Physics of Electrical Injury

2

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Introduction

Electrical injuries are common worldwide. They are responsible for an estimated 50,000 emergency department treatments per year in the USA alone [1]. In modern industrialized societies, the majority of severe electrical accidents are suffered by electrical utility employees or construction workers [2]. In other societies, where the infrastructure is less developed and there is more theft of electrical power, the majority of electrical accidents occur to amateurs. As a striking example, there were more electrical injuries than gunshot injuries in Baghdad in 2009 in males [3].

The fatality rate for serious electrical injury is about 40% [4]. There is a bimodal age distribution to electrical accidents with a high rate of accidents in children younger than 6 years old [5]. The typical incident involves a child chewing through an electrical cord or sucking on the end of an extension cord. The most common severe presentation is a second- or third-degree burn of the lip commissure.

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High- Versus Low-Voltage Classification

Electrical accidents are commonly classified as either low or high voltage as shown in Table 2.1. The arbitrary cutoff is usually set at 1,000 V. A better classification might be "indoor" vs. "outdoor" as the somewhat arbitrary voltage cutoff leads to some diagnostic errors. An even better classification is by *power* as a 1,000,000 V Van de Graaff generator does not cause injury as the power and current are almost zero.

Most Common Diagnostic Error

The most common conducted electrical weapon (CEW) delivers an averaged 600 V pulse [6]. While "low" voltage by the 1,000 V cutoff, even the 600 V value tends to cause classification errors for potential electrical injury. Most electrical injuries are burns. As will be discussed later, the electrical metric that best describes the ability of a source to cause burns is the power—measured in watts. A 7,600 V power line can easily deliver about 60 kW (kilowatts) of power to someone standing on the ground (or on an aluminum ladder) and touching the power line with a tool (Table 2.2). This level of power has the capability for significant burns and neural damage. In contrast, the handheld TASER X26 CEW delivers less than 2 W of power. Thus, the power line delivers about 30,000 times as much power.

Table 2.1 Low- and high-voltage injuries

Classification	Most common location	Typical sources	Typical injury	Typical cardiac rhythm if fatality
Low voltage	Indoors	110–220 VAC utility power 440 VAC large machine power	Burns (pediatric) or VF (adult)	Ventricular fibrillation
High voltage	Outdoors	7600 VAC power line, lightning	Burns including limb loss	Asystole

Table 2.2 Common sources of real and alleged electrocution

	TASER X26 CEW	220 VAC (single cycle)	Power line (single cycle)	Lightning bolt from cloud bottom	Lightning bolt from cloud top
Peak open circuit voltage	50 kV	310 V	7.6 kV	100 MV	1 GV
Charge	0.0001 C	0.0016 C	0.056 C	5 C	300 C
Energy per pulse	0.1 J	0.4 J	490 J	500 MJ	300 GJ
Energy in 5 s	10 J	120 J	60 J		

Occasionally, a physician will have a patient present with various complaints and a history of receiving a CEW exposure. Due to the media-reported voltage of 50 kV, the well-meaning physician will reference "high-voltage" injuries and will likely compare the CEW exposure to a long list of power-line and lightning injuries. This is impossible from a CEW exposure. This is exactly what happened in recent litigation involving a CEW [7]. The family physician provided an expert report for litigation stating:

> Type of injuries can include arrhythmias, burns, either superficially or deeper nerve damage, lacerations, muscle damage with rhabdomyolysis and subsequent renal problems and chronic muscle problems, nerve injuries with weakness, paralysis….

In fact, a battery-operated handheld CEW simply does not have the power to cause muscle or nerve damage or clinically significant rhabdomyolysis [8, 9]. Hence, the confusion regarding the "high-voltage" classification resulted in an erroneous etiology resulting in an expensive trial to finally clear it up.

Basics of Electricity

Charge

The most fundamental electrical unit is also the one least used and least recognized. Many speak of the "charge" on their mobile phone battery but few can name the unit of charge, namely, the coulomb. The coulomb is equal to the charge carried by 6.24×10^{18} electrons. A coulomb (C) of charge does not necessarily represent this many electrons as it could mean 6.24×10^{18} of any single charge (positive or negative) particles such as K^+, Na^+, or Cl^-. For obvious reasons, 3.12×10^{18} calcium ions (Ca^{++}) also represent a coulomb of charge. (Quarks can have charges of $-1/3$ and $2/3$ and thus do not follow such simple accounting rules. Fortuitously, electrical injury can be understood without dealing with quarks.)

A common error is to assume that a coulomb of charge is equal to a mole of electrons. Since a mole is 6.02×10^{23} molecules, a mole of electrons would have a charge of nearly 100,000 C. A common AA battery (actually a cell) stores about 1,000 C of charge.

Current

Electrical current is simply the rate of flow of charge. If a circuit is passing 1 C/s, the current is said to be 1 ampere (A). Since the charge carrier in a copper wire is the electron, a current of 1 A just means that 6.24×10^{18} electrons are flowing thru the wire per second.

Figure 2.1 depicts a current of 1 A flowing through a human thorax. (This is about 10 times that seen with transcutaneous pacing and about 1/20 that seen with external defibrillation.) By convention, "positive" current is defined as that flowing from the positive to the negative electrode (i.e., this assumes positive charge carriers even if the carriers are negative such as the electrons in a wire). What the generator (with the wires) actually does is to carry electrons to the *opposite* side of the thorax.

The body does not have free electrons to carry charge internally so chlorine ions carry the charge from the right to the left (of the subject) while positively charged ions (primarily sodium but also potassium, calcium, and magnesium, etc.) carry current in the opposite direction.

Electrocardiographic (ECG) and defibrillation electrodes have a gel containing a metal and a salt of that metal (typically silver or tin is the metal) to facilitate the exchange of the electron for a chlorine ion and vice versa. Without such a gel, the resistance (to low voltages and low frequencies) is extremely high on dry skin.

Average Versus RMS (Root Mean Square) Current

Household and industrial electrical power is AC (alternating current) with a frequency of 50 or 60 Hz. At any point in time, the instantaneous current is given by the blue line in Fig. 2.2. Consider the example of a 120 W bulb being powered by a 120 V hospital outlet. The instantaneous current through the bulb varies from −1.4 to +1.4 A. Thus, the average current is 0! To arrive at a simple single number to replace this "average" current, the RMS current is calculated.
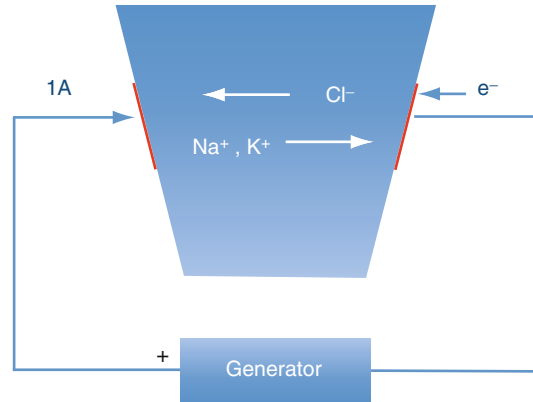
Fig. 2.1 Current through a human thorax. While convention defines the current as flowing from the positive electrode on the left (subject's right), the actual charge carriers are electrons being delivered from the wire on the right (subject's left). Internal current is carried by chlorine ions in one direction and positive ions in the opposite

First, the instantaneous current is squared giving the values shown in red. These values are all positive so their average (mean) will be positive. Finally, the square root of this mean is taken.

RMS stands for "root mean square" which unfortunately suggests a sequence of operations in reverse order of what is actually done. The correct sequence is: square, mean, root. The RMS value for an AC current with peak values of ±1.4 A is 1.0 A.

Similarly, household "120 VAC" actually has peak values of ± 170 V. The RMS "averaging" function reduces this 170 V peak down to the 120 V RMS value.

Aggregate Current

While RMS current is used for utility power safety standards, RMS current is fundamentally a predictor of heating capability. The most common misunderstanding of nonspecialists—in bioelectricity—is that RMS current somehow predicts tissue stimulation capability [10, 11].

A current composed of rapid very short pulses can have high RMS currents but be poor stimulators due to the small electrical charge carried in the short pulses. For this reason, specialized

Fig. 2.2 Utility power has a sinusoidal current (*blue line*) and thus delivers 0 net charge. This current is squared, and the average value under that curve (*red*) is used to determine the RMS current
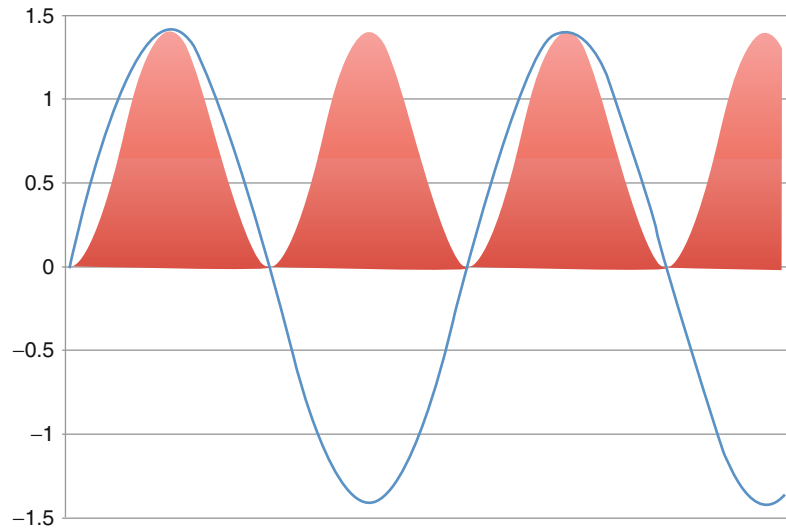
calculations were developed for ascertaining the safety of electric fences [12, 13]. However, even these calculations do not appear to be quantitatively supported by any identified published studies.

This gap has been recently filled with the identification of the aggregate current [14]. Over the range of pulse rates of 10–30 PPS, the capability of rapid short pulses to induce ventricular fibrillation is given by the aggregate current, which is the pulse charge multiplied by the pulse rate [14]. For example, the aggregate current of the popular TASER® X26 CEW is:

$$1.9 \text{ mA} = 100 \text{ } \mu\text{C} \cdot 19 \text{ PPS}$$

The ability of rapid short pulses to induce VF is approximately equal to a 60-Hz AC current with an RMS current of 7.4 times the aggregate current of the rapid short pulses [14]. For example, the aggregate current of the popular TASER® X26 of 1.9 mA can be compared to an AC source of 14.1 mA RMS. That is less than the long-application VF safety level of 35 mA of international standards [15].

Voltage

Voltage is merely the pressure pushing a current through its path. Since the original voltage sources were batteries, the definition of the "volt" was that of a standard battery (technically a single cell which does not a battery make). For

decades, the volt was set at 98% of the voltage of a mercury-cadmium cell, known as the Weston cell. The standard is now based on a solid-state circuit, based on the Josephson effect.

The other function of voltage (when high enough) is to cause the breakdown of insulators. With rounded electrodes, air typically breaks down with a voltage difference of 30 kV/cm. (This gradient of voltage is referred to as the "electric field" and is analogous to an arterial pressure gradient of mmHg/cm.) With sharp electrodes, air breaks down at about 15 kV/cm, and this can be observed with an "arc" test of a CEW. This is also why lightning rods are pointed. See Fig. 2.3 for a simple analogy with a high-pressure "squirt gun."

Resistance

Electrical resistance is simply the resistance to the flow of current. If a circuit has a resistance of 1 Ω, then for each 1 A of desired current, a pressure of 1 V is required. As another example, the human body resistance is typically estimated at 1 kΩ (1,000 Ω) from hand to foot. Thus, if someone barefoot in wet concrete touches a 220 V line, the current passed through the body will be about 220 mA (= 220 V ÷ 1,000 Ω). This definition is often referred to by the grandiose term of "Ohm's law."

Electrical resistance is highly analogous to the fluid resistance of arteries. The higher the

Fig. 2.3 High voltage
(analogous to the high water
pressure) allows arcing
through the air

resistance of the arteries, the more blood pressure is required to produce the same amount of flow. This is why a noncompliant arterial tree results in hypertension (assuming normal cardiac output). See Fig. 2.4 for the water analogy.

The term "impedance" is often used as a synonym for resistance. While troubling to some theoretical purists, these terms are now used synonymously.

A common error is to assume that a low resistance (since it results in a higher current by Ohm's law) implies greater thermal injury. Depending on the scenario, this is the opposite of the truth as will be discussed in the power section.

Power

A large water flow, say 1,000 L/min, in a very large diameter storm sewer pipe does not have much power. That is because the pressure is very low. As seen in Fig. 2.5, a relatively high flow from a large shower head (low pressure) will have minimal power.

Similarly, a high pressure of saline in a sealed percutaneous transluminal coronary angioplasty (PTCA) balloon has zero current (flow) and is hence delivering no power. The power of a flow is equal to the flow rate times the pressure. Figure 2.6 depicts a fire hose with both a high pressure and a
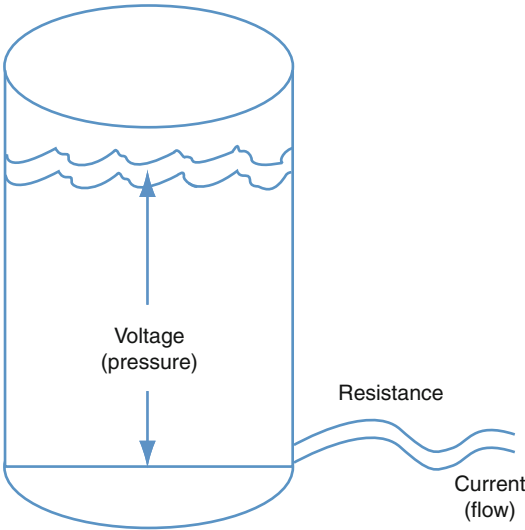
Fig. 2.4 The flow (current) is proportional to the voltage (pressure) and inversely proportional to the resistance

Fig. 2.5 A large shower head can deliver a high flow, but there is little power as the pressure is low

high flow. This can have very high power. Intuitively, there are more water molecules per second (high current) and each has more energy (high pressure). Electrically this is represented as:

$$\text{Power (watts)} = \text{pressure (volts)} \cdot \text{flow (amperes)}$$

Consider a 100 W incandescent bulb:

$$100 \text{ W} = 110 \text{ V} \cdot 0.91 \text{ A}$$

Since the voltage is given by $I \cdot R$ (current times resistance), power is often calculated as:

$$\text{Power} = I^2 R$$

Energy

The product of power and time duration gives the energy in joules:

$$\text{Energy (joules)} = \text{Power (watts)} \cdot \text{duration (seconds)}$$

or

$$\text{Energy (joules)} = \text{Voltage} \cdot \text{current} \cdot \text{duration (seconds)}$$

As an example, a typical external defibrillation shock delivers an average of 1,000 V with a current averaging around 20 A with a duration of about 10 ms (milliseconds).

The power is around:

$$20,000 \text{ W} = 1,000 \text{ V} \cdot 20 \text{ A}$$

The energy is around:

$$200 \text{ J} = 20,000 \text{ W} \cdot 0.01 \text{ s}$$

Note that the actual calculations cannot be made accurately using averages (since the voltages are changing) and the area under the curve of power (as a function) of time must be calculated. To use the calculus term, the energy is the "time integral" of the power.

Home electrical bills are paid by the energy in units of kWh (kilowatt hours). Since an hour has

Fig. 2.6 A fire hose can have both high pressure and high current, hence very high power

3,600 s, 1 kWh is equal to 3,600 kJ (3.6 MJ). The joule is about one fourth of a physics calorie or about 1/4,000 of a food calorie.

Summary of Electrical Units

The electrical units are summarized in Table 2.3. Note that the unit terms are all lower cased. Since all electrical unit terms are eponymous, it is sometimes tempting to capitalize these terms. For reasons unknown to these authors, the predilection for capitalizing electrical units is more commonly seen with the coulomb and joule but almost never seen with the volt or watt. Another common error is the assumption that "amp" is the abbreviation for "ampere." "Amp" is technically the abbreviation for "amplifier" while "A" is the abbreviation for "ampere."

The presence of these errors in reports is a sure sign of a superficial knowledge of electricity and should be avoided.

The Body as a Resistor

Resistivity Versus Resistance

While resistance is a property of an overall current path, the tissue resistivity (also referred to as "bulk" resistivity) is a property of the tissue regardless of its size. As seen in Fig. 2.7, the bulk resistivity is the resistance of a 1 cm³ cube of the tissue. The unit of bulk resistivity is the "ohm·cm" and this allows for the calculation of an arbitrary sized sample of the tissue. Note that the unit is "ohm·cm" and *not* "ohms per cm." Rather, it is ohm multiplied by cm.

Example:

Blood has a bulk resistivity of about 150 Ω·cm. What is the electrical resistance of a 20 cm section of the aorta with a 2 cm² cross-sectional area?

$$\text{Resistance} = \text{Bulk resistivity}(\Omega\,\text{cm})$$
$$\cdot\text{length}\,(\text{cm}) \div \text{cross - sectional area}\,(\text{cm}^2)$$
$$= 150\,\Omega\,\text{cm} \cdot 20\,\text{cm} \div 2\,\text{cm}^2$$
$$= 1,500\,\Omega$$

The electrical injury literature often has comments that electrical currents are carried by the blood vessels. This is clearly a very misleading statement as the 1,500 Ω resistance calculated above is very high compared to the typical 75 Ω thoracic impedance seen with defibrillation [16]. This misconception may derive from a confusion of resistance with resistivity.
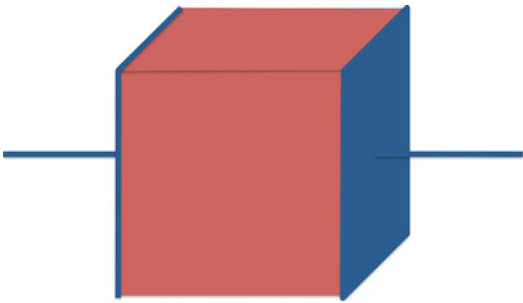
Tissue Resistivities

The tissues of the body have a wide range of resistivities and different tissue resistivities are affected by different factors as summarized in Table 2.4.

Table 2.3 Summary of electrical units

Parameter	Meaning	Units	Abbreviation	Unit definition	Physiology analogy	Common errors
Charge	Fundamental quantity	coulomb	C	6.24×10^{18} electrons (or ions)	liter of blood	Assuming a coulomb equals a mole of electrons
Current	Flow of charge	ampere	A	1 C/s	liter/minute cardiac output	Referring to current as "amperage"
RMS current	Heating capability	amperes RMS	A (RMS)		Running in place	Assuming RMS current is related to stimulation or fibrillation
Aggregate current	VF induction capability of rapid short pulses[14]	milliamperes (mA)	mA (agg)	Pulse charge · pulse rate	Blood volume per beat · heart rate	
Voltage	Electrical pressure	volt	V	98% of voltage of mercury-cadmium cell	mmHg blood pressure	
Resistance	Resistance to flow	ohm	Ω		L/min/mmHg arterial resistance	Assuming that a low resistance implies more injury
Power	Ability to do work	watt	W	volt-ampere	Product of BP and cardiac output	
Energy		joule	J	watt-second	calorie	

Note: The definitions given above are the easiest to visualize. There are various used and proposed definitions for differing applications and standards

These electrical units are placed into context with a listing of common real and alleged sources of electrocution in Table 2.2

Fig. 2.7 Tissue resistivity is defined as the resistance of a 1-cm³ cube of the tissue. Square 1-cm² electrodes are attached to opposite faces

Table 2.4 Tissue resistivity from lowest to highest

Tissue	Resistivity (Ω · cm)	Notes
Blood	150	Lower in females and with high frequencies
Dermis	500	
Muscle	180/1,700	Along fiber vs. transverse to fiber
Lungs	1,100	Lower with edema and deflation
Fat	2,200	
Bone	10,000	Lower in pediatric ribs
Epidermis	1,000,000	Dramatically lower with hydration, higher voltages, and abrasion

Note: Values shown are typical and vary with species, frequency, and methodology

Fig. 2.8 Blood resistivity increases directly with the hematocrit level
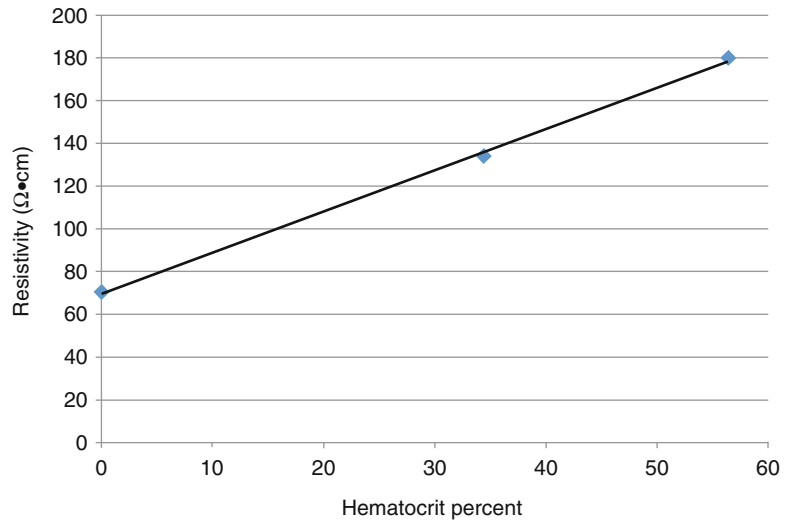
Dermis and Fat

The dermis and fat are the easiest tissues to classify electrically. The resistivity is minimally affected by various factors that change other tissue resistivities. A typical value for the dermis resistivity is $500\,\Omega$. cm while that of fat is around $2,200\,\Omega$. cm [17].

Lungs

A nonedematous and inflated lung has a fairly high resistivity. Obviously, the inspired air is not a conductor and electrical current is essentially carried by the blood from the pulmonary and systemic circulation. Typical values are $1,100\,\Omega \cdot$ cm [17]. At exhalation, the resistivity drops by about 20–30% [18, 19].

Blood

Blood is the best significant electrical conductor in the body. A typical resistivity value is $150\,\Omega \cdot$ cm [20]. The resistivity of blood is about 2× that of normal physiological saline ($70\,\Omega \cdot$ cm) as the red cells are insulators. (Ironically, some have stated that red cells actually carry current.) The resistivity of blood varies in a direct affine relationship with the hematocrit level as seen in Fig. 2.8 [20]. Hence, the blood of females is a better conductor than the blood of males.

At higher frequencies (such as those seen with RF ablation), there is capacitive coupling across the red cells; the hematocrit no longer affects the resistivity and the resistivity drops to near that of physiological saline [17, 20, 21].

Even though blood is a good conductor, it is generally irrelevant to electrical injury as the large vessels are centrally located and the skeletal muscle and dermis end up carrying the current. In addition, the largest blood vessel (inferior vena cava) runs vertically so it could not carry current from side to side or front to back anyway. Urine and amniotic fluid are better conductors than blood but generally not relevant to electrical injury.

Bone

Bone is generally the best electrical insulator in the body with a high resistivity of around 10,000 $\Omega \cdot$ cm. This varies with age as cartilage is a better conductor than hardened bone [21].

Muscle

The resistivity of both cardiac and skeletal muscle depends dramatically on the orientation of the current flow with respect to the fiber orientation. The resistivity of muscle "with the grain" can be much lower than the resistivity against the grain (transverse to the fiber orientation). The property is referred to as anisotropy. Typical resistivity values along and against the muscle grain are 130–230 $\Omega \cdot$ cm and 1,500–1,900 $\Omega \cdot$ cm, respectively [22, 23]. The ratio between the longitudinal and transverse resistivity is 7–15:1 [22, 23].

The net effect of this high anisotropy is that current (from external sources) tends to flow around the outside of the thorax and is resistant to penetration within the thorax. The profound anisotropy of muscle can lead to surprising results such as simultaneous clockwise and counterclockwise current flow in the cardiac ventricle.

Epidermis

The epidermis is the most complex electrical conductor in the human body [24]. The resistance varies dramatically depending on the surface treatment, hydration, frequency, and voltage.

In the simplest analysis, the epidermis is almost an electrical insulator with an extremely high bulk resistivity of about 1 M$\Omega \cdot$ cm. At low voltages, current is carried by the sweat glands from the dermis to the outer layer, the stratum corneum. Numerous studies—using small-tipped electrodes—have found that the skin surface resistance is low only at about 2–6 spots per square millimeter, which corresponds to the density of sweat glands [25, 26]. These sweat glands tend to bypass the high resistivity of the rest of the epidermis. Normal ECG electrodes will cover hundreds of sweat glands allowing signal passage. However, the stratum corneum has an extremely high resistance when dry. Merely applying and removing adhesive tape several times can reduce the resistance of the stratum corneum by a factor of 300 [27]! This is why surface roughening is often used to produce better ECG signals.

At about 500–600 V, the stratum corneum is broken down electrically [28, 29]. Hydration also significantly reduces this resistance [30]. Thus, the high resistance of the dry stratum corneum is generally relevant only for electrical injuries from household voltage such as 110 or 220 VAC.

Note that the high resistivity of the epidermis, the effects of hydration, and the effects of abrasions are irrelevant to CEW current as the probes penetrate beyond the epidermal layer. These factors are almost insignificant even for "drive-stun" applications as the peak voltages tend to break down the epidermis as well. In spite of this, the opinion is often heard that a CEW had more of an effect due to a subject having wet skin.

There is a common adage that electrical current takes the shortest path. With knowledge of the differing tissue resistivities, this can be seen to be sometimes misleading and even false. Figure 8.4 in the Legal chapter shows the region that passes most of the current between 2 drive-stun electrodes on the skin. The shortest path would be through the epidermis. However, due to the high resistivity of the epidermis, the current tends to mostly flow through the low resistivity dermis layer.

This is a major reason why drive-stun studies find no interference with breathing or induction of arrhythmias [31–35].

Systemic Resistance

The resistance R of a cylindrical probe of length L, diameter d, in a large medium of bulk resistivity ρ is given by [36]:

$$R = \frac{\rho}{2\pi L} \ln(4L / d)$$

For a typical CEW probe of length 0.9 cm, diameter 0.08 cm, in dermis of resistivity 500 $\Omega \cdot$ cm, the calculated resistance is 337 Ω. Thus, the resistance between 2 probes should be twice this or 674 Ω. This value is very close to the measured human average interprobe resistance of 600 Ω [6].

Tissue Injury

There are two ways that an electrical current can injure tissue. The first is electroporation and the second is thermal.

Electroporation

The electrical field is the gradient of the potential and is quantified by volts per cm. For example, a field of 1,600 V/cm means that the voltage changes by 1,600 V over a 1 cm distance in tissue. This high field level can directly cause a cellular injury called electroporation in which the membranes are polarized to over 500 mV and damaged [37–39]. Electroporation can occur in 100 μs and is thus very rapid compared to thermal injury.

Electroporation is usually only temporary with fields of around 500 V/cm, and this has been explored for numerous medical therapies. For example, by causing temporary electroporation around a tumor, the chemotherapeutic agent bleomycin can pass through the cell membranes and significantly improve chemotherapy results [40, 41]. Electroporation is also being researched for gene therapy to replace viral vectors [42–44].

However, a simple calculation shows that electroporation is essentially irrelevant in typical electrical injuries. Consider an electrical exposure lasting only long enough (0.1 s) for a spinal reflex reaction time to pull away from the current. If the field was high enough (1,600 V/cm) for permanent electroporation and the tissue bulk resistivity was 500 $\Omega \cdot$ cm, then the current density would be 3.2 A/cm^2 by using the bulk version of Ohm's law. The power density would then be:

$$5,120 \text{ W} / \text{cm}^3 = 1,600 \text{ V} / \text{cm} \cdot 3.2 \text{ A} / \text{cm}^2$$

For the exposure of only 0.1 s, the energy density would be:

$$512 \text{ J} / \text{cm}^3 = 5,120 \text{ W} / \text{cm}^3 \cdot 0.1 \text{ s}$$

With a typical tissue specific-heat of 3.8 J/g°C (1 calorie/g°C) and mass density of 1 g/cm^3, the temperature rise would be:

$$135^\circ \text{C} = 512 \text{ J} / \text{cm}^3 \div 3.8 \text{ J} / \text{g}^\circ \text{C} \div 1 \text{ g} / \text{cm}^3$$

Such a temperature rise would cause cellular thermal injury. Note that this calculation ignores heat flow away from the injury, which would be minimal for such a short exposure.

Due to the short pulse durations of CEWs, electroporation must be considered. This is clearly not a concern for the body tissue between the probes as the fields are significantly below those required [45]. However, the electrical field very close to (within <1 mm) CEW probe is higher and has been modeled to determine if there is a possibility of electroporation as shown in Fig. 2.9 [46]. The field was high enough to cause temporary but not permanent electroporation.

Fig. 2.9 The electric field at the tip of a CEW probe (*red*) is sufficient to cause temporary but not permanent electroporation effects (From Panescu et al. [46])
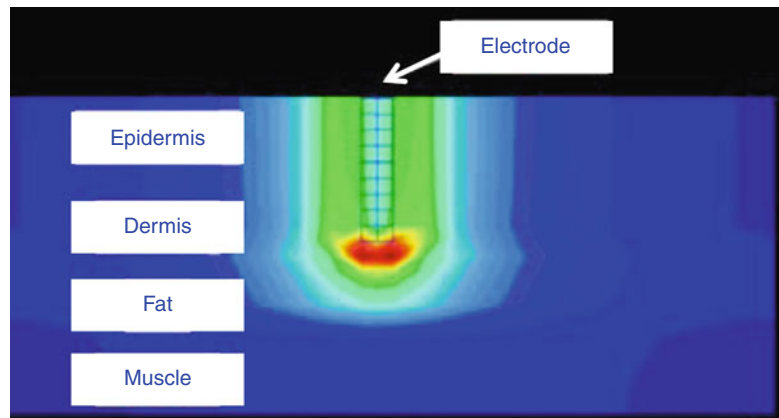
Thermal Injury

Electrical injuries are essentially burn injuries. In an elegant study, Takamiya randomly delivered the same energies to rat dorsal skin from either an electrical current or from a small heater [47]. Over the range of energies tested (100–790 J), the burns from electrical current were the same as the burns from heating. The classic epidermal nuclear elongation—once thought to be characteristic of electrical vs. conventional burn injuries—was found in both. The only difference was that the elongation from electrical injuries was found more frequently near the external root sheath.

Time and Current

Sances delivered current to anesthetized swine with a 2.5 cm diameter metal disk at various low voltages (50–450 V) [48]. After a certain time period, there was sufficient necrosis so that the resistance increased to near infinity and current was no longer passed. As seen in Fig. 2.10, the time to necrosis varied exponentially with the current density ($T \sim e^{-\text{current}}$, $r^2 = .88$, $p = 0.006$). This demonstrates the dramatic effect of current density on tissue injury and why high-current injuries occur in less than a second.

The approximate current density shown is the actual current divided by the 4.9 cm^2 surface area of the disk. Extrapolations from these estimates must be done with caution due to the fringe effects of current being concentrated along the perimeter of the disk.

Paradoxically, the lower the current density, the higher the peak temperature achieved as depicted in Fig. 2.11. This is presumably due to the longer exposure duration allowing more energy transfer as seen in Fig. 2.12.

The Confusing Role of Resistance

It is often said that it takes a low resistance to produce an electrical injury. This seems intuitive as it seems to follow from Ohm's law, which is the first thing that most learn about electricity. *However, it is usually wrong.*
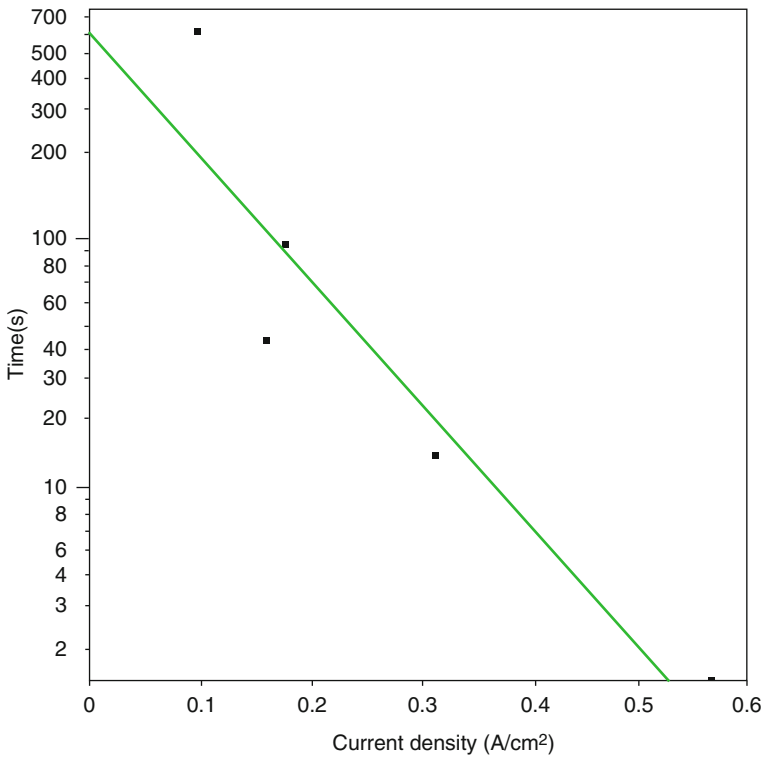
Fig. 2.10 The time to epidermal necrosis varies inversely with the exponent of the current density
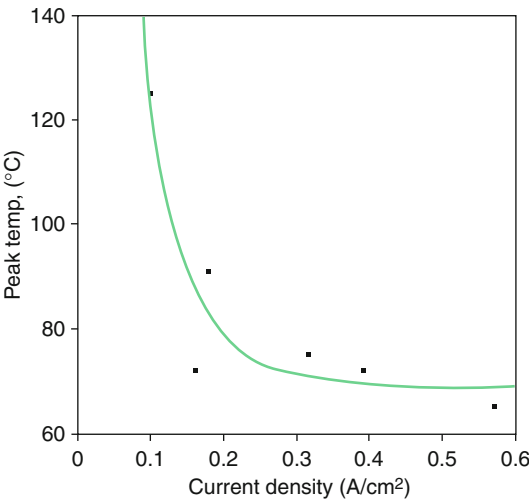
Fig. 2.11 The peak skin temperature actually increases with lower current densities
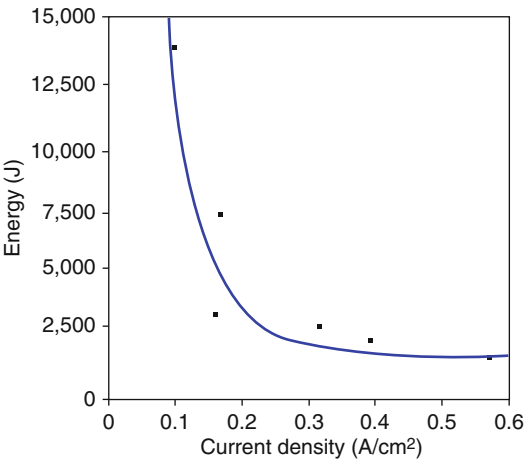
Fig. 2.12 The energy required for necrosis decreases with current density

Consider a case in which a CEW probe is embedded in a subject's leg and the other is embedded in a leather jacket arcing to dry skin over the pectoralis major. At the initiation of the current flow, the total resistance is about 10,260 Ω. This is broken down as:

200 Ω	Resistance from leg probe to surrounding tissue within 1 cm
50 Ω	Resistance from leg tissue to pectoralis major
10 Ω	Resistance from pectoralis major to stratum corneum
10,000 Ω	Resistance of area of stratum corneum under arc

Assuming that the CEW is able to deliver a 2 A current during its short pulse, then the power is given by I^2R:

$$\text{Power} = 41,040 \text{ W} = (2 \text{ A})^2 \cdot 10,260 \text{ } \Omega$$

However, that is the total power delivered to the body. The power delivered to the stratum corneum is given by:

$$\text{Power} = 40,000 \text{ W} = (2 \text{ A})^2 \cdot 10 \text{ k } \Omega$$

Thus, 97.5% of the power is dissipated at the stratum corneum. Of course, this power concentration soon disappears after the stratum corneum is removed. This explains why mild currents can leave small burn marks on the skin. This is also why one must be careful connecting low resistance to injury. In this case, the greatest injury was to the highest resistance region.

Arcing and Polarity

In an arc, electrons carry charge from the cathode (−) to the anode (+) while positive ions carry charge in the opposite direction [49]. Since the electrons move so rapidly, they arrive at the anode before the positive ions arrive at the cathode. This implies that the initial, transient current density is highest at the point, on the cathode, where the arc initiated and launched the electrons as seen in Fig. 2.13 [50]. Note that this effect is very short lived (~10 μs) so it is more relevant to short-pulse devices such as the TASER M26 CEW (40 μs pulse) than the Stinger ST-200 CEW (350 μs pulse).

Consider a CEW arcing discharge where the anode has a good contact with the skin and the
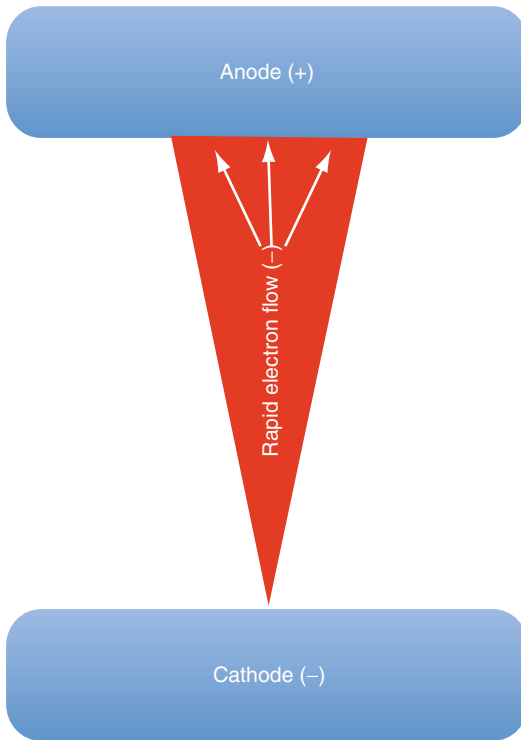
Fig. 2.13 In an arc, the initial, transient current density is greater at the cathode

arc is from the cathode to the skin. This would be expected to produce a larger but milder injury. If the cathode had a good contact, then the arc would be between the anode and the skin and a more concentrated injury would be expected.

Arrhythmia Induction

The electrical injury of greatest concern is a lethal ventricular arrhythmia. The signature rhythm of electrocution is VF (ventricular fibrillation). It was long thought that there were only two means of inducing VF in the healthy heart with electrical currents. The first is the "shock on T" which involves delivering a single strong electrical pulse during the time of the T-wave to instantly cause VF [51, 52]. The second method requires causing extremely rapid cardiac capture—typically >450 BPM (beats per minute)—which induces VF within a few seconds in a normal heart [53–55]. This is classical

"electrocution." This electrically induced VF mechanism takes far less current than "T-shock" induction but also many pulses (typically at least 6 pulses) [56]. It has recently been recognized that there is a third method of inducing VF, namely, with long-term cardiac capture.

Single Pulse

The T-wave is the part of the ECG signal that represents the ventricles beginning to relax. The T-wave represents the time when the heart cells are returning back to their "resting" state. In the middle of the T-wave, about half of the cardiac cells are back to rest and about half are still active. Because of this, an electrical shock, of appropriate strength, delivered during this time will lead to waves going in unpredictable paths throughout the heart. This leads instantly to VF. That is why the T-wave is referred to as the "vulnerable" portion of the heartbeat. For blunt trauma, mechanical energy delivered into the T-wave can also induce VF with a condition referred to as "commotio cordis" [57, 58].

Dorian et al. reported that delivering electrical charge into the T-wave sufficient to induce VF took a mean of 19 J with external patches [59]. One can calculate that this corresponds to an electrical charge of about 100,000 μC assuming typical external defibrillator capacitances. Swerdlow had a patient (unpublished) that he induced with only 1 J which (assuming typical capacitances) corresponds to about 20,000 μC of electrical charge [60]. The value of 5,000 μC is what the IEC (International Electrotechnical Commission) considers to be the 50% probability of VF risk with unidirectional impulse currents of short durations into the T-wave [61].

With electrodes inside the human heart, it is possible to induce VF with a single perfectly timed T-shock of 72 ± 42 V from an implantable defibrillator [51]. (Obviously internal defibrillation requires higher voltages on the order of 400–800 V.) With typical capacitance values, this corresponds to a charge of $7{,}920 \pm 4{,}620$ μC. Since all these values, including the IEC-recommended thresholds, are far higher than the

Fig. 2.14 UL and international standards for VF risk suggest that VF is either induced or not in the first few seconds (From Reilly [49]. Reprinted with permission)
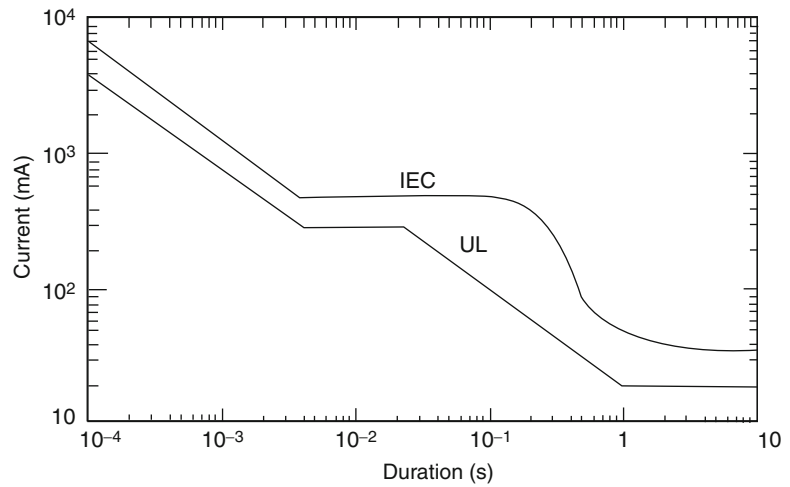
typical CEW pulse, it does not appear possible to induce VF with a T-shock even with a probe touching or inserted into the heart. The direct induction of asystole, by a lighting strike landing outside of the T-wave, is not relevant here and will not be discussed further.

Multiple Pulses

Sufficiently strong repetitive external currents will capture epicardial cells. According to the multiple wavelet hypothesis of VF, formation of new wavelets occurs through the process of wave break (or wave splitting), in which a wavelet breaks into new (daughter) wavelets. Wave break occurs at sites of electrophysiological inhomogeneity, where regions of refractoriness provide opportunities for reentry to form. Epicardial cells are intrinsically heterogeneous in their repolarization properties [62]. This intrinsic heterogeneity provides a substrate for reentry formation during rapid (~450 BPM) capture. When a portion of the incoming wave front encounters refractory tissue while other portions continue to propagate, wave break occurs leading to VF.

The VFT (VF threshold) is the amount of current required to induce VF in a particular subject with a particular connection configuration. Reilly,

in his authoritative text "Applied Bioelectricity," compiled all known studies on the effects of time duration on the direct electrical induction of VF [49]. The VFT goes down, with increasing durations, until the exposure duration reaches 1–5 s. This is reflected in recognized standards, as shown in Fig. 2.14. In other words, if an electrical current does not fibrillate within about 5 s, it will not fibrillate with longer durations (except as shown below by the third mechanism of extended high-rate capture and ischemically lowered VFT).

The ability of rapid short pulses to induce VF is approximately equal to a 60-Hz AC current with RMS current of 7.4 times the aggregate current of the rapid short pulses [14]. For example, the aggregate current of the popular TASER® X26 of 1.9 mA (= 100 μC · 19 PPS) can be compared to an AC source of 14.1 mA RMS. That is less than the long-application VF safety level of 35 mA of international standards and the 20-mA UL standard.

Long-Term Cardiac Capture

Current densities of about 40% of the threshold for the direct induction of VF will lead to VF after 90 s [63]. These current densities are well above the threshold for continuous hypotensive capture [64, 65]. Cardiac capture at rates of >220

Table 2.5 Mechanism of electrocution for various shock durations

Duration	Mechanism	Conclusions
1–10 ms	Shock on T	Requires very strong current
1–5 s	Direct induction of VF	Possible with strong electric current
5–80 s	*No known mechanism*	Unlikely with electrical current unless current is at the edge of the VFT
90–300 s	High-rate capture leading to ischemia-lowering VFT	Possible with weaker current

Fig. 2.15 There are three distinct shock duration periods with which VF can be electrically induced (From: Kroll et al. [63])
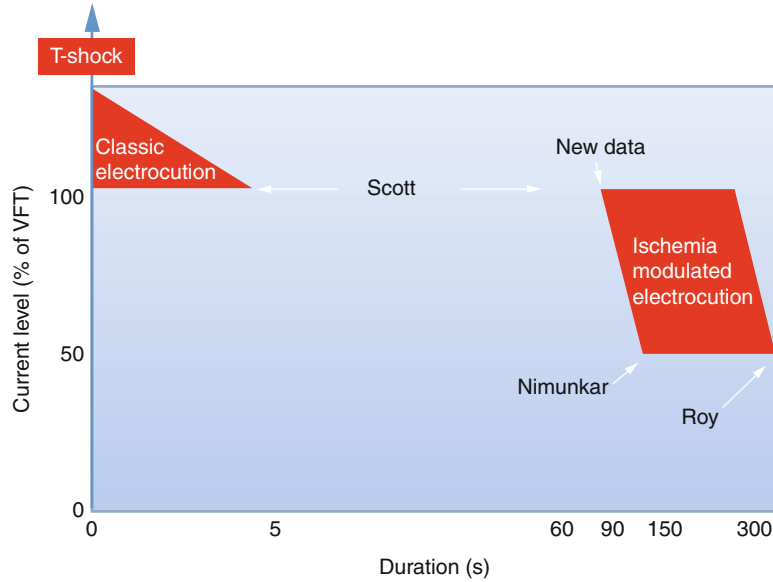
BPM, in swine, can eventually lead to VF. The required durations for this are on the order of minutes rather than seconds.

Prolonged rapid capture reduces cardiac output at the same time that the heart muscle continues to need blood. This causes ischemia sufficient to lower the VFT in about 90 s in swine. In the presence of ischemia, the VFT is cut to about 40% (of the direct-induction VFT) in large mammals. This method of VF induction is compared to the others in Table 2.5.

Figure 2.15 shows the three distinct time scales for the induction of VF by electrical current. The T-shock induction occurs instantly and is shown by the vertical line at 0 s. Direct (multiple pulse) induction of VF occurs typically in 0.1–5 s with the current required decreasing rapidly. Long-duration high-rate capture (with current densities close to the VFT) leads to an ischemically reduced VFT after ~90 s, or longer, of rapid pacing. It is important to note—and clearly shown by Scott—that there is no known mechanism for VF induction taking 5–60 s [66]. In fact, the same is true for durations between 5 and 80 s by our CEW swine data [63]. The Nimunkar swine results, based on a 17 mm dart-to-heart spacing, reflect lower current densities and required a median 150 s to induce VF [67]. Finally, Roy showed that a cardiac arrest would always occur within 300 s with hypotensive capture in canines [68].

VF Risk from a CEW

The possible risk of VF induction by a CEW is obviously of concern. Several animal studies have examined the required spacing from the tip of a CEW probe tip to the epicardium required to induce VF in swine [31, 69, 70]. These data were fit by logistic regression and are shown in Fig. 2.16

Fig. 2.16 Probability of VF induction in swine decreases rapidly with increasing spacing from CEW probe to the epicardium
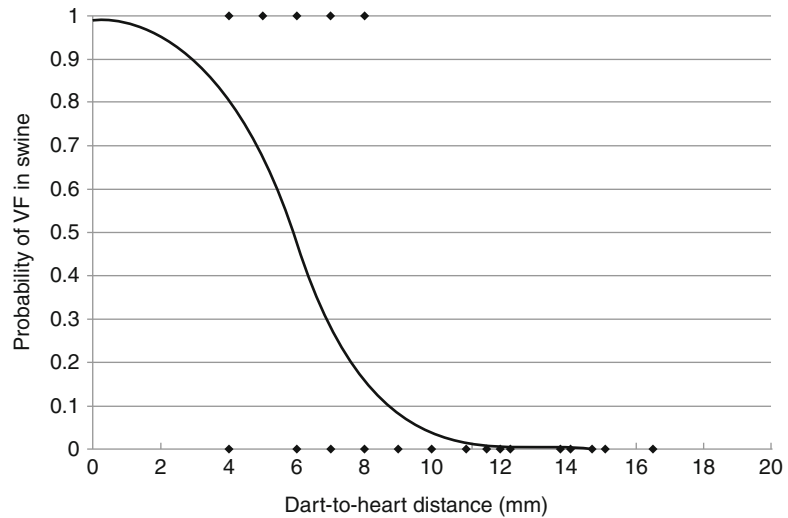
[71]. The median "dart-to-heart" distance was 6 mm in order to induce VF. Since swine are more susceptible to VF than are humans, this corresponds to a distance of 4 mm in humans [71].

The VF risk, for a typical field application, has been modeled by extending swine VF studies into humans by means of finite-element human thoracic models [69, 72]. The early models suggested a risk of 6 PPM (or a probability of 6 VF inductions per million applications). This calculation was based largely on female thoracic echocardiography studies. When refined for male subjects, and corrected for the increased susceptibility of swine to VF induction, this risk was lowered to less than 0.4 PPM [71].

Nonfibrillation Rhythms

Ventricular Tachycardia

A rapid unstable VT (ventricular tachycardia) is occasionally but not commonly induced from steady electrical stimulation. Even in that case, an unstable VT will degenerate into VF within 34 ± 7 s in humans [73]. An unstable VT has such a rapid rate that it almost always leads to immediate syncope [73, 74].

A stable VT cannot be induced absent significant scarring from a previous myocardial infarction [75–78]. This is also not expected from a CEW application as VT induction generally requires specialized pulse timings [75].

Asystole Versus VF

There are many challenges in deriving epidemiological estimates of the VF risk from electrical devices. One problem is that VF will eventually deteriorate into asystole even though asystole is not a rhythm that is electrically inducible. Massive electrical insults such as lightning exposure can lead to asystole (see Table 2.2) due to central nervous system damage but that is not relevant to this discussion. Asystole is also the first arrhythmia in about one-third of spontaneous cardiac arrests [79, 80].

Animal studies by Robertson (swine) and Worley (canine) showed that VF never deteriorated into asystole in 20-min studies [81, 82].

The literature has few human cases of VF deteriorating to asystole. There are seven well-documented cases in five sources [83–87]. The times of VF deteriorating to asystole were 13, 16, 18, 20, 21, and 42 min. The 42-min case was the only one for electrically induced VF and is consistent with the animal results showing no deterioration (to asystole) during 20 min of VF [81, 82]. The logarithm of the deterioration times was well fit by a normal

Fig. 2.17 Log-normal fit of published cases of VF deteriorating to asystole
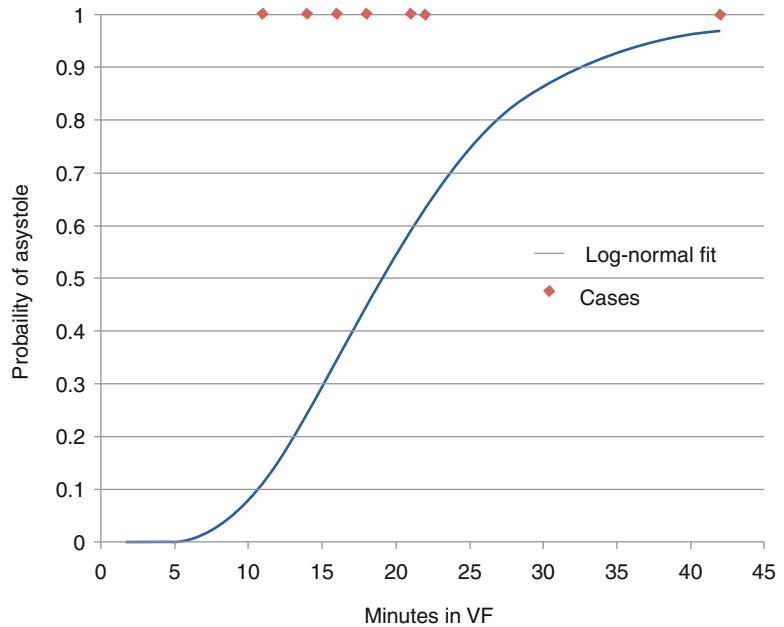
distribution ($p = 0.64$ by Shapiro-Wilk test where a large p is good). The data and fit are shown in Fig. 2.17. The median time was 19 min. Since most of these data were from spontaneous VF (instead of electrically induced VF), we would expect that the deterioration time for electrical accidents would be larger in keeping with the animal results. With cardiopulmonary resuscitation (CPR) this deterioration time might be extended to over 60 min [88].

A presenting rhythm of asystole within 20 min of an electrical current exposure is most likely not due to electrically induced VF deteriorating to asystole. Regardless of the time to presentation, a presentation of asystole was probably not due to deterioration from VF if there was intercurrent CPR. New animal data has demonstrated that the median time for electrically-induced VF to deteriorate to asystole is 35 minutes.

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