THE ELECTRICAL INJURY ENIGMA

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ABSTRACT

A chronic electrical injury model employing documentary and diagnostic techniques was designed in the primate to enable investigation of wound evolution for up to 10 days post burn. A standardized 40 kJ, 3500 V, 4.2 A, 2.5 sec bilateral, symmetric upper extremity electrical burn was done. Gross observation studies documented tissue injury extending more proximally on the deep surfaces of individual muscles and between muscle layers. Specific regions or "choke" points in the forearm exist in which decreased cross-sectional areas and highly resistant tissue composition resulted in increased heat production and more severe tissue injury. Muscle injury revealed characteristic patterns of injury and showed deep tissue damage extending proximally beneath viable muscle. Digital subtraction angiography demonstrated segmental narrowing and "pruning" of large vascular trunks with a significant decrease in nutrient vessels in affected areas. Ulnar nerve conduction studies showed loss of conduction proximal to the cubital fossa with no recovery. Although characteristic patterns of injury were documented in skin, muscle, vessels and nerves, no experimental evidence was found for progressive necrosis. These findings were compared with electrical injuries seen clinically and showed a high degree of similarity. A management protocol for the treatment of electrical injuries is discussed based on experimental findings and clinical correlates.

SOMMAIRE

Un modèle de brûlure chronique causée par électrocution fut élaboré en utilisant des primates afin de permettre l'étude de l'évolution de la plaie pour une période allant jusqu'à dix jours après la brûlure. On utilisa une electrocution quantifiée de 40 kJoules, 3500 Volts, 4.2 Ampères pendant 2.5 secondes. La tension fut appliquée bilatéralement aux extrémités des membres supérieurs. L'étude expérimentale comporte cinq points: 1) observation visuelle grossière, 2) microscopie, 3) angiographi , 4) études de distribution des temperatures et 5) analyse de la conduction des nerfs. Les différents modèles de blessures sont décrits pour la peau, le muscle, les vaisseaux sanguins et les nerfs: ceux-ci sont analysés de façon chronique. Aucune étude expérimentale permet de soutenir la thèse de la nécrose progressive Les études expérimentales furent appuyées par trois cas cliniques de brûlures causées par électrocution. Les similitudes entre les primates et humains sont abordées. Les méthodes cliniques décrivant les soins à apporter aux brûlures par électrocution des membres supérieurs sont soulignées.

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INTRODUCTION

Few injuries are more traumatic and incapacitating than severe electrical burns. Contact with high voltage is characterized by explosive entry and exit lesions and massive thermal damage to tissue. Clinical case reports and accident commission statistics confirm that 40% of high voltage burn victims suffer amputation of an extremity, prolonged hospitalization, and permanent disability. In the home, low voltage burns to the mouth of young children may result in severe scar contractures and tragic oral deformities.

Despite continual introduction of new techniques in burn care, the fundamental concepts of managing electrical burns have not changed significantly in the past fifty years. Periodic evaluation, sequential debridement, and delayed reconstruction remain the standard clinical approach Although the concept of immediate resection and skin grafting was recommended as early as 1929 by Wells[58], the difficulties inherent in differentiating damaged tissue from normal tissue, and the significant risks of "progressive necrosis" and infection have prevented widespread implementation of early reconstruction

Our understanding of electrical injuries is hindered by many obstacles: a) a lack of experimental investigations on high voltage electrical injury, b) the virtual absence of a clinically applicable animal model, and c) no studies which assess chronic wound evolution.

With the aim of solving these challenging problems, a fully isolated, high voltage testing facility has been installed in the Microsurgery Laboratories at the Royal Victoria Hospital. Using this highly specialized equipment, the goal of this work was to answer the following questions:

1. Can a clinically applicable animal model be developed to study high voltage electrical injuries and will this model permit chronic evaluation?

2. Are there characteristic patterns of tissue injury following high voltage electric injury?

3. Does the phenomenon of "progressive necrosis" exist?

4. Can the experimental findings, if any, be correlated with high voltage injuries seen clinically?

5. Is the current protocol for managing high voltage electrical injuries valid, or should it be reappraised?

LITERATURE REVIEW

The earliest reference to electricity was etched in a crypt of the 5th dynasty Egyptian architect Ti in the form of the Nile catfish, capable of generating nasty discharges to unwitting fishermen. In 1929, fifty years after the first reported fatality due to electricity, two reports were published which characterize traditional attitudes towards electrical injury. In a paper on electrical shock, MacMahon forgoes a review of the literature, describing it as "already voluminous".[34] In the same year, Wells recommends complete resection and immediate grafting of third degree electrical burns, but in application of the technique advised "discretion (as) the better part of valour".[58]

These commentaries are representative of a literature replete with clinical case reports and treatment regimes that are, ironically, not based on an understanding of the pathophysiology of electrical injury Experimental studies on the effects of electricity on living material have been intermittent and largely unfocussed. There are several reasons for this apparent paradox.

To early investigators, the properties of electricity acting in vivo were unknown. Jex-Blake reports that accurate instrumentation for the measurement of voltage, current, and resistance was largely unavailable until 1880.[27] Experimental designs consequently produced highly speculative conclusions of limited value to the clinician.
In the twenties, electrical burn research was encouraged by the

utility companies who were concerned by the mounting dangers of electricity to the public. These studies concentrated on the mechanism of death and the problems of resuscitation. More importantly, however, they shifted emphasis from qualitative discussions of esoteric electrical phenomena to quantitative studies of electrical parameters in vivo. Experimental studies from the 1930's to the present have focussed increasingly on the local lesion in an attempt to elucidate the specific physical and physiological effects of electricity on man. Unfortunately, very few investigators have examined the physical factors that account for heat generation in tissue and the critical temperatures that cause irreversible tissue damage.

Jaffee demonstrated that anatomic damage to arteries subjected to current passage was produced by heat and not by specific electrical effects.[24] The arteries of dogs were exposed to low tension alternating currents administered through saline- soaked gauze electrodes. Structural damage to the arteries was produced only when the vessels were clamped and circulation interrupted. It was concluded that the endothelial damage observed was caused by heat dissipation in the clamped artery. Under similar conditions, dry heat, transmitted through an aluminum steel wire, resulted in structural alterations markedly similar to those observed when current was the damaging agent. It was concluded that arterial damage was due not to specific electrical effects, but to heat

By the 1940's, studies of electrical injury had been reported by innumerable investigators. Exhaustive literature reviews were prepared successively by Jellinek (1905)[26], Jex-Blake (1913)[27], Jaffee (1928)[25], Pearl (1933)[38], and Alexander (1938)[2].

Weeks and Alexander studied the distribution of 60 cycle alternating current through blood vessels, muscle and nerve in the cat by insertion of a concentric electrode measuring circuit.[57] Current distribution along four separate pathways from point of entry to point of exit was registered by clamping the transformer around structures in the animal body - the sciatic nerve, certain blood vessels, small bones, and various muscles. Their recordings indicated that electric current passes along a pathway through an animal body as though it were passing through a structureless gel. The anticipated deflection of current along selective pathways - suggested by the variable resistances of different tissue - was not observed

Henriques and Moritz, following WW II, studied the thermal tolerances of living tissue and the nature of cellular changes induced by hyperthermia.[17] In a series of experiments on swine, they defined quantitatively the effects of intensity, duration, and the rate process inherent in irreversible changes to epidermis injured by heat. For each degree rise in surface temperature between 44°C and 51°C, the time required to produce irreversible damage to epidermal cells was reduced by approximately half

Studies on electrical injury that have followed these pioneer surveys have been, in the majority, clinical case reports. These

papers have attempted to report conclusively on the incidence of electrical injury, accident reconstruction, wound management, and associated complications. The case studies of Dale (1954)[10] and Lewis (1958)[32] are comprehensive reviews of physical principles and pathology. More recently, reports by DeVicenti (1969)[12], Baxter (1970)[4], Skoog (1970)[50], Artz (1974)[3], and Luce (1978)[33] are notable for the lucidity they bring to the predominant controversy surrounding appropriate wound management.

The first experimental investigation of electrical burn pathophysiology in almost thirty years was conducted by Hunt in 1976[21]. He reported on critical values for heat generation in tissue and on the interdependency of physical variables. Electrodes (3/8" diameter, 1/8" thick) were affixed to the distal end of one fore and one hind limb of the rat. The animals were shocked with a constant source of 250 volts AC until arcing occurred. Amperage, muscle and bone temperature, and time were recorded in selected experiments. Tissue temperature, registered through thermocouples implanted proximal and distal to the contact sites, always exceeded 60°C; this value was associated with significant deep muscle damage. Currents from 1 to 8 amperes were recorded. Hunt made the following conclusions: 1) tissue temperature is the critical factor in determining the magnitude of tissue injury, 2) the volume of tissue traversed by the electric current was more closely related to the extent of tissue injury than the internal resistance of the individual tissues.

Experimental research on electrical injury in general has not been conducted in response to clinical problems and needs. Investigations that have originated from science and industry have often reflected mutually exclusive concerns and objections. In contrast to this tendency a 1979 overview by Sances integrated the findings of electrical engineering studies related to consumer product safety with the clinical experience of the medical community.[46]

In an elaborate and sophisticated 1981 study, Sances demonstrated different current density values for tissues of different resistivity utilizing voltages up to 14,000 V.[48] Arteries and nerves passed the largest current densities followed by muscle, fat, bone marrow, and bone cortex. Current densities were approximately inversely proportional to the resistivities of the respective tissues. Although artery and nerve transport the largest current densities, the greatest percentage of current was passed by muscle because of its large cross-sectional area. Although variable resistivities give rise to differential current flow and heat generation, for practical purposes, in high voltage injuries, this effect is negligible and not relevant.

Recently, Buchanan et al applied 300 volts AC for 10 seconds through wire mesh electrodes attached to the opposite fore- and hindlimbs of rats.[6] In arteriograms followed over a 90 day period, neither retrograde thrombosis nor progressive necrosis occurred. Subsequently, Robson et al utilized the same model to biochemically assess wound progression.[42] They concluded that thromboxane excess occurred due to a shunt in arachidonic acid metabolism which led to

progressive muscle damage. They concluded that blockage of thromboxane production by pharmacological treatment led to significant tissue salvage as evidenced by a doubling of the average limb survival length. In both chronic experiments the physical parameters of energy delivered (voltage, current, energy), its passage through the affected tissue (current density), and its conversion to thermal energy (temperature) were not measured. Two investigators using the same small animal model came to opposite conclusions as to whether progressive necrosis occurred.

Previous contributions to electrical burn management have been characterized by an a posteriori approach to electrical injury mechanisms. Multi-disciplinary studies, which first provide a clear interpretation of the various physical factors operative in current passage through tissue, are requisite to any definition of pathophysiology, and consequently, to any significant advances in burn care.

From this review of the literature the following important points are evident: 1) there are very few experimental studies, especially those employing high voltages in large animal models Physical laws governing high voltage passage through a small animal (conductor) dictate dissimilar patterns for current distribution than can be generated in a primate model characterized by an upper limb of conical configuration closely resembling human anatomy, 2) there are virtually no reports of sophisticated physical measurements in experimental models which were allowed to progress to chronic wounds, 3) the

patterns of injury to the cutaneous, muscular and neurovascular structures have not been adequately addressed, and 4) the question of progressive necrosis remains unanswered. To ensure that the pathophysiology of electrical injury is fully elaborated, it is imperative that comprehensive wound evolution models be developed in a controlled study to separate the facts of electrical burn etiology from the fiction of 'progressive necrosis'. Such an aura of mystery surrounds this phenomenon of progressive necrosis, that the term and its significance are now regularly employed by practically all, and understood by virtually none.

THE NATURE OF ELECTRICAL INJURY

It might be useful at the outset to explain the meaning of basic electrical quantities, voltage, current, and resistance, by analogy to a closed water circuit.

Without venturing into quantum mechanics, it is simplest to say that all matter seeks to achieve an electrically neutral state, the positive charge of the atomic nucleus, balanced by the negative charge of the electron. A body overcharged or undercharged with electrons is said to be at an excited potential relative to a neutral body. This difference in potential is responsible for all electric phenomena and is called the ELECTROMOTIVE FORCE. To provide the hydraulic analogy, the pressure or voltage which drives electrical current through a current corresponds to the pressure required to overcome inertia or to pump water uphill. The pump is required to overcome the resistance of the circuit.

Electric current represents the rate, or amount of charge per second which moves past a given point in the circuit It is measured in AMPERES (1 ampere = 1 coulomb/second) and corresponds to the stream of water flowing at so many gallons per second. The resistance met by a current in flowing through the circuit is measured in OHMS The hydraulic equivalent could be the negligible frictional resistance of the pipe through which the water flows or a significant resistance represented by a constriction of the pipe.

The relationship between electric current, voltage, and resistance is given by Ohm's Law: $I = V/R$. This is identical in form to the liquid volume flow rate relationship. The electric current in amperes can be increased by either increasing the voltage (pressure) or decreasing the resistance (by using a larger pipe).

The severity of electrical lesions is related to several complex and variable factors. The principal pathological changes observed in electrical injury are believed to be produced by heat generated from the passage of current through a conductor. Joule's Law states that the amount of heat produced is directly proportional to the square of current strength, the resistance of the structures transversed, and the duration of current flow ($J = I^2RT$). Commonly in accident reconstruction, only the voltage is known. The amperage values, which correlate with tissue damage, are usually indeterminate since the resistance is unknown The initial resistance offered to current is a function of the condition and surface area of contacting skin. Factors such as moisture content, cleanliness, thickness and vascularity will affect the distribution and density of current flow and hence, the severity of injury The resistance offered by dry skin may be as high as 1,000,000 ohms; that of moist skin as low as 300 ohms

The specific resistance which internal body tissues offer to current flow is also variable. Relative tissue resistance in ascending order includes. nerve, blood vessels, muscle, skin, tendon, fat, and bone. Highly resistant conductors transfer electrical energy

to thermal energy efficiently. Therefore tissues of poor conduction and high resistance - bone, skin, and muscle - will generate more heat than will tissues of higher conductivity, nerve and blood vessels. Natural conductors such as blood vessels and nerve offer the least resistance to current flow and yet may sustain irreversible injury, often far from the area of gross local injury. Bone, as the poorest conductor, has the greatest capacity for heat production. These two factors - conductive properties and capacity for heat production - may be operative to varying degrees according to the voltage.

INCIDENCE

Reliable statistics on the incidence of electrical injury are difficult to obtain and are plagued with problems of interpretation. Commonly, electrical burn cases are inconsistently listed in hospital patient records as "burn victims" without distinguishing electrical from thermal injury. When electrical burns are specifically identified in the records, low voltage domestic injuries are often grouped with high tension industrial accidents. It is important to remember that the range of severity of injury in electrical trauma is considerable. Accident statistics which fail to differentiate between high and low voltage contact tend to perpetuate controversy surrounding the etiology, evolution and management of electrical lesions.

Sances reports the number of deaths from electrical injury in the United States at approximately 1500 per year.[47] DeVicenti estimates that electrical trauma constitutes 3 per cent of admissions to major burn centers.[12]

Clinical case reports, electric utility prevention briefs, and accident commission statistics confirm that high tension electrical linemen sustain injury more frequently than persons in other occupations, comprising 50% of reported fatalities. A Canada Manpower occupational analysis of power linemen indicates that 75% of this work force handle high voltage conductors in repair, maintenance or construction work at least once a day. The Workman's Compensation Commission of the Province of Quebec (pop. 6 million) reports 472

electrical contact accidents from 1977 to 1981 inclusive. Over this period, 17,731 work days were lost; compensation payments to disabled victims exceeded five million dollars.

In the home, low voltage burns to the mouths of young children may result in severe scar contractures and tragic oral deformities. The U.S. Consumer Product Safety Commission estimates 4000 extension and appliance cord injuries each year which are serious enough to require hospital emergency room treatment. In addition, injury to children from high tension power sources is depressingly frequent. Young boys between the ages of 9 and 16 may contact high tension lines while climbing trees or utility poles. The result is often amputations and permanent disability.

Accurate epidemiological studies are required to identify the major at-risk groups and to provide for comprehensive preventive education programs.

PATHOPHYSIOLOGY

In a 1913 lectures series on the "spectacular science" of electricity, Jex-Blake summarized research on the pathophysiology of electrical injury: "the old principle of explaining obscurium per obscurius dies very hard".[27] With few exceptions, contemporary studies on the fundamental mechanisms of current passage through tissue have confirmed Jex-Blake's dictum.

Since the 1920's the mechanism of injury in electrical contact has been the subject of controversy. The theory of specific electrical effect, first proposed by Jellinek[26], suggests physiological alterations at the cellular level due to the effects of electric current acting in an electrolyte medium, thereby potentially creating irreversible ionic changes. It is more commonly believed that the etiological agent in electrical injury is, at least, primarily, a thermal effect. According to Joule's Law heat is generated when current flows through a resistive path, the amount of heat proportional to the square of the current and the combined resistances along the path (I^2R). The nature of this transformation from electrical energy to thermal energy and its consequent effect on tissue damage requires a discussion of physical and physiological parameters.

From the instant of electrical contact through to the final demarcation of the electrical lesion, physical and physiological processes are in operation which to date are undefined. No adequate

mechanisms have been proposed to fully account for how the specific properties of tissue affect current flow; how current intensity generates heat in tissue; and how the subsequent rise in temperature results in tissue necrosis.

In contact with an energized conductor, skin serves as an insulator, impeding the flow of current. The barrier represented by skin resistance is dependent upon several variables: the physical state of the skin - dry, wet, sweaty or traumatized; the contact surface of the conductor - area, shape and composition; magnitude and frequency of the voltage applied; and the direction of the passage of current. The amount of current generated within the biological conductor is therefore dependent on alterations in skin resistance. Upon application of 250 V AC through electrodes attached to the fore and hind limb of the rat, Hunt noted an initial slow rise in amperage and correlated this with a progressive decrease in skin resistance. In the second phase, current values rose sharply, representing complete skin resistance breakdown and free current flow through internal tissues. In the final stage, current values reached a peak and abruptly fell to zero as the current arced, carbonizing tissue over the electrode-skin interface and breaking contact
Despite the several variables which may alter pre-trial values for resistance in experimental models, the critical point Hunt makes is that the resistance of skin is changing as current flows.[21] Unlike pure ohmic conductors (metals), where resistance is independent of amperage, the values for skin resistance are shifting, transforming it

alternately from insulator to conductor and back again. The capacity of the skin as an insulator affects the distribution and density of current flow, and hence, the severity of the injury.

Upon penetration of the skin, the resistance which internal body tissues offer to current flow varies. In order of increasing magnitude, different resistance is offered by nerve, blood, muscle, skin, tendon, and bone. There is some debate concerning the importance of different internal tissue resistances in the distribution of current, the generation of heat, and the ultimate severity of tissue damage.

Weeks and Alexander applied 40 ma AC directly to various tissues in the cat through a clamp transformer.[57] They found that electric current did not follow the path of least resistance, but passed through the body as though it were passing through a structureless gel, always taking the shortest path from contact to contact without deflection by anatomical landmarks.

In a model more closely approximating accident conditions, Sances applied voltage externally across the hind limbs of hogs and measured current density with sensitive probes inserted into muscle, fat, and bone.[48] In applications up to 415 V Sances observed that current densities were approximately inversely proportional to the resistivities of internal tissues. Relative tissue resistivity however, is not the most critical factor in tissue temperature rise. Sances notes that bone passes little current due to its high resistivity, and blood vessels and nerves carry a relatively small

percentage of total current owing to their minimal cross-sectional diameter. Sances' work has served to expand the current distribution argument. As relatively good conductors, nerves and blood vessels convey more current per unit area, although the greatest percentage of current will be carried through the large cross-sectional diameter represented by muscle, fat, skin, and tendon. Those who hold that the internal milieu of the animal body acts as a single uniform resistance during current flow have been disproved at least, theoretically from a practical standpoint, this paradigm may be inoperative at higher voltage levels.

In both low and high voltage contact the amount of heat generated in tissue is a function of the voltage drop and the current flow per unit of cross-sectional diameter. While these two principles - specific resistance and current density - govern the ultimate extent of tissue damage, their role and relative importance may be determined by magnitude of the applied voltage.

Sances measured current density in various tissues, applying up to 415 V to hogs, and observed preferential current pathways along the lines of least tissue resistance.[48]

Laberge et al on histological examination of swine tissue immediately post-shock (500 V) observed endothelial damage distant from the burn lesion in areas of normal surrounding tissue [30]
It appears that for low voltage contact, current dissipates along the path of least resistance. In these cases, the intensity of the current, the specific conductivity of the tissues involved and the

cross-sectional diameter of the conductor will be critical factors. The path of the current accounts for selective damage to nerves and the vascular network. The concept of cross-sectional diameter accounts for the frequency of severe injury to the extremeties and the rarity of major injury to the trunk, even in high tension contact.

Henriques and Moritz demonstrated an inverse relationship between duration of contact and temperature.[17] Thermal injury to skin was produced with constant surface temperatures of 51°C for approximately 2 minutes, 55°C for 20 sec, and 60°C for 3 seconds. Hunt recorded tissue temperatures proximal and distal to contact sites in the rat. He found that tissue temperature adjacent to the contact always exceeded 60°C (range 60-95°C) and was associated with deep muscle damage in the rat model.[21]

Sances inserted 0.4mm glass bead thermistors into swine for temperature measurements of peroneus tertius muscle, subdermal fat, and marrow of the tibia.[48] He suggests that vascular and nerve alterations probably commence at approximately 45°C. He concludes with a modification of Joule's Law, stating that the tissue temperature rise is proportional to the square of the current flowing in each of the tissue compartments multiplied by the resistivity times the cross-sectional area multiplied by the time of current application. While this may be a complex formula for heat, as an answer for the pathophysiology of electrical injury, it accounts for most clinical observations.

A major high-voltage electrical burn is one of the most devastating injuries for both the patient and the plastic surgeon. The initial sense of hopelessness is confirmed by early amputations, repeated sequential debridements, and an extended rehabilitation. At the center of the surgeon's uncertainty lies the dilemma of progressive necrosis - a widely accepted though poorly defined hypothesis. Progressive necrosis implies an ongoing inexplicable death of tissue within an electrically injured extremity. In many cases, the plastic surgeon may amputate a mummified hand, debride the forearm to viable tissue, and return 48 hours later to find the forearm necrotic and an above elbow amputation a necessity. Often, this sequential debridement will be prolonged over several weeks and further compounded by chronic infection. This cycle of continuing compromised viability can only be broken by acquiring a greater understanding of the pathophysiology of electrical injury. As noted by Muir[37], this obscure phenomenon must be elucidated before significant advances are possible in wound management:

> It would indeed be no exaggeration to say that the gloomy prospect of progressive necrosis overshadows the whole approach to the management of these injuries, and that a better understanding of this phenomenon is urgently needed What is the nature of this phenomenon? The answer has certainly not been found in the laboratory.
>
> I.F.K Muir
> The Treatment of Electrical Injuries
> Liverpool, England, 1957

In the Microsurgery Laboratories at the Royal Victoria Hospital, a multidisciplinary research team has attempted to determine the nature of progressive necrosis in order to validate its existence or discard this poorly understood myth. The methods of energy delivery and data collection have adopted a new approach to energy administration, instrumentation, and computer-controlled data acquisition and analysis (Fig. 1-3).

Fig. 1 Fully isolated experimental high voltage test area. Computer activated breakers control energy source generated by custom made dry-type and distribution step-up transformers. Measuring instrumentation linked to multichannel fiberoptic relay system.

Fig. 2 Data acquisition and control area Z-80 based single board computer with 64 Kbytes of RAM and double-density floppy disk drives with combined storage capacity of 2 4 Mbytes. 16 channel A/D converter with 8 or 12 bit resolution. 256 Kbyte buffer memory card or RAM disk

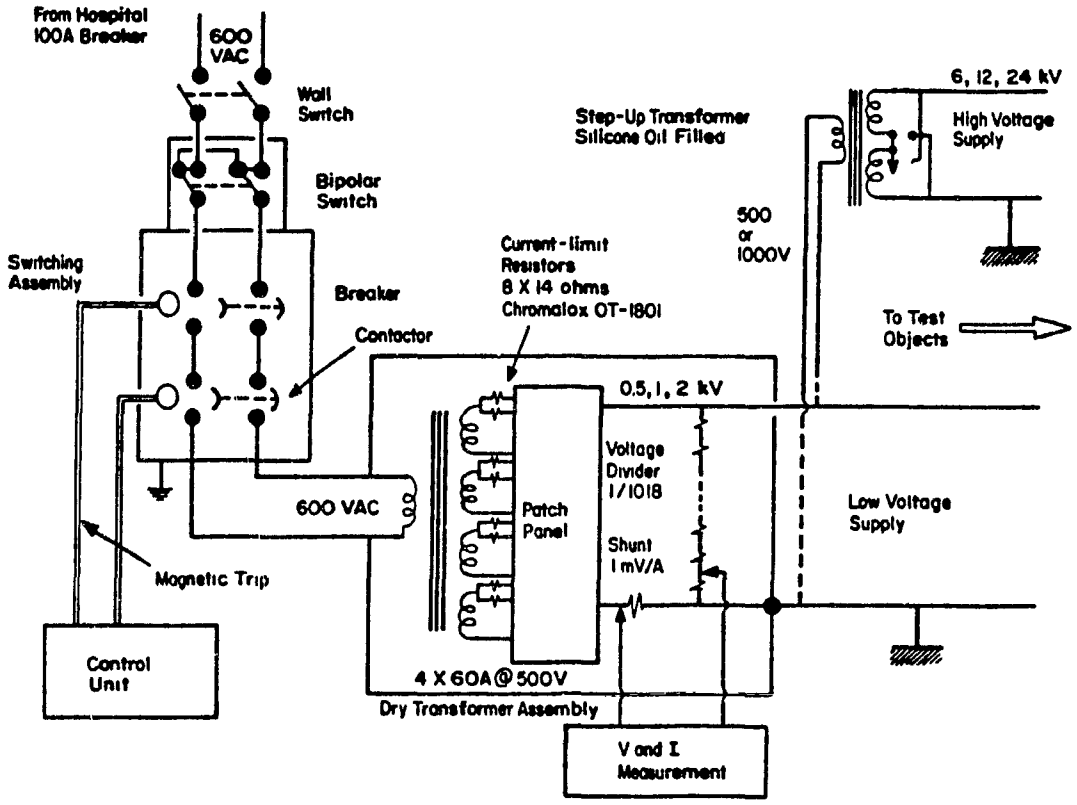


Fig. 3 The power source consists of a pair of transformers located in a fully isolated test area, with discrete voltage settings of 0.5, 1, 2, 6, 12, and 24 kV, together with control hardware. A computer-activated contactor (600 VAC bipolar, 270 amp capacity) controls power to the test object. Breaker switches in the control area energize a custom made dry type transformer (600 VAC primary and 4500 VAC secondaries rated at 60 A each) Each secondary feeds through a pair of 14 ohm resistors in parallel Connections from the resistors are made to a patch panel which allows parallel or series connection of the winding to obtain the desired voltage configuration. The output voltage and current of the dry transformer are measured respectively by a megohm voltage divider and by a 1 mV/A current shunt. Signals from those devices are directed to the data system The output voltage and current are multiplied by a distribution step-up transformer by a factor of 12 or 24 according to position of a switch on the unit With these ratios, voltages of 6, 12, and 24 kV can be generated using the dry-type transformer as the supply for the primary

THE CHOICE OF AN ANIMAL MODEL

The choice of an experimental animal model was critical for high voltage electrical burns as minor variations in model design may result in major variations in the pattern of injury As well, a model was needed which would allow easy and unobstructed access to evaluations for a considerable period post injury. Since a truly clinically applicable "chronic" model did not exist in the literature, our initial efforts were directed towards devoloping such a model.

Traditionally, in generating a wound for study, investigators have delivered a designated voltage through electrodes affixed to the anatomical areas under study This approach is dictated by Ohm's Law which states the relationship of voltage to current as $V = IR$ (where V = voltage, I = current, R = resistance). At constant temperature, all metals and semiconductors pass a steady current when a constant voltage or electric field is applied to them. Such materials are said to obey Ohm's Law. While physical laws pertaining to current passage do not always seem operative in biological environments, strictly speaking, tissue has specific physical properties that obey these laws It is the number of variables acting in conjunction which render measurements in situ difficult to obtain and interpret. In tissue, the resistance of each separate component is different and variable - changing with temperature as current flows. Thus, the actual current delivered to the model is unknown and the wound is not reproducible These conditions have been responsible for undermining the quantitative

results of past investigations.

Hunt's 1976 study of pathophysiology was conducted in the rat model at low voltage.[21] He cited electrode contact as an important variable for the severity of damage produced. Yet from the wide range of temperatures recorded (60-95^{o}C), we may conclude that the tiny (3/8") electrodes taped to the bony rat limb provided variable contact. Of note, Hunt himself concluded that the recorded tissue temperatures 'may represent only a fraction of those occurring in human electric injuries' Hunt's gross, microscopic and angiographic observations of the rat hind limb revealed no evidence of 'progressive or de novo' muscle necrosis. He conceded that his findings 'may be attributed to the use of low voltage in a small animal'.

In contrast, the pig's thick, rigidly adherent, and well-vascularized skin has physiologic and anatomic similarities to man and has been the preferred experimental model for localized burn lesions However, measurements of physical parameters, especially temperature, must be considered in the context of the significant volume expansion of the pig upper thigh. If the animal limb is perceived as a volume conductor, the amount of heat produced per unit volume of tissue - the current density - will be a function of the cross-sectional diameter of the limb The consequent reduction of current density in the thigh region and across the trunk will affect all measured physical parameters to an extent proportional to the 'flaring' of the upper hindlimb. Thus, similarity of anatomy and a reproducible energy source are critical factors in deriving an

experimental burn model which will be clinically relevant.

In contrast to delivery systems utilizing a voltage standard, the computer-controlled administration system used in our studies is based on **energy**. The computer is programmed to sample and compute values for voltage (V), current (I) and time (t). At the preselected value for energy (E = VIt) expressed in kilojoules, the computer deactivates the breaker stopping current flow. The three critical variables may be programmed separately or in combination. Thus, physical parameters directly governing wound severity are precisely controlled, and a standard, electrical injury model can be consistently reproduced. Sixteen input channels to the computer permit the use of a wide variety of surgically implanted computer-compatible instrumentation (thermistors, current sensors, potential probes). This allows precise temporal and dynamic information on physical parameters (voltage, temperature, current) as current flows. At high voltage, implantation of instrumentation into electrically-active biological systems poses problems of isolation and insulation. To isolate surgically implanted instruments (measuring kilovolts) from the computer circuitry (operating in millivolts), a 16 channel fiber optic relay system was designed. With an effective resistance in the million ohm range, accuracy, safety and reliability were ensured.

The primary characteristic of high voltage electrical injury is massive soft tissue damage to the extremities Injury to the hands and arms occurs in 75% of clinical cases. The primate upper extremity, due to its anatomical similarity to man, represents the ultimate

experimental model. The grasping hand, glabrous palmar skin, thin bony wrist, and proximal forearm musculature all contribute to a comparable clinical model. The size of the limb and its relative composite tissue volumes allow valuable translation of experimental data to the clinical situation.

CHRONIC ELECTRICAL INJURY MODEL

A chronic bilateral upper extremity electrical burn model was developed in the African Green monkey (Cerocopitheus aethiops). It consists of three stages: 1) a partial decerebration procedure to eliminate all pain perception, 2) 24 hours later, a standardized hand to hand electrical injury of 40 kilojoules and 3) follow-up observation for eight to ten days post electrical injury.

CRANIOTOMY AND PARTIAL DECEREBRATION

The animals were sedated with an intramuscular injection of a ketamine/xylazine (70 mg ketamine/6 mg xylazine) solution preoperatively. Operative anesthesia was maintained with intravenous pentobarbital. The animal received an intramuscular injection of 50,000 U penicillin G and 125 mg dihydrostreptomycin base (veterinary preparation) and 50 mg Netilmicin preoperatively and every twelve hours for the first 48 hours. Preparation for the procedure included the following: insertion of venous and arterial catheters via cutdowns, continuous electrocardiographic and rectal temperature recording, endotracheal intubation with assisted ventilation, insertion of a nasogastric feeding tube, and urinary bladder catheter. The head of the animal was positioned in a stereotaxic frame. Under sterile conditions, bilateral craniotomies were created. Using standard neurosurgical technique, the cerebrum was removed with suction. All cortical regions responsible for the processing and

interpretation of pain were removed. These areas include the entire parietal lobe, the insular and retroinsular cortex, upper temporal lobes and most of the frontal and occipital lobes. The midbrain was left intact Following hemostasis with bipolar cautery, the cranium and skin were closed. Postoperative management involved hourly fluid management, assisted ventilation and continuous pentobarbital drip (30 mg/hr) for seizure prophylaxis.

ELECTRICAL INJURY

Twenty four hours post decerebration, the animal received a bilateral upper extremity electrical injury. Copper rod electrodes coated with electroconductive jelly were secured in both palms and fastened to high tension cables with alligator clips. A 40 kilojoule standardized electrical injury was then delivered with an average voltage of 3550 volts, current of 4.2 amperes and duration of 2.5 seconds. Arcing was not observed during the delivery of electrical energy. Electrical injuries of 20 to 96 kilojoules were produced during the acute pathophysiology phase of this project and the resulting patterns of injury evaluated. A 40 kilojoule injury was chosen as the standard for the chronic portion of the study because it provided a high energy injury with an interface between viable and nonviable tissue in both the forearm and arm (Fig. 4).

Four hours following the electrical burn, bilateral fasciotomies were performed. An incision extending through the fascia was made from the carpal tunnel along the volar forearm and medial arm to the axilla,

decompressing the volar muscles in the forearm as well as the anterior and posterior compartments of the upper arm. Twenty four hours post electrical injury, the animal was extubated and daily intragastric infusions of Ensure were begun, providing 350 to 400 kcal/day. Forty eight hours post injury the pentobarbital drip was stopped. The animals remained stable until the eighth to tenth day with no pharmacologic intervention required (Fig. 5). However, when experimental protocol required an invasive procedure (cutaneous or muscle biopsy), an intravenous injection of pentobarbitol insured the elimination of spinal reflexes.

EXPERIMENTATION PERIOD

Experimentation of the electrical wound began at or before the time of injury and continued up to 10 days. Depending on the type of investigation in progress, the wounds were evaluation at specified times using varied techniques and redressed in a sterile fashion. On the final day of evaluation, extensive and detailed bilateral upper extremity dissections were completed and the extent of injury to individual muscles and muscle groups recorded on templates.

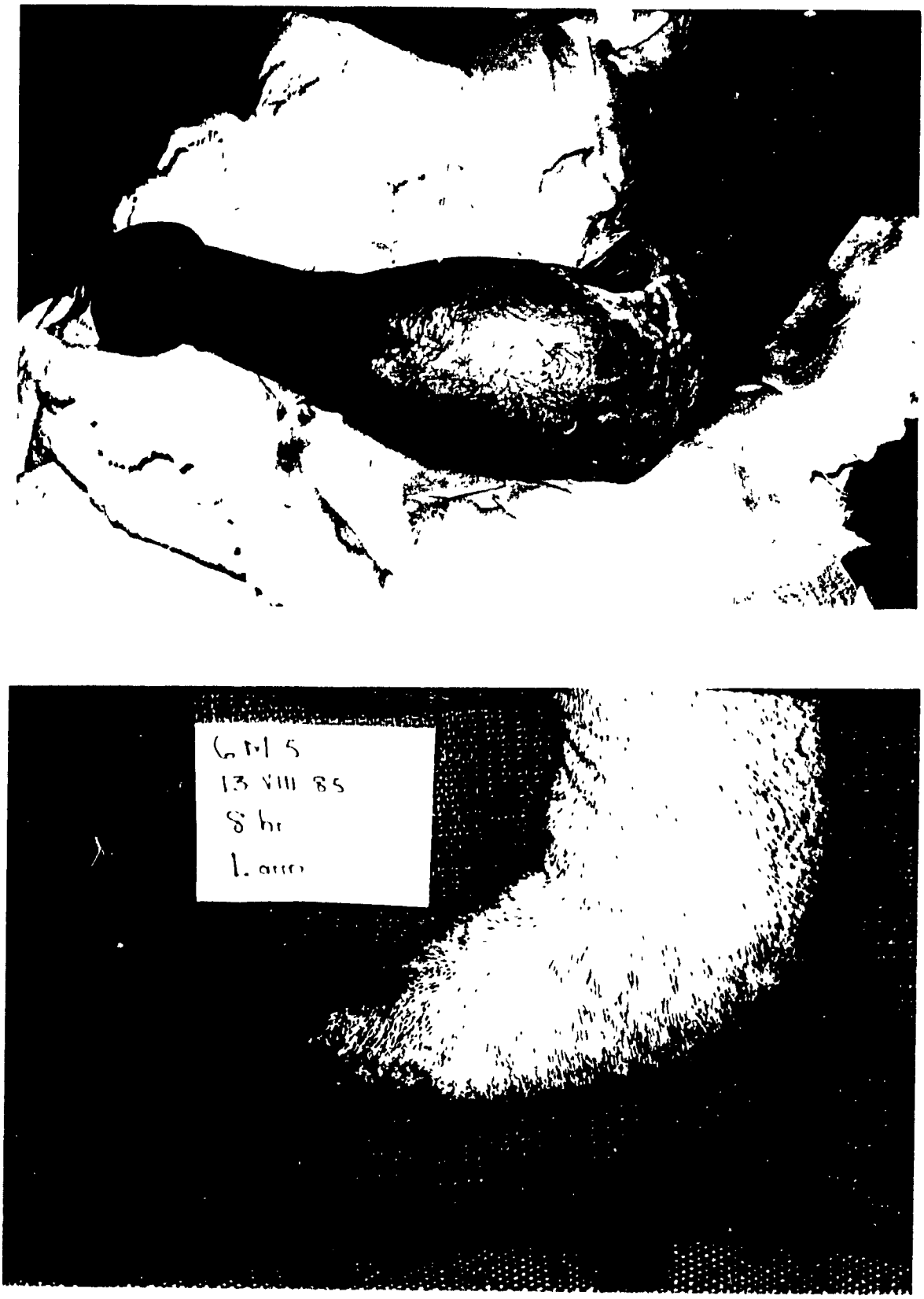


Fig. 4 High voltage electrical injury in the human extremity (above) and the primate (below).

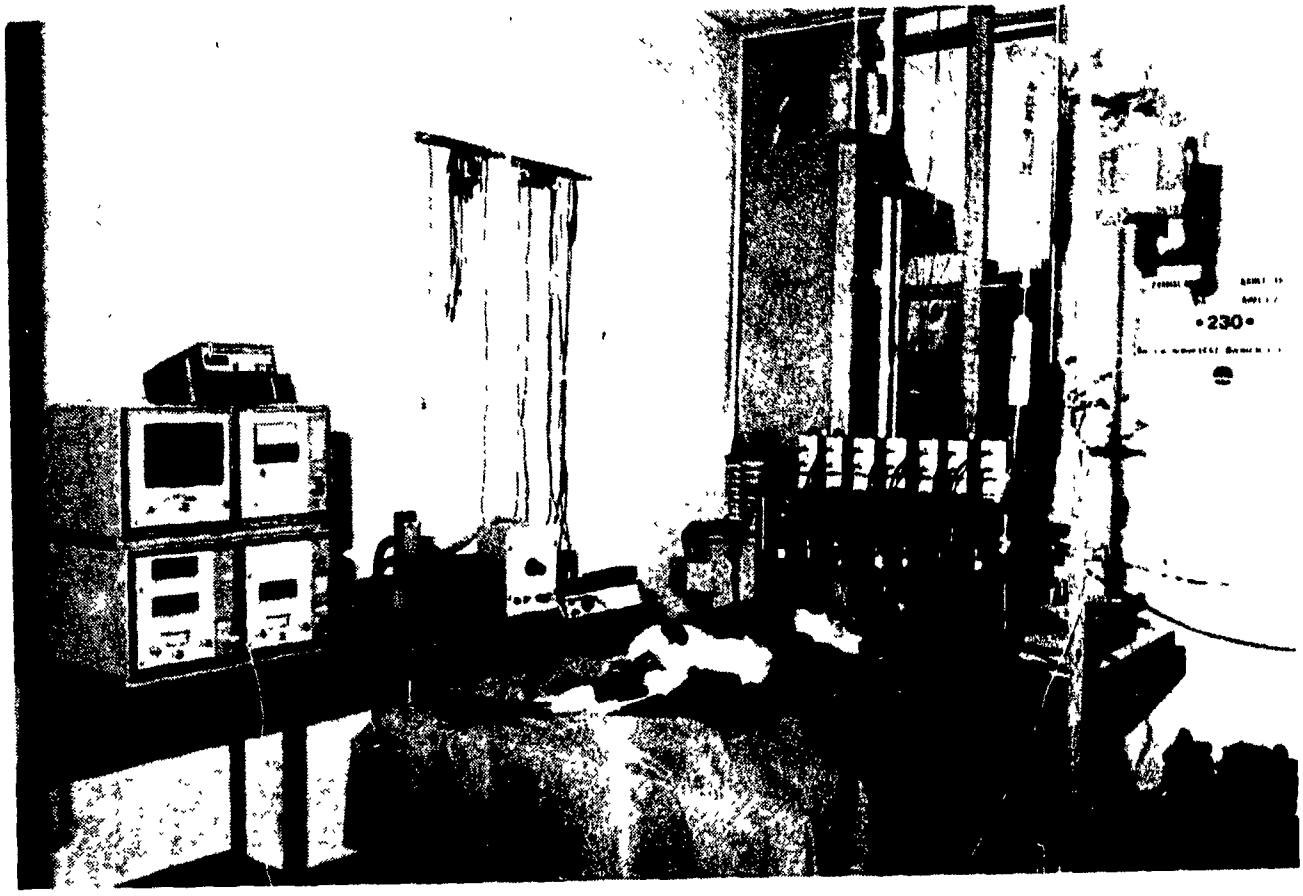


Fig. 5 Animal positioning and monitoring equipment used for the chronic electrical injury model.

EXPERIMENTAL STUDIES

METHODS AND MATERIALS

Our experimental evaluation can be divided into 6 separate sections: 1) pilot studies, 2) gross observation, 3) light microscopy, 4) digital subtraction angiography, 5) temperature recordings and 6) electrophysiologic nerve conduction studies. A total of 38 animals were utilized, each weighing 5.2 to 6.0 kg. Seven animals were used in the pilot studies and provided the following information. 1) development of a reliable decerebration procedure, 2) establishment and management of numerous monitoring techniques, 3) detailed dissections of normal upper extremities to clearly define pertinent anatomical relationships, 4) design of life-size skin and individual muscle templates for assessing injury patterns, and 5) evolution of a management protocol to insure survival of the animal to the tenth day post injury.

GROUP I. GROSS OBSERVATION

General characteristics of skin, muscle and neurovascular injury were evaluated in 6 animals bilaterally over an eight day period. Prior to fasciotomy, the patterns of skin injury were recorded on individualized upper extremity templates. Selected muscles were examined for gross appearance, color, bleeding when stabbed with a #11 scalpel blade, and response to pinching with forceps and stimulation with a nerve stimulator (2 mv) These parameters were selected as they are used by clinicians intraoperatively to assess viability and to

estimate the extent of debridement required. Specific muscles for evaluation were chosen by size and anatomic location. They included the flexor carpi ulnaris (FCU), flexor digitorum superficialis (FDS), flexor carpi radialis (FCR), biceps, (BIC), and medial head of triceps (TRIC) The pattern of injury to these muscles was recorded on individualized templates. Following injection of fluoroscein dye (500 mg), patterns of skin and muscle perfusion were recorded under Wood's lamp illumination and marked with sutures. Antiseptic tulle gras dressing (chlorhexidine acetate) was applied and covered with sterile Kling dressing. On the eighth day, detailed upper extremity dissections were completed bilaterally. The extent of injury to each of the 15 flexor and extensor muscles of both the forearm and arm was evaluated both longitudinally and on serial cross sectioning and recorded on individual templates (Fig 6 through 9). The patency of the major vessels was noted as well as the gross appearance of the median, ulnar and radial nerves.

GROUP II: LIGHT MICROSCOPY

In 3 animals, tissues were harvested bilateraly preburn and postburn at 4 hours and every 2 days until the eighth post burn day. Biopsy sites were based on a "line of demarcation" (hereinafter LOD) between grossly viable and nonviable tissue, as established immediately post burn. In each animal, 12 skin biopsies were harvested bilaterally. A total of 20 muscle samples were taken bilaterally from the five selected muscles, 2 cm above and below the

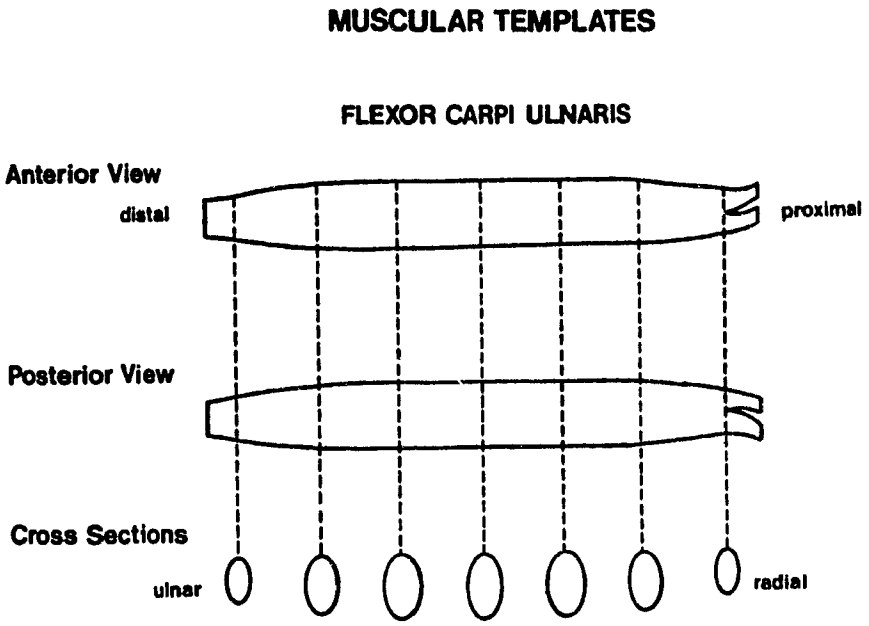


Fig. 6 Diagram of individual muscle template used for recording gross patterns of muscle injury.

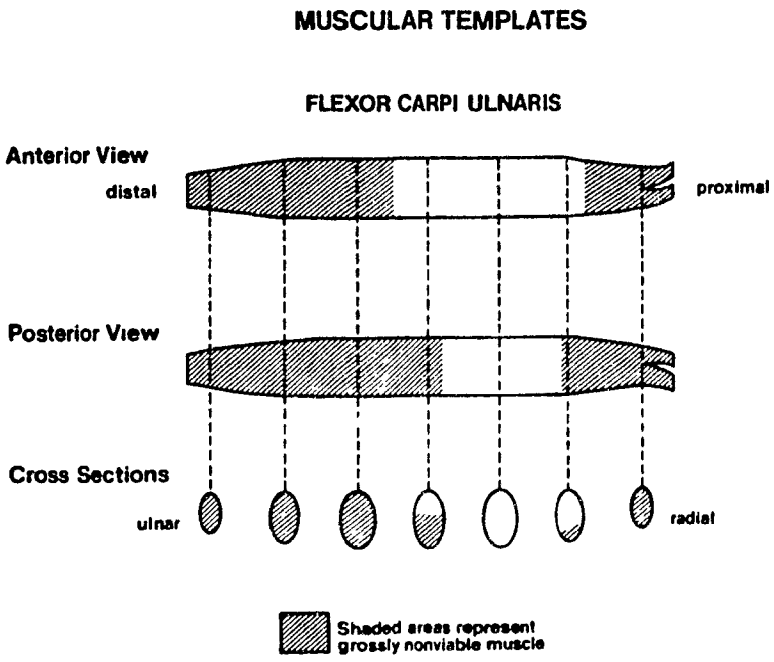


Fig. 7 Recording of grossly viable (nonshaded) and nonviable (shaded areas) data at the time of final evaluation 8 days post burn

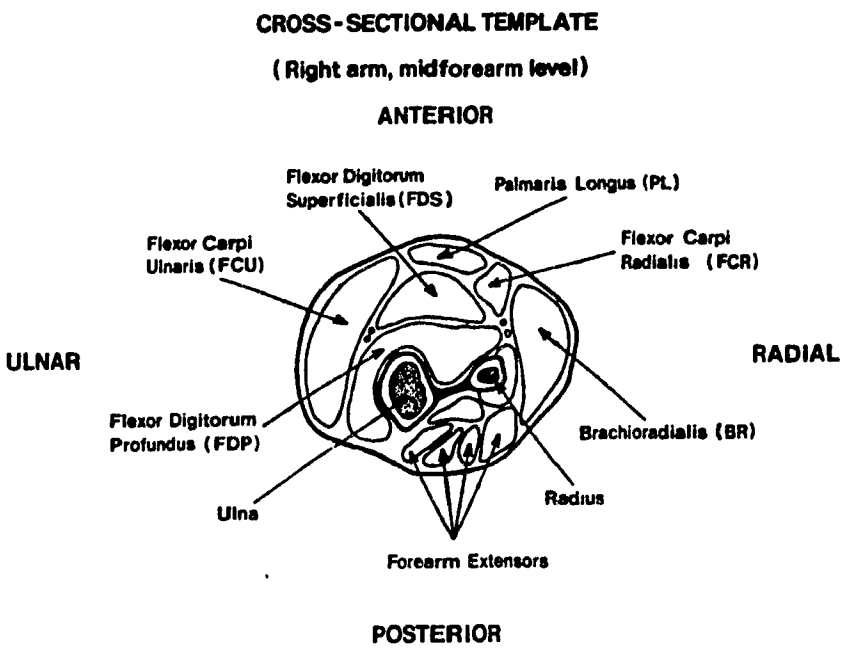


Fig. 8 Example of a cross sectional template used during the final evaluation of extremity muscles.

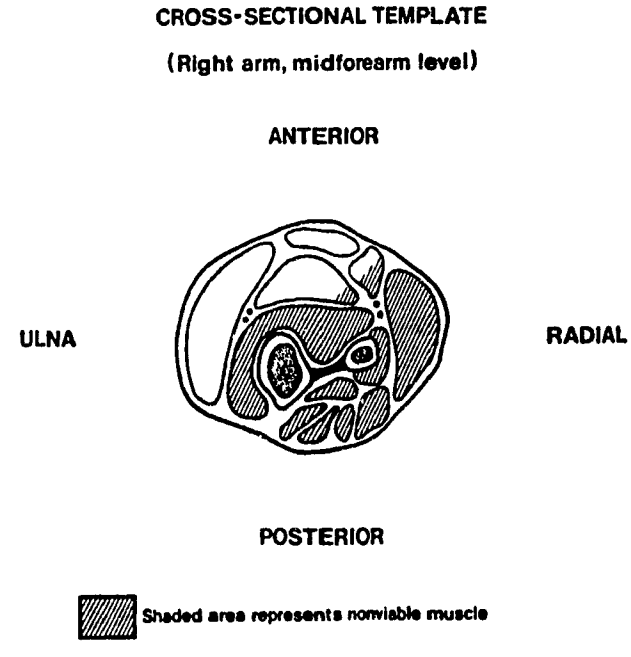


Fig 9 Example of gross observation data on a cross sectional template (shaded areas represent grossly nonviable tissue).

individualized line of demarcation between viable and nonviable muscle. Tissues were processed in the standard histological fashion and stained with hemotoxylin and eosin. Histological samples were harvested at the time of final evaluation from over 20 animals with over 1,100 light microscope slides assessed.

GROUP III: ANGIOGRAPHY

Digital subtraction angiography was used to identify characteristic vascular lesions of the acute electrical injury and to document their evolution through the 10th day post injury. Selective bilateral upper extremity digital subtraction angiograms were completed for pilot and experimental studies in 10 animals. All studies were completed using Omnipaque contrast media and standard angiographic techniques. Selective catheterization of the axillary or brachial arteries was achieved at each time interval through an indwelling 5 French Cordis catheter placed in the femoral artery prior to electrical injury. In these primates, the brachial artery bifurcates into the ulnar and radial arteries 3 to 5 cm proximal to the medial epicondyle of the humerus. With this exception, the arterial branching within the forearm and arm was similar to the human extremity.

Following pilot studies, one group of 4 animals had baseline upper extremity angiograms completed three days prior to electrical injury and again at 6 hours, then at 2, 6, and 10 days post injury Gross observations (muscle colour, stimulation, and bleeding) of the

burn at 2 day intervals confirmed the findings of the group I animals and allowed serial comparisons of gross pathology with angiographic findings. On the tenth postburn day, detailed upper extremity dissections were completed bilaterally. Gross patterns of vascular injury were determined and correlated to muscle and skin pathology. In all studies, the brachial, radial and ulnar arteries were then harvested for light microscopic study.

GROUP IV. TEMPERATURE RECORDING

Paramount to our studies was to determine whether the primary injury caused by the flow of electrical current through biologic tissues was thermal in etiology. Data from previous investigations on the primate model was examined and served as a guideline for the critical placement of the temperature probes within the arm and forearm.

In six animals, unilateral temperature recordings were performed using specially designed thermistor probes. A thermistor contains a semicondutor in which a slight temperature change causes a pronounced change in electrical resistance. The YS1 530 thermistor selected for these studies is encapsulated in a thin flexible teflon tubing. The probe measured 1 04 mm in diameter and can be inserted into tissue through a No. 14 Jelco catheter. The exposed thermistor tip has a response time of 0 1 second with an accuracy of $\pm$ 1^{o}C in the critical temperature range of 37^{o}C to 80^{o}C. Temperature probes were tunnelled into superficial areas of muscles through the bore of a No. 14 gauge

needle. Seven temperature probes were placed in the upper extremity for each experiment (Fig. 10). Representative muscles were chosen for each study, as the number of probes was limited. In the flexor carpi ulnaris (FCU), four probes were placed at 2 cm intervals from its insertion on the medial epicondyl of the humerus. One probe was placed in the brachioradialis (BR) and another in the FCU, both equidistant from the wrist. These probes were placed to assess temperature rise differences between the ulnar and radial aspects of the forearm. Finally, two probes were place in the biceps muscle. One probe positioned 2 cm below the expected line of demarcation between viable and nonviable tissue and one 2 cm above. Probe leads were plugged into amplificaton, isolation and transmission systems connected to the computer. Temperature recordings were begun prior to electrical injury and restarted immediately after the injury. Temperatures were recorded at 1 second intervals and continued for 15 minutes. The animals underwent gross evaluations of injury daily until the tenth post injury day. At that time, detailed, bilateral upper extremity dissections were performed to correlate ultimate tissue survival with the recorded temperatures on the day of initial injury.

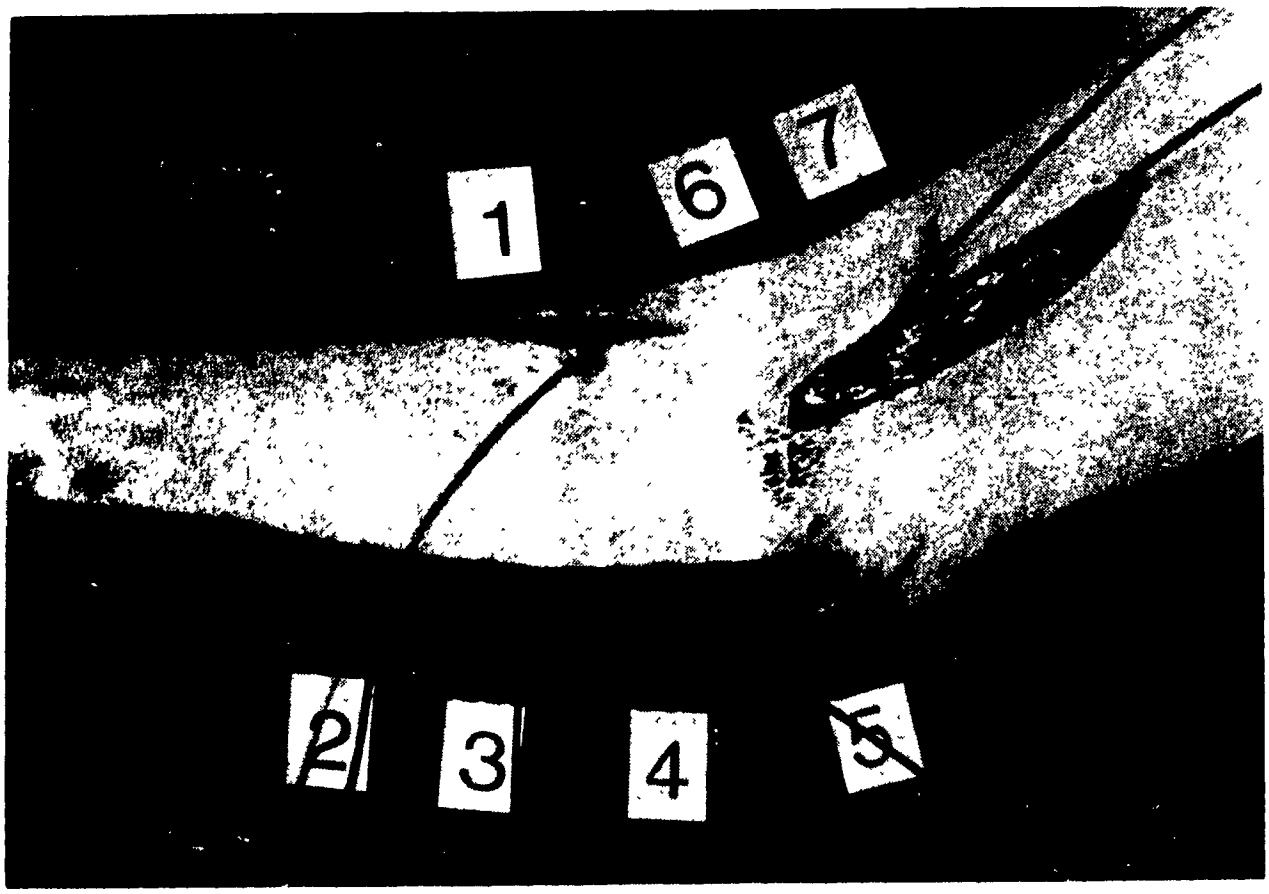


Fig. 10 The right extremity of the primate with thermistors implanted prior to electrical injury.

GROUP V: ELECTROPHYSIOLOGIC NERVE CONDUCTION RECORDINGS

In four animals, unilateral sequential ulnar nerve recordings were completed in the standard fashion beginning 1 hour post injury and then at 4, 12 and 24 hours and on days 4 and 10. With the stimulating electrode positioned high in the axilla, compound action potentials were recorded at approximately 1 cm intervals beginning at the mid-arm level and progressing distally until conduction loss was documented (Fig. 11). Levels of conduction loss and conduction velocities were determined at each time interval. On the tenth day post injury, the ulnar nerve was harvested for light microscopic study. Standard techniques were used for tissue processing and staining with hemotoxylin and eosin. In two noninjured animals, bilateral ulnar nerve recordings were done at times 0, then at 3, and 9 hours in order to serve as control recordings.

Fig. 11 The left extremity of the primate showing setup for recordings of ulnar nerve compound action potentials.

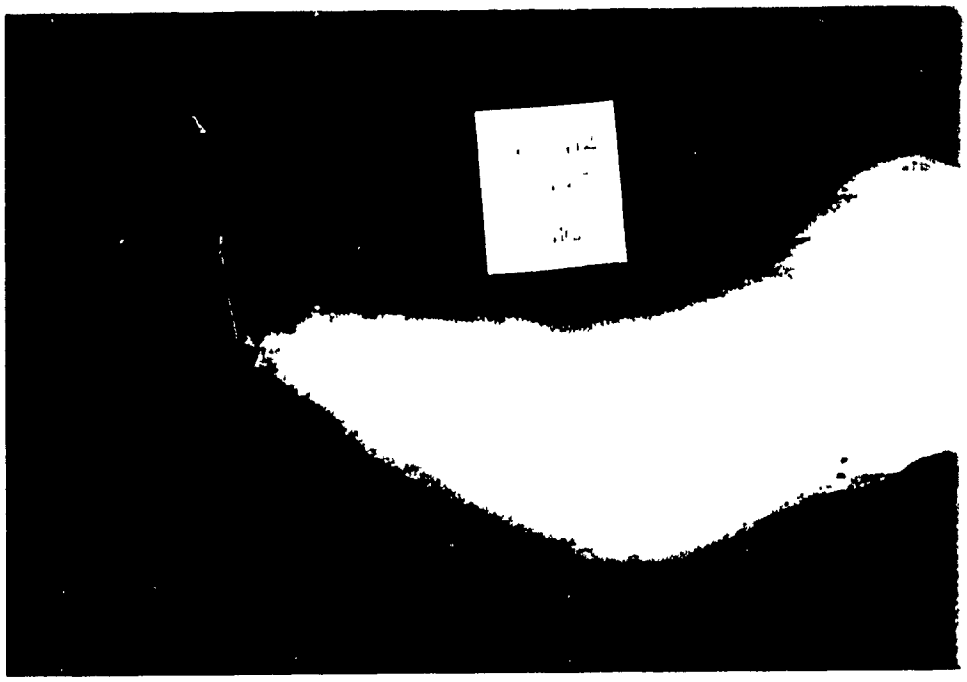
RESULTS

GROUP I: GROSS OBSERVATION

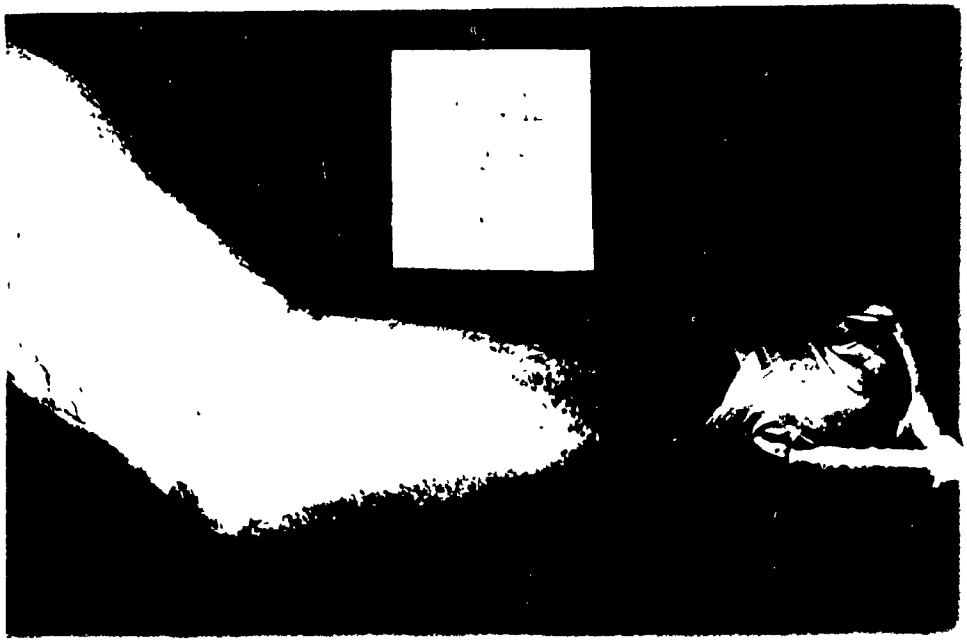
Twelve extremities were studied in depth beginning preburn and continuing daily until the 8th day post injury.

SKIN Skin involvement in all extremities showed a consistent pattern immediately following electrical injury (Fig. 12 and 13). The distal extremity was mummified up to the midforearm level. Nonperfused skin extended proximally along the extensor surface, radial aspect of the flexor forearm, and circumferentially through the cubital fossa. An island of perfused skin was seen directly over the forearm flexor bellies. The extensor surface of the forearm was severely affected, with a viable territory spared in the uppermost ulnar aspect. Axillary "skip" lesions were occasionally seen. The above pattern of cutaneous injury was observed for eight days post injury with no significant local or proximal advancement of the wound margins.

Fig. 12
Normal extremity prior to electrical injury.



Cutaneous injury on flexor surface, 4 hours post injury.



Cutaneous injury on extensor surface, 4 hours post injury.

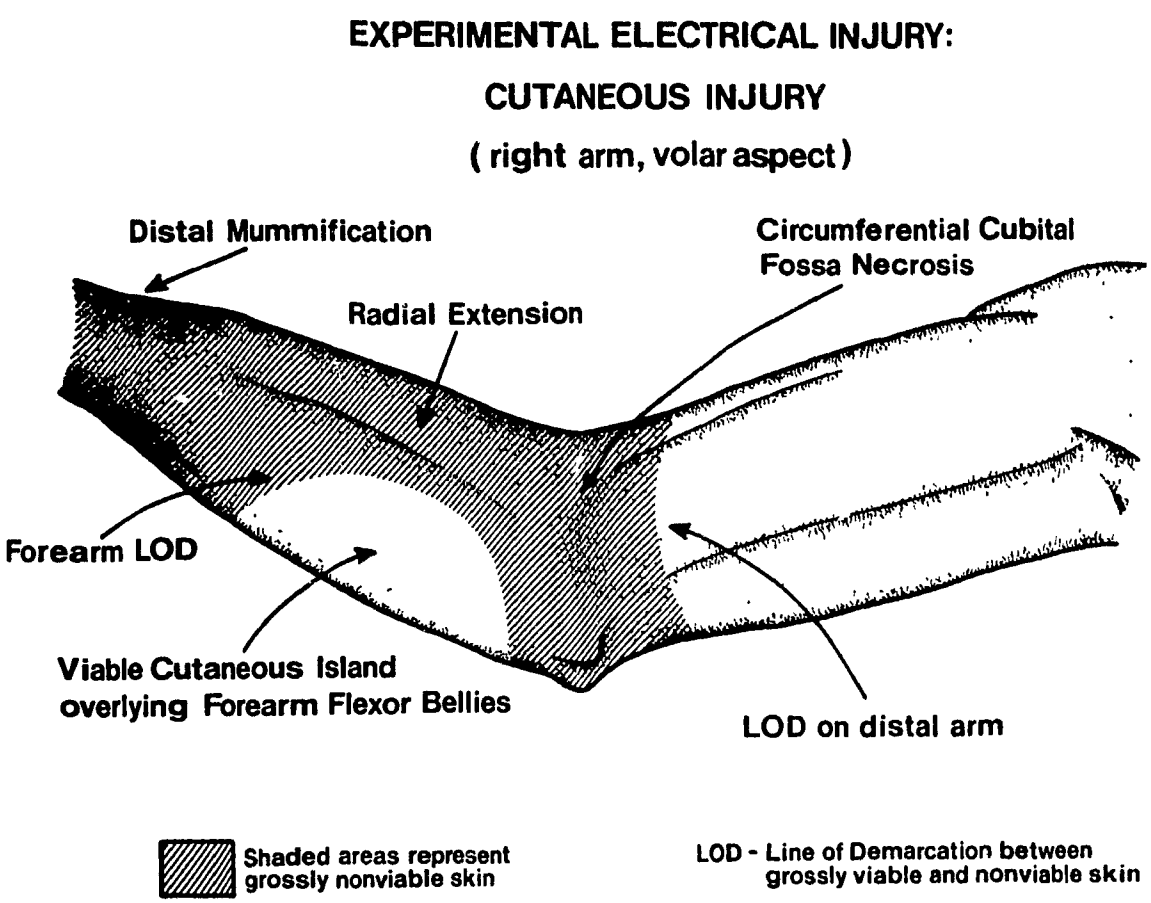


Fig. 13 Schematic illustration of cutaneous pattern of injury on right volar surface of upper extremity.

MUSCLE A reproducible pattern of muscular damage was found in all animals (Fig. 14 through 16). When first observed following the fasciotomy, an obvious line of demarcation (LOD) was apparent between nonviable pale muscle and viable pink muscle. Grossly necrotic muscle appeared pale brown to tan in colour, did not respond either to pinch or to nerve stimulation, and did not bleed. In contrast, "viable" muscle appeared healthy both in colour and texture, and bled well after being stabbed. This pattern corresponded closely to the pattern of cutaneous injury. Radially, significant portions of the FCR and the entire brachioradialis were nonviable in all instances. Within the superficial flexors, the distal two thirds were necrotic, the central belly grossly viable, but the proximal origin nonviable. Equally, the distal 2 to 3 cm biceps and triceps were nonviable whereas the remainder of the arm musculature appeared normal. Response to pinch and electrical stimulation was different in the forearm and arm While no response was found in the forearm, the "viable" muscle in the arm responded to both pinch and electrical stimulation up to 1 cm proximal to the LOD.

Evolution of the wound revealed no advancement of muscle necrosis in both the forearm and arm. In the sixty selected muscles evaluated in this study, there was no evidence of progressive necrosis Necrotic portions of the extremity underwent liquefaction during the post burn period Dissections of the musculature on the eighth day revealed distinct patterns of injury when evaluated on longitudinal and cross sectional templates In general, individual

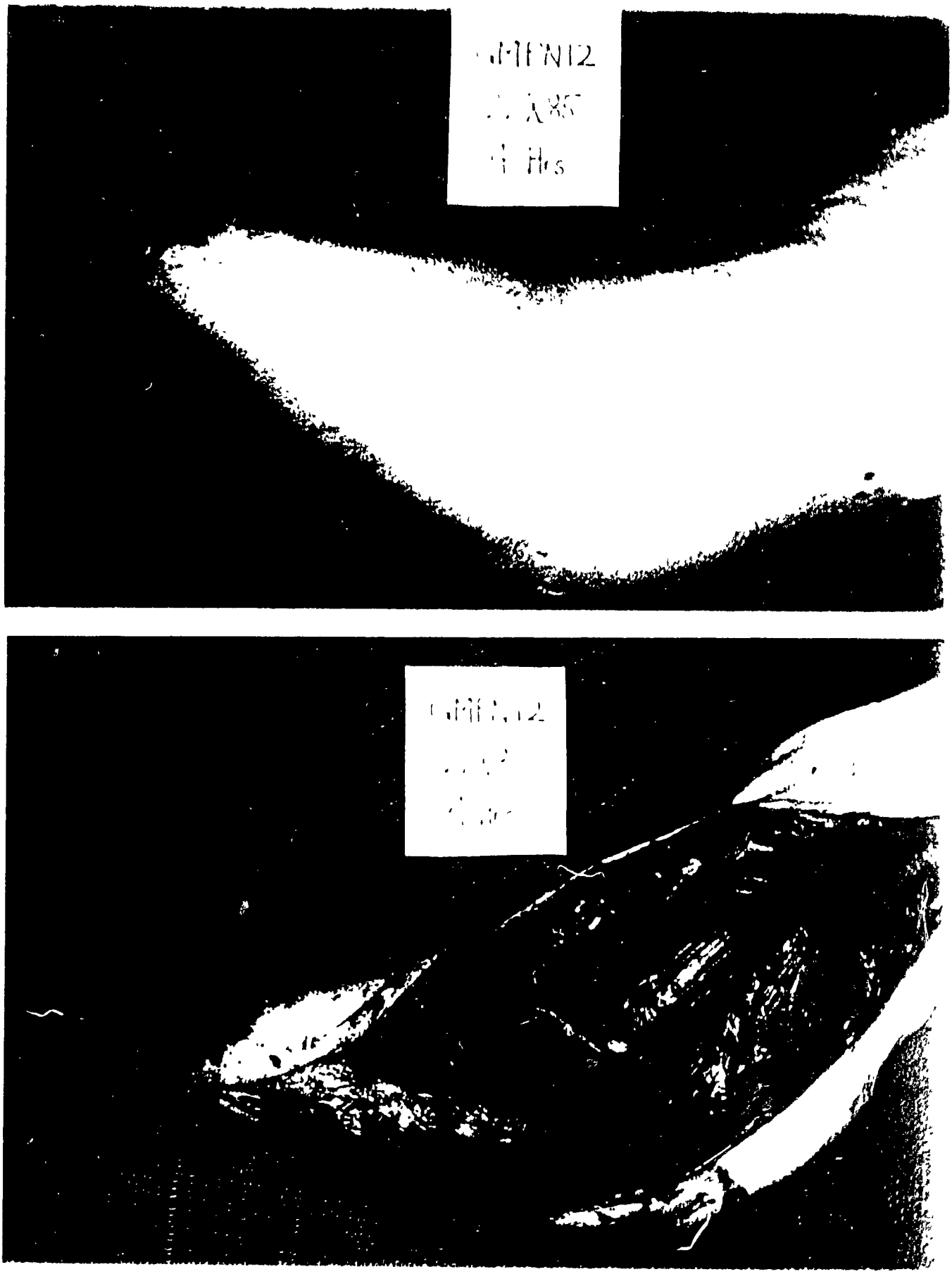


Fig 14 Cutaneous (above) and muscular (below) injury 4 hours post electrical injury

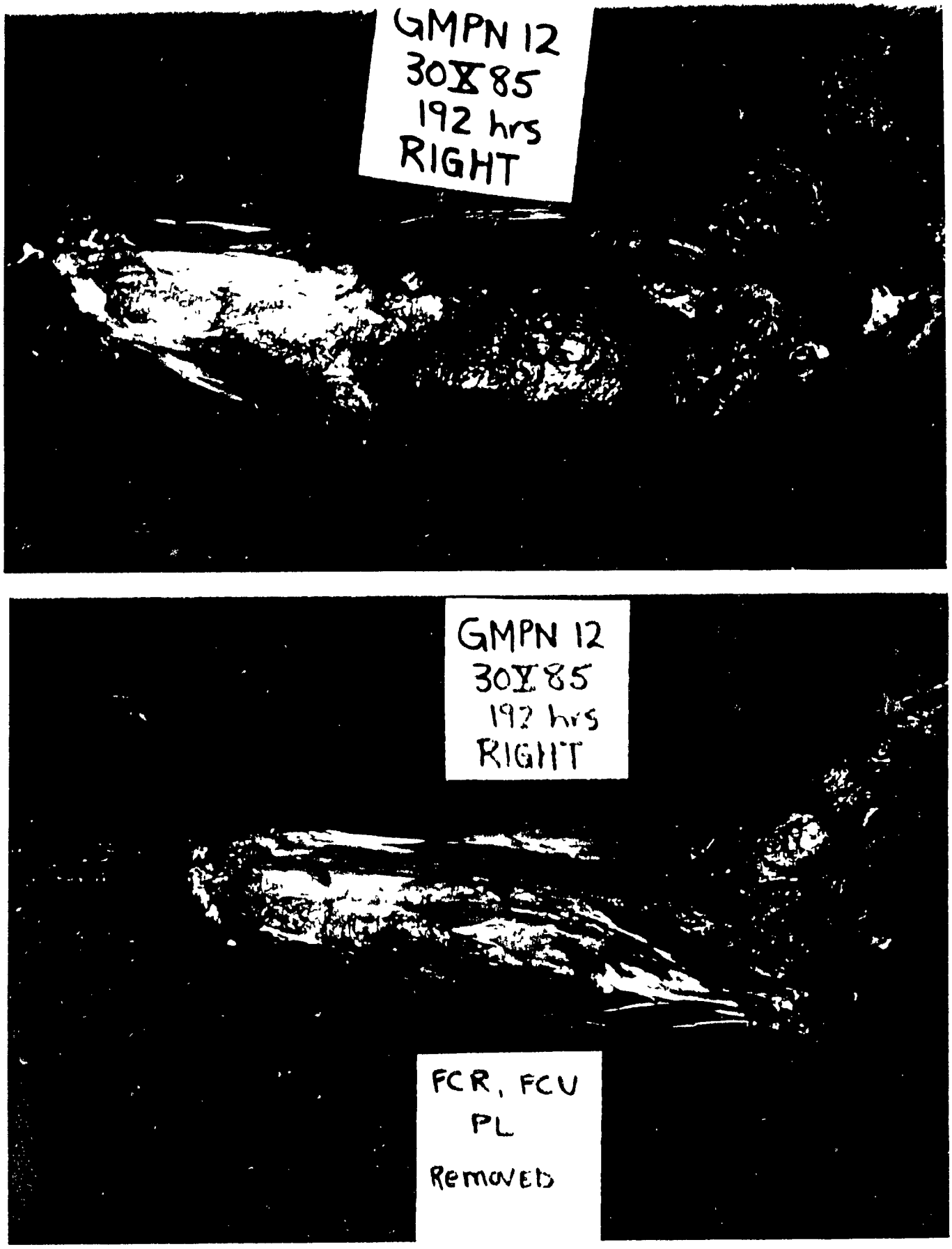


Fig 15 Muscular injury 8 days post electrical injury Above, skin removed for demonstration of viable and nonviable muscles Below, FCU, FCR, and PL removed revealing necrotic areas of deep musculature

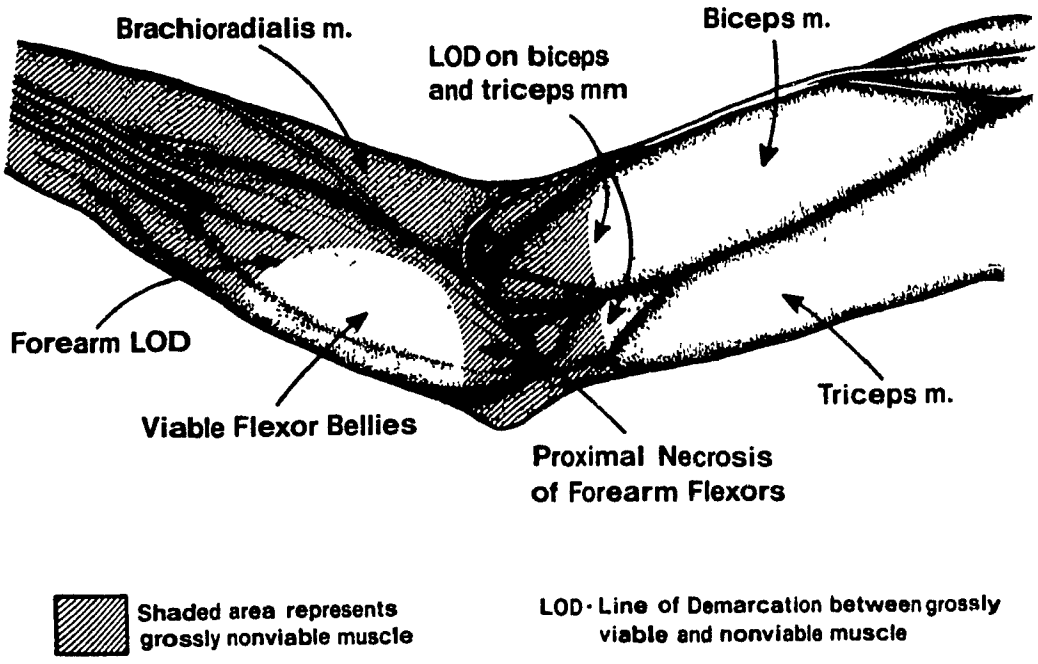


Fig. 16 Schematic illustration of muscular injury on volar surface of the upper extremity

muscles displayed more extensive injury on their deep surfaces than was evident on their superficial surfaces. In addition, muscles found in a more central location near the bone sustained significantly more proximal damage (Fig. 17). Collectively, necrosis of the muscle extended beneath well perfused cutaneous territories in both the forearm and arm Within the larger muscles of the arm, a variant form of tissue damage was found. a striking centrally located core of grossly abnormal muscle extended within the biceps and long head of triceps (Fig 18) This injured muscle was surrounded circumferentially by apparently normal muscle, extended significantly more proximal to the line of demarcation on its superficial surface.

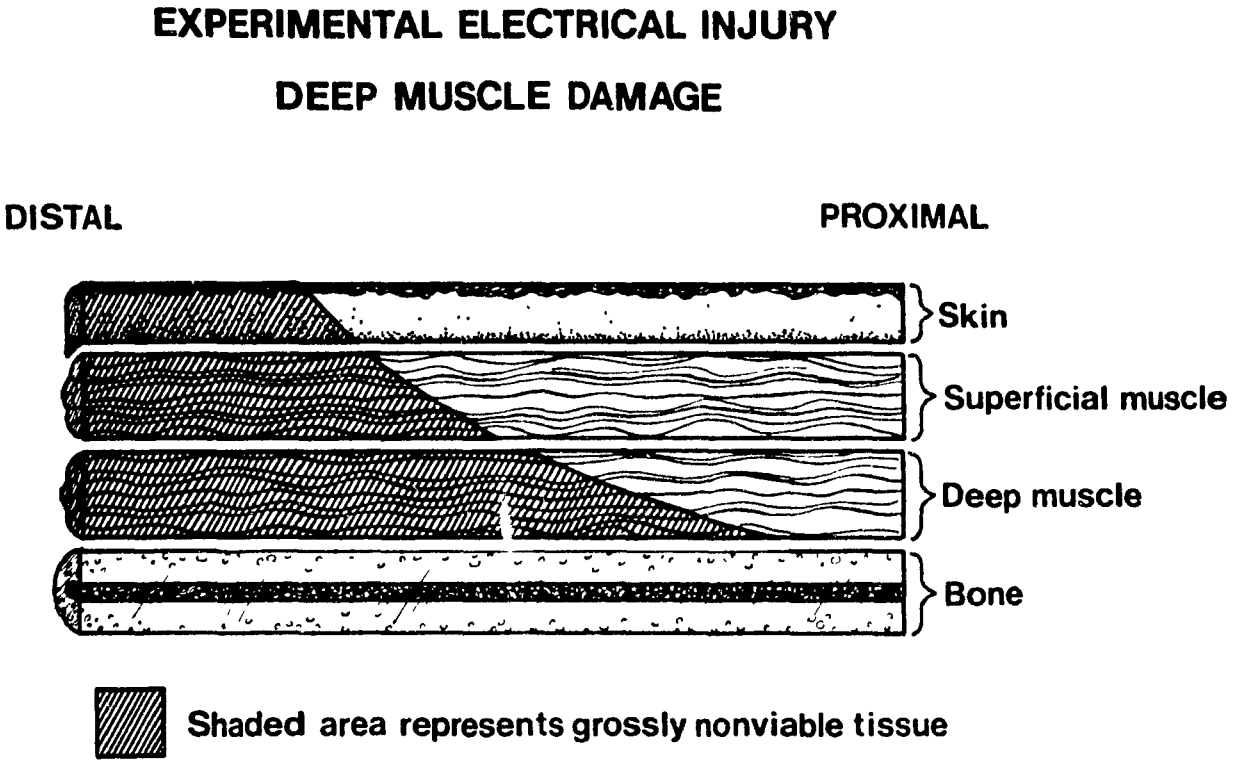
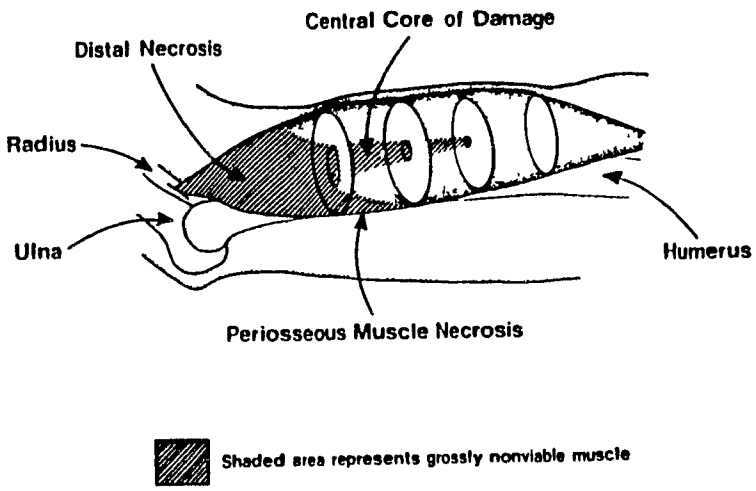


Fig. 17 Illustration of deep muscle injury. Note how periosseous muscle injury may extend proximally beneath viable skin.

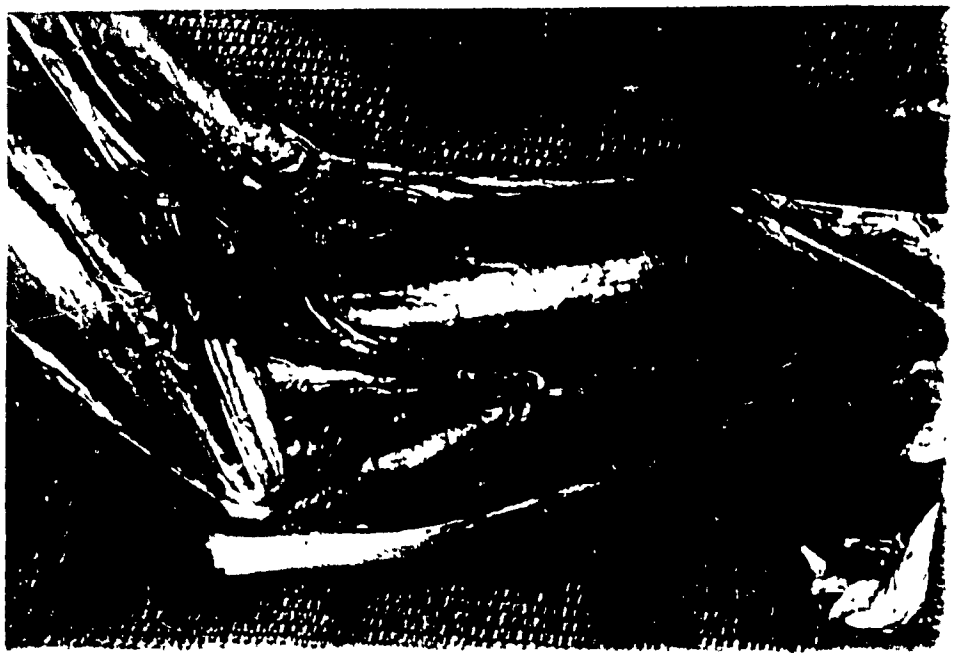
Fig. 18
Injury to biceps muscle 8 days post injury. Suture denotes line of demarcation on biceps between grossly viable and non-viable tissue.

EXPERIMENTAL ELECTRICAL BURNS:
INJURY TO BICEPS MUSCLE

Diagram of biceps injury illustrating hidden damage centrally and in the periosseous region.



Normal anatomy of the medial arm region



GROUP II: LIGHT MICROSCOPY

A total of 480 tissue samples were harvested from 6 extremities, providing 1120 histological slides for review.

SKIN Minimal changes were found in the epidermis and dermis in areas appearing grossly normal. The junction between normal and necrotic skin was distinct. As seen microscopically, cutaneous layers of the grossly abnormal skin were undergoing degeneration. Viable skin immediately adjacent to these areas showed mild edema, minimal to absent cellular infiltration, and intact skin appendages.

MUSCLE Histologically, the grossly nonviable muscle underwent an intrinsic degeneration with hypercontracted and fragmented myofibrillar elements visible at 4 hours post burn. By 48 hours, myonuclei were karytotic and undergoing karyolysis. The basal laminae were intact and highlighted by shrunken muscle fibers. At 4 days post injury, dissolution of myofibrillar material was demonstrated by the structureless, homogeneous granular appearance within cells (Fig. 19). Only nuclear remnants remained and were completely absent at 6 days post injury. Vessels in this area were thrombosed at 4 hours post injury and also underwent marked degenerative changes during the post burn period. No cellular infiltration was noted due to the lack of blood flow to these areas. Extensive necrosis of the nonviable regions prevented further tissue sampling past 6 days post burn. The changes described correspond to findings in the proximal and distal forearm flexors, as well as in the distal portions of biceps and triceps.

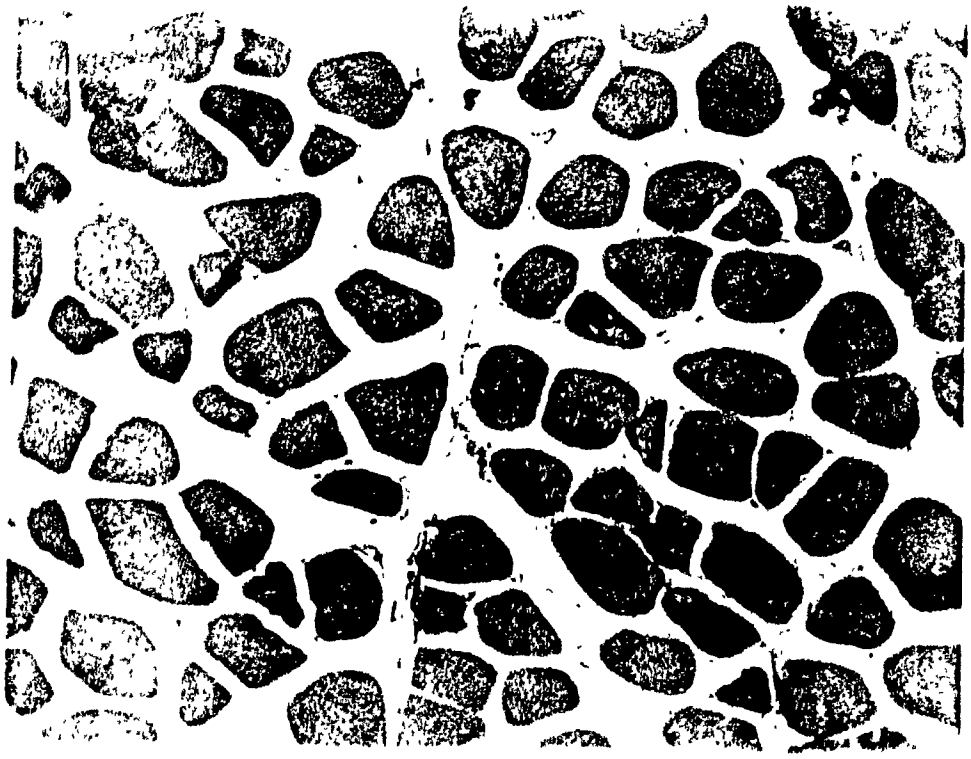


Fig. 19 Muscle from grossly necrotic areas 4 days post injury. Note shrunken cells, absence of nuclei, structureless cytoplasm with intact basal laminae.

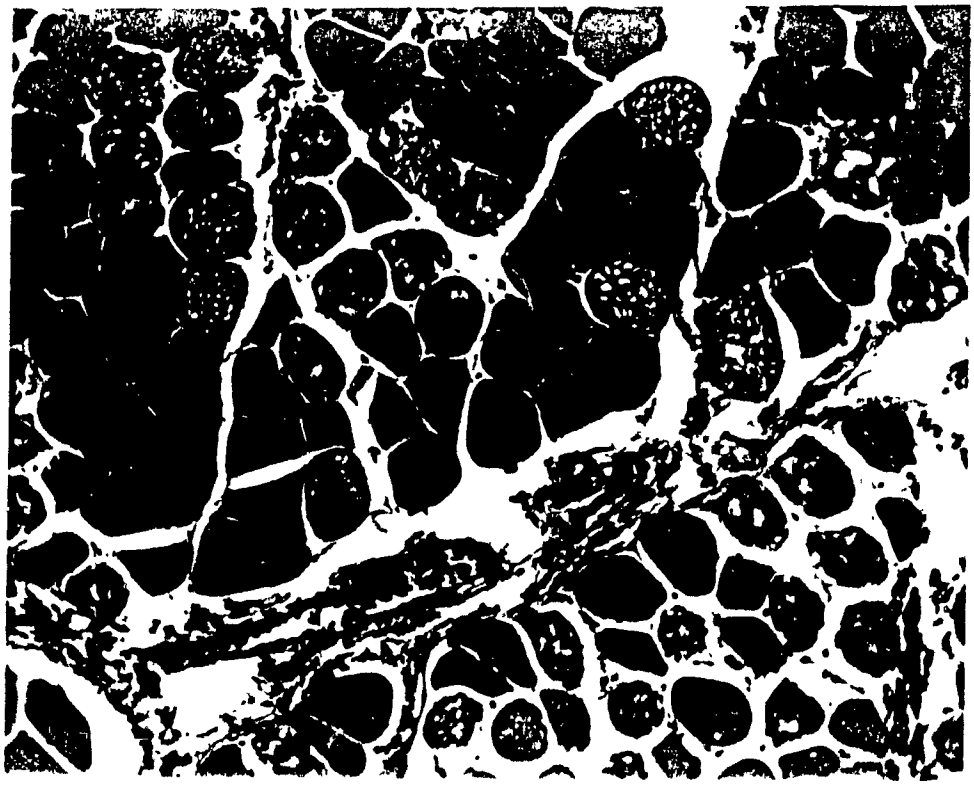


Fig 20 Muscle sample taken 4 days post injury from areas appearing grossly normal. Note close proximity of normal and affected cells.

In contrast, the grossly viable areas underwent two phases of injury and repair: an initial intrinsic degeneration, followed by a later phase of cell-mediated destruction and repair. Affected regions of muscle were not uniform and were most severe in areas adjacent to bone or grossly nonviable muscle. Sections of muscle examined at increasing distances from these critical areas revealed progressively less tissue damage. At 4 hours post burn, muscle fascicles and cells showed an uneven pattern of injury. Affected cells, in proximity to normal cells, were generally found towards the periphery of the muscle fascicle. They were enlarged and pale, with swollen vesicles present within the cytoplasm. Moderate inter- and intrafascicular edema was also present at this time. Affected cells had undergone marked degenerative changes at 4 days post burn (Fig. 20 and 21). Cellular infiltration was pronounced, increasing in severity daily until the final samples on the eighth day were examined. Macrophages were found within or surrounding necrotic cells. The amount of macrophage infiltration was again not uniform, often with minimal to severe cellular replacement noted in adjacent fascicles (Fig. 22) On the final day of evaluation, normal cells appeared intermixed with those undergoing cell- mediated destruction. At this time, regenerative changes were also evident with the appearance of myoblasts in areas adjacent to cells being phagocytosed (Fig. 23). Cross sections of the biceps and triceps were examined for evidence of a central core of damage. A dramatic increase in the number of damaged muscle fibers and

the amount of cellular infiltrate was noted in these areas. Proximal areas of biceps and triceps appeared relatively normal (Fig. 24).

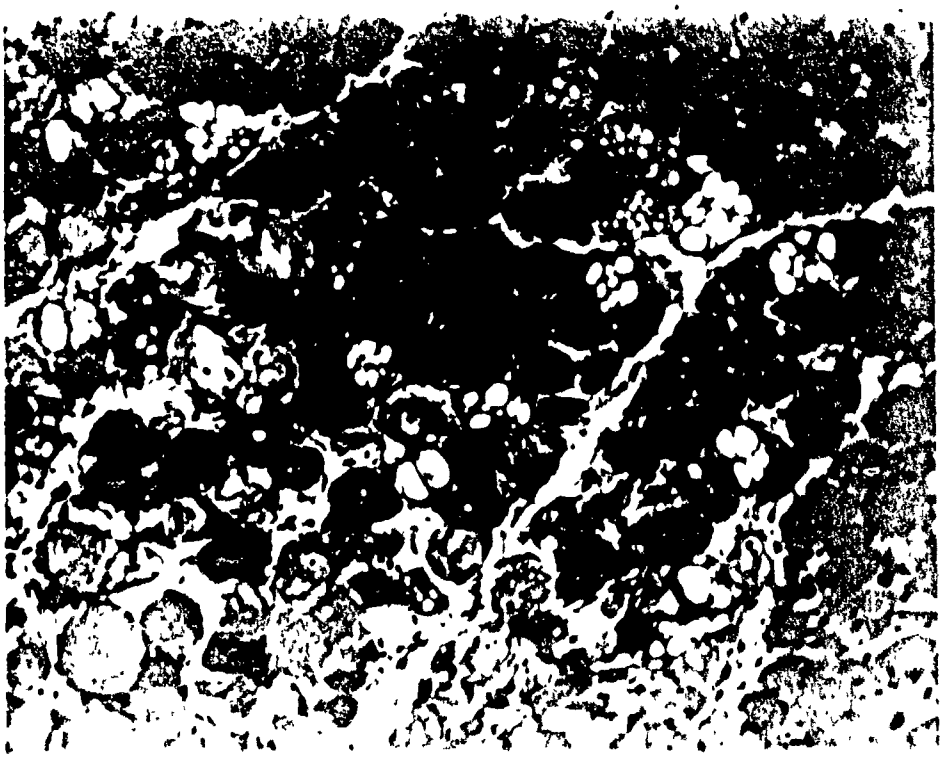


Fig. 21 Muscle sample from viable area of FCU taken 2 days post burn Note extensive fascicular damage adjacent to relatively undamaged areas.

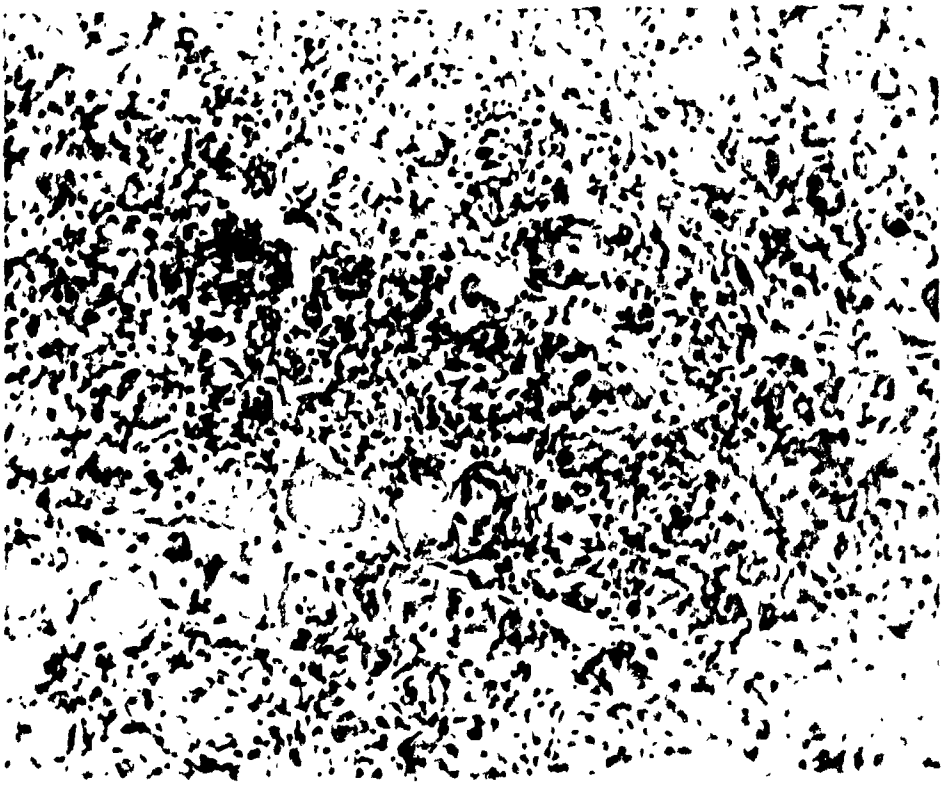


Fig. 22 Extensive cellular infiltration in viable muscle sample taken at 8 days post burn from distal biceps above LOD Note the surviving cells in this severely damaged area

Fig. 23 Magnified view of viable muscle sample taken 8 days post injury. Note marked cellular infiltration centrally surrounded by normal cells. Arrow indicates probable myoblasts and thus early evidence of cellular regeneration.

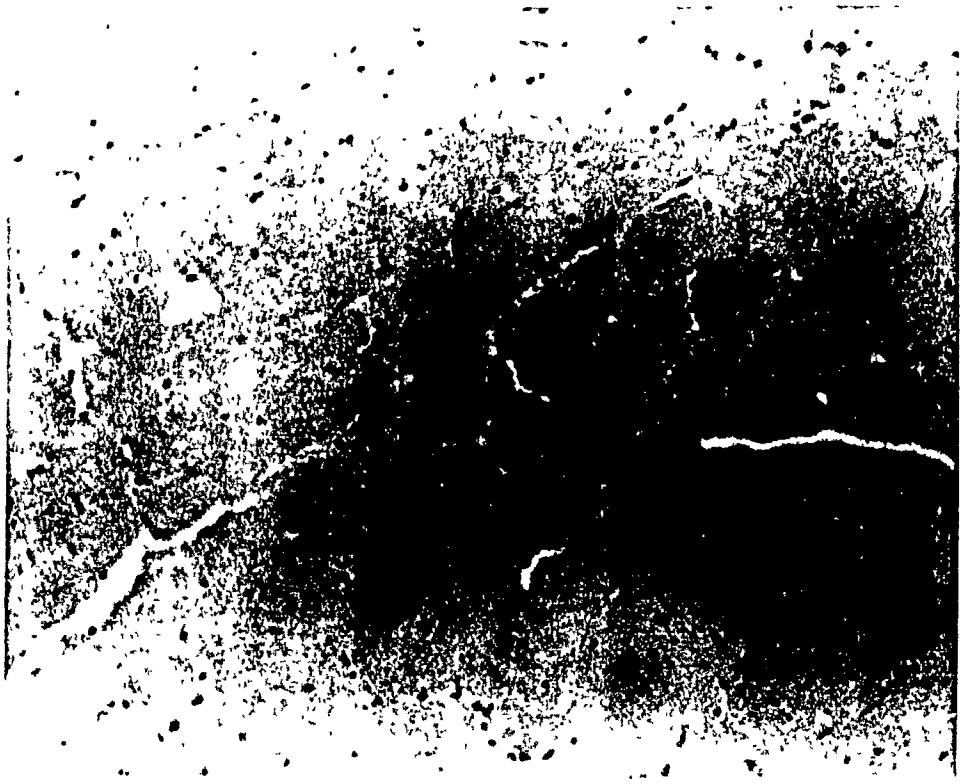


Fig 24 Muscle sample taken from proximal biceps at 8 days post injury Note minimal damage of cells in this area.

VESSELS The evaluation of the larger vessels from the Group II animals was limited to the samples examined from the eighth day following electrical injury. Microscopic analysis of the brachial, radial and ulnar vessels was completed bilaterally in animals sacrificed preburn and at 6 hours, 6 and 10 days following electrical injury from the ANGIOGRAPHY GROUP III animals Smaller vessels however, seen at the times of muscle biopsies, were examined serially post burn.
The radial, ulnar and anterior interosseous arteries were all thrombosed distally to approximately the level of demarcation between grossly viable and nonviable muscle Grossly thrombosed vessels showed an absence of endothelial cells and mild to moderate cellular swelling and degeneration within the tunica media (Fig. 25). Smooth muscle nuclei showed mild pyknosis. By the sixth day following injury, the vessel wall had undergone necrosis with dissolution of nuclei and loss of normal architecture in the vessel wall layers Remnants of the internal and external elastic membranes were evident up to the tenth day in an otherwise necrotic vessel

The transition zone between thrombosed and patent vessel in the ulnar and radial arteries revealed marked cellular swelling, vacuolization and edema in the tunica media An absence of endothelial lining associated with polymorphonuclear cell carpeting was evident at the transition from affected to normal vessel (Fig 26). These findings were consistently present post injury with a marked arteritis in the distal patent vessels on the tenth day Findings within the patent arteries in the forearm were not uniform A

mild to moderate arteritis was inconsistently found, being more prevalent in regions closest to thrombosed vesses. These areas were characterized by thickening and vacuolization of the tunica media, mild cellular infiltration, thickening of the internal elastic lamina and loss of intimal lining. They were found segmentally however, with normal and abnormal areas seen in the forearm and cubital fossa region. In the arm above the region of distal biceps and triceps necrosis, the vessels appeared normal

The patent ulnar artery showed a peculiar zone of injury in the cubital fossa. For a variable length of 2 to 4 cm, there was a mild to moderate arteritis characterized by loss of endothelium, mild infiltration of PMN's and vaculization in the media. This injury site corresponded to the segmentally narrowed areas as seen on angiography. The smaller vessels are completely thrombosed in areas of grossly necrotic muscle. In areas of the forearm with grossly viable muscle , patent arterioles and capillaries are present but in reduced numbers and intermixed with damaged muscle fascicles. These small vessels show a slight increase in density within areas of marked cellular infiltration

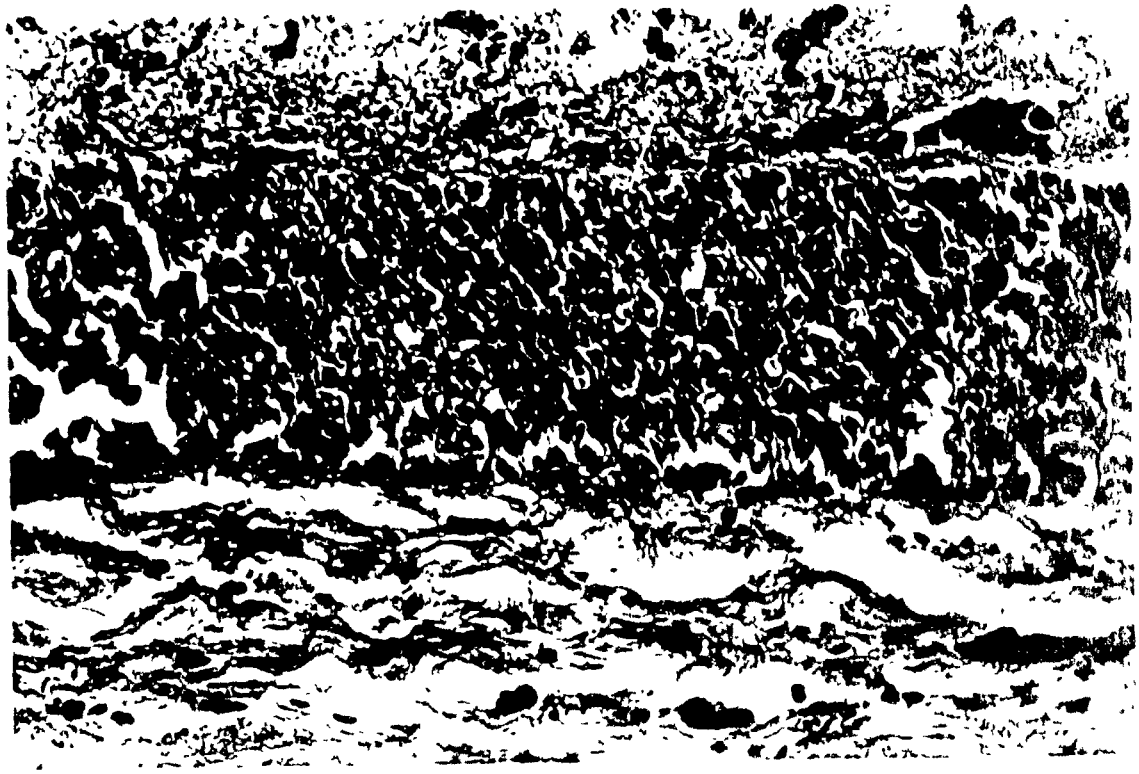


Fig. 25 Longitudinal micrograph from ulnar artery taken from thrombotic region in the distal forearm. Note marked degeneration of tunica media (lumen at top)

Fig. 26 Longitudinal micrograph of same ulnar artery immediately proximal to thrombosis in distal forearm. Note mild cellular infiltration of tunica media and loss of intimal lining

NERVE As with the vasculature, histological assessment was limited to a single evaluation at 8 days post injury. Nerves distal to the line of demarcation between viable and nonviable muscle were grossly friable and necrotic, and were not assessed microscopically. In the forearm, marked damage was sustained to the radial, median and ulnar nerves. Schwann cell degeneration was widespread with pyknotic nuclear remnants interspersed with degenerating cellular fragments (Fig 27). Axons were also severely damaged showing demyelination and degeneration at this level The perineurial and epineurial fascicular sheaths however remained intact Mild cellular infiltrate was evident at this level.
More proximally at the level of the cubital fossa, damage appeared less severe with the majority of axons and myelin sheaths intact. As serial sections were examined more proximally in the arm, the nerves appeared normal in structure (Fig 28). The nerves appeared normal beginning at the mid arm level

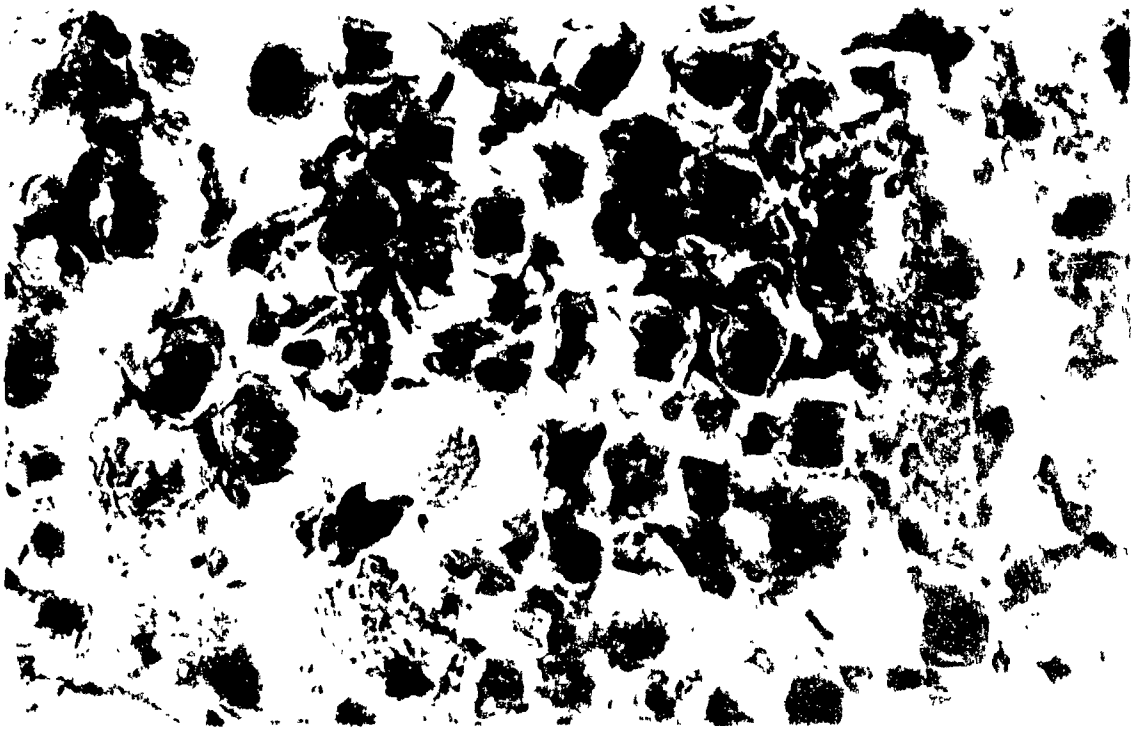


Fig. 27 Cross section of median nerve in distal forearm harvested 10 days post injury showing extensive degeneration and architectural disorganization.

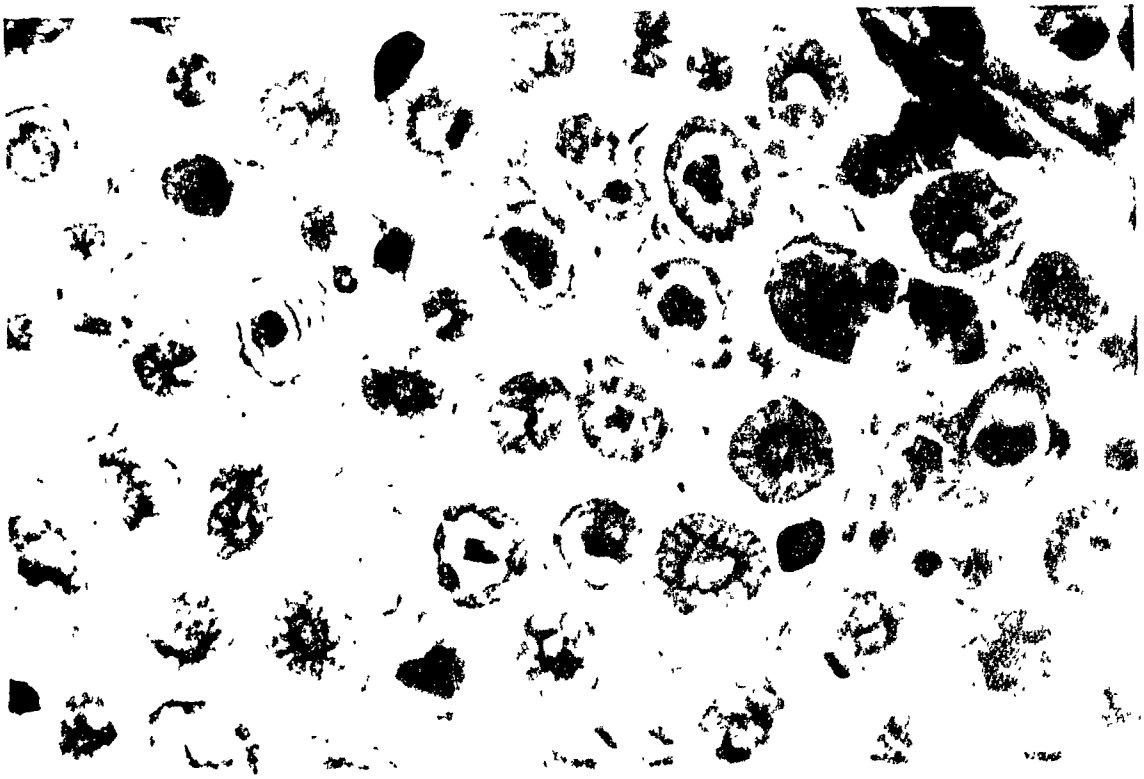


Fig 28 Cross section of same median nerve from the midforearm level revealing minimal disruption of neural structures

GROUP III: ANGIOGRAPHY

A total of 20 extremities were available for this study. Twelve extremities were used for pilot studies in order to determine a reliable protocol for obtaining these difficult films consistently. Eight extremities were used for the chronic study and provided pathological films pre-injury, at 6 hours, then at 2, 6, and 10 days post injury Normal angiograms were available from all 20 extremities and revealed a vascular supply to the arm and forearm which correlated well to that seen in the human upper extremity

The first pathological angiograms taken at 6 hours post injury demonstrated marked changes and were similar in all animals. The interface between patent and nonpatent vessel was distinct and related closely with the line of demarcation between viable and nonviable muscle. The ulnar and radial vessels were abruptly thrombosed in areas corresponding to grossly nonviable tissue and demonstrated a preocclusive dilatation in most instances (Fig 29). The ulnar artery was patent in the region of the forearm flexor bellies and appeared to be their dominant blood supply However, the radial artery was patent for only 2 to 3 cm beyond its origin and was thrombosed in or just distal to the cubital fossa Large vascular trunks showed segmental narrowing or "beading" Nutrient arteries were dramatically reduced in number in the forearm and were present only in the area of the grossly viable flexor bellies In the arm, the demarcation between patent and nonpatent nutrient vessels was distinct Proximal to this,

the brachial artery, large muscular branches and nutrient arteries appeared normal.

Serial films taken on days 2, 6 and 10 post electrical injury revealed a significant increase in the density of perfused small vessels in the forearm flexor region (Fig. 30). This increased vascularity or "blushing" was also apparent along the nutrient vessels in the distal upper arm. The cutaneous vessels of the forearm increased in density significantly by the eighth day The segmental narrowing in the cubital fossa region resolved almost completely with only slight residual narrowing present by day 10 (Fig. 31) Detailed dissections demonstrated a high degree of correlation between the gross margins of muscle injury and perfused regions as seen on angiogram.

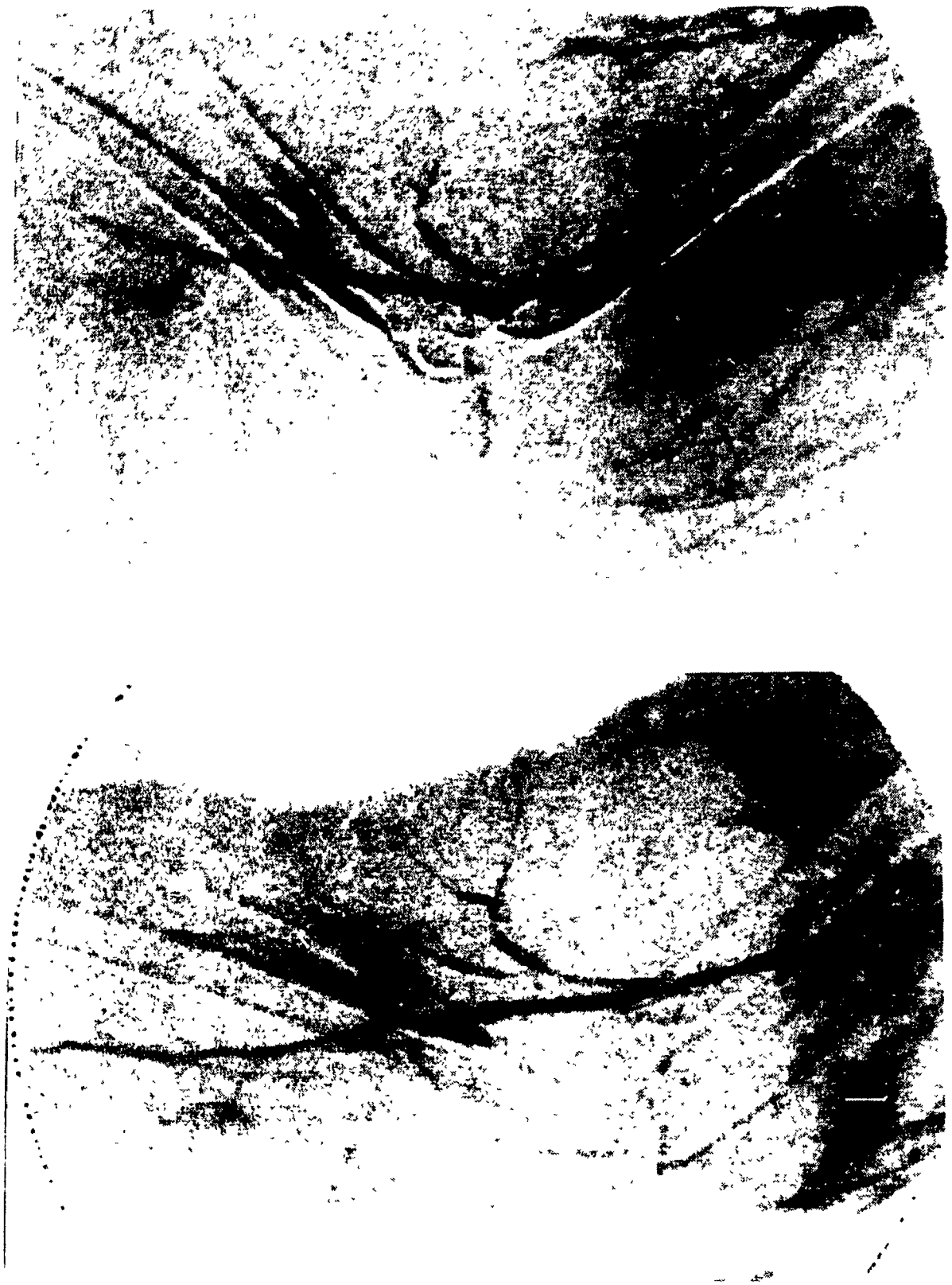


Fig. 29 Serial digital subtraction angiograms taken 4 hours (above) and 2 days (below) post burn. Note minimal change in pattern of vascular injury.

Fig. 30 Angiogram taken 10 days after electrical injury. Note marked increase in small vessel patency adjacent to necrotic areas

Fig. 31 Enlargement of right cubital fossa 4 hours (above) and 10 days (below) after injury. Note the significant luminal narrowing of the proximal ulnar artery immediately post injury. Serial films reveal a dramatic resolution of arterial spasm as evidenced by normal patency on the tenth day.

GROUP IV: TEMPERATURE RECORDING

The temperature recordings for the 6 extremities used in this experiment are shown in Table 1 and Figure 32. Four significant findings were found in this experiment. Firstly, the temperature elevations in an individual muscle (i.e. FCU) do not necessarily decrease from distal to proximal. Previous temperature studies revealed temperatures at the wrist in excess of 100°C and were not repeated due to frequent destruction of the thermistors from the high temperatures. In this study, temperatures in FCU were low in the central aspect and higher at its insertion onto the humerus. Therefore, the temperature pattern in the forearm muscle(s) appears to be high distally and proximally, with lower, more biologic temperatures in the central, viable portion of the muscle. Secondly, temperatures taken at equal distances from the wrist on opposite sides of the forearm revealed entirely different findings. In the brachioradialis, the high-low-high pattern of injury does not exist. At the midpoint of the forearm, significantly higher temperatures were found on the radial aspect of the forearm as compared to the ulnar side. In all cases, the BR was completely nonviable as assessed on the day of injury and at the time of final evaluation. Thirdly, the cubital fossa, which repeatedly sustained significant injury, was found to have elevated temperatures measured from probes in the forearm flexor insertion (FCU) and distal biceps. As no arcing was observed in our studies, the reasons for these temperature elevations appear to contradict the present-day understanding of "skip" lesions. And finally, the biceps muscle was

found to have significantly higher temperatures in its distal, nonviable portion than in the viable portion, only 2 cm proximal to the line of demarcation. All temperature values decreased to baseline at 15 minutes post injury.

Table I. Temperature elevations in the primate upper extremity

Probe site #	FCR 1	FCUa 2	FCUb 3	FCUc 4	FCUd 5	BICa 6	BICb 7
Experimental Animal							
GMT1	54	51	46	44	49	53	47
GMT2	59	*	42	39	61	64	47
GMT3	51	43	41	41	52	61	43
GMT4	61	46	40	39	54	59	46
GMT5	52	46	43	40	50	51	44
GMT6	65	*	47	43	57	66	54
Mean T °C	57	46	43	41	54	59	47

*Based on hand-to-hand contact subject to 40 KJ (V = 3525, I = 4.5, T = 2 5)

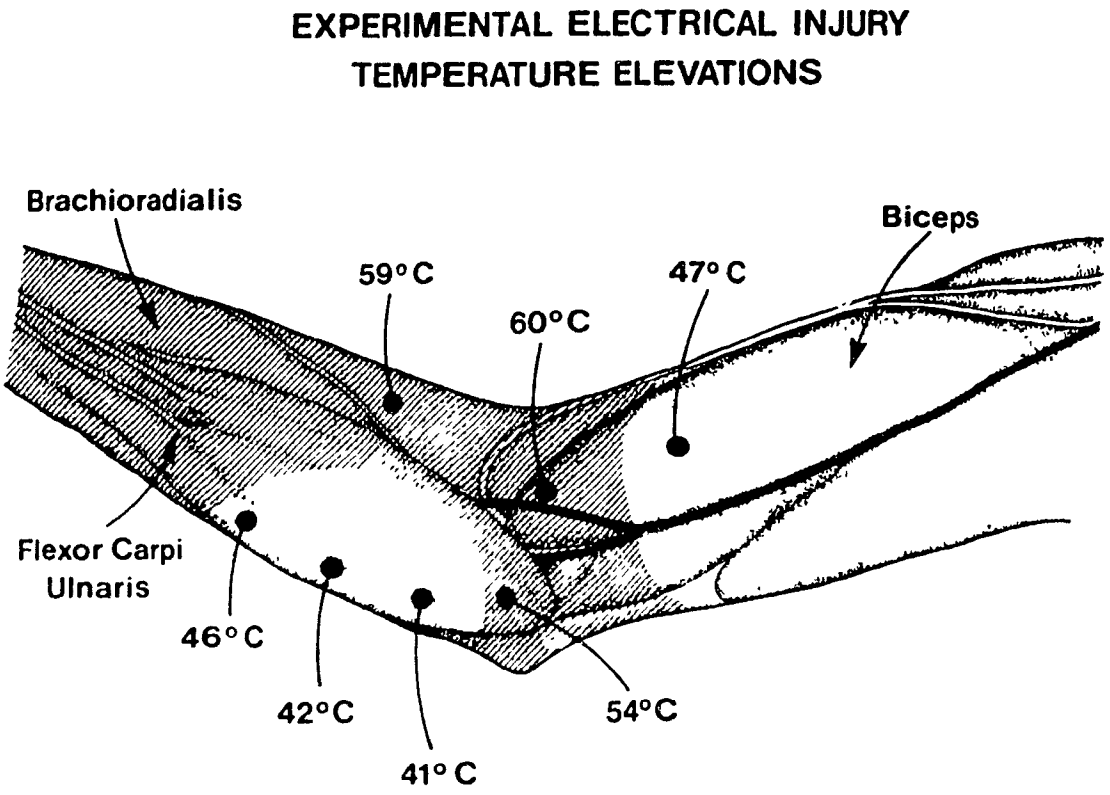


Fig. 32 Average temperature values generated in the upper extremity (n = 6)

GROUP V: PERIPHERAL NERVE INJURY

A total of 4 extremities were used in the experimental group and 4 in the control group. The injury to the ulnar nerve was consistent in all extremities The nerve trunk appeared grossly normal throughout its length, but by the fourth day a distinct differentiation occurred at the level of the cubital fossa Distally, the nerve showed gross evidence of necrosis, becoming friable and structureless Proximally, the nerve became edematous from the time of injury to approximately four days post injury Perineural edema and fibrous thickening was evident by the tenth post injury day. Sequential ulnar nerve recordings were completed on the experimental group of three extremities at times 1, 4, 12 and 24 hours and then on days 4 and 10 post injury. The findings in all animals were consistent and revealed a complete loss of conduction just proximal to cubital fossa The level of conduction loss corresponded to the line of demarcation between nonviable and viable muscle in the surrounding area This level of conduction loss did not change in the post injury period and was found to be at the same levels at times 1 hour, 24 hours and 10 days post injury. Average conduction velocities along the proximal nerve trunk were found to be affected uniformly in both the experimental and control groups (Table 2 and 3) Average conduction velocities at times 1 hour, 24 hours and 10 days were 63 3 $\pm$ 6 6 m/s, 64.4 $\pm$ 9 2 m/s and 66 0 $\pm$ 11 2 m/s respectively These values were not significantly different from each other nor from the average control of 64 5 $\pm$ 7.5 m/s

Table 2: Data from ulnar nerve recordings of experimental group (n = 4).

	1 HOUR		4 HOUR		12 HOUR		24 HOUR		4 DAY		10 DAY	
DISTANCE	ONSET	PEAK	ONSET	PEAK	ONSET	PEAK	ONSET	PEAK	ONSET	PEAK	ONSET	PEAK
ANIMAL T3												
43 mm	0.159	0.238	0.159	0.238	-----	-----	0.198	0.317	0.198	0.476	0.159	0.317
52	0.222	0.286	0.190	0.254	-----	-----	0.238	0.397	0.238	0.556	-----	-----
60	0.270	0.349	0.254	0.302	-----	-----	0.278	0.397	0.397	0.635	0.357	0.595
68	0.333	0.381	0.317	0.365	-----	-----	0.357	0.476	0.635	0.754	0.635	0.754
83	0.413	0.508	0.413	0.508	-----	-----	0.476	0.595	0.794	1.030	0.794	1.070
ANIMAL T4												
45 mm	0.357	0.556	0.397	0.556	0.278	0.437	0.238	0.437	0.238	0.397	0.238	0.397
55	0.437	0.635	0.476	0.675	0.397	0.627	0.317	0.556	0.317	0.516	0.317	0.516
64	0.595	0.794	0.635	0.794	0.556	0.675	0.476	0.635	0.397	0.595	0.357	0.595
73	0.794	0.952	0.754	0.952	0.714	0.833	0.635	0.794	0.556	0.754	0.515	0.714
84	0.833	1.110	0.833	1.150	0.873	1.030	0.754	0.913	0.754	0.923	0.714	0.873
ANIMAL T5												
39 mm	0.238	0.516	0.278	0.516	0.278	0.516	0.278	0.516	0.278	0.437	0.278	0.437
47	0.317	0.675	0.317	0.635	0.357	0.595	0.317	0.635	0.317	0.556	0.437	0.595
57	0.476	0.833	0.515	0.754	0.476	0.714	0.556	0.794	0.437	0.675	0.476	0.675
65	0.635	0.992	0.714	0.952	0.635	0.873	0.714	0.913	0.556	0.794	0.635	0.873
74	0.833	1.190	0.912	1.190	0.794	1.110	0.873	1.150	0.675	0.913	0.833	1.030
ANIMAL T6												
38	0.238	0.516	0.278	0.595	0.317	0.675	0.278	0.516	0.278	0.476	0.278	0.516
46	0.317	0.635	0.476	0.714	0.556	0.873	0.397	0.556	0.317	0.556	0.397	0.635
55	0.437	0.714	0.635	0.873	0.794	1.030	0.516	0.714	0.437	0.714	0.556	0.794
63	0.556	0.873	0.714	0.952	0.873	1.150	0.595	0.794	0.556	0.754	0.675	0.913
73	0.794	1.270	0.873	1.390	1.190	1.590	0.794	1.030	0.754	0.952	0.913	1.190

Note

Distance = distance in mm from stimulating electrode in axilla to recording electrode along ulnar nerve.

Onset = time in msec on onset of compound action potential.

Peak = time in msec to peak of compound action potential.

Table 3: Data from ulnar nerve recordings of control group (n = 4).

	BASELINE		3 HOUR		9 HOUR	
DISTANCE	ONSET	PEAK	ONSET	PEAK	ONSET	PEAK
CONTROL 1A						
40 mm	0.238	0.556	0.278	0.516	0.278	0.475
50	0.317	0.635	0.397	0.635	0.317	0.635
60	0.476	0 833	0.516	0.754	0.476	0.754
68	0.714	0.952	0.635	0.873	0.595	0.873
75	0.794	1.070	0.794	0.952	0.714	0.992
85	1.030	1.230	1.030	1.190	0.873	1.070
94	1.110	1.350	1.190	1.350	1.070	1.230
102	1.230	1.510	1.230	1.470	1.230	1.390
CONTROL 1B						
45	0.278	0.516	0.278	0.516	0.278	0.516
53	0.397	0.635	0.397	0.635	0.397	0.635
64	0.516	0.794	0.516	0.833	0.516	0.794
74	0.754	0.952	0.754	0.992	0.714	0.913
85	0.992	1.190	1.030	1.190	0.913	1.070
98	1.270	1.510	1.190	1.390	1.070	1.270
108	1.430	1.630	1.350	1.630	-----	-----
CONTROL 2A						
33	0.278	0.437	0.278	0.437	0.238	0 397
42	0.357	0.595	0.357	0.595	0 317	0.556
51	0.476	0.794	0.437	0.794	0.437	0.635
62	0.675	0.952	0.635	0.992	0.635	0.794
73	0.883	1.150	0.794	1.150	0.675	0.873
CONTROL 2B						
32	0.278	0.476	0.278	0.476	0.278	0 476
40	0.357	0.556	0.357	0.595	0.357	0.556
50	0.476	0.714	0.476	0.794	0.397	0 675
63	0.714	0.952	0.754	1.030	0.675	0.913
75	0.833	1.190	1.030	1.230	0.873	1 110

DISCUSSION

CUTANEOUS INJURY

Patterns of cutaneous injury following electrical injury are poorly documented in the literature and are often described simply as extensive, circumferential or full thickness. Clinical investigators have formulated theories regarding the patterns of damage seen in the skin Typical low voltage electrical burns showed an abrupt transition from normal to burned skin, with microvesicles in the epidermis and layer separation at the deeper epidermis Quinby et al assessed microscopic changes as a clinical index for primary excision in high tension electrical burns [41]

Traditionally, extensive damage seen at the wrist, cubital fossa and axilla has been attributed to external high-voltage current arcing [8,33] Burke et al speculated that thin, moist skin accounted for external arcing leading to cutaneous damage as the current exits the deep structures [7] Skip lesions at sites other than a skin crease are believed to represent thermal injury secondary to external arcing. However, it is evident from our studies that preferential damage to the cubital fossa results from two factors: 1) an increase in current density through the small cross-sectional diameter of this region and 2) the high resistance to flow through the predominantly tendinous and bony configuration of the cubital fossa It is our opinion that the lesions found at the wrist and cubital fossa are formed by the same mechanism and are a thermal injury produced from the underlying

structures and not the result of arcing. However, lesions found in the axillae are an enigma. In contrast to reproducible patterns of injury in the remainder of the forearm, axillary lesions are infrequent and not uniform. They are characteristically small superficial burns confined to the skin within the anatomic confines of the axillary fossa. Their exact location and number cannot be predicted. Careful examination of the underlying muscular and tendinous structures of the axilla fail to reveal any damage either grossly or microscopically The resistance to current flow through the skin may be diminished due to the presence of moisture on the overlying skin in this region As a result, small amounts of current may pass through small localized areas of opposed skin

MUSCULAR INJURY

Patterns of muscular injury are poorly documented in both the clinical and experimental literature. Luce[33] and Mann[35] have reported more extensive damage to deeper and periosseous muscles within the upper extremity This pattern of injury is ill defined in the experimental studies completed on rats, rabbits and swine However, in the 100 experimental burned extremities which we studied, the overall pattern of muscular injury was consistent as both superficial and deep muscles showed more proximal damage on their deeper surface The resulting pattern of injury revealed extensive muscle damage, often 6 to 8 cm proximal beneath viable appearing muscle, resulting in a "cone" of injured muscle

Microscopic changes in skeletal muscle following electrical injury have been described clinically by Quinby et al.[41] Areas of total muscle necrosis revealed fragmented and disrupted myocytes and hypereosinophilic fibers. In areas adjacent to nonviable regions viable muscle showed normal myocytes, while fibers adjacent to necrotic cells had pyknotic nuclei. These observations were confirmed in our primate model In areas appearing nonviable immediately after the electrical injury, uniform coagulative-type necrosis was evident in association with thrombosis of all vessels. This damage progressed to complete dissolution of intracellular structures in the absence of any cellular infiltration This pattern of injury is indistinguishable from that seen following a thermal insult to skeletal muscle Temperature elevations in the forearm were investigated in the primate model Average recorded temperatures as shown in Figure 32 correlate well with the observed pattern of injury. In transitional areas, necrotic muscle fibers were intermixed with normal cells The relative density of necrotic fibers was greatest immediately adjacent to nonviable zones and progressively decreased with increasing distance from the nonviable areas The greatest number of damaged muscle cells were found in the periphery of the individual fascicles Explanations for this pattern of injury are uncertain but the effects of electric currents on membrane structure, permeability and depolarization would be valuable to clarify these findings

Several investigators postulated that progressive necrosis of tissues occurs but few attribute it to skeletal muscle. In 1957, Muir suggested that bacterial infection might cause progressive necrosis [37] Wilkinson found patchy areas of myonecrosis and attributed it to decreased vascularity from small vessel thrombosis.[59] Lewis confirmed the presence of progressive necrosis in the soft tissues but did not venture a possible etiology.[32] Robson believed the periosseous muscle necrosis seen following high voltage injuries is secondary to the delayed release of arachadonic acid metabolites from the periosseous musculature [42] The remaining authors speculated that progressive necrosis is due to a delayed or ongoing vascular thrombosis Our experiments did not demonstrate any progressive necrosis of the skeletal musculature Grossly, the demarcation between viable and nonviable muscle remained unchanged. Muscle which initially appeared viable and bled when stabbed, remained viable up to the 10th post injury day. Light microscopy revealed a varied amount of cellular infiltration surrounding healthy muscle cells in areas adjacent to nonviable regions. Frequent sequential analysis revealed a consistent anatomically determined electrical injury whose true extent could be determined immediately post injury Extensive muscle damage was located deep in the forearm and arm; areas which the surgeon would not explore at the time of routine fasciotomy.

VASCULAR INJURY

Clinically, Jaffe et al described the loss of vessel endothelium and marked damage to the media of the vessel wall following electrical injury.[24] The mechanism of injury to the vascular system is unknown, but Skoog and others suggest that vascular damage is caused primarily by heat and not related specifically to electricity.[50] He stated that injured vessels prompt thrombus formation, resulting in ischemic necrosis of surrounding tissues. Permanent narrowing of vessels due to scarring was noted up to 3 months following injury Ponten has shown evidence of arteritis and aneurysmal formation in a detailed case report of upper extremity electrical injury.[40] Baxter reported that progressive vascular necrosis may occur up to 8 to 10 days following the initial injury, leading in some cases to delayed haemorrhage.[4] In the experimental rat model, Hunt found immediate vascular occlusion in the hindlimb vessels corresponding to cutaneous and muscular injury [21] No evidence of progressive vascular occlusion was identified Using the same model, Buchanan et al reported similar findings of immediate vascular thrombosis up to the level of limb destruction and no progression for 90 days post burn clinically.[6] Hunt and other investigators have described several angiographic patterns complete vascular occlusion associated with massive tissue necrosis, arterial 'pruning', 'skip' areas, 'beading', decreased nutrient vessels, and partial vascular occlusion.[20,21]

In our studies, serial digital subtraction angiograms revealed characteristic lesions in the upper extremity electrical burn model·

1) thrombosis of the major vessels in the forearm at levels corresponding to obvious muscle damage, 2) a marked decrease in the density of small nutrient vessels in the forearm, 3) a segmental narrowing of vascular trunks in the cubital fossa, and 4) loss of nutrient vessels at the level of necrosis in the distal biceps and triceps. Careful evaluation of serial angiograms over a ten day period revealed little change in this pattern of injury. Areas of segmental narrowing resolved in many areas although not in all. These persistent areas of vascular narrowing may be critical areas of concern for the clinician. With microscopic evidence of arteritis in these areas even at day 10 post injury, they may serve as the nidus for late thromboses. However, this was not observed in our model, leading us to speculate that late vascular occlusion is associated with a secondary initiating event, such as decussation from debridement or infection. Of great significance was the relation between the level of vascular occlusion, and the surrounding muscle damage The ulnar artery was consistently patent to the level of deep muscle injury in the distal forearm. However, extensive muscle damage was found more proximally within the forearm, in the cubital fossa and up to the mid-arm level This difference in the level of injury between skeletal muscle and vessels was represented by distances of approximately 10 to 12 cm Thus, it is important to differentiate between occlusion of the major vessels, and pruning of the nutrient artery, the latter being a more accurate indicator of muscle damage These findings contradict those of previous investigators who state that the most proximal damage is

found in the vascular and peripheral nervous system.

PERIPHERAL NERVE INJURY

Pioneering experimentation of electrical damage to peripheral nerves was conducted by Ugland on sciatic nerve preparations in cats [56] Low voltage studies indicated that changes in nerve function were reversible to a great extent and changes observed were most likely caused by direct neuronal damage induced by the current itself. In clinical publications, Ugland stated that persistent loss of nerve function was a characteristic of high voltage injuries, with damage being primarily from the thermal effects of current passage. Subsequent fibrosis and scar formation resulted in only a partial return of protective sensation. As noted by Skoog, partial recovery was often noted, but permanent damage was most common.[50] Etiologically, some investigators felt nerve damage is caused by a pure electrical effect related to the passage of electricity through a highly ionic medium.[14]

From our experimental investigations, several findings are notable First, there was a complete loss of conduction distal to the cubital fossa which did not show any evidence of recovery by the tenth post burn day. Its location was adjacent to muscle regions where temperatures of over 60 ^{o}C were recorded. One may postulate that the nerve had sustained a thermal injury at this level and that recovery of conduction ability was impossible. Second, the demarcation between the non-conductive and conductive areas was distinct without evidence

of a transition zone. Third, the level of conduction loss within the proximal nerve did not progress over the ten days post injury. No previous mention of progressive necrosis in the peripheral nervous system has been made in the literature and our data suggest that none occurs.

The level of injury within the nerve and its relation to the surrounding structures is important. Nerve conduction studies show that the ulnar nerve is functional to the level of the demarcation between viable and nonviable muscle in the distal arm. Deep muscle damage to the triceps and biceps appears to extend more proximally, although only over a short distance. The ulnar artery is patent to the midforearm level, i.e. 7 to 9 cm distal to the level of nerve conduction loss. These findings are again in direct contradiction to the literature which suggests that neurovascular structures are always affected at comparable levels.

PROGRESSIVE NECROSIS

The concept of "progressive necrosis" dominates the surgeon's treatment of high voltage electrical injuries. The desire to debride radically and cover the wound early is tempered by the realities of liquefaction and infection which would follow inadequate debridement. In hundreds of cases, numerous surgeons have concluded that progressive necrosis does indeed occur. Therefore, how do we explain our experimental findings which show unequivocally that progressive necrosis does not occur?

In a typical high-voltage electrical burn, the patient usually reaches a major hospital within several hours where resuscitation is followed by extensive fasciotomies. On Day 2, a reassessment is done in the operating room which often involves amputation of distal mummified parts and radical debridement proximally until viable tissue is identified It is upon return to the operating room at Day 4 that the spectre of progressive necrosis is invariably seen - tissues which were obviously viable on Day 2 are now necrotic on Day 4. What is the explanation? At least five factors are responsible: (1) fasciotomy incisions are often limited and do not pass from carpal tunnel to axilla as they should, (2) identification of muscle damage is often a gross determination rather than assessment of individual muscles, out of fear of injuring the nutrient vessels and/or motor nerve branches, (3) debridement is done from a "top-down" exposure, (4) damage to the deep muscles is often considered equal to that of the superficial

muscles, and (5) the methods of assessment are crude subjective techniques. At this point, the clinician is surprised that necrosis exists more proximally and extends the debridement. In the forearm, patent nutrient arteries may be easily transected which can produce ischemic necrosis. Thus, on Day 6, it is evident that an above-elbow amputation is now necessary at the juncture of lower third - middle third of the humerus The wound is left open, the surgeon is confident that early closure will be possible. However, on Day 8, it is evident that progressive necrosis of the muscles has occurred again. Further, debridement is then carried out and infection may occur due to contamination, ischemia, and deficient wound coverage. With this typical scenario, one is left to conclude that high voltage electrical injuries are more often characterized by progressive awareness and progressive production than by progressive necrosis. Unquestionably, tissue damage is present immediately at the time of injury and is of far greater extent than generally accepted or easily defined. From the hand to midforearm, the level of mummification is obvious, but the discrepancy between skin and muscle damage is not. In cross-section, there is far more extensive damage on the deep surface of the individual muscle despite its viable visible superficial surface Longitudinally, one often progresses from nonviable to viable to nonviable portions within the same muscle: i.e. flexor carpi ulnaris. Within the arm, the dilemma is even greater. After all, how can a surgeon tell that a bicep or tricep muscle has a central core of necrosis when the overlying skin is viable and the

muscle on its superficial surface appears externally to be normal to every subjective criteria available? Histologically, the vessels supplying even the forearm flexors are patent and obviously the muscle will bleed on debridement. It is only as debridement progresses up the arm that the surgeon becomes aware of the original injury. Transection of nutrient arteries, release of toxic mediators or secondary infection may lead to additional injury. Ultimately, we have concluded that progressive necrosis is not an inherent component of electrical injury nor should it dominate surgical management.

CONCLUSIONS

1. THE PRIMATE UPPER EXTREMITY ELECTRICAL INJURY MODEL HAS BEEN SUCCESSFULLY CREATED

Although demanding, this model demonstrates many of the needed characteristics of the ideal model for electrical injury studies, i.e. it is standardized, reliable, reproducible, allows easy and accurate data recording and, above all, has anatomical relationships identical to those in human extremities allowing direct clinical correlation.

2 PROGRESSIVE NECROSIS DOES NOT OCCUR FOLLOWING ELECTRICAL INJURY

Also, it should not play a dominant role in patient management. Our investigation of the effects of electrical injury on a model demonstrating a high degree of clinical similarity using gross observation, light microscopy, temperature, angiography and nerve conduction studies have failed to support the concept of progressive tissue injury.

3 TISSUE INJURY EXTENDS MORE PROXIMALLY ON THE DEEP SURFACE OF BOTH INDIVIDUAL MUSCLES AND BETWEEN LAYERS.

This is a significant finding because this pattern of injury is consistent and can be predicted with accuracy in the upper extremity. The deep injury found beneath viable appearing muscle

and skin probably accounts for the "delayed necrosis" most often cited by treating physicians. The etiology of this phenomenon may be related to the nature of current passage through the anatomic structures of the forearm or possibly due to the slower heat dissipation of the poorly conductive bone located centrally.

4. PATTERNS OF ELECTRICAL INJURY IN COMPOSITE TISSUES ARE ANATOMICALLY DETERMINED.

For a given energy and site of contact, identical patterns of injury will likely result if the anatomy is the same As the amount of energy delivered is increased, progressively more proximal damage will occur. This principle has allowed us to propose and demonstrate a reliable, reproducible electrical injury model in the primate. During the acute phase of the project, injuries were produced in separate animals while the total energy delivered was varied. Although progressively more extensive injuries were produced at higher energies, the injuries were identical for each energy level tested. A standard 40 kilojoule injury was chosen for the chronic phase of the project. This reproducible injury allowed easy and unobstructed evaluation of skin, muscle and neurovascular structures and showed a high degree of clinical correlation.

5. THERE EXIST IN THE FOREARM SPECIALIZED REGIONS OR "CHOKE" POINTS

In these areas, decreased cross-sectional areas and highly resistant tissue composition result in increased heat production and more severe tissue injury. The wrist, cubital fossa and possibly the axilla are examples of such regions. This significant finding directly contradicts the current literature. Consistently found in almost all published descriptions of electrical injuries is the so called, "skip lesion" which authors attribute to external arcing of electrical energy. Examination of electromagnetic theory however casts doubt on this well accepted theory. Two important criteria for external arcing are absent. First, for energy to "jump" from the wrist to the elbow, the flow of current must encounter and bypass a segment of considerably higher impedance The forearm region, with its predominant muscular composition, has an impedance which is less than the wrist and elbow These areas are composed of highly resistant bone and tendon Second, extremely high energies are required for electrical energy to travel externally (in the order of 2 million volts/m) and these energies are certainly not found with the more common injuries encountered with distribution lines. Arcing of current does not occur at sites other than the points of entry and exit

6. THE PREDOMINANT MECHANISM OF TISSUE INJURY APPEARS TO BE THERMAL.

Although we are unable to confirm a thermal etiology for the entire electrically injured tissue, it appears from our temperature recordings that significant temperature elevations consistently occur in regions destined to necrose Unknown at this time are the direct effects of current passage, toxic mediators or infection on ultimate tissue survival

7. DIAGNOSTIC AIDS ARE INVALUABLE FOR TISSUE ASSESSMENT

Diagnostic aids, such as angiography and nerve conduction studies, are essential in the assessment of tissue injury Objective tests, including tissue impedance have demonstrated the ability to accurately predict tissue viability in laboratory studies and are currently being investigated by other researchers in our laboratories.

CLINICAL CORRELATIONS

Despite the continual introduction of new techniques in burn care, the fundamental concepts of electrical burn management have scarcely changed in fifty years. Then, as now, periodic evaluation of damaged tissue and sequential debridement was the clinical course routinely observed. As early as 1929, Wells recognized the unique pathological aspects of electrical injury and accordingly advocated, what was, for the first time, a radical method of treatment - immediate resection and skin grafting.[58] Wells however, was also the first to emphasize the limitations of this approach Where the line of demarcation was not sufficiently clear, he advised discretion Without experimental validation, the concept of early definitive coverage was subordinated to the conventional medical reliance on clinical experience.

Clinicians have continued over the years to report on the advances in techniques that have, in their experience, led to adequate electrical burn management The fundamental concepts, however, remain unchanged - and basically conservative

The central clinical problem in electrical injury is the devastating extent of composite tissue damage The technical advances in distant transfer of compound tissues for reconstructive purposes has made early definitive coverage of electrical burns an exciting possibility Unfortunately this clinical option could not be confidently pursued until experimental studies defined the etiology of electrical injury and established reliable diagnostic methods for predicting the extent of tissue damage. Without an understanding of

the pathophysiology of electrical injury based on definitive experimental research, the clinical promise of reconstructive surgery for advances in burn management, until now, could not be realized. The majority of electrical injuries involve the upper extremity, often leading to marked functional loss or amputation. Concurrent with ongoing experimentation, electrically injured patients were assessed and treatment clinically. Thus, the opportunity existed to correlate clinical findings with observations and to modify our management protocol. Our series was comprised of five high voltage electrical injuries involving 8 extremities. Three patients had representative patterns of injury and are discussed in detail. This paper will attempt to answer three critical questions. Is there a predictable pattern of injury in high voltage electrical injuries of the upper extremity? Does progressive necrosis occur? How should these patients be managed during the acute period?

CASE NO. 1

P P , a 42 year old male, contacted a 12,500 volt, 800 A transformer while painting in a restricted shed and fell 10 to 15 feet from scaffolding On admission to our emergency room, the patient was found to have a circumferential injury of the left hand extending proximally to the mid forearm level (Fig.33). There was a "skip area" of skin, overlying the forearm flexor bellies, and a circumferential injury at the cubital fossa The patient had normal sensation above the cubital fossa and a palpable brachial pulse. There were small

Fig. 33 (above) High voltage electrical injury with mummification to the midforearm level. Note near circumferential injury at cubital fossa "choke point" (below) High voltage injury to primate upper extremity revealing anatomically similar pattern of injury

superficial "skip" lesions in the left axilla. The other entry/exit point was a large area on the right lateral portion of the thoracoabdominal wall (Fig 34) Preoperative digital subtraction angiogram revealed complete occlusion of the ulnar artery in the proximal 1/3 of the forearm, while the radial artery thrombosed just distal to the cubital fossa There was no blood flow beyond the midforearm level Tetanus prophylaxis and preoperative antibiotics were given

The patient was taken to the operating room and the hand, forearm and upper arm were decompressed. The fasciotomy incisions extended up to the axilla (Fig. 35) The hand appeared nonviable and the forearm flexor muscles were necrotic superficially to the midforearm level. The brachioradialis appeared nonviable in its entirety. The forearm extensors appeared severely damaged as visualized through the fasciotomy incisions along the dorsal forearm. There was evidence of injury to the proximal aspects of the forearm flexors and the distal portions of the biceps and triceps The wounds were dressed in a sterile fashion and the patient returned to the intensive care unit. Forty-eight hours following injury, the patient underwent further debridement of the left forearm. Obviously necrotic skeletal muscle was removed The superficial forearm flexors (FDS, FCU, PL, FCR) sustained extensive damage up to the midforearm level as well as proximally in the cubital fossa (Fig 36). Interestingly, the forearm flexors were damaged more proximally on their deep surfaces. The brachioradialis was nonviable The deep forearm flexors (FDP, FPL) were viable only in a

Fig. 34 (above) Exit lesion on the lateral thoracoabdomen (below) This area has been excised full thickness and skin grafted.

Fig. 35 Complete upper extremity fasciotomy similar to that performed in the primate was done to decompress all muscle groups.

Fig. 36 Examination of superficial and deep muscle layers in the forearm reveals a similar pattern of injury to that seen in the primate The muscle bellies were viable centrally with both proximal and distal portions necrotic.

small central region. The distal viable margins of FDP corresponded to the margin of viability on the deep aspect of the superficial flexors. The extent of damage was measured from the radial styloid and recorded on templates of the individual muscle. The ulnar artery was occluded at the level of the forearm flexor bellies. The radial artery was occluded just distal to the cubital fossa, i.e. more proximal than the ulnar artery In the arm, the distal 4 cm of biceps appeared nonviable with the deeper aspect of the muscle damaged 4 to 5 cm more proximally (Fig. 37). Surprisingly, the brachialis muscle was found to be nonviable on its superficial aspect corresponding to the level of injury on the deep surface of the biceps muscle. The level of injury on the deep surface of brachialis was again 4 to 5 cm more proximal than was appreciated superficially, making the proximal level of injury at the mid-arm level It should be emphasized that this muscle was damaged 30 cm proximal to the level of skin injury and 15 cm proximal to the level of arterial thrombosis. The brachial artery appeared normal. The majority of the eschar from the right side of the thoracoabdomen was debrided. All wounds were dressed and the patient returned to the intensive care unit.

On the 4th day following injury, the patient underwent definitive debridement and wound coverage. Prior to induction of general anesthesia, the level of cutaneous sensation was unchanged from previous evaluations The level of cutaneous and muscular injury was also found to be unchanged at the mid-arm level. There was no evidence of progressive necrosis. Due to the extensive amount of skeletal

Fig. 37 Examination of the arm musculature revealed the deeper muscles to be injured more proximally. In the above photo, the biceps muscle is being retracted to reveal deep muscle damage to the brachialis muscle.

muscle damage at this level, a mid-arm amputation and primary closure was performed. The remaining eschar from the right side was debrided and skin grafted The patient remained in intensive care for 6 days and was discharged from hospital 5 weeks after the initial injury.

CASE NO. 2

A.G., a 53 year old male electrical field worker, contacted a 14,400 volt line with major entry/exit injuries to the upper extremities bilaterally. On initial examination, the right hand was found to be mummified extending proximally to the distal third of the forearm On the left extremity, there was a 10 X 15 cm portion of exposed muscle and tendon over the volar forearm (Fig. 38). There were full thickness "skip" lesions at both cubital fossae and superficial lesions in the axillae bilaterally. Tetanus prophylaxis and preoperative antibiotics were given. Angiograms revealed both the ulnar and radial arteries were occluded on the right. The interosseous vessels was patent 3 to 4 cm distal to the cubital fossa. On the left side, the ulnar and interosseous were occluded but filled partially in a retrograde fashion from a patent radial artery. The patient was taken to the operating room where bilateral decompressing fasciotomies of the hand, carpal tunnel, forearm and arm were performed (Fig. 39).

Three days following injury, the patient returned for debridement and wound closure. On the right side, the superficial forearm flexors (FCU, FDS) demonstrated a critical pattern of injury. Centrally, the muscle belly appeared viable, i.e. the muscle was of normal color and

Fig. 38 Left upper extremity injury demonstrating large volar defect with circumferential cubital fossa damage.

Fig. 39 Complete upper extremity fasciotomy complete on day of admission.

consistency and bled actively when stabbed. However, both muscles revealed complete necrosis proximally and distally. The deep musculature, beneath the island of viable flexor bellies, were necrotic (FDP). Radially, the brachioradialis was completely necrotic as were the forearm extensors Significant skeletal muscle damage to the distal biceps, brachialis and triceps warranted an amputation 6 cm distal to the surgical neck of the humerus. The wound was left open to assess any possible ongoing necrosis On the left side, the flexor compartment sustained extensive damage along with the distal biceps and brachialis. The FDS, FCR, and PT muscles appeared necrotic and were debrided. Flexor carpi ulnaris and FDP were necrotic from the midforearm distally. On the extensor surface, the brachioradialis and ECRL were necrotic and debrided The ECRB appeared edematous but viable. The radial artery appeared grossly normal throughout its length as did the three major nerve trunks The ulnar artery was thrombosed proximally. In the arm, the distal third of biceps and brachialis were debrided. After debridement, a 12 X 35 cm defect was present on the volar aspect of the forearm and was covered with a free rectus abdominus myocutaneous flap anastomosed end-to-side with the brachial artery proximally in the distal arm (Fig 40).

On the 5th day following injury, the right stump was debrided minimally and the wound closed primarily On the 11th day, the stump was opened and necrotic portions of the triceps and deltoid not previously explored were removed. After the injury, a small wound opened in the left axilla at the sight of the "skip" lesions. On the

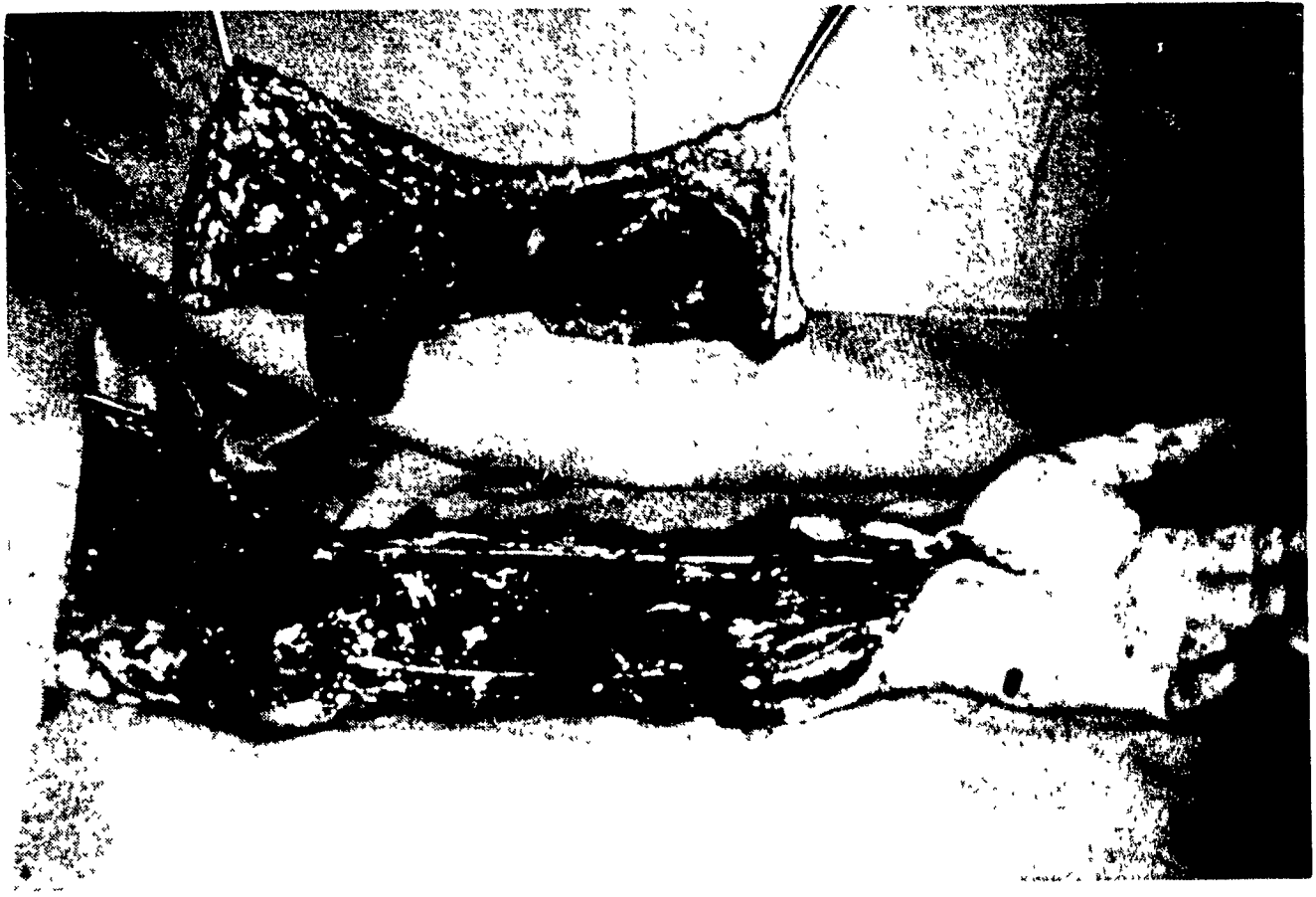


Fig. 40 Following debridement of necrotic tissue, a free rectus abdominus flap was harvested and used for reconstruction of the remaining defect.

Fig. 41 Final result of upper extremity prior to discharge

22nd day, this area was debrided together with necrosed margins along the edge of the free flap. The axilla was skin grafted.

The patient required daily dressing changes and minor debridements for the draining wound in the left forearm and axilla. The patient was discharged from hospital 4 1/2 months after the initial injury EMG's performed 2 and 5 months following electrical injury revealed complete loss of function of the ulnar, median and radial nerves in the forearm and hand There was no voluntary movement of the biceps muscle although good function remained in the triceps and deltoid (Fig. 41).

CASE NO. 3

C.L , 69 year old male, was pruning trees when he sustained a high voltage injury after his branch cutter touched a 14,400 volt distribution wire. He fell backwards to the ground approximately 20 feet below and sustained a brief loss of consciousness. He was taken to a peripheral hospital and immediately transferred to a critical care facility On initial examination, entry/exit wounds were present in the hand and forearm bilaterally and extensive coagulative necrosis of the left lower extremity, just distal to the knee. Second and third degree thermal burns were present over the anterior neck and lower half of his face Preoperative resuscitation included nasotracheal intubation and assisted ventilation, Swan-Ganz catherization, chest tube, tetanus prophylaxis and antibiotic coverage

The patient was taken immediately to the operating room where complete upper extremity fasciotomies were performed bilaterally. At

this time, extensive damage to the musculature of the hand and distal half of the forearms were found. Due to the extensive nature of the lower extremity injury, a below knee amputation was performed. The patient was then returned to the surgical intensive care unit for monitoring. Due to persistent hemodynamic instability, definitive debridement and wound coverage was delayed until the sixth day following the initial injury. At this time, attention was directed to the left upper extremity, the forearm muscles were debrided to the level of the proximal third of the forearm The majority of the forearm tendons were preserved. Necrotic tissue over the palm was also removed The median nerve was observed and appeared normal grossly Stimulation proximally failed to elicit any function in the hand musculature The ulnar nerve also appeared grossly normal Following debridement, a latissimus dorsi myocutaneous free flap was harvested and anastomosed to the radial artery proximally. The flap was inset over the flexor surface of the forarm and all wounds dressed sterilely. Due to the critical condition of the patient, debridement and coverage of the right upper extremity was delayed

The patient initially did well post operatively but by the tenth day following injury, he showed evidence of sepsis Blood cultures were positive for Enterobacter species and yeast Concurrent with worsening sepsis was a rapidly progressive renal failure necessitating hemodialysis Despite aggressive antimicrobial therapy and continued hemodialysis, the patient's condition deteriorated rapidly and he

succumbed to overwhelming sepsis and renal failure 15 days after the initial injury.

DISCUSSION

The inherent difficulties in treating electrically injured patients results from a basic uncertainty of the pathophysiology of the wound, and the spectre of progressive necrosis. Systematic tissue evaluation, the timing of debridement and methods of wound closure and coverage have evolved almost empirically.

CUTANEOUS INJURY

Cutaneous injury must be evaluated regarding accuracy of diagnosis, arc lesions and progressive necrosis.

DEFINING VIABILITY The demarcation between viable and nonviable skin is poorly documented in the experimental and clinical literature. Robinson found that within 12 hours, the demarcation of injured and uninjured skin was sharp.[43] In a review of 182 cases of electrical injuries, Butler described cutaneous injuries ranging in size from pinpoint to large surface areas.[8] He describes the "burns" as being characteristically dry, cold and insensitive which tend to maintain their appearance without suppuration for days or weeks Other investigators have also shown the skin injury to remain unchanged[53] or simply compare the cutaneous injury to a thermal burn[7,16] suggesting well defined margins. Our findings, both experimentally and clinically, also revealed definable margins at the time of fasciotomy post injury.

"ARC LESIONS" Arc lesions are frequently considered to be the cutaneous lesions overlying the flexion creases. These lesions, also known as "skip" lesions for their appearance proximal to viable regions, are universally thought to be the result of external arcing of electrical energy.[7,33,40,41,50,52,60] Our experimental findings however, contradict this well accepted theory. In over 100 experimental injuries extensive injury at the wrist and cubital fossa occured which was similar in appearance and composition to those seen clinically At no time was arcing observed in our experimental studies. Electromagnetic theory also suggests that skip injuries secondary to electrical arcing would be an unlikely occurrence. In order for electric energy to travel outside a conductor, at least two important criteria must be observed. First, external arcing occurs when the impedance to current flow between the points of arcing is several thousand fold greater than the media in which it is flowing. In effect, the current "jumps" over an obstruction rather than preferentially flowing across the shortest distance. Second, the energy needed for electric current to travel externally is in the order of 2 million volts/meter. In other words, for the current to arc from the wrist to the cubital fossa, a distance of 30 cm, an electric potential of at least 600,000 volts is needed. From our clinical studies, and those of other investigators, both of these critical factors are absent The midforearm level, with its predominant muscular composition, has an impedance to current flow which is much less than the wrist and cubital fossa, both of which show a predominance of

highly resistant bone and tendon. Also, the most common electrical injuries involve contact with distribution lines which have potentials of only 12,000 to 25,000 volts. It would therefore be impossible for external arcing to occur over such relatively great distances

PROGRESSIVE CUTANEOUS INJURY A progressive necrosis of skin would be of major concern to the reconstructive surgeon. At least one investigator has reported that skin which at first appears unaffected may become necrotic.[10] Quinby has shown irregular damage and depth of injury to skin histologically.[41] He found apparently viable skin areas mixed with areas of clearly total thermal destruction. Left untreated, he felt these lesions would demarcate slowly and deepen with infection. In our experience and in those of other investigators, the lesions found in the skin following electrical injury remain unchanged.

MUSCULAR INJURY

DEEP INJURY Universal in all clinical reports is the obvious lack of correlation between the damage to skeletal muscle and the overlying cutaneous injury Investigators have demonstrated the frequent occurrence of small skin lesions in association with more extensive muscle involvement.[7,8,10,22,33,40,43,44] Both experimentally and clinically, this has been a consistent observation in our studies and cannot be over emphasized. Lesions involving the hand or distal forearm may extend proximally into the arm beneath viable skin resulting in significant patient morbidity and mortality if not detected. Comparable lesions were observed in our experimental and

clinical studies revealing viable skin over the forearm flexor bellies in association with necrotic biceps and brachialis muscles to the midarm level. This represented a distance of up to 30 cm in our clinical cases. These findings are confirmed in a case report by Ponten who found necrosis of muscle occurring under intact skin at a distance of up to 25 cm from the entrance point of the current.[40]

Also of clinical importance was the extensive injury to the deep aspect of muscles or muscle groups. As seen through the fasciotomy incisions, the superficial muscles of the forearm demonstrated an interface between viable and nonviable tissue in both our experimental and clinical cases. The deeper aspects of individual muscles however, were often damaged more proximally than their superficial surfaces. This undoubtedly represents what is referred to as the deep, "hidden" component of muscle injury.[7,8,22,33,44] More specifically, Hunt describes a central "core" of necrotic muscle in association with relative sparing of superifical muscles.[23] This pattern of muscle injury dominated our experimental and clinical cases. Although also seen in the forearm musculature, this type of injury was more clearly seen in the larger muscles of the arm (biceps, brachialis and triceps) where the undersurface of these muscles was damaged 4 to 6 cm more proximally.

Deep muscle groups in the forearm (FDP, PT, SUP) and in the arm (brachialis) also revealed more proximal damage. Interestingly, the level of injury on the superficial aspect of the deep muscle corresponded to the level of injury on the deep aspect of the overlying

muscle (Fig. 42). The result is a "cone" of tissue injury with its apex extending proximally for considerable distances beneath viable skin and muscle. The high degree of correlation between the patterns of muscular injury seen in our primate model and clinical cases suggests that the flow of electrical energy within the upper extremity is anatomically determined and not simply a random event.

RADIAL PREDOMINANCE Since most electrical injuries of the upper extremity result from grasping a charged object, the points of contact are frequently the hand or palm. The pattern of tissue injury which extends proximally from this point is most often more severe along the radial aspect of the forearm. This is evident in our cases by the consistent and often complete destruction of the extensor muscles, brachioradialis, and the radial aspects of FCR, FDS, FDP. Also, the vascular injury is often more predominant radially with the ulnar artery patent more distally than the radial artery. Other investigators have observed this phenomenon in cases where the point of contact was the hand or palm.[40] As demonstrated angiographically, more extensive injury was observed in the radial artery. Explanations for this phenomenon are not addressed in the clinical literature and may be related to the distribution of muscle mass or tissue composition in these areas. Radially situated muscles (BR, ECRL and ECRB) also provide the only pure muscular bridge across the predominantly tendinous and bony cubital fossa. This may act as a "sink" to channel a large portion of current flow along this route and consequently sustains the greatest effects of this current, i.e. heat production.

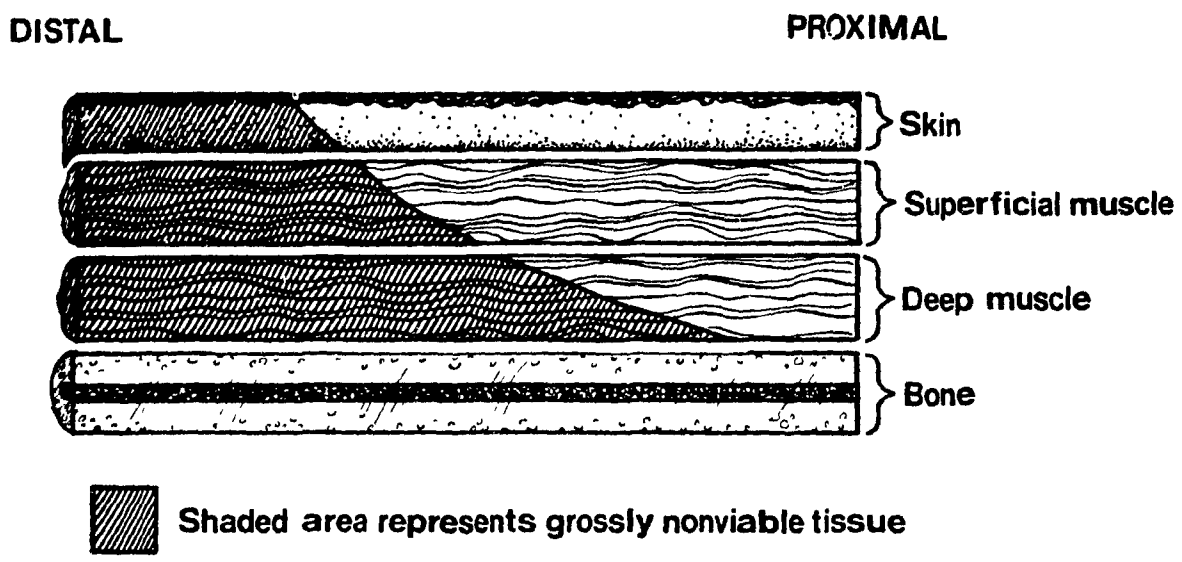


Fig. 42 Diagram illustrating deep muscle damage which extends proximally within the forearm beneath viable muscle.

CHOKE POINTS From our experimental studies, it was apparent that there existed in the upper extremity areas showing more extensive injury than the surrounding tissue. We identified these areas as the wrist, cubital fossa and possibly the axilla. Common to all areas is a decreased cross sectional area composed predominantly of highly resistant bone and tendon. Clinically, these areas showed a propensity for increased tissue injury. Clinical investigators have referred to these areas as "skip" lesions resulting from external arcing of electric energy.[12,13,28,33,41,50,53] Our experimental and clinical cases demonstrate three significant findings with regard to these critical areas. First, muscular injury extends both distally and proximally from these areas. This is seen in our cases where the superficial forearm flexor bellies appeared viable whereas their origins and regions immediately proximal to the wrist displayed marked tissue injury. As well, the distal portions of the biceps, brachialis and triceps showed consistent damage. This pattern resulted in an island of viable skeletal muscle in the forearm, completely surrounded by necrotic tissue. Second, these areas do not correspond to the level of vascular injury. In both the experimental and clinical cases, muscle damage was noted in the cubital fossa with patent vascular trunks (radial and ulnar arteries) extending distally for varying distances Significant segmental narrowing of dominant vascular trunks was occasionally noted in these areas. Third, the most proximal "choke" point appears to correspond to the level of peripheral nerve injury In the cases where marked cubital fossa injury was noted, the level of sensation was

consistently absent from the level of the cubital fossa distally. A lack of conduction along the ulnar nerve distal to the cubital fossa in experimental studies where a lesion was present in this area has been demonstrated.

PROGRESSIVE MUSCULAR INJURY The uncertainty of progressive necrosis has plagued the treatment of electrically injured patients and has provided the foundation for two contradictory management protocols. Some investigators feel there is an ongoing tissue necrosis of muscular or vascular origin leading to prolonged sloughing of tissues following the initial insult [10,15,29,33,44,52] Strategies for treatment concentrated on multiple debridements and deferral of definitive treatment as long as possible to enable the progressive necrosis of tissues to stabilize. Others believe that the injuries sustained by the passage of electrical energy occurred as the result of the initial contact. Proponents of this theory advocate early excision and wound coverage.

In the chronic model, detailed dissections of upper extremities 10 days following electrical injury revealed a consistent and reproducible composite tissue injury We failed to observe any sign of an ongoing, progressive necrosis of skeletal muscle. Our most significant finding however, was deep muscular injury extending for considerable distances proximally beneath both viable skin and muscle. These findings were mirrored in our clinical cases. In all 8 extremities examined in detail, no progressive necrosis of muscle was observed. More importantly, deep muscular injury was detected in both the forearm and arm in cases where the predominant cutaneous injury was at distal

forearm level. The authors strongly believe that the inability to appreciate and detect this deep muscular injury results in delayed tissue sloughing and the appearance of "progressive necrosis".

VASCULAR INJURY

The pattern of injury to the vascular system is a critical determinant of ultimate tissue survival and limb reconstruction. Investigating vascular lesions in the rat limb model, Hunt found abrupt thrombosis of large vascular trunks and pruning of nutrient vessels.[20] Clinically, he determined the major vascular injuries were confined to the smaller nutrient muscular arterial and arteriolar branches [23] Quinby believed the slow demarcation of nonviable tissues is vascular in origin, resulting from progressive small vessel thrombosis.[41] Similarly, many investigators state that the progressive nature of electrical injuries is the result of an ongoing vascular thrombosis.[7,8,24,40,50,52]

The vascular lesions seen in our clinical cases correlated well with the findings of our experimental studies Major vascular trunks thrombosed abruptly with little pre-obstructive aneurysmal formation This level of occlusion did not change post injury However, there was marked pruning of small vessels in areas sustaining significant muscular injury. There was often significant segmental narrowing in the large vessels In our experimental studies, these areas showed mild to moderate vasculitis microscopically Experimentally and clinically, we found no evidence for progressive vascular occlusion

PERIPHERAL NERVE INJURY

Peripheral nerve injuries are found in 13 to 22% of cases of electrical injuries.[4,12,51] Although some recovery is often noted, permanent damage is common. Skoog believed the heat produced by high voltage injuries, associated with edema and scarring led to an unfavorable prognosis of neural recovery.[50] In cases of severe nerve injury associated with total loss of function, the heat produced by current passage was thought to be the predominant cause of injury.[56] Some investigators however, feel nerve injury is caused by a pure electrical effect related to the passage of electricity through a highly ionic medium.[12]

Our experimental investigations of peripheral nerve injury using gross observations and serial electrophysiologic nerve recordings revealed a sharp demarcation of conduction loss along the ulnar nerve which did not recover nor show signs of progression by the tenth day following injury There was no correlation between the level of vascular and peripheral nerve injury, i.e. the level of vascular patency was consistently more distal that the level of nerve conduction Conduction loss was consistently found just proximal to the extensively damaged cubital fossa corresponding roughly to the line of demarcation between viable and nonviable muscle in the surrounding area Our clinical cases showed similar evidence of peripheral nerve injury Grossly, injury to the nerves was difficult to assess accurately at the time of initial presentation Although not performed in all cases, intraoperative conduction studies confirmed the lack of

conduction in affected nerves at the time of initial fasciotomy. Because of this difficulty in determining the level of conduction loss in relation to the surrounding tissue, the use of conduction analysis intraoperatively appears mandatory. When observed immediately postinjury adjacent to areas of obvious muscle necrosis, nerves may appear normal in size and consistency and yet be nonfunctioning.

IMPLICATIONS

By demonstrating a significant correlation between experimental and clinical patterns of tissue injury, we can formulate methods of wound evaluation We feel that the evaluation of an electrically injured patient can be completed in four stages: 1) initial resuscitation, 2) primary operative evaluation, 3) diagnostic evaluation and 4) definitive operative debridement and wound coverage.

I. INITIAL RESUSCITATION

Achieving cardiopulmonary stability dominates the primary management of high voltage electrical injuries. Similar to thermal burns, the injured extremity is initially of secondary importance. Following the establishment and maintenance of an adequate airway, cardiovascular evaluation should include continuous EKG monitoring and cardiac enzyme analysis Venous access must be established early to provide the appropriate fluid resuscitation As demonstrated by clinical experience, the use of fluid regimes established for thermally injured patients correlates poorly with the extent of injury and fluid requirements of the electrically injured patient. Urine outputs of 60 to 100 ml/hr must be maintained for the initial 24 hours following electrical injury to protect the kidneys from hemochromogens and possible acute tubular necrosis Fluid and electrolyte requirements should be tailored to serial blood chemistries. Initial management also includes tetanus immunization and management of coexisting injuries.

The resuscitation of the patient is completed as soon as possible in preparation for the initial evaluation in the operating room.

II. INITIAL EVALUATION

The patient is taken to the operating room immediately following resuscitation and stabilization. In order to decompress affected muscle and neurovascular structures, regardless of whether a circumferential skin injury or distal pulses are present, fasciotomies of appropriate muscle compartments must be completed. Since muscle damage and edema may occur for great distances proximally beneath viable skin, the proximal extent of the fasciotomy incision is of critical importance. Certainly injuries of the hand must be decompressed into the forearm If the wrist in involved, the complete forearm fasciotomy is essential. Once the injury involves the distal third of the forearm, the possibility of involvement of the distal biceps, triceps and brachialis muscles must be anticipated. This necessitates decompression of the upper arm compartments.

The evaluation of the affected extremity is begun distally in a systematic fashion. Critical muscles and muscle layers are assessed in both the forearm and arm Attention is directed towards the known choke points (wrist, cubital fossa and axilla). Peripheral nerves should be decompressed if nerve entrapment from edema is suspected especially at the carpal tunnel for decompression of the median nerve as advocated by Salisbury.[45] Once completed, wounds are dressed sterile and the patient monitored in an acute care facility.

III. DIAGNOSTIC EVALUATION

During the subsequent 3 or 4 days following operative decompression, objective testing and general treatment options are considered.

Angiography is done with digital subtraction angiography preferred for its ability to better define small vessels. The assessment of vascular injury includes the demonstration of large and nutrient vessel patency, and identification of areas of segmental narrowing. These affected vessel segments are isolated areas of vasculitis based on our light microscopy from experimental studies. The loss of endothelium in these areas may predispose to vascular thrombosis or bacterial invasion. However, there is no evidence for a progressive vascular injury in our experimental and clinical cases. In addition, early angiographic evaluation may also serve as a blueprint for possible microvascular reconstruction.

An accurate assessment for both motor and sensory function is recorded based on clinical examinations. Treatment options are evaluated based on the findings at the time of initial evaluation and angiography.

IV. DEFINITIVE DEBRIDEMENT AND WOUND COVERAGE

Demonstrating the absence of progressive necrosis in our experimental research has enabled early definitive wound management with confidence. Depending upon the severity of initial injury, the definitive debridement need not be delayed longer than the third to

fifth day. Debridement of the injured extremity should begin distally with proper identification and evaluation of skin, and individual muscles, nerves and vessels.

The skeletal musculature of the upper extremity should be evaluated through the fasciotomy incisions in a systematic order. hand musculature, superficial and deep forearm flexors, superficial and deep forearm extensors, and the arm musculature. During the evaluation of both muscles and muscle groups, five critical questions must be answered: 1) Is there deep injury in the muscle or muscle group being examined? 2) Does the proximal portion of the muscle or muscle group show evidence of injury? 3) Has the entire radial aspect of the forearm been evaluated (i.e. BR)? 4) Have all muscles in the periosseous areas been evaluated? and 5) Have all choke points been evaluated carefully for muscular injury both distal and proximal to the joint being examined? Grossly necrotic muscle will appear pale in color, not bleed when stabbed with a scalpel and does not contract on electrical stimulation. Although viable muscle may not contract when stimulated, the muscle is normal in color and bleeds bright red blood when stabbed. Objective testing for an instantaneous assessment of muscle viability is lacking at present. However, the key to debridement of these injuries is the proper identification of areas at highest risk for electrical injury. The authors feel that the patterns of injury are anatomically determined and therefore, these critical areas of skin, muscle, nerve and vessel injury can be predicted In our experimental model, we were able to assess muscle viability using

these subjective techniques with great accuracy after pilot studies revealed specific, reproducible and consistent patterns of composite tissue injury showing a high degree of clinical correlation. Our pilot studies using magnetic resonance imaging preoperatively and electrical impedance intraoperatively suggest they are both an accurate objective means of evaluating muscular injury.

Angiographic evidence of vascular injury will outline areas of severe muscular injury. Distally thrombosed vessels should be debrided and the more proximal segmentally affected areas assessed. Subjective assessment of peripheral nerves often revealed minimal abnormalities in the early post injury period and conduction studies are indicated to appreciate the level of conduction loss in the major nerve trunks. Immediate recovery does not occur as demonstrated in our experimental studies although longterm recovery cannot be excluded. Debridement of peripheral nerves will be determined by the extent of distal injury to surrounding tissues and their reconstructive potential.

Wound coverage should be completed at the time of definitive debridement to minimize bacterial colonization and cover vital structures Skin grafting is sufficient for coverage of nonvital structures. Exposure of neurovascular tissues and tendon will most often require skin or muscle flap coverage. The design of local flaps is most often hindered by injury to the surrounding tissues. Free tissue transfers in the form of a muscle or myocutaneous flaps are ideally suited for reliable coverage of large defects. Particular

attention must be directed towards the site of microvascular anastomoses in relation to angiographic evidence of segmental narrowing. Where possible, the anastomosis should be placed above the most proximal area of narrowing.

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