room-body	289	freshly excised
Arm	D.C. pulses	37 (body)	160	corrected for bone and fat
Forearm	D.C.	37	330	geometrical mean
Finger & hand	D.C.	37	280	
Finger	D.C.	37	235	
Neck	D.C.	37	280	
Trunk	D.C.	37	415	along axis of body
Head	D.C.	37	840	trans-temporal
Head (scalp)	D.C.	37	230	
Thorax	D.C.	37	463	

TABLE 5. Resistance and Resistivity of the Human Body

Segment	Resistance (ohms)	Resistivity (ohm-cm)	Reference*
Total body (criminal electrocution)	218		Kennelly
Hand to hand	550-1,970 610-1,870		Bullard Jelliffe
Bone	900,000		Jellinek
Brain	2,000		Jellinek
Liver	900		Jellinek
Muscle	1,500		Jellinek
Skin (dry) (per cm ²)	100,000 50,000 40,000		Brandon Galinary & Kratter Chapuis
(per 100 cm ²)	500		Jaffe
Skin (wet) (in water)	2,500-3,000 1,200-1,500		Cardieu Jaffe
Skin (current passing for 1 min)	380		Jolly & Gaertner
Cell protoplasm (mammalian)		300	Cole (28)
Surface membrane (most probable range)		1,000(ohm/cm ²)	Cole (28)
Medium for mammalian cells		100	Cole (28)

*References for resistances are given in reference 1.

TABLE 6. Electric Current (60 Hz) and Current Density Thresholds

Event	Current (mA)	Current Density (mA/cm ²)	External Field Strength (60 Hz)	Reference
Sensation	1	0.1-1	1 MV/cm ²	(29)
Let go	10	0.1-1	1 MV/cm ²	
Fibrillation	100	1	10 MV/cm ²	
Air breakdown			30 kV/cm	
Perception	1			(6)
Pain	5			
Tetanic muscle contraction	15			
Respiratory muscle tetanically contracted	30			
Cardiac fibrillation	60-5,000			
Respiratory failure,	10,000			
Perception	0.1-0.5			(31)
Let go	5-9			
Fibrillation	>10			

TABLE 7. Ionic Equilibria in Human Body Fluids

A. Human Plasma (81)

Cation	mEq/l*	Anion	mEq/l*
Na	138	Cl	105.0
K	4.5	HCO ₃	25
Ca	5.2	Pr ³	16
Mg	2.0	PO ₄	2.2
		SO ₄	0.5
		Other anions	1.0

B. Human Erythrocytes (81)

Na	16	Cl	55
K	96	HCO ₃	15
Ca	0.5	Other anions	
Mg	4.6	Other anions	47

C. Nerve Cell (82)

Ion	Outside (mEq/l)	Inside (mEq/l)
Na	150	15
K	5.5	150
Cl	125	9

D. Muscle of Dog (1 kg of fat free muscle)(81)

Na	146	12
K	3.8	137.4
Ca	3.2	3.2
Mg	1.3	38.5
Cl	124.4	-
P	2.6	117.3

E. Sweat Constituents (81)

Cl ion, NH₄ ion, urea, uric acid

*mEq/l means one thousandth of the molar solution which can react or replace the corresponding amount of hydrogen ion in 1/1000 of 1 normal solution.

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GLOSSARY

Action potential - when an excitation impulse propagates through the nerve system, there accompanies this action a potential difference which is called action potential.

Atropine - a poisonous, white, crystalline alkaloid, used to increase heart rate by blocking the vagus nerve.

Atrophy - arrested development of a part or organ of an animal.

Biofluid - a fluid contained in living matter.

Bond energy - the energy required to break a chemical bond which holds atoms together to form a molecule.

Cardiac muscle - heart muscle.

Cerebrospinal fluid - the fluid in the brain and spinal cord system.

Chemical affinity - the tendency for a chemical reaction to occur.

Clostridial myositis - an inflammation of a voluntary muscle caused by some kinds of bacteria.

Conduction velocity - the velocity of the conduction of excitation along a nerve fiber.

Decalcification - the action of the removal of calcium ion from a calcium containing chemical substance.

Dermis - the layer of skin beneath the epidermis.

Edema - an abnormal accumulation of fluid in the intercellular tissue spaces of the body.

Epidermis - the outermost layer of the skin.

Erythrocytes - red blood corpuscles.

Femoral artery - the artery in the thigh.

Histology - tissue study.

Humerus - the bone that extends from the shoulder to the elbow.

Hyperemia - an excess of blood in a part.

in situ - in its natural position.

Intima - innermost.

in vitro - within a glass; observable in a test tube.

in vivo - within the living body.

Ionic association - the formation of an ionic aggregate from free ions in a solution of electrolyte.

Ionic dissociation - molecules of an electrolyte in a solution dissociate into free ions.

Ischemic - pertaining to the deficiency of blood in a part.

Mucosa - a mucous membrane.

Myelin - a fatlike substance forming a sheath around certain nerve fibers.

Opisthotonos - a form of tetanic spasm.

Parietal thrombi - the formation of clot in walls of cavity.

Peripheral nerve - external nerve; away from the central nervous system.

Perivascular - situated around a vessel.

Redissociate - to dissociate into free ions from a previously formed ionic aggregate or neutral molecule in an electrolyte solution.

Respiratory paralysis - the loss or impairment of the function of breathing.

Sciatic nerve - the nerve in the region of the hip.

Tetanic - tending to produce a state of a muscle when undergoing continued contraction.

Thrombosis - the formation of clot.

Tuberosity - an obtuse prominence on bones for the attachment of ligaments.

Vagus nerve - the tenth cranial nerve.

Vascular - pertaining to vessels.

Vasoconstriction - the diminution of the vessels leading to decreased blood flow.

Vasomotor - affecting the caliber of a blood vessel.

Vasoparalysis - partial paralysis of vasomotor nerves.

Ventricular fibrillation - a condition characterized by fibrillary twitching of the ventricular muscles; the impulses traversing the ventricles so rapidly that coordinated contraction cannot occur.

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6. ABSTRACT (A 200-word or less factual summary of most significant information. If document includes a significant bibliography or literature survey, mention it here.) This report reviews and discusses the frequency, severity and mechanisms of the electrical injury as reported in the literature. Detailed national statistics on the frequency and severity of specific types of electrical injury are not available, however, limited data from the literature and from a survey of a few randomly selected hospitals are summarized and presented. The mechanisms of injury involve the physical and physiological parameters of the electrical source and the human body. These parameters are discussed according to known principles. Injury to the human body, initiated by the flow of electrical current, is the consequence of disturbance, change or destruction of physiological entities such as nerves, blood vessels, organs, muscles and bones. For interpreting the electrical injury phenomena two theories exist in the literature. One theory suggests that electrical injury is thermal in nature and is caused by the Joulean heat (I^2R) resulting from current flow through the body. The other theory suggests that injury is caused by specific electrical effects such as electrolyte shifts that are induced by the current flow. Evidence to support or dispute both theories was found in the literature. Due to the lack of quantitative data, however, the validity of these theories can only be assessed qualitatively.				
17. KEY WORDS (six to twelve entries; alphabetical order; capitalize only the first letter of the first key word unless a proper name; separated by semicolons) Arc burn; contact burn; current threshold; electrical injury; electrolyte shift; heat effect; injury mechanism; mouth burn; specific electrical effect; injury statistics.				
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Electrical injuries - a literature review

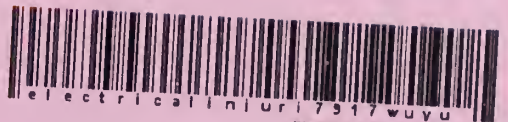
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