ELECTRIC SHOCK

MICHAEL MORSE
JENNIFER MORSE
University of San Diego
San Diego, California

1. INTRODUCTION

However human beings came to be designed, we are lucky enough to have a design that protects the internal human machine from the external electrical environment. Perhaps it is this natural protection that has made it possible for our society to so extensively use electricity in almost every aspect of our daily lives. It is to our benefit that human skin has proven to be a naturally protective barrier to the flow of electricity (1,2). Still, under the right conditions, humans can become part of the electric circuits or processes found within their environment. The resulting injury can run the gamut from momentary pain to extensive injuries impairing multiple-organ systems to electrically initiated ventricular fibrillation (3–5). The nature of human contact with electricity will be discussed herein.

Most important to understanding the risks associated with any particular shock is to consider any electrical contact in the context of other similar shocks. To do so requires that electric shocks be delineated and compared based on a set of descriptive parameters. To understand electric shock, one needs to consider each of the following (6–9):

1. Energy Source: AC, DC, or Impulse (static, lightning, capacitive)
2. Differential voltage
3. Entry and Exit Points
4. Theoretical Current Pathway
5. Pathway Resistance
6. Amount of Electrical Current
7. Shock Duration
8. Energy transferred into and dissipated by the body

Throughout this chapter, we will focus on a consideration of the nature of electric shock and how these parameters predict the risk of injury associated with electrical contact.

2. HUMAN RESPONSE TO ELECTRICITY

The scientific knowledge base for human contact with electricity is ever-expanding. The *traditional model* of electrical contact is based on the assumption that injury must be linked to pathway, current level, and energy imparted during the shock. The *modern model* for electric shock injury considers that electrical injury may also be dependent on the electric field and also recognizes that evidence exists of pathway-independent mechanisms. No discussion of electrical injury is complete without also considering *secondary* impact associated with electrical contacts.

2.1. Response to Electrical Contact (Traditional Model)

Although a broad range of effects exist that electrical contact can have on the human body, the parameters of the shock help to characterize the risks associated with the shock. These effects might be called primary effects, as they occur as the direct result of current flow within the body. Table 1 provides an overview of the primary effects of electric shock based on the current level, assuming other shock parameters (pathway, duration, energy source) are fixed (6,8,10–12). As a result of human variability as well as the variability associated with any given shock scenario, the information in Table 1 should be viewed as a good starting point for assessing shock risk rather than as an absolute guideline.

1. *Sensation*: At a current of approximately 1 mA, an average human will begin to note a tingling sensation associated with current flow through their body (6,8,10).
2. *Let-go Current*: Current flow through muscles will cause those muscles to contract. When electrical current traverses a motor nerve, the nerve will be stimulated and the muscle fibers innervated by that nerve will contract. The let-go current is reached when the effects of the electric current exceed one's voluntary ability to counter those effects. In essence, one cannot voluntarily let go of an energized current source until that source is de-energized or the muscles so fatigue that they fail to maintain the grasp. For an average male, the let-go current is approximately 14 mA. For an average female, it is 10 mA (6,8,10). A common misconception is that the force of contraction is somehow superhuman. In fact, it is just maximal muscle contraction, also known as tetanic contraction attained when the current level

Table 1. Human Response to Electrical Contact

Response	Average Minimum AC Current level
Sensation - tingling (first perception)	1 Milliamp
Painful shock but no loss of muscle control	9 milliamps
(Can't) Let go	14 milliamps for males (10 milliamps for females)
Painful muscle contraction/ difficulty breathing	23 milliamps
Ventricular Fibrillation	50 milliamps (hand to hand contact)
Myocardial sustained contraction	Greater than 1 ampere (>1000 milliamps)
Burns (Thermal Injury)	Greater than 1 ampere (> 1000 milliamps)
Typical Household Circuit Breaker	20 Amperes

Note: These "average" values are broadly used and are based on well-established research, but for the sake of creating this type of simple table, human variability has largely been set aside. Individual human response can vary considerably from these numbers. As such, these numbers should be viewed as *typical* but certainly not definitive.

and stimulation frequency cause all muscle fibers to be maximally recruited to contract. Tetanic contraction can only be reached when the muscle stimulation frequency is between 40 and 100 Hertz (2,13). Beyond 100 Hertz, muscles will be overstimulated and fatigue very rapidly. Early research demonstrated that commercially generated AC electricity at 60 Hertz caused maximal muscle impact (6).

3. *Pain Threshold*: The threshold of pain is felt when sensory nerves are so electrically stimulated as to cause pain. It is also caused by the direct mechanical stimulation of pain sensors associated with substantial contraction of muscles being electrically stimulated.
4. *Ventricular Fibrillation*: Table 1 indicates that a current of at least 50 mA is necessary in a 3-second, hand-to-hand contact (in which the heart is in the theoretical current pathway) for ventricular fibrillation to occur. In practice, even at currents exceeding 50 mA, fibrillation is frequently not triggered. Ventricular fibrillation is a somewhat random process linked to the parameters of the shock, including current pathway and shock duration as well as to such human-linked parameters as the phase of the shock victim's heart and the victim's health. For brief shocks, the probability of fibrillation is increased if the shock coincides with the sensitive S-T and T wave intervals of the electrocardiogram(EKG) (14). The risk of ventricular fibrillation can be assessed in any shock scenario but is never absolutely predictable. When fibrillation does occur, medical defibrillation is essential to save the shock victim's life. Based on animal studies, the minimal fibrillation current (a 1 in 200 chance) can be predicted for electric shocks between one-half of a 60 Hertz electrical cycle (8.3 ms) and 5 seconds using the formula (6,15):

$$I = K/T^{.5},$$

I = fibrillation current in milliamps

T = time in seconds

K = Constant ranging from 100 to 185 dependant on the victim's weight ($K = 165$ for a 70 kg victim.)

Except in instances of head involvement in the shock, individuals who suffer electrically induced ventricular fibrillation most likely have an immediate cognizance of the shock, associated pain, and perhaps even impending death for as long as 15 seconds before they become hypoxic and lose consciousness (16).

5. *Respiratory Arrest*: When the electrical current pathway traverses the respiratory muscles (diaphragm and intercostals) or the nerves that innervate those muscles, those muscles can be stimulated to tetanic contraction such that voluntary respiration ceases and death by asphyxiation will ensue absent cessation of the electrical current. Electrical current can also impact the respiratory center of the brain, causing respiratory arrest. Medical triage for electric shock requires immediately assessing if there has been respiratory impact and providing proper treatment until breathing can be restarted (11,12,17).
6. *Sustained Myocardial Contraction*: As with all other muscles stimulated by AC (in the frequency range of 40 to 100 Hertz), when sufficient electrical current traverses the heart muscle, it will maximally contract and stay contracted until the current ceases. Per Table 1, to reach such a current level at the heart requires a hand-to-hand contact current level of 1 ampere or more. Upon release of the current, the heart can, in some instances, return to normal pacing and, in others, it may fibrillate.
7. *Tissue Burns*: Burning occurs because energy has been imparted to the tissues. The amount of energy is a function of electrical current level, tissue resistance, and shock duration. The process is called "resistive heating," and the energy is imparted to tissues along the current pathway. It is a localized effect typically observed in areas where the current density is highest (such as the current entry or exit points) or areas of greater tissue resistance (such as dry skin). If enough energy is imparted either by way of current level or shock duration, any organ system within the body can be impacted. Entry or exit burns will tend to occur before internal burns. Human tissue will experience first-degree burns when raised to 50°C for a period of 20 seconds (18).

2.2. Response to Electrical Contact (Modern Model)

In recent years, researchers have found many instances where the symptomatology following electrical contact differs greatly from the anticipated response, as defined in Table 1, or by the traditional model. These responses have been attributed to other mechanisms of electrical injury, which until recently, were unknown or have not yet been explained:

1. *Electroporation:* Electroporation is a path-related electrical phenomenon in which the injury is neither the product of the current level nor of the energy level of the shock. Frequently, electrical injury is observed absent a contact capable of generating enough tissue heating to cause damage. Lee and Kolodney (19) reported an alternative to thermal heating that explains how electrical injury occurs in circumstances where the energy of the contact is less than the injury would suggest. In the presence of a significant enough electric field, cell membranes will rupture. This rupturing or "electroporation" disrupts the metabolic functioning of the cell and can cause cell death. Per Lee and Kolodney, an electric field of 200 V/m in the direction of a 1-cm long skeletal muscle should be enough to rupture the membrane (19). This theory recognizes that significant

injury can occur in low-voltage contacts if the electric field is high enough. As an example, in a 120-volt contact, where the entry and exit points are very close, the electric field can be as high as 10,000 volts/meter, many times the field strength necessary to cause significant cellular injury (19). Electroporation can cause slow cellular death that is consistent with the often noted delayed onset of neurological sequelae (20) following contact and might also serve to explain the delayed onset of some neuropsychological symptomatology. Electroporation explains how significant and apparently disproportionate responses can be observed following low-voltage contacts, even when the contact is brief because the injury is proportionate to the electric field and not proportionate to the energy of the contact. As electroporation can only occur along the current path where an electric field gradient exists, electroporation would not explain neurological injury that has been observed remote to the theoretical path of the current.

2. *Diffuse Electrical Injury* (*DEI*): One of the rarest responses to electrical contact is "Diffuse Electrical Injury." Multiple researchers have reported similar symptomatologies found in individuals who have suffered electric shock. The symptoms include an array of neurological, physical, and neuropsychiatric symptoms that occur with statistical significance but are not explained by the energy, current, electric field, or theoretical pathway of the electrical current. DEI is best defined as being a statistically grouped set of symptoms that are chronologically linked to an electric shock but may be remote to the theoretical current path and disproportionate with the shock parameters. The traditional view held that only organ systems in the path or proximity to the path of the current could be impacted during an electrical injury. Such phenomena, where the response is not proportional to the known parameters of the contact but where the response is repeated among a large sample of shock victims, has come to suggest that mechanisms of injury associated with electrical contact still exist that are as of yet not explained and that fly below the diagnostic radar defined by our societal level of technology (21–25).

2.3. Secondary Responses to Electric Shock

Secondary effects from electrical contacts are those that happen independent of the current flow within the body. Common secondary effects are as follows:

1. *Injury from Falls or Impacts*: The natural (fear) response associated with any electrical contact is to pull away from the current source. As muscles are being contracted by the force of the current, the response of jerking backwards to get away from the source can lead to significant tissue injury that is not caused by electrically induced muscle contraction. The risk of injury is heightened once the let-go current is exceeded. In some instances, broken bones or other significant injuries result from falls or impacts that occur secondary to the actual electrical contact (26,27).
2. *Electric Arc Injury:* Electric arcs occur when air breaks down and ionizes, forming a plasma that can reach temperatures of 4000 degrees centigrade (28). For air, the dielectric strength is approximately 30,000 volts per cm (29). An electric arc will be generated when there are two points separated by air such that the difference in voltage between those two points is greater than 30,000 volts times the number of centimeters of separation. Associated with the rapid generation of the arc is heating and expansion of the surrounding air and vaporization and expansion of the metal vapor. Even if the current path does not include a person in proximity to the arc, the extreme temperature of the arc can cause substantial flash burns. Flash burns will manifest in the form of superficial skin carbonization (30,31). When an arc occurs in a closed space, such as found in high-voltage switching gear, the speed with which the arc is drawn and the rate at which air is heated can cause an explosive blast resulting not only in extensive flash burns but also in concussive blunt force tissue injury and secondary injuries from impacts and falls. A 25 kA arc at a range of 2 ft produces a directed force of 160 lbs/ft^2 (32).

3. ANALYSIS OF ELECTRICAL CONTACTS

The analysis of any electric shock begins with the quantification of the parameters of the shock. Absent such quantification, shocks would have no more individual distinction than as if we referred to all wheeled vehicles on this planet simply as transporters. Clearly, bicycles are not in the same category as freight trains. Such is the case for the spectrum of electrical contacts. Grouping all types of shocks into a small number of big categories is the mistake most often made by those with limited experience in the study of electrical injury.

3.1. Energy Source

The source of the electrical energy can vary significantly, and the resultant effects on the body can vary based on the type of power source. Sources might be characterized as either *sustained energy* or *impulse energy.* Sustained sources are those where the reservoir of available energy does not limit the duration of the contact. Where the source is a generator or, in some instances, a battery, the reservoir of energy far exceeds the energy of the electrical contact. When the energy source is a stored charge, such as a capacitor or a static charge, the available energy frequently sets the limits of the contact.

1. *Alternating Current* (*AC*): Virtually all household and industrial electricity is AC, which explains why most electrical injuries are from AC sources. As a result, most of what we know about electric shock injury is applicable predominantly to AC, but, by extrapolation, may also apply to other types of

sources. Alternating current is produced using a rotating generator, which, by virtue of its cyclical nature, produces an electrical sine wave that alternates equally between positive and negative values at a constant rate (frequency). The amplitude of AC sources can vary from very low voltage (as in garden or pool lighting) to household (120/240 volt) to industrial (480 volt) to distribution line (7 kV) to transmission line voltages (ranging to several 100 of kilovolts). As such, the electrical current associated with an AC shock can vary from milliamps to hundreds of amps. Common to AC systems is that the power source (generator) most often has the driving capability to keep delivering electrical energy almost indefinitely relative to the shock duration (33).

AC electricity is parameterized by the frequency of repetition of the waveform and by the amplitude of the waveform. For the purpose of calculations, the value used for amplitude is the Root Mean Square (RMS) voltage. Household electricity in the United States has an RMS voltage of 120 volts and a repetition frequency of 60 cycles per second (60 Hertz).

2. *Direct Current* (*DC*): DC is produced from a constant voltage source such as a battery. Although commercial and scientific applications exist that use high-voltage DC (34), electrical injuries are rare when compared with AC. Like AC, most DC sources have the capacity to deliver energy for sustained period of times. In our world, commonly available DC sources are fairly low voltage (such as car batteries) or have little available energy (such as flashlight batteries.) As a result, research into DC-type electrical injury is somewhat limited and more myth than fact may exist about the difference in injuries that can result from comparable AC and DC contacts. Although AC contacts can cause injury by multiple mechanisms, including thermal damage and fibrillation, the most likely mechanism of DC damage is by thermal injury (33). Research has also shown that greater amounts of DC current are required to have the same effect (such as sensation and pain thresholds) (28). In the classic work by Dalziel, he found that AC differed from DC in that test subjects experienced a burning pain during a DC shock and that significant pain was only experienced upon release of the contact (35).
3. *Static Discharge*: Static discharge is an impulse-type shock that occurs when energy built up on an insulated surface is discharged to ground. Discharge occurs when the charged surface comes into contact or proximity with ground, allowing the charge to dissipate and permitting current flow. The most common human experience with static discharge occurs when one receives a small and brief shock by touching a doorknob after walking across a carpeted floor. By walking across the carpet, charge is being stored. By touching the doorknob, the charge is brought into close proximity to ground and dissipated. Although the charged surface can be at a very high voltage, the energy available is very small and thus rapidly dissipates during the discharge. The energy transferred in such a shock is approximately .01 joules (as compared with 1–2 joules for a 120 volt, .1 second, AC shock). Although sometimes annoying, such a low-energy static shock cannot cause primary injury (28).

   Industrial static injuries can be quite significant. Energy may build up on machinery with fast-moving insulated rollers over which dry material is being passed. Static buildup is heightened in particularly dry environments. (The author saw such a condition in a paper mill.) In industrial environments, a static charge equaling 100s of thousands or even millions of volts can build up on a large surface with significant energy. When the dielectric (point of breakdown) for air (30,000 volts per cm) is reached, the air ionizes and an arc is drawn between the charged surface to ground or, in some instances, to an unsuspecting individual who inadvertently narrows the gap by virtue of stepping near the charged surface and creating a pathway to ground.

   Although typically brief, the impulse can have significant enough energy to cause primary thermal injury, although secondary injuries from falls or impacts are most likely.
4. *Lightning*: Lightning is a very-high-energy static discharge capable of causing massive injury, immense thermal tissue damage, and death. A typical lightning strike can have currents ranging to 200,000 amperes with the duration of the strike ranging from .0001 to .003 seconds (36). The amount of energy associated with a lightning strike can easily exceed several hundred million joules.

   Injury from lightning is much less predictable than from generated electricity. The brevity of the lightning discharge can often yield only superficial burns, but fractures and multisystem thermal injuries are also reported (3). As the lightning seeks ground, injury from flashover from one object to another is quite common. The energy imparted to a lightning victim can range very widely depending on a multitude of variables associated with the geometry of the environment at the time of the strike.
5. *Capacitive Discharge*: Capacitive discharge is a type of electrical impulse that is very similar to static discharge. Capacitors are energy storage devices. Once charged, capacitors can hold their energy for an extended duration of time. A capacitor bank the size of a suitcase can store 100 kilojoules of energy, although individual capacitors exist that can be charged to lethal levels. When a pathway is created through which the capacitor can discharge, the energy is released. Discharge through a resistive load (such as a human) follows an exponential decay. As large capacitors can store large amounts of energy at significant voltages, potential exists for dramatic primary and secondary injury. Capacitors should be handled very carefully and care should be taken to assure that they are discharged before touching the leads (34).

3.2. Differential Voltage

All shocks share at least one common factor. For a shock to occur, the recipient of the shock must be in a position such that at least two points on their body are subjected to voltages of different values. This differential voltage is the voltage measured between those two points. Generally, most components of the physical world that we contact are held to the same voltage, commonly referred to as the ground or earth potential. No shock can ever occur unless a differential voltage exists. One of the two voltages can be the earth potential whereas the other voltage can be the voltage from an energy source such as those described above (21).

3.3. Entry and Exit Points

The entry point is the known or approximated point of contact between the body and the source of the current (typically the higher of the two differential voltages). The exit point is the known or approximated point of contact between the body and point at which the current exits. The exit point is typically the point of lower voltage. Hence, the differential voltage is defined as the voltage drop between the point of current entry to exit.

Entry and exit points tend to be focal contact points and are often the points of highest current density (amps/cm^2) and are thus the most likely points to find burns in brief contacts (18). It is, however, known that significant injury can occur absent entry or exit burns. In low-voltage contacts ($<$1000 volts differential voltage), entry and exit wounds are observed in only 45% of the contacts (33). The greater the voltage and the greater the shock duration, the greater the energy of the contact and the greater the risk of entry and exit burns.

NOTE: In alternating current (AC) contacts, the terms "entry" and "exit" are misnomers because the current oscillates making entry and exit points oscillate with the current. Still, the convention is to call the point closest to the technology driving the current the entry point and the point closest to the earth or ground potential, the exit point.

Electrical injury can be very complex and temporally dynamic. During the duration of a shock, multiple entry and exit points may exist that can often confuse the analysis of the electric shock scenario. A typical mistake in evaluating shocks is to try to treat the shock environment as geometrically static.

3.4. Theoretical Current Pathway

The theoretical current pathway is defined as the shortest linear pathway between entry and exit points. Historically, on a systemic scale, the body has been viewed as a "structureless gel" (3,7,37). Although, on a purely theoretical level, this defies the basic laws of physics, it does allow for an excellent first approximation of the current traversing the body during the shock, which in turn provides a good basis upon which injury may be predicted.

3.5. Pathway Resistance

Pathway resistance can be calculated by adding resistances that have been experimentally determined for the different body parts along the theoretical current path (1,38). Such resistances are based on a gross view of each body segment assuming the homogenous ("structureless gel") tissue model. Table 2 contains a list of resistance values that are used to calculate current flow during an electrical contact.

When considering the local effects of electric current, however, the homogenous tissue model must be abandoned. Table 3 is a list of the resistivity (and conductivity) values for individual tissue types (39). The resistance for any given body part is made up of the parallel and serial combinations of the individual resistive tissue pathways. The resistivity values of Table 3, when taken in the context of local tissue geometry, can be used in the calculation of localized current distribution and can, in turn, be used to anticipate localized injury (40).

3.6. Electric Current

Human tissues are generally viewed as being electrical conductors. The differential voltage across the electrically conductive tissues between the entry and exit points is what drives electrical current through the body. The current delivers the energy that impacts and damages the tissues. The amount of current traversing between the entry and exit points is defined by Ohm's law, which is stated as:

$$\text{Voltage } (V) = \text{Current } (I) \times \text{Resistance } (R).$$

By solving for current, Ohm's law is restated as:

$$\text{Current } (I) = \text{Voltage}(V)/\text{Resistance } (R),$$

Table 2. Homogenous Body Resistances for Adult Humans

Body Resistance	1000 ohms IEEE STD. 80
Average Body Resistance—Hands to feet (dry entry and exit)	4838 ohms (Hamman)
Average Body Resistance—Hands to feet (wet entry and exit)	865 ohms (Hamman)
Body Resistance—Hand to hand (wet entry and exit)	1300 ohms (Hamman)
Body Resistance—Head to leg (high-voltage wet contacts)	$<$300 ohms (Morse)
Arm	200 ohms (Webster)
Leg	200 ohms (Webster)
Torso	100 ohms (Webster)
Dry Skin (1 cm of contact area)	15 kohms to 1 Mohm (Webster) 70 kohms to 100 kohms (Hammam)
Wet Skin (1 cm of contact area)	150 ohms to 10 kohms (Webster) 700 ohms to 1 kohms (Hammam)

Table 3. Tissue Resistivity and Conductivity

Tissue	Resistivity ohm-meter	Conductivity (ohm-meter)$^{-1}$
Blood	1.5	0.666666667
Plasma	0.63	1.587301587
Cerebrospinal Fluid	0.65	1.538461538
Urine	0.3	3.333333333
Skeletal Muscle	3	0.333333333
Cardiac Muscle	7.5	0.133333333
Lung	12.75	0.078431373
Fat	25	0.04
Bone	160	0.00625
Copper	1.72E-10	5800464037

where *Voltage*, measured in volts, is the differential voltage between the entry and exit. *Resistance*, measured in ohms, is the pathway resistance. The resulting *current* is measured in amperes (amps) (8,10).

3.7. Shock Duration

The duration of the electric shock is defined as the amount of time during which a differential voltage exists and during which a resultant flow of current between the entry and exit points exists. The amount of energy imparted to the body, as well as the probability of ventricular fibrillation, increase as a direct function of shock duration.

3.8. Energy Transferred in the Body

The energy imparted by the electricity causes resistive heating and resultant thermal tissue damage. Energy transferred into a body during an electric shock is defined by the equation:

$$\text{Energy} = (\text{Current}^2 \times \text{Resistance}) \times \text{Shock Duration}$$

$$\text{Energy} = (I^2 \times R) \times T,$$

or by the equivalent equation:

$$\text{Energy} = \text{Differential Voltage} \times \text{Current} \times \text{Shock Duration}$$

$$\text{Energy} = V \times I \times T.$$

When shock duration (T) is in seconds, voltage (V) is in volts, and current (I) is in amperes, the calculated energy (E) is in Watt-seconds or joules, where 1 joule equals = 1 watt-second. Energy can also be given in calories (c), where 1 calorie = 0.24 joules.

NOTE: By definition, one calorie is the amount of energy necessary to raise one cubic centimeter of water one degree centigrade. As soft tissue is largely water, one can approximate that for every calorie imparted to one cubic centimeter of tissue, the temperature will rise approximately 1 degree centigrade (18).

4. EXAMPLE: ELECTRIC SHOCK ANALYSIS

4.1. Scenario

A healthy 25-year-old male of average weight (70 kg) receives an electric shock while in the process of turning on a lamp in his household laundry/utility room. When interviewed, he reports that it was a hot, humid day and that he had just completed exercising. He describes the shock as having been excruciatingly painful and feeling as if it *grabbed* his whole body. He reports that the shock began when he attempted to turn on the lamp, and the shock lasted for *what seemed like* many seconds. Although he suffered no burns, some slight reddening of the skin occurred on his right hand at the point of contact. A family member witnessed the occurrence, and, when interviewed, reports that the contact looked like it lasted around three seconds before the shock victim was able to "jerk" himself free of the lamp. The shock victim was right-hand dominant and grabbed the lamp switch with his right hand while opening (grasping) the top lid to the washing machine with his left hand. The shock victim was wearing running shoes (rubber soles). Upon inspection, it was found that the lamp had a frayed hot lead that caused the switch and case to energize and that the washing machine was functioning properly and had a properly grounded metal enclosure. The lamp was plugged into a standard household outlet and had a 75-watt incandescent bulb. The fuse box providing the energy to the branch circuit containing that outlet had a 15-amp circuit breaker. No other significant loads were operating on that branch circuit at the time of the shock. No Ground Fault Circuit Interrupter (GFCI) exists on either the branch or the outlet.

4.2. Analysis

This scene is a typical household electric shock scenario. Before assessing the potential for injury, the parameters of the shock must be determined.

1. *Power Source*: The power source is a standard household outlet into which the lamp was plugged. Such outlets in the United States provide 120 volts AC at 60 Hertz frequency. The current available in the circuit was 15 amps minus any current drawn by any other components operating on that branch circuit. As no other components existed, the outlet could draw 15 amps before the breaker would trip.

   NOTE: Had the circuit been protected by a GFCI, the outlet would have been de-energized when a shock (fault) current of approximately 5 mA occurred. Circuit breakers are used to protect machinery, whereas GFCIs are used in circuits to protect people.
2. *Differential Voltage*: The contact was between a surface energized by a frayed hot lead and a grounded metal enclosure. The RMS voltage between the hot lead and ground is 120 volts.
3. *Entry and Exit Points*: The entry point was observed to be the right hand and the exit point was the left hand. As the shock victim was wearing rubber soled

shoes, which are insulators, it is doubtful that any other exit points existed capable of providing noninsulated pathways to ground. (We will assume that entry and exit points were each 1 cm^2, although that might be worth further examination to increase the accuracy of the analysis.)

4. *Theoretical Current Pathway:* The shock is best characterized as a "hand-to-hand" contact. The theoretical current path is the shortest linear pathway from the entry to exit point that would traverse the right-hand skin, right hand and arm, torso, left arm and hand, and left-hand skin. The line of the theoretical current path would include the heart.
5. *Pathway Resistance*: The pathway resistance is determined by adding all of the resistances along the theoretical current pathway. These resistances are based on the "structureless gel" model of the human body but provide an excellent first estimate of current. When considering skin resistances, one must consider the moisture present at the time of the contact. In this instance, it is a hot, humid day and the victim has been exercising. In the worst case, one can assume that the victim's hands are very wet, which is validated to a degree by the absence of entry or exit burns even though the shock duration was several seconds. Resistance values are taken from Table 2. The resistance is calculated as follows:

$$R_{\text{pathway}} = R_{\text{skin right}} + R_{\text{arm}} + R_{\text{Torso}} + R_{\text{left arm}} + R_{\text{skin left}}$$

$$R_{\text{pathway}} = 150 \text{ ohms} + 200 \text{ ohms} + 100 \text{ ohms} + 200 \text{ ohms} + 150 \text{ ohms}$$

$$R_{\text{pathway}} = 800 \text{ ohms}.$$

6. *Current Flow:* Current is determined by Ohm's law as follows:

$$I = V/R$$

$$I = 120 \text{ Volts}/800 \text{ Ohms}$$

$$I = .15 \text{ Amps (or 150 mA)}.$$

7. *Shock Duration:* It has been reported that shock victims will experience distortion of time such that their estimate of shock duration can be significantly in error (3). As such, it is best to rely on other sources to determine shock duration. Shock duration can range from instantaneous [defined as approximately .1 second, which is the typical human nerve and muscle response time to an electrical contact (41)] to very long durations in scenarios where the let-go current has been exceeded and the victim cannot voluntarily let go of the current source. The witness, in this instance, places the shock duration at 3 seconds, which seems reasonable under the circumstances.
8. *Minimum Fibrillation Current:* Although fibrillation did not occur, it is worth looking at the minimal fibrillation current as the heart was in the theoretical current path. The purpose is to determine if the victim was at risk of fibrillation in this scenario. Applying the equation:

$$I = K/T^{.5},$$

where $K = 165$ for a 70 kg male and $T = 3$ seconds, yields a minimum fibrillation current of 95 ma. The shock victim was at risk of ventricular fibrillation during his contact.
9. *Shock Energy:* The energy imparted into the body is calculated using the formula:

$$\text{Energy} = \text{Voltage} \times \text{Current} \times \text{Time}$$

$$\text{Energy} = 120 \text{ Volts} \times .15 \text{ Amperes} \times 3 \text{ Seconds}$$

$$\text{Energy} = 54 \text{ Watt}-\text{Seconds } (= 54 \text{ joules}),$$

which can be converted to calories by multiplying by .24

$$\text{Energy} = 12.96 \text{ calories}.$$

4.3. Discussion of the Shock

By comparing the current level of 150 mA to the information in Table 1, we see that the current was significantly in excess of 14 ma, the average human let-go current for a male. As such, it is not surprising that the shock victim could not let go of the source of the current. The current would have caused the muscles in the hand to contract, thus causing the victim to grasp both the light and the lid of the washing machine. Also, we note that the current exceeds the threshold for pain and, as reported by the victim, the shock was very painful. Current this high at 60 Hertz is expected to cause tetanic muscle contractions mimicking the worst muscle cramps one could experience. Although the current did exceed the Table 1 current at which ventricular fibrillation could occur (50 ma) as well as the calculated minimum fibrillation current of 96 ma, it should be noted again that ventricular fibrillation is a random process linked to many factors, and it is not surprising that there was no fibrillation. The risk still existed. If it did not occur at the time of the shock, it is generally not a future health concern. As to respiration, the shock could have and might have impacted the respiratory (intercostals and diaphragm) muscles. The brevity and pain of the shock precludes awareness on the part of the victim of breathing difficulties in many instances. Note that, in longer shocks, respiratory impact can be horrific, leading to the sense that the victim is suffocating and helpless. As with ventricular fibrillation, impact on the respiratory center of the brain is also dependent on many variables and was not observed in this particular shock.

As to the energy of the shock, enough energy was imparted to the body to heat one cc of water 15 degrees centigrade, but because the energy is dispersed through a much greater volume of tissues, internal burning is certainly not possible. In cases where the skin of the entry or exit point is dry and poses a risk of greater resistive heating, the skin in a shock similar to this could experience very minor entry or exit burns. Such burning is not expected for wet skin.

This shock yielded exactly the results that one might anticipate. As a result of the small amount of energy imparted to the body, and based on known mechanisms of injury (traditional model), little expectation exists that the shock victim will experience any future effects from the shock.

It is also worth noting that when considering circuit protection, the current level of the shock was but a small fraction of the value of the 15-amp circuit breaker protecting the circuit. As such, there would have been no expectation that the circuit breaker would trip. A GFCI, which is intended to protect individuals in just such a scenario as this, would have tripped, had it been present in the circuit. GFCIs are designed to trip at high fault currents in less than .03 seconds and are responsive to fault currents as low as 5 ma, making the probability of fibrillation virtually nonexistent (42).

5. LOCALIZED EFFECTS DURING AN ELECTRIC CONTACT

Up until this point, the focus has been on a global understanding of electric shock, which has allowed the body to be treated as homogenous and generally "structureless." Although these simplifications provide for an excellent estimate of the generalized effects of an electrical contact, no discussion of electric shock should end without considering the reality that most shock injuries *are* localized. Most significant among those considerations is the question of how the theoretical current path differs from the actual current path, and to which tissues the energy is actually imparted during an electric shock.

5.1. Theoretical Versus Realistic Current Pathway

To understand the localized impact, it is important to look more deeply at what truly defines the current pathway. On a global level, the current pathway is characterized only by the known electrical entry and exit points as in a "hand-to-hand" or "hand-to-foot" contact. The internal current pathway can never truly be known. History has proven that it is generally the rule in the majority of electrical contacts that electrical injury will occur along the theoretical current path. Recent study of electrical injury has found many exceptions to this majority rule.

A view that comports correctly with the laws of physics (and less with the "structureless gel" theory) must begin by recognizing that many different tissues exist between the entry and exit points. Each tissue defines a separate current pathway. The current must be divided among all of the pathways. The percentage current distribution to each separate tissue pathway must be inversely proportionate to that pathway's resistance, as is dictated by the current divider rule extension of Ohm's law. Treating each tissue pathway separately recognizes that, as Ohm's law was applied to the "structureless gel," it must apply as well to each separate pathway. By Ohm's law, $I_{\text{pathway}} = V_{\text{pathway}}/R_{\text{Pathway}}$ for each pathway. The "structureless gel" must then truly represent the cumulative effect of all the individual pathways. The least resistive path may not always be the physically shortest linear path. In theory, some current will flow in all pathways (where $R_{\text{path}} <$ infinity) between entry and exit points. This analysis assumes that tissues are purely resistive, lacking capacitive interaction, which is generally true at the low frequency of commercially generated electricity (41).

The resistance for any pathway is given by the following formula, which recognizes that resistance is a function of the geometry (length and area) as well as the parametric characteristics (material resistivity) of the pathway.

$$R_{\text{tissue}} = \rho L/A,$$

where:

$$\rho = \text{Resistivity of the tissue in the pathway (Units : ohm} - \text{meter)}$$

$$L = \text{Length of the pathway through which current flows (Units : meter)}$$

$$A = \text{Cross}-\text{sectional area of the pathway through which current flows (Units : meter}^2\text{)}$$

Applying this formula and recognizing that bulk tissues (such as muscle) have cross-sectional areas that can be orders of magnitude larger than conductive pathways offered by tissues with lower resistivity values (such as neural and vascular paths) (38) yields the result that bulk tissue pathways will have dramatically lower resistance than other pathways made of more conductive material. Bulk tissues would thus conduct the overwhelming percentage of current in any electrical contact, which gives rise to validation of the appearance of the broadly accepted "structureless gel" theory. The pathways made of more conductive tissues, even if conducting less current can still have dramatically higher current densities, which creates the reality that localized tissue damage is possible in tissues conducting at higher current densities even if surrounding tissues have current densities below the injury threshold. It also suggests that nonpath-related injury may be the result of current deviation to pathways other than the shortest linear path.

Given the multitude of conductive pathways and the extensive resistive proximity among pathways, solving for localized current density is a daunting task. The finite element method (FEM) has proven effective in the solution of such problems (40). Figures 1a to 1d depict a localized solution to model current density in a hand-arm involved electrical contact. Data from the visible human project provided a tissue template cross section (Fig. 1a) to which

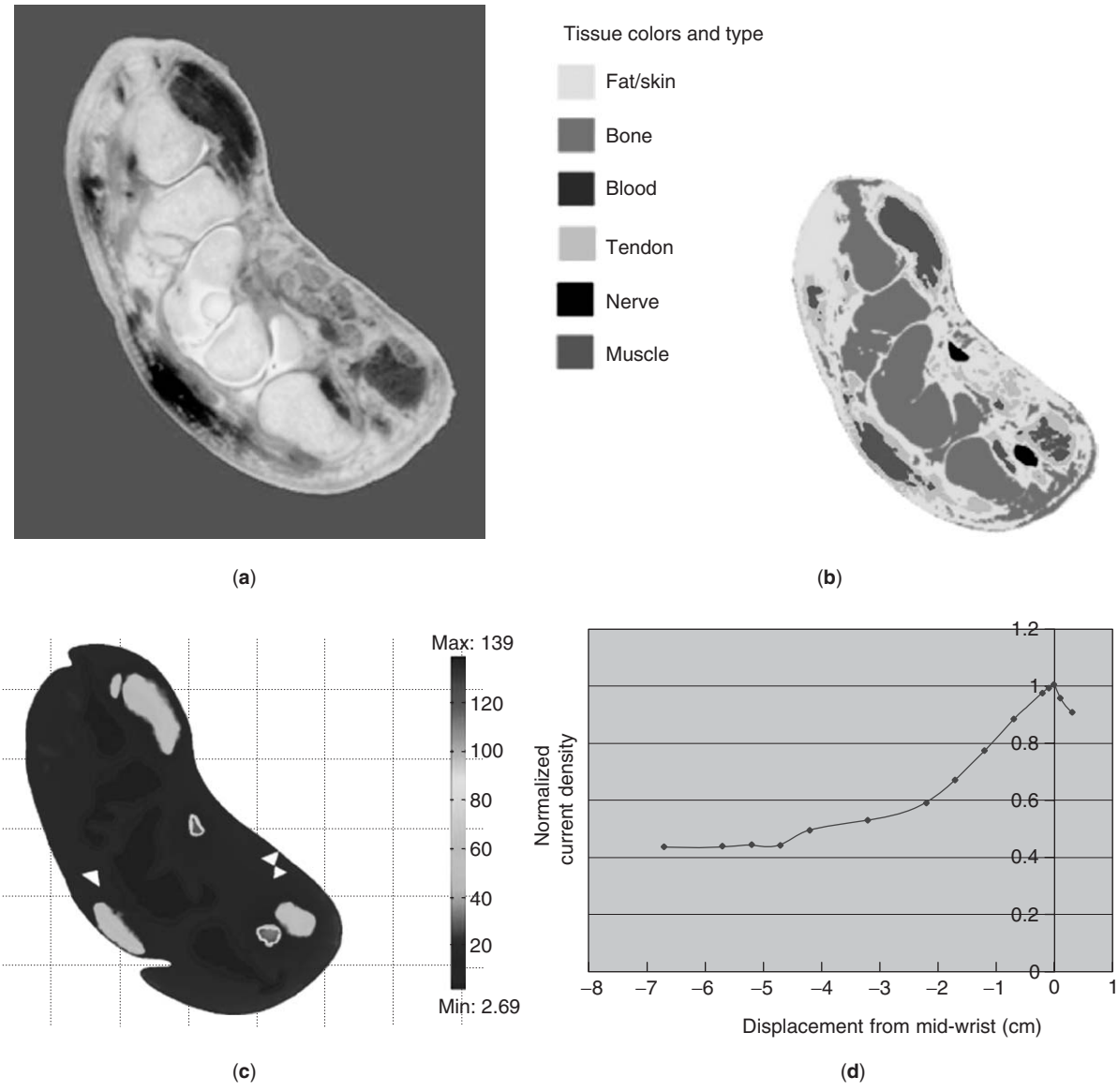

Figure 1. (a) Cross section of carpal tunnel region. (b) Cross-section characterized by tissue type. (c) Finite Element Model of current flow demonstrating increased current density in the nerve tissue. (d) Nerve current density as a function of distance from carpal tunnel region.

known tissue resistivities (Table 3) were applied yielding a tissue-type diagram (Fig. 1b). A stimulus current is applied, yielding an FEM solution (Fig. 1c). Figure 1d is a plot of current densities in nerve tissue along the current pathway. The results indicate that the current density is highest in nerve tissue when compared with surrounding bulk muscle tissue and that current density peaks in the region of the carpal tunnel, which may explain the frequent diagnosis of carpal tunnel syndrome (CTS) found in individuals following hand-involved electric shocks.

5.2. Localization of Electrical Burns

When enough energy is added to biological tissue (beyond any amount removed by cooling mechanisms such as air or blood flow), the tissue will denature and burn. For each calorie added to any specific cubic centimeter of soft tissue, that tissue will increase approximately 1 degree centigrade if the energy is added fast enough to make the effect of the cooling mechanisms negligible. Although the discussion thus far allows for the determination of total energy added to the body, that energy is not evenly dispersed. Some tissues will barely change temperature whereas others will rapidly heat and burn as is seen by the presence of entry and exit burns even in fairly low-energy contacts. If enough energy is added to the body, gross systemic heating will occur along and near the pathway of the current with the result that any organ system can suffer thermal damage from the added energy (43–45).

When considering which tissues will burn first, always look to the entry and exit points where the current density

is usually the greatest. Although the current will disperse through the volume of bulk tissues, all current must enter and exit the body through the entrance and exit points. If the entry or exit points are small, the current density can be quite large, even for a low-voltage contact.

5.3. Example: Localized Tissue Heating

For the electrical contact example given earlier, the current entered and exited through $1\,cm^2$ areas. Each area posed a resistance of 150 ohms. The current density would be $150\,mA/cm^2$. The energy dissipated at the entry (or exit) would be calculated as:

$$\begin{aligned}\text{Energy} &= I^2 \times R \times T \\ &= (.15\text{ Amps})^2 \times (150\text{ Ohms}) \times (3\text{ Seconds}) \\ &\quad \text{Watt seconds} = 10.125\text{ Watt seconds} \\ &= 10.125\text{ joules} \\ &= 10.125\text{ joules} \times .24\text{ calories/joule} \\ &= 2.43\text{ calories.}\end{aligned}$$

Assuming the tissue at the entry point was .2-cm thick, the total volume of the tissue absorbing the 2.43 calories would equal $1\,cm^2 \times .2\,cm = .2\,cm^3$.

The temperature increase is calculated as:

$$\begin{aligned}\text{Temp. Increase} &= (2.43\text{ calories})/(.2\,cm^3) \\ &\quad \times 1\text{ degree Centigrade/calorie/cm}^3 \\ \text{Temp Increase} &= 12.15\text{ degrees centigrade.}\end{aligned}$$

If the skin was at 37°C prior to the shock and assuming cooling effect was negligible, the final temperature at the entry (or exit) point would be $37^\circ C + 12.15^\circ C = 49.15^\circ C$.

The resultant temperature is just below that needed for the tissue to burn, but was not sustained long enough to actually inflict a burn. The additional energy could explain the observed tissue reddening.

6. THE FUTURE OF ELECTRIC SHOCK RESEARCH

Research into electrical injury has been going on since we started generating electricity for commercial purposes and has now spanned more than one century. (The first death related to commercially generated electricity occurred in 1879.) Until the last decade of the twentieth century, it was largely assumed that this was a field where the great advances were in the past. Today, we realize how little we truly know about the effects of electricity on the human body. Our ability to engineer barely touches the level and complexity at which humans have been engineered. The great unanswered question of what pathway(s) current truly follows during an electrical contact still remains unanswered. Although we know the gross mechanisms of electrical injury, recent research suggests that multiple mechanisms may exist that are undetectable by our current diagnostic technology. Such mechanisms are yet to be discovered and explained. The effects of electricity on the human body reach far beyond that which we now understand or can even anticipate.

BIBLIOGRAPHY

1. M. S. Hammam, A range of body impedance values for low voltage, low source impedance systems of 60 Hz. *IEEE Trans. Power Apparatus Syst*. 1983; **PAS-102**(5):1097–1105.
2. M. A. Cooper and T. Price, Electrical and lightning injuries. *Emerg. Med. Clin. North Am*. 1984; **2**:489–501.
3. F. Panse, Electrical trauma. In: *Handbook of Clinical Neurology - Injuries of the Brain and Skull, Part I,* vol. 23. 1975, pp. 683–729.
4. T. Skoog, Electrical injuries. *J. Trauma* 1970; **10**(16):816–830.
5. E. Somogyi and C. G. Tedeschi, Section 4. Injury by electrical force. *Forensic Medicine, A Study in Trauma*, vol. 1. pp. 645–676.
6. C.F. Dalziel, Dangerous electric currents. *AIEE Trans.* 1946; **65**:579–584.
7. R. C. Lee, E. G. Cravalho, and J. F. Burke, *Electrical Trauma, the Pathophysiology, Manifestations, and Clinical Management*. New York: Cambridge, 1992.
8. D. W. Bodle, A. J. Ghazi, M. Syed, and R. L. Woodside, *Characterization of the Electrical Environment*. Toronto, Canada: University of Toronto Press, 1976, Chapter 4, pp. 208–250.
9. I. Damjanovv and J. Linder, eds. *Anderson's Pathology,* 10th ed. St. Louis, MO: Mosby-Year Book, 1996.
10. J. G. Webster, *Medical lnstrumentation*. Boston, MA: Houghton Mifflin, 1978, Chapter 13, pp. 667–688.
11. T. Bernstein, Effects of electricity and lightning on man and animals. *J. Forens. Sci*. 1973; **18**(1):3–11.
12. J. C. Keesey and F. S. Letcher, Human thresholds of electric shock at power transmission frequencies. *Arch. Environ. Health* 1970; **21**:547–552.
13. P. Reilly, Scales of reaction to electric shock: thresholds and biophysical mechanisms. *Ann. NY Acad. Sci*. 1994; **720**:21–37.
14. M. A. Chilbert, et al. Fibrillation induced at powerline current levels. *IEEE Trans. BME* 1989; **36**(8):864–869.
15. T. Bernstein, Electrical shock hazards and safety standards. *IEEE Trans. Educ*. 1991; **34**(3):216–222.
16. C. W. Walter, Questions and answers: is death from accidental electric shock instantaneous? *JAMA* 1972; **221**(8):922.
17. A. Bradford and R. G. O'Regan, The effects of low voltage electric shock on respiration of the anaesthetized cat. *Quart. J. Exper. Physiol*. 1985; **70**:115–127.
18. R. K. Wright and J. H. Davis, The investigation of electrical deaths, a report of 220 fatalities. *J. Forens. Sci*. 1980; **25**(3):514–521.
19. R. C. Lee and M. S. Kolodney, Electrical injury mechanisms: electrical breakdown of cell membranes. *Plast. Reconstr. Surg*. 1987; **80**(5):672–679.
20. D. F. Farrell and A. Starr, Delayed neurological sequelae of electrical injuries. *Neurology* 1968; **18**:601–606.
21. M. S. Morse and J. S Morse, Diffuse electrical injury: a study of 89 subjects reporting long-term symptomatology that is remote to the theoretical current pathway. *IEEE Trans. Biomed. Eng*. 2004; **51**(8):1449–1459.

22. M. K. Kelly, N. H. Pliskin, G. Meyer, and R. C. Lee, Neuropsychiatric aspects of electrical injury. *Ann. NY Acad. Sci*.1994; **720**:213–218.
23. N. H. Pliskin, G. J. Meyer, M. C. Dolske, R. L. Heilbronner, K. M. Kelley, and R. C. Lee, Neuropsychiatric aspects of electrical injury – a review of neuropsychological research. *Ann. NY Acad. Sci.* 1994; **720**:219–223.
24. M. Primeau, G. Engelstatter, and K. Bares, Behavioral consequences of lightning and electrical injury. *Sem. Neurol*. 1995; **15**(3):279–285.
25. M. Morse and D. K. Weiss, An evaluation protocol for electric shock injury supported by minimal diagnostic evidence. *Proc. IEEE Engineering in Medicine and Biology Conference,* San Diego, CA, October 1993.
26. C. F. Dalziel, Electric shock hazard. *IEEE Spectrum* 1972; Feb:41–50.
27. M. A. Cooper, Emergent care of lightning and electrical injuries. *Semin. Neurol*. 1995; **15**:268–276.
28. T. A. Bernstein, Investigation of alleged appliance electrocutions and fires caused by internally generated voltages. *IEEE Trans. Industry Appl*. 1989; **25**(4):668.
29. P. A. Tipler, *College Physics.* New Jersey: Worth, 1987, p. 467.
30. G. Yan, et al., A clinical analysis of 836 cases with electrical injury. *Ann. NY Acad. Sci*. 1999; **888**:88–99.
31. M. Capelli-Schellpfeffer, G. Miller, and M. Humilier, Thermoacoustic energy effects in electrical arcs. *Ann. NY Acad. Sci.* 1999; **888**:19–31.
32. R. H. Lee, Pressure developed by arcs. *IEEE Trans. Industry Appl*. 1987; **IA-23**(4):760–764.
33. R. K. Wright, Death or injury caused by electrocution. *Clin. Lab. Med*. 1983; **3**(2):343–353.
34. L. B. Gordon, Electrical hazards in the high voltage laboratory. *IEEE Trans. Ed*. 1991; **34**(3):231–242.
35. C. F. Dalziel, Electric shock. *AIEE Trans*. 1941; **60**:1073–1078.
36. B.W. Blount, Lightning injuries. *Am. Fam. Phys*. 1990; **42**:405–415.
37. W. Weeks and L. Alexander, The distribution of electric shock in the animal body; an experimental investigation of sixty cycle alternating current. *J. Indust. Hyg. Toxicol*. 1939; **21**(10):517–525.
38. L. A. Geddes and L. E. Baker, The specific resistance of biological material- a compendium of data for the biomedical engineer and physiologist. *Med. Bio. Eng.* 1967; **5**:271–293.
39. L. Baker, Principles of the impedance technique. *IEEE Eng. Med. Bio*. 1989: 11–13.
40. M. S. Morse, J. S Morse, and R. L. TenWolde, Analysis of current density in the carpal tunnel region during an electrical accident by way of the finite element method. *Proceedings of the 25th IEEE Engineering in Medicine and Biology Conference*, Cancun, Mexico, September, 2003.
41. R. C. Lee, Physical mechanisms of tissue injury in electrical trauma. *IEEE Trans. Ed*. 1991; **34**(3):223–230.
42. R.W. Beausoliel and W. J. Meese, Survey of ground fault circuit interrupter usage for protection against hazardous shock. Government Report: NBS-BSS81. July 1976.
43. K. Diller, The mechanisms and kinetics of heat injury accumulation. *Ann. NY Acad. Sci*. 1994; **720**:38–55.
44. R. C. Lee, G. Russo, and G. Kicska, Kinetics of heating in electric shock. *Ann. NY Acad. Sci*. 1994; **720**:56–64.
45. B.I. Tropea and R.C. Lee, Thermal injury kinetics in electrical trauma. *J. Biomech. Eng.* 1992; **114**:241–250. *bsol*; *rmWatt – seconds*