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Biophysical injury mechanisms associated with lightning injury

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1. Introduction

The passage of damaging quantities of electrical current through the body can produce tissue injury through multiple distinct biophysical energy transduction mechanisms. These mechanisms include the direct action of direct electrical forces on proteins, membranes and other biomolecular structures, as well as the indirect action mediated by the generation of heat. Adding to this complexity are the multiple modes of frequency-dependent tissue-current interactions, the variation in current density along the path through the body, as well as variations in body size, body position and use of protective gear. The dominant mode of injury for any particular trauma victim and how it manifests depends on several different factors. As a result, in no two cases of accidental electrical injury are the injury manifestations identical.

The purpose of this chapter is to review the biophysical pathways and mechanisms of electrical trauma injury and their relationship to clinical injury manifestations. The focus will be on frequency-dependent effects because of the relevance to lightning injury. It was written with the hope to provide insight into some of the unusual neurophysiological manifestations.

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2. Electrical energy transport in tissues

In order to understand the mechanisms of lightning injury it is essential to discuss the transport of electrical energy in tissues. The human body consists of about 60% water, in which 33% is intracellular and 27% extracellular [1]. Body fluids in both the intracellular and extracellular compartments are highly electrolytic. These two compartments are separated by a relatively impermeable and highly resistive plasma membrane. Current conduction in tissues is carried by mobile ions in the extracellular and intracellular fluids. These mobile ions provide a conductivity of approximately 1.4 Sm^{-1} in physiological saline.

In response to an imposed voltage difference across metal or an electrical arc, current passes from a high potential to the lower potential. The particles that carry the charge in metals, and in an electrical arc, are electrons. This conversion from mobile ions to electrons occurs at the skin surface through an electrochemical reaction driven by the voltage drop [23].

After penetrating the outer layer of skin, the electrical current density distribution depends on the relative electrical properties of various tissues and the frequency of the electrical current. Frequency is important for two reasons. Current flow across insulating boundaries like the epidermis or cellular membranes (1) is by capacitive coupling; and (2) is by magnetic forces acting between adjacent moving charges. Capacitive current (or more formally "displacement current") moving across cellular membranes govern current distribu-

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tion around individual cells. Magnetic forces tend to concentrate the current near the surface of conductors and can be described by a characteristic "skin depth". The skin depth effect governs the distribution of current across the entire current path whether it is the full cross-section of a finger, extremity, or torso.

Practically, the skin depth in tissues becomes important in objects the size of adult human at frequencies above 10^7 Hz. At low frequencies (i.e., below radiofrequencies), it distributes so that the electric field strength is nearly uniform across any plane perpendicular to the current path [16,39,52]. The higher the frequency is the shorter the skin depth. At gigahertz frequencies the skin depth is on the order of a centimeter. Above this frequency the current is concentrated near the body surface. Sances and co-workers measured very low frequency (60 Hz) current distribution in the hind limb of anesthetized pigs [52]. They suggested that large arteries and nerves experienced the largest current density because of their higher conductivity. However, they observed that skeletal muscles carried the majority of the current due to its predominant volumetric proportions.

On the microscopic level in tissues the current distribution is frequency-dependent as well. In frequencies below roughly 10 kHz, the current distribution around cells within tissues is determined by the density, orientation, shape and size of cells. Because the cell membrane is a good insulator, electrical current is shielded from the interior of cells. The presence of cells diminishes the area available for ion flux and, in effect, makes tissues less conductive. As cell size increases, the membrane has less impact on a cell's electrical properties because the volume fraction of the cell occupied by the membrane is proportionately decreased [22]. With a similar argument, the resistivity of muscle that is measured parallel to the long axis of the muscle cells is less than what is measured perpendicular to the axis. Free water content is also important. In cortical bone and epidermis, the resistivity is higher because their free water content is lower.

The current distribution at higher frequencies is quite different. The cell membrane is no longer an effective barrier to current passage, as the current is conducted into the cytoplasm as a displacement current as illustrated in Fig. 3. At radiofrequency (RF) and above, the current passes through cells as if the membrane did not exist and the tissue was a uniform conductor. Of course, when the frequencies are in the microwave region or higher, the skin depth presents a constraint and results in non-uniform current.

Lightning produces fairly broadband fields and currents ranging between kilohertz and above. Typically, the majority of the current passes along the surface as expected for high-frequency current. Also, because the lightning plasma is a much better conductor, most of the current should remain in the arc running parallel to the body.

3. Biophysics of tissue injury

Electric forces cause tissue damage by altering molecular and cell organelle structures. This can involve multiple biophysical modes of injury including heat-mediated damage, cell membrane electroporation, electrochemical interactions at the contact points [5,8, 40], and blunt mechanical trauma due to the thermoacoustic blast (i.e. thunder) from a high-energy arc [9]. While these forces can alter all tissue components, it is the membranes of cells that have the greatest vulnerability. Thus, the plasma membrane of the cell appears to be the most important structure in determining the rate of tissue injury accumulation.

The most important function of the cell membrane is to provide a diffusion barrier against free ion exchange. Ion traffic takes place through a tightly regulated system of pumps and channels. Because most of the cellular energy of mammalian cells is ultimately invested in maintaining the electrochemical potential across the cell membrane, the importance of the structural integrity of the lipid bilayer is apparent [45].

3.1. Electrical forces acting on the cell membrane

A cell in an applied DC or low-frequency electrical field will experience electrical forces acting around and in the cell membrane. The magnitude of the induced V_m across the membrane depends on a variety of factors, such as the intra- and extracellular medium conductivity, cell shape and size, the external electric field strength E, as well as how the electric field vector orients with respect to the point of interest on the cell membrane [30,47,60].

Considering typical physiologic conditions, the magnitude of the induced V_m at the electrode-facing pole caps of a spherical cell (Fig. 1) can simply be expressed as:

$$V_m(r,\theta) = 1.5ER\cos\theta \quad (1)$$

with R being the radius of the cell and θ is the angle between the applied field vector and the position vector identifying the specific membrane section defined by r, θ. For elongated cells such as muscle and nerve cells

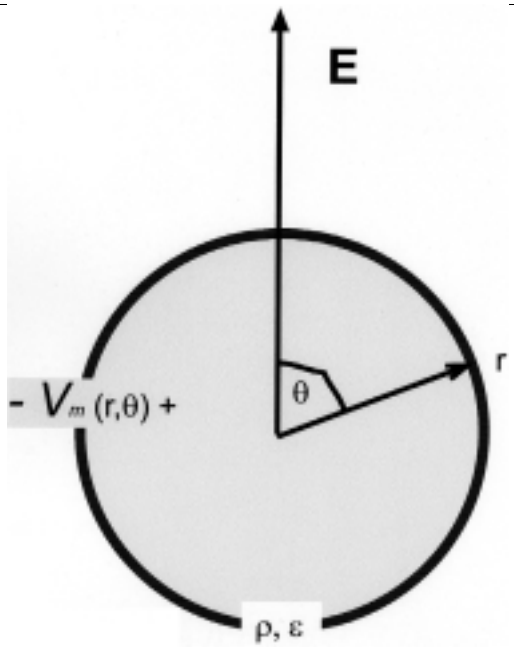

Fig. 1. Transmembrane potential (ΔV_m) induced by an external D.C. electric field E_o acting on a typical eukaryotic cell in a physiologically conducting bath. The field lines do not penetrate the cell because the pathway of least resistance for the current is around the cell.

Eq. (1) can be rewritten with L as the length of the cells projection in the field:

$$V_{m,\max} \approx 0.5\, EL \tag{2}$$

when the cell is aligned in the direction of the applied field [30]. Figure 1 illustrates schematically the dependence of V_m on the cell size.

Keep in mind that at frequencies much higher than the reciprocal charging time of the cell, the membrane is no longer a barrier to current passage. Therefore, the electric field strength in the cytoplasm is equivalent to the external field. Under these conditions the induced V_m is simple the product of the external field E times the membrane thickness. Typical cell membrane thickness is on the scale of nanometers, whereas the length of the cell L is typically microns to centimeters.

Intermembrane molecular changes in response to direct electrical forces (i.e. electroporation and electroconformational coupling) occur on the time scale of milliseconds. These two damage mechanisms lead to an altered structure and compromised functional integrity of the cell plasma membrane. It is only with prolonged interactions, on the scale of seconds, that thermal damage begins to dominate in the injury accumulation processes.

3.2. Membrane electroporation

The natural transmembrane potential difference (V_m) of a cell, typically 50–70 mV in magnitude, originates from the difference in ionic strengths of the cell's intra- and extracellular fluids [10]. When the transmembrane potential of a cell is in excess of two to three hundred millivolts, structural breakdown of the membrane occurs. This event is called electroporation, which is mediated by re-organization of lipids in a lipid bilayer and electroconformational denaturation of membrane proteins [12,41,60]. Recent reviews and books published have extensively treated this subject [11,30,44,47,48,55,60,61].

Electroporation can be either transient or stable, depending on the magnitude and the duration of the imposed transmembrane potential. Because physical properties of the membrane are extremely temperature dependent, tissue temperature is also important. The field-induced generation and growth of pores result in increased membrane electrical conductance, causing the imposed V_m to fall. This reduces the effort of water leaving the membrane pores and increases the probability of spontaneous bilayer sealing. If sealing occurs, sealing kinetics can be orders of magnitude slower than the field relaxation because pore closure requires removal of the membrane fragments and associated water molecules within the membrane pore. This is a time and energy consuming process [6,20,21].

The threshold transmembrane potential for induction of membrane electroporation is remarkably similar across cell types and ranges from 250–350 mV [6, 20,21,26]. Several authors have developed reasonably accurate models based of chemical energies of membrane-water interactions to explain the experimentally observed values of V_m required for electroporation [14,25].

After the effect of ionic concentrations is included in the model, it is even able to confirm asymmetries in V_m observed with respect to the hyperpolarized (anode facing) and hypopolarized (cathode-facing) pole caps of a cell [20,21,28]. Although the pore sealing time (time needed for pores to close) in the range of seconds predicted by the model is in agreement with some published experimental results [49], others have found longer sealing times in the range of several minutes [6, 20,51]. This might be explained by the fact that: (1) this model is based on pure lipid bilayers instead of cell membranes embedded with proteins, and (2) it only considers primary pores (pores formed during shock) formed by V_m and not those formed after the exter-

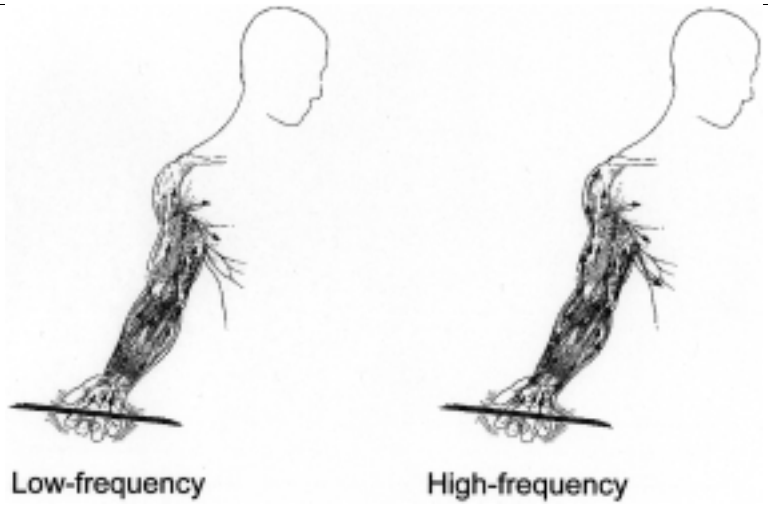

Fig. 2. The effect of frequency on the spatial distribution of current in the body. At high frequencies the magnetic interactions between moving charges constrains the distribution of current to the surface of conductors.

nal field pulse ends (secondary pores), which provide transport routes for macromolecules.

Typical DC field strengths required for electroporation are in the range of 1 kV cm^{-1} for most tissue cells that have a characteristic length scales of 10–40 μm. On the other hand, specialized cells that are designed to communicate via small electric fields are typically much larger. These are nerve and striated muscle (skeletal and cardiac) cells. Due to their relatively long lengths, skeletal muscle cells are up to 8 cm long in large animals and nerve cells range upwards of 2 meters long, these cells have much lower electroporation thresholds. Figure 2 shows an anatomical sketch of the median nerve as an example. The magnitude of the transmembrane potential induced by an externally applied electric field scales with the dimensions of the cell in the direction of the applied field as shown in Fig. 1. Therefore, muscle and nerve cell membranes can be damaged with electrical fields as small as 60 V cm^{-1}. Skeletal muscle cells within the human upper extremity can reach up to several centimeters in length and peripheral nerve cells are substantially longer. These dimensions are enormous compared to cells of other tissue that have characteristic dimensions in the range of 10–100 microns. Therefore, the strength of the electric field required to electroporate skeletal muscle and peripheral nerve cells is typically 100–1000 fold less than that required to electroporate smaller cell types.

The distribution of electropore formation in a large cell placed in an applied field was recently addressed by DeBruin and Krassowska [17,18]. Expanding from previous mathematical models based on the traditional "Cable" distributed RC circuit model, and including the fact that the membrane charging time of about 1 μs is very short compared with a 1-ms field duration, they concluded that supraphysiological V_m at the pole caps is large enough to create pores, and thereby effectively preventing a further increase in V_m in these areas. This confirms early experimental findings that show a saturation of V_m, which is independent of the field strength for high-voltage shocks [29,36,38].

If the membrane is permeabilized, the work required in maintaining the transmembrane concentration differences increases proportionately. The conductance of electroporated cell membranes can increase by several orders of magnitude. When ATP-fueled protein ionic pumps are not able to keep pace with the diffusion of ions through the membrane defects, metabolic energy exhaustion results. If the membrane is not sealed, the cell will progress to biochemical arrest and then to necrosis. Thus, in discussing tissue injury resulting from electrical shock, the principal focus is directed at the kinetics of cell membrane injury and the reversibility of that process.

3.3. Thermal "Burn" injury

Thermal burn is the term most often used to describe tissue denaturation due to a damaging suparphysiological temperature. It is generally appreciated that the effects are related to the damage to proteins, which are followed by grossly visible changes in tissue.

The phospholipid bilayer is a supramolecular assembly of surfactant molecules maintained by hydrophobic

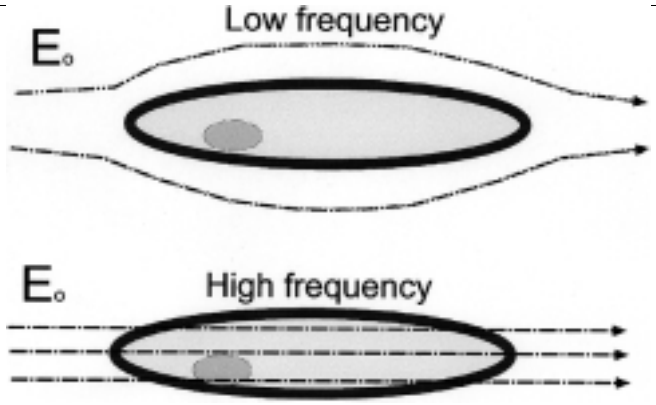

Fig. 3. The effect of frequency on the microscopic distribution of current in and around the cell. At low frequencies (upper image) the electric field and corresponding current lines are mostly shielded from the cytoplasm of the cell because the membrane is contains so few charge carriers. At high frequencies, the current passes across the membrane by capacitive coupling. At high frequencies the membrane is not an effective barrier.

and hydrophilic forces, and it is probably the part of the cell membrane that is the most vulnerable to thermal damage [24]. Even at temperatures of only 6 °C above normal (i.e. 43°C) the kinetic energy of the molecules in the cell membrane can exceed the hydration energy barrier that holds the phospholipids in the membrane as a supramolecular assembly [46]. In effect, the warmed membrane goes into solution in the surrounding water, rendering the membrane freely permeable to small ions.

3.4. Electrochemical denaturation of membrane proteins

The composition of cell membranes consists of 40–60% proteins. These membrane proteins function as ion channels, transporters and signal receptors. These proteins are acted upon by the electric field within the membrane. Under natural conditions, the membrane electric field ranges between 10^6–10^7 volts/meter. Each peptide unit of the typical protein represents an electric dipole moment of about 3.5 Debye (D). For each α-helical structure, which is oriented perpendicular to the cell membrane, many small peptide dipoles are aligned to form a larger dipole on the order of 120 D [31,32,59]. An electric field-induced membrane potential will strongly affect the orientations of these electric dipoles. Thus, the intramembrane electric field has a powerful allosteric effect on membrane proteins.

For voltage-dependent membrane proteins, many charged side groups are movable in response to changes in membrane potential. This gating action allows them to function as voltage sensors. One example is the ion channel. Segments of the four inter-repeat domains that line the channel consist of repeated motifs of positively charged amino acid residue followed by two hydrophobic residues. They have been suggested to be a voltage sensor in the proteins' gating. These voltage sensors are obviously susceptible to an applied intensive electric field.

Many other membrane proteins are sensitive to the membrane potential by virtue of their intramembrane charged particles. For example, dihydropuridine receptors in the transverse (T) tubular membrane of skeletal muscle fibers function as voltage sensors involved in excitation-contraction coupling. Intensive electric field-induced supramembrane potentials may cause these intramembrane charged particles to move along or reorient the equivalent dipoles to align with the direction of the external electrical field. As a result, the ionized side-chain in the membrane proteins may dissociate from the main α-helix, or the electrically charged subgroup may separate from the proteins. Moreover, physiologically moveable charged particles in the membrane proteins may lose their moveable capability by sticking to or separating from the neighboring amino acids.

Tsong and Teissie examined the effects of a strong field pulse exposure on the membrane protein Na/K ATPase in erythrocytes [56,57]. After using a microsecond pulsed intense electric field to shock red blood cell membranes, they found that in a low ionic medium at least 35% of membrane pores induced by the shock pulse were linked to channels in denatured Na/K ATPase in the cell membrane [37]. They attributed this ionic leakage to the electroporation of the Na/K ATPase. Abramov et al. [1] performed *in vivo* studies on changes in the functions of rat sensory nerves and skele-

tal muscles after an intense electrical shock [1]. They found that 4 ms shock pulses of 500 V/cm significantly reduced the magnitude of the action potential and increased its latency period.

In a series of experiments performed to study the effects of field-induced large transmembrane potentials on ion channel proteins in skeletal muscle fibers [12, 13], both the open channel currents from the voltage-dependent Na^+ channels and the delayed rectifier K^+ channels were reduced by an intense electric shock. An electric shock by a single 4 ms, -500 mV pulse may decrease about 20% of the Na^+ channel currents and 30% of the delayed rectifier K^+ channels currents. The channel conductance of the delayed rectifier K^+ channels were also substantially reduced by the above electric shock. The decrement varies from 10 to 40% depending on the type of cells.

A question was raised whether the intense electrical field-induced functional reductions in the membrane proteins, such as K^+ and Na^+ channels, resulted from field-induced Joule heating or from electroconformational damages. It is necessary to identify which one is the dominant mechanism involved with the membrane protein damage in electrical injury. The characteristic of Joule heating-induced damages in cell membrane is that the degree of damage is proportional to electrical energy dissipated in the cell membrane, which is directly related to the field-induced transmembrane currents [7,42,43]. In contrast to Joule heating damages, field-induced conformational changes in membrane proteins are primarily due to the high voltage across the cell membrane.

According to Joule's Law, the electric field-induced evolution rate of heat on the cell membrane should be equal to the shock pulse-induced electric energy consumed on the cell membrane. Studies have estimated the consumed energy by integrating the transmembrane currents with time during the shock pulse and then multiplying by the applied membrane potential difference [13].

3.5. Radiofrequency and microwave burns

Although relatively rare, each year several cases of RF or microwave field injuries occur in the US and the World. The victims are usually industrial workers. Above the low frequency regime, tissue response strongly depends upon the field frequency. In the 10–100 megahertz RF range, two types of tissue heating occur: Joule and dielectric heating, where Joule heating outweighs dielectric heating. Small charged molecules in the intra- and extracellular solution, such as unbound water molecules, are able to follow the field up to the gigahertz range (10^9 cycles/sec) [15]. However, at microwave frequencies (100 MHz to 100 GHz), dielectric heating is more significant than Joule heating because both bound and free water are excited by microwave. A water molecule has a small size but a large dipolar moment; hence water has a strong susceptibility to microwave, whereby water molecules are induced into rapid oscillatory rotation that heats up the whole tissue. If the amplitude of the field strength is constant, the heating rate increases with field frequency until the viscous drag on the rotating molecule makes it lose pace with the field. Molecular dipoles of larger sizes oscillate more slowly, so that their most efficient induction frequency is in the radiofrequency range. For example, coupling of RF energy into proteins and DNA's is the basis of recent concerns about cellular phones. The therapeutic use of RF hyperthermia is based on the same mechanism.

Exposure to ambient microwave fields is known to cause burn trauma. Microwave burns have clinical manifestations different from low frequency electrical shocks [2,50,53,58]. At low frequency the epidermis is a highly resistive barrier. However, in the microwave regime, electrical power readily passes the epidermis in the form of "capacitive" coupling with very little energy dissipation. Consequently, the epidermis may not be burned unless it is very moist. The microwave field penetration into tissue has a characteristic depth in the range of 1 cm, resulting in direct heating of sub-epidermal tissue water. The skin depth effective is illustrated in Fig. 2. The rate of tissue heating is dependent not only on the amplitude of tissue electric field, but also on the density of dipoles. For example, microwave heating is much slower in fatty tissues [54].

3.6. Lightning injury

Lightning is a common term for the phenomenon in which the voltage difference between clouds and other objects reaches such a level (in air exceeding 2 million V m^{-1}) that dielectric breakdown ("arcing") occurs. The current through an arc can be enormous but the duration is quite brief (10–100 ms). The primary current is confined to the surface of conducting objects connected by the arc. Peak lightning current falls into the 30,000–50,000 A range, which is able to generate temperatures near 30,000 K°. This abrupt heating generates a high-pressure thermoacoustic blast wave known as thunder.

When lightning strikes the ground it spreads out radially from the contact point, setting up large currents travelling along the ground surface. A substantial voltage drop can occur between the feet of a person walking nearby. For example, with an average lightning current of 20,000 A, a step length of 50 cm, and an individual located 10 meters away from strike point, the voltage drops between the legs can reach 1500 V. This can induce a 2 to 3 A current flow through the body between the legs for a 10 μs period.

An individual directly struck by lightning will experience much higher current for this brief period of time. Initially, the surface of the body is charged by the high electric field in the air. During this charging, the body surface potential difference can reach several thousand volts between the upper and lower body. This can cause breakdown of the epidermis, and several hundred amperes to flow through the body for a 1 to 10 μs period, which is certainly long enough to induce electroporation. Following this, a much smaller current persists for several milliseconds, in which time the body is discharging into the ground. The duration of current flow is relatively short, so there is no substantial heating except a breakdown of the epidermis. However, disruption of cell membrane can wreck havoc on nerve and muscle tissues. Fortunately, spontaneous sealing of membranes can occur, which may account for the high survival rate in victims of lightning strikes.

The tremendous current flow in lightning creates a large surrounding magnetic field. This pulsed magnetic field can induce current flow in conducting objects such as the human body. A person standing in the immediate vicinity of a lightning strike can experience enough magnetically induced current through the body to produce destructive effects. Also, electrical currents will be induced through any electrical circuits penetrated by the magnetic field. Consider the scenario of a person standing on wet ground and leaning on a metal golf club. The golf club links the upper body to the soil, which in effect, forms a closed loop consisting of golf club, body, and wet soil. A large magnetic pulse can drive large currents through this circuit. Large four legged animals are also at high risk for this problem.

The thermoacoustic blast can also be very destructive in the immediate vicinity of a lightning strike, and hence it may also contribute to tissue injury in lightning strike victims. This force can split trees. It is well known that ear damage occurs with only 1 atmospheres of overpressure. Lightning associated thermostatic blast pressure can reach 100 atmospheres or greater. This will generate air pressures ranging from 4 or 5 atmospheres in the immediate vicinity of a lightning strike, to 1 or 2 atmospheres 1 meter away [33]. Clearly, these pressures are sufficient to cause physical damage, particularly, to the eardrums of the victim of a lightning strike.

3.7. Microwave and RF burns

According to the few reports on microwave injuries in the literature, a large portion of the injuries occurs in radiofrequency electrocautery surgical units. This is where microwave is delivered to heat up and denature proteins in order to control bleeding or to rapidly vaporizing tissue water in order to cut tissue [35]. Unintentional tissue injury may occur. Most of the injury occurs below the epidermis, with the fat tissue largely spared. However, fat does not attenuate the field well. Skeletal muscle has a high level of hydration, and hence it carries a heavy load of thermal input under microwave. Clinical experience indicates that a characteristic "layered burn" occurs: burned skin and muscle with fat spared in between.

Radiofrequency burns show similar patterns as the higher frequency microwave burns. However, there is more Joule heating component in RF heating, hence fat is not as well spared as in microwave heating. Another more common complication of RF hyperthermia procedures is nerve injury [15]. Therapeutic guidelines on RF hyperthermia require maintenance of tissue temperature at 42°C to 43°C. Exposure to this temperature for 0.5–1 hour will generate substantial heat-mediated membrane permeabilization. Patients with RF burns usually have deep fourth degree burns penetrating all tissues. Most of these injuries occur as a side effect of either a tumor-treating therapy or a healing-enhancement physical therapy.

3.8. Lightning injury

The superficial surface burn on the skin from a lightning strike does not correlate with the severity of the injury. The immense current pulse in lightning induces a proportionately large surrounding magnetic field pulse. This sharp magnetic field pulse penetrates through the body, setting up secondary electrical currents inside. The secondary currents, forming closed loops around the penetrating magnetic field lines, are large enough to cause cardiac arrest, seizures and other harmful effects. The duration of the primary lightning current pulse and its induced secondary electric currents are so brief that heating is not responsible for the internal

damage, rather it is a pure electrical phenomenon. As previously described, there are some exceptions to this, such as when lightning flashes over to charge up a car or truck to which a victim is in contact. Thus the electrical discharge of the car or truck through the victim takes long enough for heating and burning to occur in addition to the damage from electrical effects.

Keraunoparalysis or complete neurological and muscular "stunning" is the primary clinical feature immediately after the shock in survivors of lightning strikes. Initially, this is manifested by cardiorespiratory arrest and mental obtundation. The condition is transient and usually clears in a few minutes if life support is administered. Subsequent amnesia and "neurotic" behavior often occurs and can last for one week. Usually within 24 to 48 hours there is less confusion, but muscular weakness and pain, visual photophobia, and other disturbances of neurologic control are the most prominent clinical features that usually persist.

Transient nerve dysfunction can have a variable course of recovery depending upon the severity of field exposure. Nerves can regain function in hours or require months. The mechanism of recovery is unknown and thus, it is difficult to relieve anxieties in victims. Permanent sequelae have been correlated with demonstrable anatomical lesions [34].

4. Summary and conclusions

The impact of electrical injury on society is quite substantial, and hence a swift diagnosis, cure, and rehabilitation of electrical trauma patients will have great socio-economical benefit. However, a prompt, accurate clinical diagnosis of electrical injury is one of the most difficult tasks in the medical field because it usually calls upon an understanding of the interaction between electric current and human tissue. Specifically speaking, the difficulty involves the following:

1. The exact tissue damage mechanism and damage level depend on a host of parameters: the characteristics of the power source (DC or AC current, voltage, frequency, etc.), path and duration of closed circuit, area and impedance of contact spot, etc. Correspondingly, there is a whole spectrum of damage characteristics depending on the values of these parameters. The physician needs to do 4-dimensional (spatial plus temporal) detective work in order to arrive at a correct diagnosis.
2. Electrical damage to the tissues is not easily detectable by visual inspection or physical examination. Often times its sequelae will not manifest themselves after a certain period of time: electrically injured tissue may initially appear viable only to become visibly necrotic at a later point (in a number of days) [3,4,27].
3. Due to the multidisciplinary complexity of the electrical trauma problem, there is no standardized diagnostic and treatment protocol for it. A discussion of the pathophysiology of electrical injury often involves aspects of biophysics, electrochemistry, cell biology, and neurology to name a few. The subtle biological changes of the tissue caused by electrical shock often elude conventional diagnostic protocols.

The continuing development of uses for electricity mandates a better understanding of the potential harmful effects of direct electrical contact on biological systems. The molecular structure of biological systems can be severely altered by high-energy commercial frequency electrical power. The basic biomolecular process involved in electrical injury is mostly misunderstood because of the interdisciplinary nature of this problem. The mechanisms of damage include cell membrane electroporation, Joule heating, electroconformational protein denaturation, and others. Effective diagnosis and treatment for electrical injury patients requires understanding and addressing each of the direct and indirect modes of tissue-field interaction.

Acknowledgments

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