High voltage electrical injuries – clinical and operative observations

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Summary. Seven cases of high voltage electrical injuries seen between 1984 and 1991 are presented. The clinical and operative findings in some of these cases can be explained only partly by the intensive experimental studies which have been undertaken over the past two decades to elucidate the pathophysiology of these mutilating injuries.

Key words: High voltage – Electrical injury

The first reported electrical injury was in 1746 when two Dutch physicists accidentally discharged a Leyden Jar between themselves. The first death was not reported until 1879 when a stagehand in Lyons, France, died while installing some lighting for a theatrical production.

Baxter [2] postulated that these injuries are thermal. This thermal injury is proportional to the resistance of tissue through which the current passes. He also postulated that the heat is generated in increasing increments by nerves, vessels, muscle, skin, tendons, fat and bone. Bone offers more resistance to the passage of current and, thus according to Ohm’s law, the greatest heat will be generated by bone. Hunt et al [4] on the other hand found that the internal milieu of an experimental animal’s body acts like a volume conductor of a single resistance and not as if it were composed of tissues of varying resistances. With the onset of current flow, amperage and temperature rose in parallel throughout. By the time of current arcing, they found both muscle and bone temperatures to be equal. However, they observed it took bone longer to dissipate the heat and felt that it was this prolonged elevation in temperature that accounted for the perosseous “Core” of necrotic muscle seen clinically. Hunt et al. [4] also postulate that involved muscle and vessels sustain irreversible damage at the time of the current passage with immediate microscopic muscle coagulation necrosis and small nutrient artery thrombosis. Heggers and Robson [5] have stated that cellular integrity depends on a homoeostatic relationship between PGE$_2$ and PGE$_{2a}$.

Injury to the cell disrupts this, causing a shunt in arachidonic acid metabolism toward thromboxane production. Production of this vaso-active prostandoid in large amounts leads to vasoconstriction, thrombosis, progressive ischaemic necrosis and further thromboxane production. In the experimental electrical injury presented, the elevated thromboxane production by the injured cells could easily provide the impetus for converting areas of patch necrosis into areas of complete tissue loss.

Lee and Kolodney [6], using an axisymmetric unidimensional model containing bone, skeletal muscle, fat and skin in coaxial cylindrical geometry, have found that the concept previously reported by Baxter [2] and Hunt et al. [4] was not supported using this well accepted physical model. They found that when the tissues are electrically in parallel, skeletal muscle sustained the largest temperature rise and heated adjacent tissues. Thus, when bone is not in series with other tissues, joule heating of bone is unlikely to be responsible for thermal damage of adjacent tissue. In addition, the effect of tissue perfusion on the thermal response was found to be essential for rapid cooling of centrally located tissues. The specific magnitude and kinetics of the tissue temperature response to electrical current vary along the current path with change of the area through which the current passes.

Since the cross-sectional area of the soft tissue and the ratio of soft tissue to bone would be less near joints than, for example, in the arm, an extreme temperature rise would be expected at the joints with resultant tissue damage.

Robson [8] has also suggested, following Lee and Kolodney’s work [6], that no interpretation is required to explain the presence of deep necrosis beneath unaffected skin. He states that skin temperature may never rise to the lethal range in an electrical injury. The fact that...
this apparently uninjured skin may progressively necrose, could be due to progressive ischaemia and not the electrical event itself.

It has also been reported that tissue injury has occurred in the absence of obvious thermal effect, which implies that electric current can produce damage through non-thermal effects. This has resulted in delayed neurological sequelae [3, 7].

Recently, Zelt and co-workers [9] investigated chronic wound evolution in high voltage electrical injury in African Green Monkeys. They performed a standard 40 KV, 3500 V, 4.2 A, 2.5's bilateral symmetrical upper extremity electrical injury. Multidisciplinary investigators focused on three objectives:
1. To develop a chronic electrical burn model,
2. To document the pattern of tissue injury and its evolution over a ten day period,
3. To determine experimentally whether progressive necrosis is a critical factor in electrical injuries.

The electrical injury was precisely controlled by a computer-controlled administration system. Four hours after this controlled high voltage electrical injury, bilateral fasciotomy was done from carpal tunnel to axilla. The evaluation was done on:

- gross observation
- light microscopy
- digital subtraction angiography
- electrophysiologic nerve conduction

All of the above evaluated criteria were performed from a few hours to ten days post-injury, except for angiography, which was undertaken a few days prior to injury. They concluded from this experiment that:
1. Patterns of electrical injury are anatomically determined for a given energy and site of contact; identical patterns of injury will result if the anatomy is the same. As the amount of energy delivered is increased, progressively more proximal damage will occur.
2. There exists in the forearm specific regions, or “Choke” points, in which decreased cross-section areas and high resistant tissue composition result in increased heat production and more severe tissue damage. The wrist, cubital fossa, and possibly the axilla, are examples of these regions.
3. In the upper extremity, tissue injury extends more proximally on the deep surface of both individual muscles and between layers.
4. The predominant mechanism of tissue injury appears to be thermal. Unknown at this time are the effects of current passage, toxic mediators, or infection on ultimate tissue survival. Arcing of current does not occur at sites other than the points of entry and exit.
5. Progressive necrosis does not occur following electrical injury and should not play a dominant role in patient management. Efforts should be made towards identification of tissue damage in the acute stage. Diagnostic aids, such as angiography and nerve conduction studies, are valuable in the assessment of tissue injury. Objective tests, such as tissue impedance, are needed.

Case reports

Case 1

A 30-year-old man sustained an extensive electric injury when working in a high tension electrical field 250 km away. He arrived at the hospital four hours later. He had lost consciousness following the injury but was successfully resuscitated after one hour of fluids and oxygen.

On examination, his general condition was satisfactory, and he was fully conscious without neurological symptoms or signs. His pulse was regular at 60/min with good volume, and his blood pressure was 120/80. Examination revealed deep burns of the left axilla and arm, and both index and middle fingers of the left hand; the distal interphalangeal joints were open and the distal phalanges were flail. There were deep burns over the right ankle joint extending to the dorsum of the right foot; the big toe was charred, and the fifth metatarsal bone was exposed.

Baseline investigations were within normal limits, and the ECG was normal. He was resuscitated with the appropriate fluids and sedation and was treated conservatively with closed dressings, waiting for the slough to separate and the gangrenous part to demarcate.

A month later, serial desloughing was performed on the axilla, arm, ankle and dorsum of the right foot. The right great toe was disarticulated, as were terminal phalanges of the index and middle fingers, together with a sequestrectomy of the fifth metatarsal bone. The raw areas were covered with split thickness grafts. He left hospital after 21/2 months and attended the outpatient clinic for eight months.

Case 2

A 36-year-old man sustained high voltage injury in the same accident as the patient in Case 1. He was admitted to hospital with this patient; he had lost consciousness for a shorter period and was similarly resuscitated.

On examination, he was fully conscious, had a regular pulse of 60/min with good volume. His blood pressure was 160/90, and he had no neurological symptoms or signs. The baseline investigations were normal, as was the ECG.

Examination showed deep burns of both palms and a circumferential burn of the right ankle and dorsum of the foot; all toes were blue. There were deep burns on the left big toe.

The right ankle was released by escharotomy, and the remainder of the burns were treated conservatively.

A month later, the palms and front of the right ankle and dorsum of the foot were debrided and grafted. The gangrenous toes of the left foot and the right great toe were disarticulated after demarcation. The stumps were covered with healthy granulation tissue and subsequently covered with split skin graft. The patient was discharged after 12 weeks. He had a course of outpatient hand physiotherapy.

Case 3

A 26-year-old man sustained a 22000 volt electrical shock when trying to release an electrical shutter. He was brought to hospital 5 h after the accident. He was in a state of shock and had a transient loss of consciousness at the time of the accident.

On examination, he was restless and pale with an imperceptible pulse and an unrecordable blood pressure. There were extensive deep burns of the right hand, forearm, arm and both ankles. The right forearm and arm were swollen, and the skin of the proximal forearm was dusky. There were patches of superficial burns on the right chest and face.

He was resuscitated with haemacel and appropriate fluids. Immediate escharectomy was performed along the whole of the right