

A review of hazards associated with exposure to low voltages

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OVERVIEW

This review summarizes peer reviewed papers, government reports, and regulatory group recommendations on hazards from electricity. A goal of this report is to determine a **safe voltage level** below which these hazards will not occur; the review emphasis is, therefore, on ‘extremely-low’ voltage (<50 V_{RMS} or 71 V_{PEAK}) exposure. This report is divided into five main sections dealing with human exposure. SECTION I addresses the basic mechanisms by which electric current can affect biological tissue in a hazardous manner. SECTION II summarizes experimental research studies involving application of electric current to human subjects. SECTION III reviews the epidemiology and case reports of human electrocution. SECTION IV includes a summary of previous electrical safety standards. SECTION V includes the review conclusions for human exposure. An APPENDIX deals with the electrocution of dogs.

Review Scope

This review and its summary conclusions relate only to adverse effects of transdermal current exposure¹. This review is not concerned with electric shocks that cause no long-term hazardous effects (e.g. sensory sensations such as noxious stimulation and phosphenes). Moreover, this review does not include injuries that result from humans being startled by otherwise non-hazardous electrical current (e.g. falls) or interference with medical devices. This review only includes scientific reports which: 1) appeared in scientific journals; 2) include recommendations by a (inter)nationally recognized

¹ When electricity enters the body subdermally, as for example through two needles inserted into the heart, voltages as low as 20 V and currents as low as 100 μ A can cause fibrillation (Camps et al. 1976). With electrodes placed directly on the heart, ventricular fibrillation is usually achieved with voltages of ~0.2 V and current flow of 80-600 μ A (Kugelberg 1976; Webster 1998).

scientific organization; or 3) were sponsored by a government agency. This review does not address resuscitative measures, forensic diagnosis, or electric safety measures. Lightning strikes, high-voltage arcs, and electrical fires/explosions are not considered.

SECTION I: BASIC MECHANISMS BY WHICH ELECTRIC CURRENTS AFFECT BIOLOGICAL TISSUE

Electrically Excitable Tissue / Burns

The human body will conduct electricity. If the body makes contact with an electrically ‘energized’ surface while simultaneously making contact with another surface at a different potential (or ‘ground’) then an electric current will flow through the body, entering the body at one contact point, traversing the body, and exiting at the other contact point. The magnitude of this current will increase as the voltage difference across the ‘contact points’ increases. This section introduces potential hazards associated with such currents.

Certain tissues in the body have traditionally been considered most sensitive to electricity because they normally use bio-electric signals. Cells in the central and peripheral nervous system (neurons) use bio-electrical signals to rapidly process and communicate information. Neurons regulate the contraction of cardiac cells, diaphragm muscle cells (inducing lung inspiration), and peripheral muscle cells (controlling movement). Cardiac and muscle cells, in turn, also use bio-electric signals to trigger their contraction. These cells are collectively referred to as ‘electrically excitable cells’ (Hille 2001)

‘**Electric stimulation**’, or ‘**electrical shock**’, results when a portion of the current conducted by the body passes through/polarizes excitable cell membranes. Theoretical and forensic studies examining the effects of electricity on biological tissue have thus focused on systems containing or regulated by excitable cells. For example, electric shock can lead to activation of

neurons/muscles involved in respiration or cardiac pacing. Electric shocks can acutely affect cell function without necessarily damaging these cells.

Electric currents may also heat external and internal tissue sufficiently to induce structural damage through **electrical burns**. Electrical burns affect human health through actions on both excitable (e.g. cardiac, nervous) and non-excitable (e.g. skin, blood vessels) tissues.

Electroporation refers to changes in cell membrane permeability by electric fields. Electroporation affects both excitable and non-excitable cell function and can lead to irreversible cell damage (Chilbert 1998). The role of irreversible electroporation in transdermal electrical accidents remains unclear and is thought to be limited to high voltages/currents (>200 mA; Reilly 1998).

“Electrocution” refers to fatalities resulting directly from ‘lethal’ current flow through the body.

Conventions, Metrics, and Cellular Models

The theory governing the interaction of electric current with excitable cells (electric shocks) has been well characterized by my group (Durand and Bikson 2001; Bikson et al. 2004) and others (Reilly 1998; Rattay 1999; McIntyre and Grill 1999). The consensus of these reports is that the effects of electric currents can be directly calculated using information about the detailed cell geometry/biophysical properties and detailed information about the electrical potential (induced by current flow) along this geometry; all with *micrometer resolution*². Unfortunately, this combined physiological/electric-potential data is not available for human exposure. Moreover, theoretical consideration of the detailed electric potential induced under various exposure conditions and their effects on every excitable cell in the body is intractable (but see Reilly 1998, p334).

Therefore, the approach taken by previous research reports and regulatory groups has been to assume a (quasi-) uniform electric field (E in volts/meter) across the tissue of interest (but see Reilly and Diamont 2003).³ This assumption may

² Specifically, information about the second derivative of the extracellular potential in space can be related to the ‘excitation strength’, the amount of current moving across a cell membrane.

³ Note that this assumption ignores a central concept that it is the second special derivative of voltage (i.e.

be grossly valid for currents passing across the entire body. It significantly simplifies the analysis of electric current effects because it allows standardization across disparate experimental studies where similar ‘uniform’ fields were used. In addition, this assumption facilitates the establishment of safe exposure levels using a single number, such as the uniform electric field strength.

Electric field (E in units of V/m) can be related to current density (J in $A\ m^{-2}$) by: $E = J \cdot \rho$ where a homogenous volume resistivity (ρ in $\Omega \cdot m$) is assumed. Current density can, in turn, be related to current (I in A) across the *entire* tissue through knowledge of the tissue geometry, notably cross-sectional area (A_{cs} in m^2), and current entry/exit locations. The voltage (V in V) across the entire tissue can be theoretically related to current I through a tissue with a total path resistance R by $V = I \cdot R$. Finally, for a cylindrical block, total path resistance R , can be related directly to *uniform* resistivity ρ by $R = \rho \cdot d / A_{cs}$ where d is the path length (in meters).

‘Stray voltage’ refers to unintended electrical potentials between contact points that may be encountered by humans or animals. Accidental electrocution can result when stray voltages exceed a **safe threshold voltage level**. This report focuses on determining this safe voltage level as stray voltages are readily measurable during quality assurance (Con Edison 2004). The internal electric field will depend not only on the contact voltage magnitude but also on contact geometry/tissue properties; ‘safe voltage levels’ may thus vary depending on exposure/subject conditions. Additional consideration must be exercised in considering safe voltage levels (as opposed to safe current levels); note that an individual wearing electrically insulating gear (e.g. rubber gloves) can potentially be exposed to higher voltages without hazardous effects; this is because the total resistance including the insulating gear will be much higher than the body resistance alone, and the resulting current flowing through the body will be significantly reduced. The skin itself (see *Body resistance* below) can also provide a significant amount of electrical insulation.

During exposures of high intensity and long duration, tissue heating may be a mechanism of injury. The heating of tissue by electric currents (burns) is related to the total amount of energy

the first derivative of a “*non-uniform*” electric field) that is critical in determining excitation strength.

delivered to the tissue and the thermal properties of the tissue. The rate at which energy is delivered by current flow at any instant is given by its instantaneous power ($p(t)$ in Joules/s or W) which can be calculated from $p(t) = v(t) \cdot i(t)$ (equivalently $i(t)^2 \cdot R$ or $v(t)^2/R$) where $v(t)$ is the instantaneous voltage and $i(t)$ the instantaneous current. The total energy (in Joules) over time delivered to a system can be calculated by integrating this instantaneous power over time (T).

In the case of constant voltage and current (dc), total energy can be calculated⁴ from $V \cdot I \cdot T$. In the case of alternating voltage and current (ac), a sinusoidal current, through a specific resistive path, will deliver the same amount of power as a constant (dc) current with an amplitude 0.707 times the peak sinusoid amplitude, irrespective of frequency. The root-mean-square (RMS) value of a sinusoid is calculated as 0.707 times its peak.

A majority of this energy will be converted into heat causing a rise in tissue temperature. However, the body actively regulates temperature, thus a complete theoretical analysis of electric current induced tissue heating may be complex; key bio-heat models have been previously developed at The City College of New York (Arkin et al. 1994). Large temperature rises can lead to cell death.

It is accepted that all effects of electricity are aggravated as the amplitude of the current/voltage is increased. Consistent with industry standards⁵, “low voltage” refers to voltages under 1000 V_{RMS} . “Very-low” voltage is defined here as between 50-1000 V_{RMS} (71-1414 V_{PEAK}); many household and commercial electric systems operate in this range. “Extremely-low voltages” are defined here as below 50 V_{RMS} (71 V_{PEAK}).

⁴ Or alternatively as $Q = J^2 \rho T$ where Q is ‘thermal energy density’ in $J m^{-3}$.

⁵ In the IEEE Standard Dictionary (IEEE Std 100-), “high voltage” is defined as follows: for maintenance of energized power lines, as voltage levels above 1000 V_{RMS} , and for system voltage ratings as a class of nominal system voltages from 100 to 230 kV V_{RMS} . “Medium voltage” is defined as a class of nominal system voltages from 1000 V_{RMS} to 100 kV V_{RMS} . “Low voltage” is defined as a class of nominal system voltages 1000 V_{RMS} or less.

Particular attention is paid in this review to hazards associated with extremely-low voltages.⁶

Current temporal waveform and exposure duration

Tissue heating depends on the RMS values of the current and little or not-at-all on its waveform/frequency (e.g. the RMS value of a sinusoid is independent of frequency). However, for electric stimulation (shocks) the waveform of the current can have a profound influence on current efficacy (Bikson et al. 2004). AC (e.g. 60 Hz sinusoidal) is considered more likely to induce hazardous electric shocks than dc current (Camps et al. 1976; Reilly 1998). Dalziel and Lee (1969) found 10-400 Hz currents most effective in inducing involuntary hand muscle contraction. DiMaio and DiMaio (2001) considered 39-150 Hz the most lethal. Kugelberg (1976) found frequencies between 12-60 Hz most effective in inducing fibrillation of the human heart. The two competing factors relating to stimulation frequency efficacy are: 1) as electrical excitation occurs during the rising or falling phase of the current flow, increasing frequency increase the amount of potential excitations per time (e.g. involuntary muscle contractions); 2) excitable cell membranes act as low-pass filters (Bikson et al. 2004), higher frequencies are thus less effective. Unless otherwise stated the results reviewed here refer to 50/60 Hz sinusoidal waveforms (as are used in power distribution systems).

The duration of exposure will influence both the electric shock and burning mechanisms. Increasing exposure time will result in more energy delivered to the tissue; if the tissue cannot dissipate this energy, it will continue to heat up and eventually burn (see *Conventions, Metrics, and Cellular Models*). The long term effects of electric shocks are more complex (Durand and Bikson 2001). As exposure

⁶ This review reports voltage/current values in both ‘PEAK’ and ‘RMS’. In reviewing previous reports, numbers explicitly given in RMS have been converted to PEAK. When unspecified in the original report, numbers are exactly reproduced here. This approach is consistent with: 1) an appreciation that electric shocks (rather than heating) and hence peak voltage (rather than RMS) is most relevant for extremely-low voltage electrocution; and 2) an attempt to develop a safe threshold voltage (RMS values are always equal to or less than peak values). Thus, in this review, unless otherwise specified, voltage should be read as PEAK.

time increases the chance of an electric shock precipitating a 'stochastic' reaction increases.

The shorter the exposure duration the greater the current needed to induce ventricular fibrillation (see cardiac results below; Camps et al. 1976). DiMaio and DiMaio (2001) suggest that for either 120 mA or 1.2 A exposure, ventricular fibrillation could occur in 4 s and 0.1 s respectively. Dalziel and Lee (1969), extrapolating from animal studies (APPENDIX), reported that, for exposure times (T in seconds) between 8.3 ms and 5 s, the lower 0.5% population-rank current threshold ($I_{0.5f}$ in mA) for induction of fibrillation is given by:

$$I_{0.5f} = k / \sqrt{T} \quad (1)$$

where K is a constant linearly related to victim weight (see *Individual Susceptibility* below) and presumably to contact geometry (see *Current Path* below). Dalziel and Lee (1969) concluded that "ventricular fibrillation in a normal adult [>50 kg] worker is unlikely if the shock intensity is less than $116/\sqrt{T}$ milliamperes [RMS]." In addition, for longer duration (5 to 30 s) exposures, the threshold may remain fairly steady. Biegelmeier and Lee (1980) proposed an inverse relationship between fibrillation threshold and exposure time ($I_f \propto T^{-1}$) for exposure periods between 0.2 and 2 s; and a threshold plateau above and below these times (a 'Z' curve).

The 'direct' effects of electric shocks terminate once contact with energized source ends. In some cases, induced pathologies will persist (e.g. fibrillation). In other cases, normal function resumes immediately after termination of stimulation (see respiratory arrest).

Body resistance/related susceptibility factors

Though the effects of electricity are more directly standardized by reference to current, for the reasons discussed above, it is often necessary to consider contact voltage levels. Ohm's law (voltage = current · total path resistance) establishes a simple relationship between these two factors. Total path resistance is thus a critical factor in determining voltage safety levels. As a result of body/electrode contact geometry and tissue inhomogeneity, the current density across any given tissue will vary. Regardless, for any given voltage, decreasing body

resistance will increase overall current magnitudes and hence the risk of electrocution.

Generally tissue inhomogeneity is ignored and resistance is reported as the total current path resistance, the 'body resistance', between two electrodes on the body surface. Note that measured total body resistance includes the resistance of the electrodes (see below). 'Internal body resistance' refers to the total current path resistance excluding the skin. Experimentally, the skin resistance can be removed by abrasion. For measuring 'total' body resistance, common electrode positions include hand-to-hand and hand-to-foot.

During electrocution the total current path resistance will be the sum of the body resistance including the skin, the electrode interface resistance, and the resistance of any other materials along the current path such as cloths or soil. High resistance clothing (e.g. rubber or dry leather gloves, dry shoes) provides increased protection against electric hazards; e.g. >1 M Ω (Reilly 1998). As this review will focus on worst-case scenario exposure, protective gear and insulating clothing are not considered further.

There is an expected variation in resistance across subjects. In addition, individual body resistance is a complex function of electrode size, electrode material, electrode position, skin and its surface conditions, temperature, and applied voltage magnitude/frequency (Biegelmeier 1985a; Reilly 1998; Webster 1998). Moreover, skin resistance will vary with exposure time⁷. The following section addresses the general range of resistances that would be relevant during extremely-low voltage electrocution at frequencies <100 Hz. In experimental studies, "wet" conditions are usually achieved using conductive NaCl or saline solution.

The presence of moisture from wet clothing, high humidity, or perspiration will decrease the electrical resistance of the electrode interface and of the skin; this will increase current magnitudes and thus the risk of electrocution. High-humidity is a common feature in very-low voltage electrocutions and ubiquitous with extremely-low voltage electrocution (SECTION III). The increased frequency of low-voltage electrocution in summer

⁷ The total opposition to the flow of ac current is denoted impedance, which in general is a complex quantity with a magnitude and phase. For evaluating electrocution only the magnitude of the body impedance (rather than the phase) is critical; this magnitude is a real number.

has been suggested to relate to sweating (Wright and Davis 1980; Fatovich 1992). Also, the resistance of the skin is significantly reduced by abrasion/cuts.

The passage of significant current can reduce skin resistance to negligible values (>200 V, Biegelmeier 1985a; >240 V, Webster 1998; 100-250 V, Reilly 1998; Camp et al. 1976). Thus with higher voltages, skin conditions are considered to play no significant role (DiMaio and DiMaio 2001). Gettman (1985) noted that below 12 V skin resistance would not break down.

Skin can provide a significant source of insulation. Skin has a *surface resistivity* (in $\Omega\text{-cm}^2$). Skin *resistance* is a function of contact area; larger contact areas result in reduced effective total skin resistance. Camp et al. (1976) noted that if the palms are thickened by manual labor the dry skin resistance can reach 2000 k Ω . DiMaio and DiMaio (2001) concluded that for 120 V, dry skin may have a resistance of 100 k Ω ; dry and calloused skin up to 1000 k Ω ; moist skin 1 k Ω or less; and moist, thin skin as low as 100 Ω . Wright and Davis (1990) considered the minimum internal resistance of the body 500 Ω and minimal hands/feet resistance 1 k Ω ; dry skin easily reaching 100 k Ω .

Webster (1998) noted dry skin resistance (one square centimeter area) may range from 15 k Ω to 1 M Ω ; if the skin is wet or broken this resistance drops to as low as 1% of the value of dry skin. Webster (1998) considered the internal resistance of the body (between any two limbs) 500 Ω , with each limb contributing ~200 Ω and the trunk ~100 Ω .

Using large-area (>80 cm²) electrodes and for low voltages, Biegelmeier (1985a) reported a 'initial' (analogous to internal) body resistance of 781 \pm 114 Ω and 637 \pm 98 Ω for 25 V and 15 V respectively (average \pm standard deviation; hand-to-hand path). Reviewing data on the distribution of internal resistance, Biegelmeier (1985a) concluded that ~50% of total body internal resistance (limb-to-limb) resides in the wrists and ankles (e.g. 25% of hand-to-hand total path resistance is across each wrist); the body trunk accounted for <10% of total limb-to-limb internal resistance. Using a simplified model, Biegelmeier (1985a) considered 2 hand-to-2 feet and 2 hand-to-trunk internal resistance 50% and 25% of total hand-to-hand resistance, respectively. As contact area decreased, the total body resistance increased significantly (for a 0.01 cm² electrode resistance was >1000 k Ω at 100V).

Osyka (1963) reported a total-body-impedance of ~2 k Ω (hand-to-hand) and 500 Ω (2 hands-to-2 feet) under wet conditions (60 Hz, ~10V).

Freiberger (1934) reported an average hand-to-hand and hand-to-foot internal body resistance (skin ablated) of ~1 k Ω ; these measurements were conducted on cadavers which could result in an overestimate of resistance in living persons (Reilly 1998). Freiberger (1934) examined the distribution of internal resistance; he concluded that ~50% of the total internal body resistance resides in the wrists and ankles. The body trunk resistance was <10% of the total internal body resistance. Freiberger's statistical data was incorporated by the International Electrotechnical Commission (Table 1).

Table 1. Statistical data for total body impedance as adopted by the International Electrotechnical Commission (CEI-IEC 479-1; 1994). Large area contacts, hand-to-hand, dry skin, ac 50/60 Hz.

Touch voltage (V)	Total body impedance (Ω) not exceeded by indicated percentile rank		
	5%	50%	95%
25	1,750	3,250	6,100
50	1,450	2,625	4,375
75	1,250	2,200	3,500
100	1,200	1,875	3,200

Using short-duration high-voltage discharges in living people, Taylor (1985) found similar resistance distributions across the body as Freiberger (1934) and Biegelmeier (1985a). Taylor (1985) reported a total body resistance of 470 \pm 36 Ω (mean \pm S.D.; hand-to-hand) and 516 \pm 55 Ω (left hand-to-left foot). Taylor (1985) considered 70-100 of these resistances coming from wet skin.

Hart (1985) reported an internal resistance of 400-500 Ω (hand-to-hand) and 450-500 Ω (hand-to-foot). He found internal body resistance from hand-to-forearm was 140 Ω and from finger-to-forearm 700-800 Ω .

In the majority of the above studies contact area was relatively large for the purposes of reducing electrode resistance (see below) and reproducing 'worst-case' condition. Using data from animal models (100 V, dry contact), Prieto et al. (1985) found total body resistance increased with decreasing contact area (total path resistance \propto area^{-0.32}) and contact circumference (total path resistance \propto circumference^{-0.51}).

Statistical impedance data was developed by Underwriters Laboratories (12 V_{DC}, relatively large electrodes, 'wet' conditions; Whitaker 1939)

for children (3-15 years; 14-58 kg) and for adults (18-58 years; 45-95 kg). For children they found a resistance variation from the 5% to 95% rank of 1.7 to 4.47 k Ω (hand-to-hand) and 0.9 to 2.04 k Ω (two hand-to-two feet). For adults they found a resistance variation from the 5% to 95% rank of 1.28 to 2.45 k Ω (hand-to-hand) and 0.63 to 1.16 k Ω (two hand-to-two feet). 60 Hz AC resistance values can be 60-90% of these DC resistance values (Reilly 1998).

Thus the majority of research reports consider worst-case (large contact area, wet conditions) total body resistance (limb-to-limb) to be slightly greater than 500 Ω . Worst-case resistance across the chest can be less than 100 Ω . Under non-worst-case conditions (e.g. small contact size, dry skin) total body resistance values quickly increase to greater than 2 k Ω .

Electrodes / Metal-body interface

Metal in contact with tissue is referred to as an 'electrode'. In the metal electrode and in attached electrical circuits, charge is carried by electrons. In the body, charge is carried by ions, notably sodium, potassium, and chloride ions. During the passage of current between the metal electrode and tissue, as occurs during electrocution, the central process that occurs at the 'electrode-body interface' is a transduction of charge carriers from electrons in the metal electrode to ions in the body. We have recently reviewed this process (Merrill et al. 2004).

Key to the present review is that this interface can 1) have a significant resistance and 2) can alter the waveform of the passing current. The magnitude of this resistance/distortion is a complex function of metal electrode composition, the magnitude of both the applied current and voltage, and effective electrode contact area (roughness); this later dependence would contribute to the increased total body resistance noted with small contacts above. To my knowledge the effect of the electrode-body interface on waveform has not been considered in the context of electrocution; changes in current waveform will affect electric shock thresholds. The resistance of the interface will act to increase the resistance of the total current path and thus protect from electrocution. Under 'worst-case-conditions' the interface resistance may be assumed to be negligible.

Mechanisms of injury/death by electrocution

Electrocution by extremely-low voltages (<71 V_{PEAK}) is significantly less common than by very-low (71-1414 V_{PEAK}) or higher voltages (SECTION III). Literature on the pathophysiology of electrical injury is therefore dominated by higher voltages. Unless otherwise specified, the comments in the following section do not necessarily apply to extremely-low voltage exposure.

The **cardiac** results of electrical injury may be fatal (Leibovici et al. 1995). Low-voltage exposure can induce **ventricular fibrillation**. High voltage/current electrical exposure can lead to **astole**, which renders the heart electromechanically silent. These heart pathologies can be elicited by stimulation of cardiac muscle cells or pacemaker neurons. Both pathologies may persist even after termination of current flow. Because normal blood pumping stops, if the victim is not actively resuscitated (e.g. defibrillated), unconsciousness can follow in 10-15 s (Wright and Davis 1980), irreversible damage can occur within 3 minutes (Geddes and Baker 1989), and death in 5-10 minutes (Dalziel and Lee 1969; Wright and Davis 1980). The brain and heart are particularly sensitive to anoxia (Cabanes 1985). The mechanisms of ventricular fibrillation have been reviewed elsewhere (Bridges et al. 1985).

Table 2 summarizes the currents considered sufficient to disrupt cardiac function. The threshold for inducing fibrillation is dependent on exposure duration (see *Exposure duration* above) and body weight (see *Individual susceptibility* below). Grouping all (high and low voltage) electrocutions, cardiac fibrillation is considered the most common cause of death (Fatovich 1992; Webster 1998). Blood vessels are also sensitive to electric currents (Leibovici et al. 1995).

Electric currents can induce **respiratory arrest** though both 'tetanic paralysis' of the respiratory muscles and damage to the respiratory control centers of the brain. In both cases death is asphyxial with the heart continuing to beat until death occurs. Irreversible respiratory system damage is considered rare (Camps et al. 1976); indeed the passage of several hundred milliamps through the brain during therapeutic electroconvulsive therapy rarely damages the respiratory control centers (Devanand et al. 1994). Tetanic paralysis is considered more common and requires continuous contact with the energized source (Wright and Davis 1980; DiMaio and DiMaio 2001). In contrast to cardiac results, the effects of current

on respiration have not been extensively studied. Dalziel and Lee (1969) reported that “18 to 22 or more milliamperes [RMS], flowing across the chest stopped breathing during the period the current flowed...normal respiration resumed upon interruption of the current, and no adverse after-effects were produced.” Cerebral injury, as result of hypoxia, can occur if the duration is more than 3 to 4 minutes (Cabones 1985). Table 2 summarizes the currents considered sufficient to induce tetanic paralysis of respiratory muscles.

Electric shock can induce violent **muscle contraction** and hemorrhage of muscle fibers (Leibovici et al. 1995; Karger et al. 2002). This can lead to dislocation of joints, fractures, and fatal/immobilizing falls. DiMaio and DiMaio (2001) summarize cases of fractures resulting from 50/60 Hz currents ranging from 110-440 V. In addition, violent muscle contraction can damage blood vessels; this can aggravate, in feed-forward manner, the direct effects of electric fields on blood vessels (Leibovici et al. 1995). Non-fatal hand muscle contraction is discussed below (‘let-go’ current). In the case of contact with an energized source on the floor, falls resulting from leg muscle contraction can subsequently expose a victim to currents across the chest (see *Current Path*).

Electric shock can be hazardous through actions on the **nervous system** (Leibovici et al. 1995). Stimulation of the vagus nerve (parasympathetic system) can induce slowing of the heart. Stimulation of sympathetic nerve fibers will have the opposite effect and can trigger vasoconstriction. Though occurring at relatively low current thresholds, the above effects are not necessarily fatal; autonomic influences may affect ventricular fibrillation threshold (see *Individual susceptibility*). Respiratory arrest (see above) can result from (reversible) electrical stimulation of CNS breathing centers or the phrenic/thoracic nerves which produces tetanic contraction of the diaphragm (Geddes and Baker 1989). Currents flowing through the CNS can induce cerebral edema, convulsions, coma, hysteria, amnesia, auditory and visual dysfunction, and transient loss of consciousness (Chilbert 1998); it should be emphasized that these effect has not been demonstrated for extremely-low voltages. With high currents transient or permanent neuropathy can occur, especially in limbs that were in the current’s path. Damage can result from neuronal electric shock, burns, or secondary to violent muscle contraction.

Eyes can be damaged by electric shocks when the voltage is greater than 200 volts (Leibovici et al. 1995; Boozalis et al. 1991).

It has been suggested that all high-voltage but only some low-voltage electrocutions result in visible burns on the body (Wright and Davis 1980; Fatovich 1992; DHHS 1998; DiMaio and DiMaio 2001). High-voltage (high-current) burns can result in limb amputation and death (Chilbert 1998). In deaths resulting from internal burning, external burns are expected; the exception being when the victims were under water or very wet (Karger et al. 2002; Leibovici et al. 1995). However, very-low voltage electrocutions, perhaps relating to the necessary exposure time, are generally associated with burns (SECTION III); electrical burns can also occur postmortem.

For low-voltages, local **skin burns**, usually limited to points of current entry and exit, are generally not the “vital” reaction (the direct cause of death). Wright and Davis (1980) calculated that the minimum current necessary to induce cardiac fibrillation (see above) would induce a peak temperature rise of only 0.145°C. Moritz and Henriques (1947) reported first-degree burns require exposure of the skin to 50°C for 20 s. Chilbert (1998) summarized: “cutaneous burns occur when the temperature is elevated for a sufficient length of time: 45°C requires more than 3h, 51°C requires less than 4 min, and 70°C requires less than 1s for injury.” Reilly (1998) noted that for currents passing across a limb, strong muscle contraction and severe pain due to electrical activation of nerves would occur at current levels well below those causing burning. Leibovici et al. (1995) suggested thermal injuries were the most harmful mechanism when grouping *both* low- and high-voltage exposures. However, the role (if any) of internal organ heating during extremely-low voltage contact remains unclear.

Current Path

The body acts as a volume conductor. The points of current entry and exit are important because 1) the current density will be highest nearest these points and 2) the direction of current flow (electric field) along excitable tissue will affect electric shock efficacy (Reilly 1998).

When current is applied at two points on the surface of the body only a small fraction of the total current flows through the heart (Webster 1998). Freiberger (1934) reported that for hand-to-feet and

foot-to-foot electrode contacts less than 8.5% and 0.4% (respectively) of the net current would travel through the heart.

Leibovici et al. (1995) reported that current passing through the thorax is associated with 60% of electrocutions, whereas for current passing from leg to leg it is 20%. These numbers do not address the over-all (including not fatal) chances of exposure at each geometry nor do they distinguish across high/low voltage exposure levels. Leibovici et al. (1995) note that though current density will be higher across limbs (due to smaller cross sectional area), the presence of vital organs in the torso accounts for lethality of trans-thoracic currents (see *Mechanisms of injury*).

Camps et al. (1976) concluded that for ventricular fibrillation the most dangerous current path is left arm-to-opposite leg; from arm-to-arm being 60% less lethal. Bailey et al. (2001) found a majority of victims died from current flow from upper-to-lower extremities. In contrast, Alexander (1941) noticed that more victims die from current flow from upper-to-upper extremities. As noted above, these findings would be more relevant if the prevalence of the current exposure pathway, *including survivors*, was known. The role of current path has been examined systematically in dogs (APPENDIX).

The resistance of the current path is highly dependent on contact location (see *Body Resistance*). A majority of extremely-low voltage electrocution cases (SECTION III) involved electrode contacts to the chest.

Demographics patterns, Individual susceptibility

Fatal electrocutions, particularly occupational, occur predominantly among 20-34 year old males (DHHS 1998; Taylor et al. 2002; Bailey et al. 2001; Fatovich 1992); this reflects the prevalence of this demographic in trades with greater exposure to electrical sources and a generally higher exposure to electrical equipment and machinery.

It has been suggested that pre-existing cardiac pathology can increase risk of death during or after electric shocks (Camps et al. 1976; Griffin 1985; Bailey 2001). As with other hazards, physical or mental fatigue or awareness will increase the chance of exposure; however it remains unclear if physical fatigue in humans increases susceptibility to electrocution. Experiments with dogs (APPENDIX) have shown that during common abnormal

physiologic conditions there is a reduced current threshold for producing ventricular fibrillation; in particular adrenergic stimulation ('flight or fight' reaction) and acidosis. In humans, physical exertion will produce acidosis while 'aggravating' electric shocks can trigger adrenergic stimulation (Wright and Davis 1980). Indeed, extremely-low voltage electrocutions are often associated with physical exertion and require prolonged contact (SECTION III). Reviewing previous studies, Griffin (1985) concluded physical stress, but *not* exercise (in the absence of ischemia) reduced fibrillation threshold, while alcohol may increase fibrillation threshold. Griffin (1985) notes that "vagal or parasympathomimetic activity tends to promote and increased ventricular fibrillation threshold opposing the decreased fibrillation threshold resulting from sympathetic activity."

Some studies using animals have shown that average fibrillation threshold increases with body weight (Webster 1998; Reilly 1998). Dalziel and Lee (1969), extrapolating from animal studies (APPENDIX), concluded that, the threshold for cardiac fibrillation is *linearly* related to weight; equation (1). Using data from 104 animals of several species (rabbits, monkeys, dogs, goats, ponies), Geddes et al. (1973) found the threshold current for fibrillation, at 5 s exposures, varies almost as the *square root* of body weight (W in kg):

$$I_f = bW^a \text{ mA}_{\text{RMS}} \quad (2)$$

where b and a are parameters based on electrode-position only (e.g. $b=29.7$, $a=0.51$ for right forelimb-to-left hindlimb path).

Increased mass/volume would be expected to decrease current density across any given tissue (SECTION I). Based on the assumption that fibrillation threshold is a function of current density and assuming a fixed shape/organ proportions across different sizes and weights, Bridges (1985) proposed a $2/3$ power weight relationship:

$$I_f \propto W^{2/3} \quad (3)$$

Dalziel's linear rule (1), Geddes' square root rule (2), and Bridges' $2/3$ power rule (3) are in reasonable agreement over a fairly wide range of body weights. Some researchers have argued that these threshold-weight rules are consistent across experimental species, thus suggesting they may be *quantitatively* extendable to man (Dalziel and Lee

1969). Other groups raise concerns that experimental animal models have radically different proportions between anatomical features, invalidating extension of these rules to human. Even a *qualitative* relationship between fibrillation threshold and weight has been challenged (Biegelmeier 1985b) based on the concept that the

fraction of current intersecting the heart will not vary with size/weight. It is generally accepted that fibrillation data from dogs (APPENDIX) may be used to establish lower safety limits for man.

See also comments on variations in *Body resistance* above.

Table 2. *Estimated effects of transdermal 60 Hz AC currents through the human body (in mA_{PEAK}, limb contact) in selected reports, reviews, and books*

	DHHS (1998)	Leibovici et al. (1995)	Bridges et al. (1985)	Camps et al. (1976)	DiMaio and DiMaio (2001)	Wright and Davis (1980)	Webster (1998)	Reilly (1998)
Barely perceptible	1		0.5-2		1	1	0.7-2	
Painful Sensation		5						
Tetanic muscle contraction / 'let-go' current	22 [@]	15	8-28 [@]	8-13 [@]	15-17	22 [@]	8-31 [@]	8-25
Paralysis of respiratory muscles	28 [@]		20-40 (debated)		50	28 [@]	25-31 [@]	25-31 [@]
Ventricular fibrillation (exposure time)	100	30-50	>70 (0.1s) 40-100 (∞)	70 (5 s)	75-100 (>5 s)	120 (0.1 s)	75-400	33 (5 s) [*] 807 (8.3ms)
Cardiac standstill / internal organ damage	2000				1000	>2000	1000-6000	

[@] (Presumably) based on work by Dalziel and colleagues; adjusted for 'Peak'.

^{*} Dalziel and Lee (1969). Lower 0.5% estimate for a small child. Values more than double for adults.

SECTION II: EXPERIMENTAL RESEARCH STUDIES INVOLVING APPLICATION OF ELECTRIC CURRENT TO HUMAN SUBJECTS.

Application of currents across the hands / 'Let-go' current

With experimental subjects, application of current either hand-to-hand or hand-to-foot can lead to: 1) direct activation of sensory fibers and hence discomfort and pain, subjects have a pain tolerance level at which they 'let-go'; 2) stimulation of involuntary contraction of both fore-arm flexor and extensors, when the stronger flexors dominate the subject may not be able to release, 'let-go', a grasped object. In studies conducted from the 1930's to 1950's, both criteria have been used to establish 'let-go' thresholds. The former is dependant largely subject motivation and is not directly relevant for accidental electrocution. Because subjects may become attached to an

energized source, the involuntary contraction 'let-go' threshold (either release grip or rotate handle) has been of interest in establishing electricity safety levels. Women have a lower average 'let-go' threshold than men; this could relate to both physical and motivational (physiological) factors (Dalziel and Lee 1969).

Gilbert (1939) used the release-grip endpoint in determining an average let go current of 21 mA. Whitaker (1939) using the release-grip endpoint current found a range of 8.4 mA to 14 mA, average 11 mA. Thompson using a rotate-handle endpoint reported an average 'let-go' current of 11.7 mA, maximum 28 mA. In the commonly referenced initial report of Dalziel (1938), using the release-grip criteria, the average endpoint was 17.7 mA, maximum 25 mA (male subjects). These reports were reviewed by Reilly (1998; Table 2). Dalziel and Lee (1969, 1972) summarized results from 124 males and 28 females; the average 'let-go' currents were 22.3 mA_{PEAK} male and 14.8 mA_{PEAK} female

while the lower 0.5 percentile values were 12.7 mA_{PEAK} male and 8.5 mA_{PEAK} female. Thus across studies 8.5 mA_{PEAK} appears a safe 'let-go' threshold.

Studies using pain criteria are relevant for electrocution from the perspective that subjects never experienced chronic adverse effects from the repeating testing. Grayson (1931) used current in range of 11.2 mA. Dalziel and colleagues used currents as high as 31 mA (Dalziel 1938; Dalziel and Lee 1969).

The usual 'let-go' test procedure had the subject firmly grip an energized conductor (often moist with saline solution). In most cases only subjects in good physical condition (circulatory, respiratory systems) were accepted. In applying these values to electrical safety standards several important points must be considered 1) the conditions of contact with a moist hand wrapped around a handle/wire represents a "worse case" scenario; 2) even at the 'let-go' current the subject may have been able to disengage their hand by moving other body parts; 3) physiological factors profoundly effect 'let-go' current levels (Dalziel and Lee 1969); most importantly, 4) in none of the above studies were repeated current exposures reported to result in acute or chronic health problems. Thus the 'let-go' current does not imply a hazardous level of current for healthy subjects⁸. Nonetheless the prospect of a subject becoming attached to an energized conductor is of concern and this value can be considered in that sense. Finally, current applied across the chest may have different thresholds for safety and mechanism of damage. The 'let-go' current does provide a *rough* indicator of the current levels necessary to tetanize muscle.

Gettman (1985), summarizing Underwriters Laboratories safety criteria for electric fences, indicated that acceptable output current decreases with increasing exposure duration. For 5 ms exposure duration, currents up to 77 mA were considered safe; whereas for 200 ms exposure, currents up to only 7 mA were considered safe. Gettman (1985) considered a current interruption of 750 ms sufficient to allow individuals to break contact with an energized wire.

Application of currents to measure resistance

⁸ Indeed in one anecdotal report a worker became attached to an energized overhanging wire which acted to break his fall and thus potentially prevent injury. Once assistance arrived and the worker was freed from the wire he suffered no severe chronic effects.

Numerous studies have repeatedly applied voltages to subjects to measure the resistive properties of the body (see also above). While these studies were not explicitly intended to evaluate the risks of electric shocks, they provide data regarding shocks that are non-hazardous. Indeed if shocks were considered reasonably likely to induce any injury, the studies would not have been conducted.

Underwriter Laboratories has routinely applied up to 17 V/21 mA (hand-to-hand, hand-to-foot) to adults and children (range 3 to 58 years; Whitaker 1939). Osypka (1963) applied voltages of ~10 V. Biegelmeier (1985a) used voltages up to 150 V (30 V routinely) for 100 ms.

SECTION III: REPORTS OF ACCIDENTAL HUMAN ELECTROCUTION.

The following section summarizes published reports and reviews of electrocution death. Only studies in which voltage levels were noted are reviewed here. It should also be emphasize that particular focus was placed in this review on extremely-low voltage electrocutions; however these represent a minority of electrocution deaths.

Based on the National Traumatic Occupational Fatalities (NTOF) surveillance systems of the U.S. Department of Health and Human Services (DHHS) National Institute of Occupational Safety and Health (NIOSH), 5,348 workers died of electrocution from 1980 to 1992 (DHHS 98-131). The NIOSH Fatality Assessment and Control Evaluation (FACE) program analyzed 211 electrocution fatalities from 1982 to 1994. The FACE reports concluded that 147 fatalities resulted from voltages over 600 V and 25 fatalities resulted from voltages between 110-120; no electrocution cases from voltages below 110 V were reported (DHHS 98-131). The FACE report concludes that most of these electrocutions "could have been prevented through compliance of existing OSHA, NEC, and NESC regulations; and/or use of adequate personal protective equipment (PPE)". Table 2 includes a summary of the DHHS (1998) estimates of the effects of 60 Hz ac currents.

Peng and Shikui (1995) reviewed cases of low-voltage electrocutions in China. Though they state that it is 'relatively rare to be electrocuted by voltages lower than 100 V', specific statistics or the methods by which they identified cases were not presented. Peng and Shikui (1995) presented 7 cases of electrocution by AC or DC voltages ranging from

25-85 Volts. In all cases, the contact site was on or near the chest, the contact time was “long”, and skin burns were observed. In addition the authors note that all victims were working in an enclosed, high humidity and high temperature environment which would 1) increase susceptibility to electric shock through decreased skin resistance; 2) decrease reaction time and ability to disengage from the voltage source; 3) increase the chance of heatstroke and unconsciousness as a result of exertion/fatigue. The victims were otherwise healthy 20-41 year old males. The authors note that autopsies showed “congestion of internal organs and some focal hemorrhage. These changes comforted asphyxial death... ventricular fibrillation might also exist.”

Bailey et al. (2001) reviewed 124 electrocution deaths in Quebec between 1987 and 1992; 25 fatalities occurred at 240-120 V (RMS), the lowest voltage range reported. Transthoracic currents (59%) and wet extremities (50%) were more common in the 240-120 V range than for fatalities resulting from higher voltage (>240 V) electrocutions, while burns were less common for low voltage electrocution.

Karger et al. (2002) re-examined 36 cases of German electrocution fatalities from 1972-1997. Twelve of these fatalities (32%) occurred in the voltage range of 65-1000 V, the lowest voltage range reported. In this lower range, burns were less common than in the higher (>1000 V) range.

Fatovich (1992) summarized 104 electrocution fatalities in Western Australia from 1976 to 1990. He reported that 88% of victims were exposed to voltages less than 1000 volts.

Wright and Davis (1980) investigated 220 deaths by electrocution in Dade County, Florida. They report that 108 deaths resulted from voltages below 1000 V.

It is important to note that in all the above reports, voltages were measured at some period after the electrical accident and with loads/geometries potentially different than during the electrocution. In some cases electrocution may be overlooked as a cause of death (Wright and Davis 1980) while in others it may be incorrectly reported as the cause of death. Thus while case reports of electrocution remain a decisive indicator of safe voltage thresholds, care must be taken in accepting their quantitative findings.

SECTION IV STANDARDS

There is no single internationally enforced electrical safety code⁹. In the United States, the Department of Labor Occupational Safety and Health Administration (OSHA) standards are federally enforced. In addition, individual states and municipal authorities have adopted specific codes, notably the National Fire Protection Association (NFPA) National Electrical Code (NEC).

Electrical safety codes often do not specify a ‘safe voltage level’ or fail to provide scientific justification for specified safe levels. In the latter case it is not possible to tell if specified ‘safe level’ incorporate an (arbitrary) ‘safety factor’. A safety factor refers to a reduction in specified exposure levels below levels scientifically established to cause harm. In addition, it is not always specified if standards are lowered to protect from non-hazardous (e.g. startling) electric shocks. In this section, underlined voltages result from codes which provide (minimal) scientific justification.

Standards for medical devices, which may be used under ‘open heart’ conditions¹, are peculiarly stringent¹⁰; these standards are not included in this review.

The 2004 OSHA “Electrical Standard; Proposed Rule” (29 CFR Part 1910) does not explicitly define a ‘safe voltage level’ but does specify: “except as elsewhere required or permitted by this standard, live parts of electric equipment operating at 50 volts or more shall be guarded against accidental contact by use of approved cabinets or other forms of approved enclosures.” OSHA specifications traditionally “draw heavily” from the NEC (NFPA 70) and the NFPA “Standard for Electrical Safety in the Workplace” (NFPA 70E).

The 2002 NEC (NFPA 70) applied reduced safety standards for exposure to extremely-low voltages (all uninsulated parts <71 V_{PEAK} / <50 V_{RMS}). The 2002 National Electrical Code specifies that under non-hazardous conditions “live parts of generators operated at more than 50 volts [RMS] to

⁹ In this review ‘standard’ and ‘code’ are used equivalently.

¹⁰ The Association for the Advancement of Medical Instrumentation developed an American National Standard on “Safe Current Limits for Electromedical Apparatus” (ANSI-AAMI ES1-1993) which limits leakage current to less than 1 mA. The International Electrochemical Commission (IEC) 601-1 standard allows a “patient auxiliary current” up to 100 μ A.

ground shall not be exposed to accidental contact when accessible to unqualified persons.” (445.14) and that “where wet contact (immersion not included) is likely to occur... V_{\max} shall not be greater than 15 volts [RMS].” The 2004 NFPA 70E standard, which is “compatible with corresponding provisions of the NEC”, summarizes that: “energized parts that operate at less than 50 volts [RMS] to ground shall not be required to be deenergized if there will be no increased exposure to electrical burns or to explosions due to electric arcs.”

The U.S. Consumer Product Safety Commission (CPSC) “Electrical Safety In and Around Pools, Hot Tubs and Spas” suggests ground fault circuit interrupters are necessary for *underwater* lighting circuits operating at more than 15 V. The CPSC “Requirement for electrically operated toys or other electrically operated articles for use by children” (CPSC 1505) specifies that “a potential of more than 30 volt r.m.s. (42.4 volts peak) shall not exist between any exposed live part in a toy and any other part or ground”. Exceptions are provided for *protected* lamps.

The International Electrotechnical Commission (IEC) has issued several reports on electrical safety. The IEC “Electrical installations of buildings” report (IEC 60634-4-41:2001) specifies that for unearthed circuits “if the nominal voltage does not exceed 25 V a.c. r.m.s. [$35 V_{\text{PEAK}}$] or 60 V ripple-free d.c., protection against direct contact is generally unnecessary; however, it may be necessary under certain conditions of external influences (under consideration).” For earthed circuits the IEC considers protection unnecessary when “nominal voltage does not exceed 25 V a.c. r.m.s or 60 V ripple-free d.c., when the equipment is normally used in dry locations only and large-area contact of live parts with the human body is not expected; 6 V a.c. r.m.s. [$8.5 V_{\text{PEAK}}$] or 15 V ripple-free d.c. in all other cases.” The IEC report also considered measures limiting current flow to 30 mA or measures limiting exposure to voltages of 50 V to 5 s ‘protective.’ No scientific justification is provided in the IEC 60634 report for any of the above values.

The IEC “Extra-low voltage-limit values” report (CEI-IEC 1201:1992) specifies 22.4 V “non-hazardous” (worst case conditions of large contact area, negligible skin and earth resistance); the report notes “higher voltages are not necessarily hazardous.” The reports also specifies that for a “non-grippable” part with contact area less 1 cm^2 the limit increases to 92.4 Volts.

The most comprehensive review of electrical safety to date by the IEC, “Effects of current on human beings and livestock” (CEI-IEC 479-1; 1994) includes an analysis on effective body resistance (Table 1). The report notes that “conductive solutions decrease the impedance considerably down to half the values measured under dry conditions [e.g. 875Ω for 5% rank at 25 V]...impedance for children is expected to be somewhat higher.” The threshold values for ventricular fibrillation, the “main cause of death by electrical shock”, were 40 mA for >3s exposure, 50 mA at 1 s exposure, and 500 mA for 0.1 s exposure (left hand-to-foot/feet). An identical threshold was set for “breathing arrest”. With one electrode on the chest the threshold for ventricular fibrillation by “only a rough estimation” reduces to 60% of the above values. The CEI-IEC 479-1 report did not explicitly specify a safe voltage level; however, a current of 40 mA across 875Ω corresponds to a voltage of 35 V (left hand-to-foot path). A new IEC report directly addressing ‘touch’ voltages is expected (IEC-61201).

The 1988 European Organization for Nuclear Research, (CERN) “Dangers due to electricity” safety Instruction (IS-28) is “essentially based on IEC publication 479-1” (see above) including statistical resistance values (Table 1). In addition the IS-28 report specifies a total body resistance of $>650 \Omega$ under moist/wet conditions and $>325 \Omega$ for ‘immersed skin’. Presumably based on minimal ‘let-go’ thresholds, the IS-28 report specifies “as a rough guide to complete safety, the current limit should be considered as 10 mA” for $<20 \text{ ms}$.

The IEEE “National Electrical Safety Code” (C2-2002) explicitly does not specify a minimum approach distance for exposed parts energized below 71 V (Table 441-1). The IEEE “Guide for safety in AC substation grounding” (80-1986) incorporates a voltage-independent body resistance of 1000Ω and further considers the insulating effect of soil between the human body and contact points; the 80-1986 standard uses ventricular fibrillation to establish current limitations.

Lee and Meliopoulos (1999) analyzed the IEEE 80-1986 standard and the IEC-479 standard *with the addition of soil resistance*. For their worst-case-conditions (soil resistivity $10 \Omega\text{-cm}$, 0.5 s shock duration, hand-to-2 feet contact) they calculate permissible voltages of 166 V and 89 V for the IEEE 80-1986 and the IEC-479 standard (with soil resistance), respectively.

SECTION V REVIEW SUMMARY

In practical situations, uninsulated contacts with an energized source will most likely involve two or more limbs. Based on the reports summarized above, assuming two limbs make large-surface-area wet-conductive contact, minimal skin resistance (but no abrasions or immersion), and negligible electrode interface resistance, total current path resistance can be as low as 550 Ω .

Tetanic peripheral muscle contraction ('let-go' current) does not directly lead to death and is not clearly linked with low-voltage electrocution. 'Let-go' currents are integrated by some regulatory agencies because exposure to higher hazardous current, in combination with loss of mobility due to muscle contraction can be fatal.

Based on the reports summarized above, respiratory paralysis can lead to death but requires *several minutes of contact*; currents as low as 30 mA_{PEAK} across the chest may induce paralysis. Assuming a low body resistance of 550 Ω this corresponds to a voltage of 16.5 V_{PEAK}; this is a *theoretical* value based on several worst case resistance assumptions and limited current threshold data¹¹. Based on the case studies summarized above the single lowest voltage reported to cause transdermal electrocution in an adult is 25 V. Note that this value is significantly above the threshold for perception/discomfort (1-5 mA; Geddes and Baker 1989; Webster 1998; Reilly 1998; Table 2) while well below the current levels causing unconsciousness (>300 mA); electrocution by respiratory paralysis may thus require physical immobilization not directly related to current flow (e.g. fall).

Based on the reports reviewed above, for *less than 1 min duration* electrical contact, currents >40 mA may be necessary to cause ventricular fibrillation, corresponding to *theoretical* value of 27.5 V_{PEAK} (assuming a 550 Ω body resistance). For a *less than 1 s* exposure these values increase to >100 mA_{PEAK} and 55 V_{PEAK}. In the case reports summarized above, there were no instances of accidental electrocution from short-term transdermal exposure to voltages below 50 V_{PEAK}. Indeed, such exposures were used routinely during experimental studies on human subjects.

¹¹ The 30 mA value, though cited in several reviews, is apparently based solely on work by Dalziel et al.

Conclusions

Only a small percentage of electric shocks have hazardous effects¹². Less than 1000 deaths per year in the United States result from (non-lightning) electrical accidents (Chilbert 1998; Bailey 2001; but see Wright and Davis 1980); one-third of these deaths are caused by voltages below 1414 V_{PEAK} (1000 V_{RMS}). Extrapolating from the reports summarized above, <200 deaths per year in the United States result from voltages below 250 V. Reports of electrocution below 50 V are rare.

Calculation of general voltage safety values from fundamental (cellular) principles is currently intractable (SECTION I). The electrocution reports summarized here (SECTION III) indicate **25 V_{PEAK}** (17.8 V_{RMS}) is the lowest lethal voltage; an extreme combination of sensitivity factors is required for extremely-low voltage electrocution. The combination of both worst-case experimental resistance and worst-case experimental current values (SECTION I/II, above) provides theoretical values below this threshold; this approach is based on limited data and speculative exposure conditions. A 25 V_{PEAK} safety threshold is conservative relative to most electrical codes and standards (SECTION IV); OSHA, NFPA 70E, and NEC, all have more stringent requirements for voltages above 71 V_{PEAK} (50 V_{RMS}).

In the unlikely event a victim's exposed chest contacts an energized source, the safe voltage threshold would be reduced. It must be emphasized that significant unknowns remain about the effects of electricity on the body and further experimental studies are necessary to better establish under what conditions extremely-low voltages can harm humans. Specific areas of concern include determining current threshold for respiratory arrest, risks under 'immersion' conditions, and the need for separate resistance/current thresholds for children.

¹² Cases of non-fatal electric shock are often not reported. There are several reports of legal electrocution incidents where voltages >1400 V applied for >17 s did not result in death (<http://hypertextbook.com/facts/NancyRyan.shtml>).

APPENDIX: ELECTROCUTION OF DOGS.

Research on the effects of electrical currents on canines has been conducted towards better understanding and treating human exposure. In this context, some studies with canines have been discussed above. This section deals specifically with the risks of dog electrocution. Similar mechanisms are thought to result in electrocution in dogs as in humans (see *Mechanisms of injury* above). As in humans, cardiac fibrillation has been the most extensively studied.

Dalziel and Lee (1969) consolidated reports by Ferris (1936), Kouwenhoven (1959), and Kiselev (1963) examining fibrillation thresholds in dogs. Dalziel and Less (1969) noted the inverse relationship, across studies, between current threshold and the square root of exposure time, equation (1) above. The lower 0.5-percentile-rank for fibrillation thresholds for 5 s, 3 s, and 8.3 ms exposures were 35 mA, 45 mA, and 910 mA respectively (5 to 27 Kg dogs); smaller dogs had on average lower fibrillation thresholds. In the above tests, electrodes were attached to the right front foot and the opposite rear limb; Kouwenhoven et al. (1932) considered current intensity more important in this configuration than from upper extremity-to-upper extremity.

Geddes and Baker conducted a series of experiments examining the effects of electric currents on dogs (summarized in Geddes and Baker 1989). They note: "A consistent sequence of events occurred with each animal as the current was increased. The first was a strong contraction of skeletal muscles; this was followed by an arrest of spontaneous respiratory movements, vagal slowing of the heart, and initially in most animals there was evacuation of the bladder and bowel. Finally, ventricular fibrillation occurred." Geddes et al. (1973) found the current needed for fibrillation using the upper extremity-to-upper extremity path is about three times greater than for when current is applied between the fore and hind limbs. Geddes et al. (1973; 1989) also found similar thresholds and duration relationship as summarized by Dalziel and Lee (1969) with smaller animals having a lower average threshold. Geddes and Baker (1989) note that the value of 60-Hz current required to initiate fibrillation in a 70-kg animal (which approximate the weight of an adult man) is 215 mA (flowing for 5 s; left arm-left leg). Geddes and Baker (1989) also examined the effect of current frequency on fibrillation threshold. The threshold in the region of

10-100 Hz was almost independent of frequency; above 100 Hz the current required to initiate fibrillation is markedly higher.

Kugelberg (1976) found frequencies between 12-60 Hz most effective in inducing fibrillation of the canine heart; this is within the range used for power distribution.

Adrenergic stimulation (Han et al. 1964) and acidosis (Gerst et al. 1966) will reduce the current threshold for producing ventricular fibrillation; these conditions would be expected during physical excursion or (electric shock induced) stress.

Practically, dogs may be prone to accidental electrocution as a result of: 1) lack of insulation, such as shoes; 2) lack of potential hazard avoidance; 3) inability to understand/separate from electrical hazard once encountered; 4) a higher incidence of >2 limbs in the current path which would reduce path resistance. Cases of accidental canine electrocution have been reported but the frequency of these events remains unknown. As in man, once initiated by a brief electric shock, ventricular fibrillation rarely stops in dogs and can lead to death in several minutes. As in man, respiratory arrest has a lower current threshold but would require constant exposure for several minutes.

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