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## **ELECTRICAL AND LIGHTNING INJURIES**

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### **PERSPECTIVE**

#### **Electrical Injury**

Injuries from artificial electricity have been reported for almost 300 years. The first recorded death caused by electrical current from an artificial source was reported in 1879, when a carpenter in Lyons, France, inadvertently contacted a 250-volt AC generator.<sup>1</sup> The first U.S. fatality occurred in 1881, when a local inebriate, Samuel W. Smith, passed out on a similar generator in front of a crowd in Buffalo, New York. The apparent painlessness of his death impressed the crowd, and electrocution became considered a “humane” mode of execution. In 1890 William Kemmeler was the first man to be put to death in New York State’s electric chair.<sup>2</sup>

Electrical burns account for 4% to 6.5% of all admissions to burn units in the United States and for approximately 1000 fatalities per year in the United States.<sup>3</sup> Most electrical fatalities and adult admissions to burn centers from electrical injury are occupationally related. Children have a predisposition to injuries from low-voltage sources, such as electric cords, because of their limited mobility within a relatively confined environment.<sup>4</sup> During adolescence, however, a more active exploration of the environment is possible and may lead to more severe high-voltage injuries or death.

At the time of presentation, documentation of injuries is important not only for the immediate resuscitation of the victim but also medicolegally. Nearly all cases of electrical injuries eventually involve litigation for negligence, product liability, or worker compensation.

#### **Lightning Injury**

Since no agency requires the reporting of lightning injuries and because many victims do not seek treatment at the time of their injury, the incidence of injury and death from lightning is unknown. In the United States, nearly 7000 deaths were reported in a 34-year period ending in 1974.<sup>5</sup> Fewer than 2000 deaths

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were reported in a 17-year study ending in 1986.<sup>6</sup> Currently it is estimated that lightning causes 50 to 300 deaths per year in the United States, with four to five times as many victims suffering non-lethal injuries.<sup>5, 6</sup> In typical years, lightning kills more people in the United States than any other natural disaster except flash floods and is consistently among the top four weather-related killers.<sup>7</sup> Lightning incidents are most common in locations where there are more thunderstorms, namely in the South, in the Rockies, along the Atlantic Coast, and in the Hudson, Ohio, and Mississippi river valleys.

Certain snowy conditions can also result in lightning and thus put skiers at risk for lightning injury. The formation of sleet and graupel (a type of frozen precipitation sometimes referred to as snow pellets or soft hail) may indicate the presence of large differences in electrical potential in the atmosphere. Winter sports enthusiasts should learn to recognize the appearance of graupel and appreciate the lightning risks associated with it.<sup>8</sup>

Although some studies show that campers, joggers, and other athletes are the most common victims, others note work-related injuries in as many as 63% of victims.<sup>6, 7</sup> The chances of being struck may be increased by wearing or carrying a metal object such as a helmet, rifle, umbrella, or golf club.<sup>9</sup> Lightning incidents often involve more than one victim when the current “splashes” to other individuals or as ground current spreads the electrical power throughout the area where a group may be sheltered in a storm.

## PRINCIPLES OF DISEASE

### *Physics of Injury*

The exact pathophysiology of electrical injury is not well understood because of the large number of variables that cannot be measured or controlled when an electrical current passes through tissue. With high-voltage injuries, most of the injury appears to be thermal and most histologic studies reveal coagulation necrosis consistent with thermal injury.<sup>10, 11</sup> Lee et al<sup>12</sup> proposed the theory of electroporation in which electrical charges too small to produce thermal damage cause protein configuration changes that threaten cell wall integrity and cellular function. The nature and severity of electrical burn injury are directly proportional to the current strength, resistance, and duration of current flow

#### **Box 1:**

#### *Electrothermal Heating Formulas*

$$P = (I^2)Rt,$$

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and:

$$I = V/R$$

where:

P thermal power (heat), in Joules

I current, in amperes

R resistance, in ohms

t time, in seconds

V potential, in volts

Current strength is expressed in amperes and is a measure of the amount of energy flowing through an object. Current is determined by the voltage and resistance.<sup>13</sup> Resistance is determined by the current's pathway through the body. The factors determining the severity of burn injury are summarized in Box 2.

### Box 2:

#### *Factors Determining Electrical Injury*

Type of circuit

Duration

Resistance of tissues

Voltage

Amperage

Pathway of current

### **Type of Circuit**

One of the factors affecting the nature and severity of electrical injury is the type of circuit involved, either direct current (DC) or alternating current (AC).

High-voltage DC contact tends to cause a single muscle spasm, often throwing the victim from the source. This results in a shorter duration of exposure but increases the likelihood of traumatic blunt injury. Brief contact with a DC source can also result in disturbances in cardiac rhythm, depending on the phase of the cardiac cycle affected, the electrophysiologic principle used with cardiac defibrillators.<sup>4</sup>

AC exposure to the same voltage tends to be three times more dangerous than DC. Continuous muscle contraction, or tetany, can occur when the muscle fibers are stimulated at between 40 and 110 times per second. Unfortunately, the frequency of electrical transmission used in the United States is 60 Hz, which is near the lowest frequency at which an incandescent light will appear to be continuously lit.

The terms entry and exit are commonly used to describe electrical injuries. The

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terms source contact point and ground contact point, however, are more appropriate when referring to AC injuries. The hand is the most common site of contact via a tool that is in contact with an AC electric source. Since the flexors of the hand and forearm are much stronger than the extensors, contraction of the flexors at the wrist, elbow, and shoulder will occur, causing the hand grasping the current source to pull the source even closer to the body. Currents greater than the “let-go threshold” (6 to 9 mA) can prevent the victim from releasing the current source, which prolongs the duration of exposure to the electrical current.

### **Resistance**

Resistance is the tendency of a material to resist the flow of electric current; it is specific for a given tissue, depending on its moisture content, temperature, and other physical properties. The higher the resistance of a tissue to the flow of current, the greater the potential for transformation of electrical energy to thermal energy at any given current. Nerves, designed to carry electrical signals, and muscle and blood vessels, because of their high electrolyte and water content, have a low resistance and are good conductors. Bone, tendon, and fat, which all contain a large amount of inert matrix, have a very high resistance and tend to heat up and coagulate rather than transmit current. The other tissues of the body are intermediate in resistance<sup>5, 14</sup>

### **Box 3:**

<i>Resistance of Body Tissues</i>
<b>Least</b>
Nerves
Blood
Mucous membranes
Muscle
<b>Intermediate</b>
Dry skin
<b>Most</b>
Tendon
Fat
Bone

Skin is the primary resistor to the flow of current into the body. Skin on the inside of the arm or back of the hand has a resistance of about 30,000 W/cm<sup>2</sup>. Deeply calloused skin can have 20 to 70 times greater resistance.<sup>11</sup>

**Table 1:**

<i>Skin Resistance</i>	
<b>Tissue</b>	<b>Tissue and Resistance</b>
Mucous Membranes	100
<b>Vascular Areas</b>	
Volar arm, inner thigh	300-10,000
<b>Wet Skin</b>	
Bathtub	1200–1500
Sweat	2500
Other skin	10,000–40,000
Sole of foot	100,000–200,000
Heavily Calloused Palm	1,000,000–2,000,000

This high resistance may result in a significant amount of energy being expended at the skin surface as the current burns its way through deep callous, resulting in greater thermal injury to the skin but less internal damage than would be expected if the current were delivered undiminished to the deep tissues. As the duration of contact increases, however, the skin begins to blister and break down, resulting in decreased resistance and a surge of current internally that can cause extensive deep-tissue destruction. Moisture also lowers resistance. Sweating can decrease the skin’s resistance to 2500 to 3000 ohms, and immersion in water causes a further reduction to 1200 to 1500 ohms.

**Amperage**

Current, expressed in amperes, is a measure of the amount of energy that flows through an object. As defined by Joule’s law, the heat generated is proportional to the amperage squared.<sup>13</sup> Amperage is dependent on the source voltage and the resistance of the conductor and must normally be estimated. Although the voltage of the source is often known, the resistance varies according to the tissues involved. Additionally, as the tissue breaks down under the energy of the current flow, its resistance may change markedly, making it difficult to predict the amperage for any given electrical injury.

Table 2 shows the physical effects of different amperages at 50 to 60 Hz, which is the AC frequency used in European countries and the United States.

**Table 2:**

*Physical Effects of Different Amperage Levels at 50 to 60*

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<i><b>Hz</b></i>	
<b>Physical effect</b>	<b>Milliamperes (mA)</b>
Tingling sensation	1–4
Let-go current	
Children	4
Women	7
Men	9
Freezing to circuit	10–20
Respiratory arrest from thoracic muscle tetany	20–50
Ventricular fibrillation	60–120

A very narrow range exists between the threshold of perception of current (0.2 to 0.4 mA) and let-go current (6 to 9 mA), the level above which a person becomes unable to release the current source because of muscular tetany. Thoracic tetany can occur at levels just above this let-go current and result in respiratory arrest owing to paralysis of the muscles of respiration. Ventricular fibrillation is estimated to occur at an amperage of 60 to 120 mA. Using skin resistances from Table 1, contact with a 110-volt source with dry skin (10,000 to 40,000 W) results in current of 2.75 to 11 mA. Wet skin submerged in a bathtub (1200 to 1500 W) subjected to the same household voltage results in a current of 73–92 mA, that can cause electrocution with cardiac arrest but no surface burns.

### **Duration of Contact**

In general, the longer the duration of contact with high-voltage current, the greater the electrothermal heating and degree of tissue destruction. Once carbonization of tissue occurs, the resistance to current flow increases. The physics are different with lightning. The extremely short duration and extraordinarily high voltage and amperage of lightning both result in a very short flow of current internally, with little, if any, skin breakdown and almost immediate flashover of current around the body.

### **Voltage**

Voltage is a measure of the difference in electrical potential between two points and is determined by the electrical source. Electrical injuries are conventionally divided into high or low voltage using 500 or 1000 V as the most common dividing lines. Although both high and low voltage can cause significant morbidity and mortality, high voltage results in greater current flow and therefore has a greater potential for tissue destruction leading to major amputations and tissue loss.

No deaths are recorded from contact with the low voltages associated with long distance communications lines (24 V) or telephone lines (65 V). Death, however,

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is reported with exposure to 110 V household current, especially in special environmental circumstances such as bathtub-related electrocutions.

### **Pathway**

The pathway that a current takes determines the tissues at risk, the type of injury seen, and the degree of conversion of electrical energy to heat. This is true whether high, low, or lightning voltages are being considered. Current passing through the heart or thorax can cause cardiac dysrhythmias and direct myocardial damage. Current passing through the brain can result in respiratory arrest, seizures, and paralysis. Current in proximity to the eyes can cause cataracts. Current passing through the trunk of a body will cause less damage than if it passes through a single digit. As current density increases, its tendency to flow through the less-resistant tissues is overcome. Eventually it will flow through tissues indiscriminately, as if the body were a volume conductor, with the potential to destroy all tissues in the current's path. Because the current is often concentrated at the source and ground contact points, the greatest degree of damage is often observed there. Nevertheless, extensive deep destruction of the tissues may exist between these sites with high-voltage injuries, and the surface damage is often only "the tip of the iceberg" for these injuries. Damage to the internal structures of the body may be spotty, with areas of normal-appearing tissue adjacent to burned tissue and with damage to structures at sites distant from the apparent contact points. Although lightning is governed by the same physical laws as artificial electricity, the rapid rise and decay of the energy makes predicting the extent of lightning injury even more complicated than artificial electrical injury. The most important difference between lightning and high-voltage electrical injuries is the duration of exposure to the current.

Lightning is neither a direct current nor an alternating current. At best, it is a unidirectional massive current impulse. Therefore lightning is classed as a current phenomenon, rather than a voltage phenomenon. The cloud-to-ground lightning impulse results from the breakdown of a large electric field between a cloud and the ground, measured in millions of volts. Once connection is made with the ground, this voltage difference between the cloud and ground disappears, and a large current flows impulsively for a very short time. The study of such massive discharges of such short duration is not well advanced, particularly with respect to the effects on the human body.

Mathematical modeling of a lightning strike to the human body is substantiated on animal models.<sup>5, 15</sup> After lightning meets the body, current is initially transmitted internally, after which skin breaks down, and ultimately external "flashover" occurs. Experimental evidence suggests that "a fast flashover appreciably diminishes the energy dissipation within the body and results in survival."<sup>16</sup> Although current may flow internally for an incredibly short time and cause short-circuiting of electrical systems such as the heart, respiratory centers, and autonomic nervous system, as well as spasm of arteries and muscles, it seldom causes significant burns or tissue destruction.<sup>5, 17</sup> Thus burns and myoglobinuric renal failure are not a common injury pattern from lightning, whereas cardiac and

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respiratory arrest, vascular spasm, neurologic damage, and autonomic instability play a much greater role.

Lightning tends to cause asystole rather than ventricular fibrillation. Although cardiac automaticity may reestablish a rhythm, the duration of the respiratory arrest may cause secondary deterioration of the rhythm to refractory ventricular fibrillation and asystole.<sup>2, 5, 18</sup> This secondary arrest occurs experimentally in sheep.<sup>17</sup> Other injuries caused by blunt trauma or ischemia from vascular spasms, such as myocardial infarction or spinal artery syndromes, may occasionally occur.<sup>19, 20, 21, 22, 23</sup>

### *Mechanisms of Injury*

#### **Electrical Injury**

The primary electrical injury is burns. Secondary blunt trauma results from falls or being thrown from the electrical source by an intense contraction of muscles.

Electrical burns can be classified into four different types.<sup>24</sup>

#### **Box 4:**

<i>Types of Electrical Burns</i>
<b>Direct contact</b>
Electrothermal heating
<b>Indirect contact</b>
Arc
Flame
Flash

Heating of tissues secondary to current causes electrothermal burns. Usually these burns are a result of a low voltage shock with a limited affected area. Severe electrothermal burns can occur, however, if a person grips a high voltage conductor. The prolonged flow of current can result in significant burns anywhere along the current path. Typically the skin lesions of electrothermal burns are well-demarcated, deep-partial to full-thickness burns.

The most destructive indirect injury occurs when a victim becomes part of an electrical arc. An electrical arc is a current spark formed between two objects of differing potential that are not in contact with each other, usually a highly charged source and a ground. Because the temperature of an electrical arc is approximately 2500° C, it causes very deep thermal burns at the point where it contacts the skin.<sup>14</sup> In arcing circumstances, burns may be caused by the heat of the arc itself, electrothermal heating due to current flow, or by flames that result from the ignition of clothing. Instead of jumping in the form of a discrete arc

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causing contact point burns, current may jump the gap by splashing across the entire body. These splash burns may cover a large portion of the body but are generally only partial thickness.<sup>24</sup>

At the time of presentation, it is often difficult to determine the mechanism of injury that caused an electrically injured patient's burns. Electrothermal heating is the main cause of muscle damage and is almost exclusively seen in high voltage accidents with prolonged (seconds) contact and current flow.<sup>24</sup>

The histologic change seen in muscle injury that results from direct contact with an electrical source is coagulation necrosis with shortening of the sarcomere.<sup>11, 14</sup> Muscle damage can be spotty, so areas of viable and nonviable muscle are often found in the same muscle group. Periosteal muscle damage may occur even though overlying muscle appears to be normal. Similar to the muscle damage, serious vascular damage usually occurs only after a high voltage accident.

Vascular damage is greatest in the media. This can lead to delayed hemorrhage when the vessel eventually ruptures.<sup>2, 14, 25</sup> Intimal damage may result in either immediate or delayed thrombosis and vascular occlusion as edema and clots form on the damaged internal surface of the vessel over a period of days.<sup>25</sup> The injury is usually most severe in the small muscle branches, where blood flow is slower.<sup>26</sup> This damage to small muscle arteries, combined with mixed muscle viability that is not visible to gross inspection, creates the illusion of "progressive" tissue necrosis.

Immediate arterial thrombosis will result in the absence of a pulse on initial examination. The absence of a pulse, however, can also be due to transient vascular spasm. Pulselessness resulting from vascular spasm should resolve within a few hours. If pulselessness persists after this time, serious vascular injury is likely.

Damage to neural tissue may also occur via several mechanisms. A nerve may demonstrate an immediate drop in conductivity as it undergoes coagulation necrosis similar to that observed in muscle. In addition, it may suffer indirect damage as its vascular supply or myelin sheath is injured or as progressive edema results in a compartment syndrome. The signs of neural damage may develop immediately or be delayed hours to days. Since the skull is a common contact point, the brain is commonly injured. Histologic studies of the brain reveal focal petechial hemorrhages in the brain stem, cerebral edema, and widespread chromatolysis (the disintegration of chromophil bodies of neurons).<sup>14</sup>

Exposure to electricity may cause immediate death from asystole, ventricular fibrillation, or respiratory paralysis, depending on the voltage and pathway.

### **Lightning Injury**

Lightning injury may occur by five mechanisms.

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#### **Box 5:**

***Mechanisms of Lightning Injury***

**Direct strike**

Orifice entry

**Contact**

Side flash, “splash”

Ground current or step voltage

Blunt trauma

The mechanism of injury with a direct strike is self-evident. It has been substantiated experimentally in sheep that lightning strikes near the head may enter orifices such as the eyes, ears, and mouth to flow internally.<sup>17, 19, 27</sup> This may help explain the myriad eye and ear symptoms and signs that are reported with lightning injury.

Injury from contact occurs when the person is touching an object that is part of the pathway of lightning current, such as a tree or tent pole. Side flash or splash occurs as lightning jumps from its primary strike object to a nearby person on its way to ground.<sup>21, 28, 29</sup> Step voltage, a difference in electrical potential between a person's feet, may occur as lightning current spreads radially through the ground. A person is a far better conductor of electricity than the earth. Thus a person who has one foot closer than the other to the strike point will have a potential difference between the feet so that the lightning current will preferentially flow through the legs and body rather than the ground. This is a common killer of large livestock such as cattle and horses because of the distance between their hind legs and forelegs.<sup>30</sup>

Blunt injury from lightning can occur from two mechanisms. First, the person may be thrown a considerable distance by the sudden, massive contraction caused by current passing through the body. Second, an explosive or implosive force occurs as the lightning pathway is instantaneously superheated and then rapidly cooled following the passage of the lightning. The heating is seldom long enough to cause severe burns but does cause rapid expansion of air followed by rapid implosion of the cooled air as it rushes back into the void.<sup>5</sup>

## **CLINICAL FEATURES**

High-voltage injury patients commonly present with devastating burns requiring prolonged hospitalization with multiple complications. Lightning and low-voltage injury patients may have little evidence of injury or, alternatively, may be in

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cardiopulmonary arrest. After the initial resuscitation of lightning and low-voltage injury patients, other conditions may be identified. These patients may have significant residual morbidity from pain syndromes or brain damage that is similar to that suffered with blunt head injury.<sup>31, 32, 33, 34, 35</sup>

### *Head and Neck*

#### **Electrical Injury**

The head is a common point of contact for high-voltage injuries, and the patient may exhibit burns as well as neurologic damage. Cataracts develop in approximately 6% of cases of high-voltage injuries, especially whenever electrical injury occurs in the vicinity of the head.<sup>36</sup> Although cataracts may be present initially or develop shortly after the accident, they more typically appear months after the injury. Visual acuity and fundoscopic examination should be performed at presentation. Referral to an ophthalmologist familiar with electrical cataract formation may be necessary.

#### **Lightning Injury**

Lightning strikes may cause skull fractures and cervical spine injury from associated blunt trauma.<sup>21, 22, 37</sup> Tympanic membrane rupture is commonly found in lightning victims and may be secondary to the shock wave, a direct burn, or a basilar skull fracture.<sup>5, 18, 38</sup> Although most patients recover without serious sequelae, disruption of the ossicles and mastoid may occur, as well as cerebrospinal fluid otorrhea, hemotympanum, and permanent deafness.<sup>18, 38</sup> Injuries to the eyes include corneal lesions, uveitis, iridocyclitis, hyphema, vitreous hemorrhage, optic atrophy, retinal detachment, and choroidoretinitis. As a result, dilated, unreactive pupils are not a reliable indicator of death.<sup>39</sup> As with electrical injuries, cataracts may develop later in some patients.<sup>5, 18, 40</sup>

### *Cardiovascular System*

#### **Electrical Injury**

Cardiac arrest, either from asystole or ventricular fibrillation, is a common presenting condition in electrical accidents. Other electrocardiographic (ECG) findings include sinus tachycardia, transient ST segment elevation, reversible QT segment prolongation, premature ventricular contractions, atrial fibrillation, and bundle branch block.<sup>2, 41</sup> Acute myocardial infarction is reported but is relatively rare.<sup>42, 43</sup> Damage to skeletal muscles may produce a rise in the CPK-MB fraction, leading to a spurious diagnosis of myocardial infarction in

some settings.<sup>43, 44</sup>

### **Lightning Injury**

In lightning injury, cardiac damage or arrest may be caused by either the electrical shock or induced vascular spasm.<sup>20, 45</sup> Numerous dysrhythmias are reported in the absence of cardiac arrest.<sup>5, 14</sup> Nonspecific ST-T wave segment changes and prolongation of the QT interval may occur, and serum levels of cardiac enzymes are often elevated.<sup>5, 46, 47</sup> Hypertension is commonly present initially with lightning injury but usually resolves without treatment within a few hours.

## *Skin*

### **Electrical Injury**

Other than cardiac arrest, the most devastating injuries that accompany an electrical injury are burns, which are most severe at the source and ground contact points. The most common sites of contact with the source include the hands and the skull. The most common areas of ground are the heels. A patient may have multiple source and ground contact points. Burns in severe electrical accidents often appear as painless, depressed, yellow-gray, punctate areas with central necrosis, or the areas may be mummified.<sup>14</sup> High-voltage current often flows internally and can create massive muscle damage. If contact was brief, however, minimal flow may have occurred and the visible skin damage may represent nearly all of the damage. One should not attempt to predict the amount of underlying tissue damage from the amount of cutaneous involvement.

A peculiar type of burn associated with electrical injury is the “kissing burn,” which occurs at the flexor creases.<sup>14</sup> As the current causes flexion of the extremity, the skin of the flexor surfaces at the joints touches. Combined with the moist environment that often occurs at the flexor areas, the electric current may arc across the flexor crease, causing arc burns on both flexor surfaces and extensive underlying tissue damage.

Electrical flash burns are usually superficial partial-thickness burns, similar to other flash burns. Isolated thermal burns may also be seen when clothing ignites. The total body surface area affected by burns in electrical injury averages 10% to 25%. Severe burns to the skull, and occasionally to the dura, are reported.<sup>48, 49</sup> The most common electrical injury seen in children less than 4 years of age is the mouth burn that occurs from sucking on a household electrical extension cord.<sup>4</sup> These burns usually represent local arc burns, may involve the orbicularis oris muscle, and are especially worrisome when the commissure is involved because of the likelihood of cosmetic deformity. A significant risk of delayed bleeding from the labial artery exists when the eschar separates. Damage to developing dentition is reported, and referral to an oral surgeon familiar with electrical injuries

is recommended.<sup>50</sup>

### **Lightning Injury**

Deep burns occur in fewer than 5% of lightning injuries.<sup>18, 51</sup> Patients may exhibit one or more of four types of superficial burns or skin changes that do not reflect actual burn injury: linear, punctate, feathering, or thermal burns.<sup>5, 12, 18, 51, 52, 53</sup>

Linear burns tend to occur in areas where sweat or water accumulate, such as under the arms or down the chest. These are superficial burns that appear to be caused by steam production from the flashover phenomenon. Punctate burns appear as multiple, small cigarette-like burns, often with a heavier central concentration in a rosette-like pattern.<sup>52</sup> They range from a few millimeters to a centimeter in diameter and seldom require grafting.

Feathering burns are not true burns because there is no damage to the skin itself.<sup>51</sup> They seem to be caused by electron showers induced by the lightning that make a fern pattern on the skin.<sup>51, 52, 53</sup> These transient lesions are pathognomonic for lightning injury and require no therapy.<sup>54</sup> Thermal burns occur if the clothing is ignited or may be caused by metal that the person is wearing or carrying during the flashover.<sup>24</sup>

## ***Extremities***

### **Electrical Injury**

In high-voltage injuries, muscle necrosis can extend to sites distant from the observed skin injury, and compartment syndromes occur as a result of vascular ischemia and muscle edema. Decompression fasciotomy or amputation is often necessary because of extensive tissue damage.<sup>55, 56</sup> Massive release of myoglobin from the damaged muscle may lead to myoglobinuric renal failure. Vascular damage from the electrical energy may become evident at any time.<sup>25, 26</sup> Pulses and capillary refill should be assessed and documented in all extremities, and neurovascular checks should be repeated frequently. Because the arteries are a high-flow system, heat may be dissipated fairly well and cause little apparent initial damage but result in subsequent deterioration. The veins, on the other hand, are a low-flow system, allowing the heat energy to cause more rapid heating of the blood, with resulting thrombosis. Consequently, an extremity may appear edematous initially. With severe injuries, the entire extremity may appear mummified when all tissue elements, including the arteries, suffer coagulation necrosis.

Damage to the vessel wall at the time of injury may also result in delayed thrombosis and hemorrhage, especially in the small arteries to the muscle.<sup>25, 26</sup> This ongoing vascular damage can cause a partial-thickness burn to develop into

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a full-thickness burn as the vascular supply to the area diminishes. Progressive loss of muscle because of vascular ischemia downstream from damaged vessels may mandate repeated deep debridements.<sup>26</sup>

### **Lightning Injury**

Lightning injury may cause transient vasospasm so severe that the extremities appear cold, blue, mottled, and pulseless. This usually resolves within a few hours and rarely requires vascular imaging or surgical intervention.<sup>4, 18</sup>

### ***Skeletal System***

Fractures of most of the long bones caused by the trauma associated with electrical injury are reported.<sup>59, 60</sup> Both posterior and anterior shoulder dislocations caused by tetanic spasm of the rotator cuff muscles are also reported, as well as spinal fractures. As with electrical injury, numerous types of fractures and dislocations are reported with lightning injury.<sup>5</sup>

### ***Nervous System***

#### **Electrical Injury**

In high-voltage injuries, loss of consciousness may occur but is usually transient unless there is a significant concomitant head injury. Prolonged coma with eventual recovery is also reported. Patients may exhibit confusion, flat affect, and difficulty with short-term memory and concentration.<sup>31, 32, 33, 34, 35, 59</sup> Electrical injury to the central nervous system (CNS) may cause a seizure, either as an isolated event or as part of a new-onset seizure disorder.<sup>59</sup> Other possible causes of seizures, such as hypoxia and traumatic CNS injury, should be considered. Neurologic symptoms may improve, but long-term disability is common. Lower extremity weakness is commonly undiagnosed until ambulation is attempted.<sup>60</sup>

In high-voltage exposures, spinal cord injury may result from fractures or ligamentous disruption of the cervical, thoracic, or lumbar spine.<sup>60, 61, 62</sup> Neurologic damage in patients without evidence of spinal injury seems to follow two patterns, immediate and delayed. Patients with immediate damage have symptoms of weakness and paresthesias develop within hours of the insult.<sup>59</sup> Lower extremity findings are more common than upper extremity findings. These patients have a good prognosis for partial or complete recovery. Delayed neurologic damage may present from days to years after the insult. The findings usually fall into three clinical pictures: ascending paralysis, amyotrophic lateral sclerosis, or transverse myelitis.<sup>62</sup> Motor findings predominate. Sensory findings are also common, but they may be patchy and may not match the motor levels.

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Although recovery is reported, the prognosis is usually poor.<sup>60</sup>

### **Lightning Injury**

On initial presentation, up to two thirds of the seriously injured lightning patients have keraunoparalysis, which is a unique temporary paralysis secondary to lightning strike. It is characterized by lower and sometimes upper extremities that are blue, mottled, cold, and pulseless. These findings are secondary to vascular spasm and sympathetic nervous system instability.<sup>18, 63</sup> Generally this clears within a few hours, although some patients may be left with permanent paresis or paresthesias.

Paraplegia, intracranial hemorrhages, cardiac enzyme elevations, seizures, and electroencephalogram (EEG) changes are reported after lightning injuries.<sup>5, 46, 47, 62, 63, 64, 65, 66</sup> Loss of consciousness for varying periods is common, and confusion and anterograde amnesia are almost universal findings.<sup>18, 67</sup> Peripheral nerve damage is common, and recovery is usually poor.<sup>68, 69</sup> A syndrome of delayed muscle atrophy caused by electrical injury of the nerves is described, even in the absence of cutaneous burns.<sup>70</sup>

### ***Other Viscera***

#### **Electrical Injury**

Injury to the lungs may occur because of associated blunt trauma but is rare from electrical current, perhaps because air is a poor conductor. Injury to solid visceral organs also is rare, but damage to the pancreas and liver is reported.<sup>71</sup> Injuries to hollow viscera, including the small intestine, large intestine, bladder, and gallbladder, are also reported.<sup>71,72</sup>

#### **Lightning Injury**

Pulmonary contusion and hemorrhage are reported with lightning injury.<sup>22, 73</sup> Blunt abdominal injuries occur rarely.<sup>5</sup> None of the other intraabdominal catastrophes commonly associated with high-voltage electrical injury such as gallbladder necrosis or mesenteric thrombosis are seen with lightning injury.

### ***Other Low-Voltage Injuries***

An accurate history is essential to ensure that an apparent low-voltage injury was not caused by the discharge from a capacitor (as in the repair of a television, microwave oven, or computer monitor) or other high-voltage source. Although burns from low-voltage sources are usually less severe than those from high-voltage sources, patients may still complain of paresthesias for an extended

period, experience cardiac dysrhythmias, or have cataracts develop if the shock occurs close to the face or head.<sup>4</sup>

## ***Complications***

### **Electrical Injury**

Cardiac arrest generally occurs only at the initial presentation or as a final event after a long and complicated hospital course. Many of the complications are similar to those of thermal burns and crush injuries, including infection, clostridial myositis, and myoglobinuria. The incidence of acute myoglobinuric renal failure has decreased since the widespread adoption of aggressive alkalinized fluid resuscitation. Fasciotomies or carpal tunnel release may be necessary for treatment of compartment syndromes.<sup>55, 56</sup> Tissue loss and major amputations are common with severe high-voltage injuries and result in the need for extensive rehabilitation.

Neurologic complications such as loss of consciousness, peripheral nerve damage, and delayed spinal cord syndromes may occur.<sup>35, 59, 60, 61, 62, 69</sup> Damage to the brain may result in a permanent seizure disorder.<sup>37</sup> Long-term neuropsychiatric complications include depression, anxiety, inability to continue in the same profession, aggressive behavior, and suicide.<sup>74</sup> Stress ulcers are the most common gastrointestinal complication after burn ileus. Abdominal injuries from ischemia, vascular damage, burns, or associated blunt trauma may be overlooked initially.<sup>14, 71, 72</sup> The most common causes of hospital mortality are pneumonia, sepsis, and multisystem organ failure.

### **Lightning Injury**

Complications of lightning injury fall into three areas: (1) those that could be reasonably predicted from the presenting signs and can be treated routinely, such as hearing loss from tympanic membrane rupture or paresthesias and paresis from neurologic damage, (2) long-term neurologic deficits similar to those suffered with blunt head injury and chronic pain syndromes, and (3) iatrogenic complications that are secondary to overaggressive management.

In the past, patients with lightning injuries were often treated like those with high-voltage electrical injuries. These injuries, however, are distinctly different. The treatment of lightning victims seldom requires massive fluid resuscitation, fasciotomies for compartment syndromes, mannitol and furosemide diuretics, alkalinization of the urine, amputations, or large repeated debridements.<sup>5, 18, 21, 46</sup>

## **DIFFERENTIAL CONSIDERATIONS**

### **Electrical Injury**

Electrical injuries are usually self-evident from historical surroundings, except in the case of bathtub accidents, instances when no burns occur, or foul play. It is important to determine the mechanism of burn injury because flash burns have a much better prognosis than arc or conductive burns. Alterations in consciousness or seizures can be caused by the electrical injury or result from an associated traumatic brain injury.

### **Lightning Injury**

The differential diagnosis of lightning injury is more complex, often because the incident is unobserved. It includes many of the causes of unconsciousness, paralysis, or disorientation of unclear etiology.<sup>5</sup>

#### **Box 6:**

<i>Differential Diagnosis of Lightning Injuries</i>
Cardiac dysrhythmias Myocardial infarction Cerebrovascular accident Subarachnoid hemorrhage Seizures Closed-head injury Spinal cord trauma

Evidence of a thunderstorm or a witness to the lightning strike may not be available, particularly when victims are alone when injured. The presence of typical burn patterns, such as feathering, may be helpful.

## **MANAGEMENT**

### ***Prehospital***

#### **Securing the Scene**

When first reaching the scene, prehospital personnel should secure the area so that bystanders and rescuers do not sustain other injuries. For high-voltage incidents, the power source must be turned off. Although many approaches to achieving this goal are recommended, the safest approach is to involve the local

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power company in high-voltage accidents. Accidents involving discrete electrical sources that are easily disconnected through a circuit box or switch are easier to manage, although rescuers should still ensure that the power is off before approaching the victim. The use of electrical gloves by emergency medical service (EMS) personnel is very dangerous. A microscopic hole in a glove can result in an explosive injury to the hand inside it, as thousands of volts from the circuit concentrate there to enter the glove.

Although a line may be on the ground and appear to be dead, it may have substantial current from it pouring into the ground. This current spreads out in a circle along and just below the surface of the ground, so the area remains electrified and dangerous, a phenomenon known as ground current. Even if the line is dead, automatic circuit reclosers may repower the line resulting in successive surges of current.<sup>75</sup> Therefore rescue vehicles should park at least one entire span away from the line.

In lightning incidents, pre-hospital personnel must remember that lightning can strike the same place twice and guard themselves against also becoming victims with appropriate and timely evacuation measures.

### **Triage Considerations**

Field evaluation of patients may involve triage of multiple victims. Traditional rules of mass casualty triage do not apply to lightning victims. The major cause of death in lightning injuries is cardiorespiratory arrest. <sup>18</sup> In the absence of cardiopulmonary arrest, victims are highly unlikely to die of any other cause.<sup>18</sup> Thus triage of lightning victims should concentrate on those who appear to be in cardiorespiratory arrest. Cardiopulmonary resuscitation should be started on those who have no pulse or respirations. When multiple victims are involved, the evaluation of those who are breathing may be delayed because they are likely to survive the incident.

For practical purposes, lightning acts as a massive DC countershock that results in asystole.<sup>5</sup> Although intrinsic cardiac automaticity may resume cardiac activity, the respiratory arrest caused by CNS injury often lasts longer than the cardiac pause and may lead to a secondary cardiac arrest with ventricular fibrillation from hypoxia.<sup>6, 14, 17</sup> If the victim is properly ventilated during the time between the two arrests, the second arrest may theoretically be avoided.

### **Initial Resuscitation**

Electrical injury victims may require a combination of cardiac and trauma care because they often suffer blunt injuries and burns as well as possible cardiac damage. Spinal immobilization is indicated whenever associated spinal trauma is suspected. Fractures and dislocations should be splinted, and burns should be covered with clean, dry dressings.

All patients with conductive injury should have at least one large-bore intravenous (IV) line established. An electrical injury should be treated like a crush injury, rather than a thermal burn, because of the large amount of tissue damage that is often present under normal-appearing skin. As a result, none of the formulas for IV fluids based on percentage of burned body surface area are reliable.

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Hypotensive patients should initially receive a bolus of 20 ml/kg of isotonic fluid, and subsequent fluid management then based on the patient's vital signs and clinical status.

### ***ED***

#### **Assessment**

The victim of an electrical injury may not be able to give an adequate history, either because of the severity of injury and accompanying shock and hypoxia or because of unconsciousness or confusion that often accompanies less severe injuries. The history obtained from bystanders and the paramedics regarding the type of electrical source, duration of contact, environmental factors at the scene, and resuscitative measures provided can be helpful.

Resuscitative efforts should be continued in the ED with adequate fluid administration and insertion of a Foley catheter for patients with high-voltage injury and extensive burns. Fluids should be administered at a rate sufficient to maintain a urine output of at least 0.5 to 1.0 ml/kg/hr in the absence of heme pigment in the urine and 1.0 to 1.5 ml/kg/hr in its presence.

Cardiac monitoring is indicated for severely injured patients and for those who have the indications listed in Box 7.76

#### **Box 7:**

<b><i>Indications for Electrocardiographic Monitoring</i></b>
Cardiac arrest
Documented loss of consciousness
Abnormal ECG
Dysrhythmia observed in prehospital or ED setting
History of cardiac disease
Presence of significant risk factors for cardiac disease
Concomitant injury severe enough to warrant admission
Suspicion of conductive injury
Hypoxia
Chest pain

All high-voltage injury victims and low-voltage victims with cardiorespiratory complaints should have an ECG and cardiac isoenzyme determinations. Although ECG changes and dysrhythmias are common with electrical injuries, anesthesia and surgical procedures performed in the first 48 hours of care can be accomplished without cardiac complications.<sup>76, 77</sup> The patient's clinical status should guide the use of invasive monitoring with central venous pressure catheters, intracranial pressure monitors, and Swan-Ganz catheters.<sup>5, 78</sup> Victims of electrical injury with altered mentation should have a computed

tomography (CT) scan performed. Most lightning victims will behave as though they have had electroconvulsive therapy, with confusion and anterograde amnesia for several days. If any neurologic deterioration occurs after an electrical injury, a CT scan is indicated to assess for intracranial hemorrhage.<sup>18, 66</sup>

Lightning victims who do not suffer cardiopulmonary arrest at the time of the strike generally do well with supportive therapy. Those who have cardiopulmonary arrest may have a poor prognosis, particularly if there is hypoxic brain damage.<sup>5, 6, 76</sup>

### ***Ancillary Tests***

#### **Electrical Injury**

The laboratory evaluation of the patient sustaining an electrical injury depends on the extent of injury. All patients with evidence of conductive injury or significant surface burns should have the following laboratory tests: complete blood count (CBC), electrolyte levels, serum myoglobin, blood urea nitrogen (BUN), serum creatinine, and urinalysis. Patients with severe electrical injury or suspected intraabdominal injury should also have obtained pancreatic and hepatic enzymes and coagulation profile.<sup>41</sup> The clinician should consider ordering a type and cross-match, particularly if major debridements may be necessary. Arterial blood gas analysis is indicated if the victim needs ventilatory intervention or alkalinization therapy.

All patients should be evaluated for myoglobinuria, a common complication of high voltage electrical injury. If the urine is pigmented or the dipstick examination of the urine is positive for blood, and no red blood cells (RBCs) are seen on microscopic analysis, assume the patient has myoglobinuria and treat accordingly. Creatine kinase (CK) levels should be drawn and isoenzyme analysis performed. Peak CK levels are shown to predict the amount of muscle injury, risk of amputation, and length of hospitalization; the clinical value of a single level in the acute setting, however, is not established. Cardiac enzyme levels should be interpreted with care when diagnosing myocardial infarction in the setting of electrical injury. The peak CK level is not indicative of myocardial damage in electrical injury because of the large amount of skeletal muscle injury. Skeletal muscle cells damaged by electrical current can contain as much as 20% to 25% CK-MB fraction, suggesting injured skeletal muscle rather than myocardial injury as a possible source of an elevated CK-MB fraction.<sup>44</sup> CK-MB fractions, ECG changes, thallium studies, angiography, and echocardiography correlate poorly in acute myocardial infarction following electrical injury. Other cardiac enzymes (such as troponin) are not well studied in electrical injury but may prove useful in determining myocardial injury.

All patients sustaining an electrical injury should receive cardiac monitoring in the ED and an ECG despite the source voltage. Indications for admission for electrocardiographic monitoring are listed in Box 7.41, <sup>76</sup>

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Radiographs of the cervical spine should be performed if spinal injury is clinically suspected. Radiographs of any other areas in which the patient complains of pain or has an apparent deformity should be performed. Angiography is not shown to be useful in the planning of debridements or amputations and is not routinely indicated.<sup>26</sup> Technetium pyrophosphate scanning to detect areas of clinically unsuspected myonecrosis can be useful.<sup>45, 55</sup> Nonviable muscle will appear as cold spots on the scan, lacking the normal level of uptake. Hot spots on the scan may consist of 20% to 80% viable muscle and should be followed clinically.<sup>55, 79, 80</sup> CT or magnetic resonance imaging may be useful in the evaluation of associated trauma and are essential for evaluation of possible intracranial injuries, particularly if the Glasgow Coma Scale score does not progressively improve.

### **Lightning Injury**

In patients injured by lightning an ECG should be obtained. Serum markers for cardiac injury are indicated in patients with chest pain, abnormal ECGs, or altered mentation. The severity or nature of the victim's injuries may require other laboratory studies. Radiographic studies, particularly CT scan of the head, may be indicated, depending on the patient's level of consciousness at presentation and throughout the evaluation and treatment.

### ***Specific Therapies***

#### **Rhabdomyolysis**

Patients with heme pigment in the urine should be assumed to have myoglobinuria until the diagnosis can be excluded by more specific testing. Alkalinization of the urine increases the solubility of myoglobin in the urine, increasing the rate of clearance. Urine output should be maintained at 1.0 to 1.5 ml/kg/hr until all traces of myoglobin have cleared from the urine while the blood is maintained at a pH of at least 7.45 using sodium bicarbonate. Furosemide or mannitol may be used to cause further diuresis. The recommended dose of mannitol is 25 g initially, followed by 12.5 g/kg/hr, titrated as needed to maintain urine flow greater than 50 ml/hr. Unlike with high-voltage injuries, rhabdomyolysis is rare with lightning injuries.

#### **Burn Wound Care**

Cutaneous burns should be dressed with antibiotic dressings, such as sulfadiazine silver (Silvadene). Mafenide acetate (Sulfamylon) is occasionally used for selected burns; however, its use may result in electrolyte abnormalities because of its carbonic anhydrase inhibitory activity.

Electrical burns are especially prone to tetanus, and patients should receive

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tetanus toxoid and tetanus immune globulin on the basis of their immunization history. Clostridial myositis is common, but prophylactic administration of high-dose penicillin to prevent clostridial myonecrosis is controversial and should not be used without discussion with the managing surgeon or burn unit. In general, systemic antibiotics are not used unless culture or biopsy proves infection is present.

### **Extremity Injuries**

Current management of electrical injuries of the extremities favors early and aggressive surgical management, including early fasciotomy, carpal tunnel release, or even amputation of an obviously nonviable extremity.<sup>56, 81</sup>

Extremities should be splinted in a functional position to minimize edema and contracture formation. The hand should be splinted in 35- to 45-degree extension at the wrist, 80- to 90-degree flexion at the metacarpophalangeal joints, and almost full extension at the proximal and distal interphalangeal joints. This position minimizes edema formation. During the first several days of hospitalization, frequent monitoring of the neurovascular status of all extremities is essential.

## **DISPOSITION**

### **Electrical Injuries**

#### *Transfer to Burn Center*

Most patients with significant electrical burns should be stabilized and transferred to a regional burn center for burn care and extensive occupational and physical rehabilitation. These severely injured victims and their families may benefit from counseling because of the extensive life changes that occur consequent to the injury.

#### *Admission*

Indications for admission for electrocardiographic monitoring are listed in Box 7. In general, when corporal conduction is suspected, the patient should be admitted for 12 to 24 hours of cardiac monitoring.

#### *Outpatient Management*

Patients who are totally asymptomatic and have a normal physical examination after low-voltage exposure can be reassured and then discharged without performing any ancillary tests.<sup>82</sup> Those patients with cutaneous burns or mild persistent symptoms can be discharged if they have a normal ECG and no urinary heme pigment. The ED physician should provide out-patient referral in the event that current symptoms persist or new symptoms (delayed cataracts, weakness, or paresthesias) develop.

Electrical injury during pregnancy from low-voltage sources is reported to result

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in stillbirth. A prospective cohort study of women receiving electric shock in pregnancy suggests that accidental electric shock usually does not pose a major fetal risk.<sup>83</sup> Nevertheless, obstetric consultation or referral is advisable for all pregnant patients reporting electrical injury, regardless of symptomatology at the time of presentation. Placental abruption, the most common cause of fetal death after blunt trauma, may result from even minor trauma such as may be associated with electrical injuries. Patients in the latter half of pregnancy should receive fetal monitoring if there has been even minor blunt trauma and be considered high-risk patients for the remainder of their pregnancy.<sup>84, 85</sup> First-trimester patients should be informed of the remote risk of spontaneous abortion and, if no other indications for admission exist, may be discharged with instructions for threatened miscarriage and close obstetric follow-up evaluation. The prognosis for fetal survival after lightning strike varies.<sup>5, 18</sup>

Pediatric patients with oral burns may generally be safely discharged if close adult care is assured. There is no evidence that an isolated oral burn correlates with cardiac injury or myoglobinuria. In general, these patients require surgical and dental consultation for oral splinting, eventual debridement, and occasionally reconstructive surgery. After appropriate consultation, if hospitalization is not deemed necessary, the child's parents should be warned about the possibility of delayed hemorrhage and receive instructions to apply direct pressure by pinching the bleeding site and to immediately return to the ED.

### **Lightning Injuries**

Many of the signs of lightning injuries, such as lower extremity paralysis and mottling and confusion and amnesia, resolve with time. After spinal cord and intracranial processes are excluded, observation is the mainstay of treatment. Consultation with other specialists may be indicated for otic and ophthalmic damage, although these are usually not emergent considerations. More severely injured lightning patients require both trauma surgeon and cardiology consultations, although with lightning injuries medical pathology predominates. If there is a history of loss of consciousness or if the patient exhibits confusion, hospital admission and observation are suggested. After evaluation, if the ECG is normal, asymptomatic patients (including those with feathering burns) may be discharged home with referral for follow up from ophthalmology and other specialties as indicated.

## **KEY CONCEPTS**

- Exposure to AC is three times more dangerous than DC of the same voltage because of the potential for muscular tetany and prolonged contact.

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- Nerves, muscles, and blood vessels have low resistance and are better electrical conductors than bone, tendon, and fat.
- Electrical burns are usually most severe at the source and ground contact points. It is not possible to predict the amount of underlying tissue damage based on the amount of cutaneous involvement.
- Traditional rules of triage do not apply to lightning victims. Triage of lightning victims should concentrate on those who appear to be in cardiorespiratory arrest.
- The most common presenting signs of lightning injury (keraunoparalysis, mottling, confusion, amnesia) resolve with time. After spinal cord and intracranial processes are excluded, observation is the mainstay of treatment.

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