

Emergent Injuries To Children And Adolescents Due To Electricity And Lightning Strikes

A 13-month-old male presents to the ED with a small, deep burn of about 7 millimeters in diameter to the left oral commissure. The child's mother states that her son had been playing under a table, where he was found unconscious on the floor, adjacent to the connection of an extension cord and a table lamp cord. There was some saliva on the connector. The mother administered cardiopulmonary resuscitation, and the child responded. In the ED he is alert and moving all extremities. His pulse is 100 beats per minute, and the cardiac monitor shows a normal sinus rhythm. The child is admitted to telemetry for 24 hours, due to a possible cardiac arrest. He is discharged the next day, and the parents are counseled about surgical follow-up, local mouth care, and the possibility of delayed bleeding.

THOUGH children and adolescents with an injury due to electricity are uncommon in the pediatric ED, because of the broad range of injury severity and the multisystem effects of electricity, the emergency practitioner must be knowledgeable about electricity's effects on the body. In this issue of *Pediatric Emergency Medicine PRACTICE*, we will examine the impact of both generated electricity (low- and high-voltage sources) and lightning strikes on the pediatric population, and detail treatment options that have been proven to decrease the potentially devastating negative outcomes we see when the human body comes into contact with this potent force of nature.

Abbreviations Used In This Article

AC — Alternating current
 AED — Automatic external defibrillator
 Amp — Ampere (unit of current)
 CK — Creatinine kinase
 CK-MB — Creatinine kinase, myocardial band

August 2005
 Volume 2, Number 8

Authors

Floyd S Ota, MD, FAAP

Clinical Assistant Professor of Pediatrics, University of Texas Southwestern Medical Center—Dallas, TX.

Gary F Purdue, MD, FACS

Professor of Surgery, University of Texas Southwestern Medical Center; Director, Parkland Memorial Hospital Burn Center—Dallas, TX.

Peer Reviewers

Kenneth T Kwon, MD, FACEP, FAAP

Director of Pediatric Emergency Medicine, Associate Clinical Professor, Department of Emergency Medicine, UC Irvine Medical Center/University Children's Hospital—Orange, CA.

Carl H Schultz, MD, FACEP

Professor of Emergency Medicine, Department of Emergency Medicine; Co-Director, EMS and Disaster Medical Sciences Fellowship, UCI School of Medicine; Director, Disaster Medical Services, UCI Medical Center—Orange, CA.

CME Objectives

Upon completing this article, you should be able to:

1. Discuss the pathophysiology of electrical injuries in children;
2. Describe the difference between injuries from lightning and injuries from other high-voltage electrical sources;
3. List internal injuries that may arise from electrical contact; and
4. List the indications for surgical intervention following electrical injuries.

Date of original release: August 29, 2005.

Date of most recent review: August 3, 2005.

See "Physician CME Information" on back page.

Editor-in-Chief

Lance Brown, MD, MPH, FACEP, Chief, Division of Pediatric Emergency Medicine; Associate Professor of Emergency Medicine and Pediatrics; Loma Linda University Medical Center and Children's Hospital, Loma Linda, CA.

Editorial Board

Jeffrey R. Avner, MD, FAAP, Professor of Clinical Pediatrics, Albert Einstein College of Medicine; Director, Pediatric Emergency Service, Children's Hospital at Montefiore, Bronx, NY.

Beverly Bauman, MD, FAAP, FACEP, Assistant Chief, Pediatric Emergency Services, Oregon Health & Sciences University, Portland, OR.

T. Kent Denmark, MD, FAAP, FACEP, Residency Director, Pediatric Emergency Medicine; Assistant Professor, Departments of Emergency Medicine and Pediatrics; Loma Linda University Medical Center and Children's Hospital, Loma Linda, CA.

Michael J. Gerardi, MD, FAAP, FACEP, Clinical Assistant Professor, Medicine, University of Medicine and Dentistry of New Jersey; Director, Pediatric Emergency Medicine, Children's Medical Center, Atlantic Health System; Department of Emergency Medicine, Morristown Memorial Hospital.

Ran D. Goldman, MD, Associate Professor, Department of Pediatrics, University of Toronto; Division of Pediatric Emergency Medicine and Clinical Pharmacology and

Toxicology, The Hospital for Sick Children, Toronto.

Martin I. Herman, MD, FAAP, FACEP, Professor of Pediatrics, Division of Critical Care and Emergency Services, UT Health Sciences, School of Medicine; Assistant Director Emergency Services, Lebonheur Children's Medical Center, Memphis TN.

Marilyn P. Hicks, MD, FACEP, Director, Pediatric Emergency Medicine Education, Department of Emergency Medicine, WakeMed, Raleigh, NC; Adjunct Assistant Professor, Department of Emergency Medicine, University of North Carolina, Chapel Hill, Chapel Hill, NC.

Mark A. Hostetler, MD, MPH, Assistant Professor, Department of Pediatrics; Chief, Section of Emergency Medicine; Medical

Director, Pediatric Emergency Department, The University of Chicago, Pritzker School of Medicine, Chicago, IL.

Alson S. Inaba, MD, FAAP, PALS-NF, Pediatric Emergency Medicine Attending Physician, Kapiolani Medical Center for Women & Children; Associate Professor of Pediatrics, University of Hawaii John A. Burns School of Medicine, Honolulu, HI; Pediatric Advanced Life Support National Faculty Representative, American Heart Association, Hawaii & Pacific Island Region.

Andy Jagoda, MD, FACEP, Vice-Chair of Academic Affairs, Department of Emergency Medicine; Residency Program Director; Director, International Studies Program, Mount Sinai School of Medicine, New York, NY.

Brent R. King, MD, FACEP, FAAP, FAAEM, Professor of Emergency Medicine and Pediatrics; Chairman, Department of Emergency Medicine, The University of Texas Houston Medical School, Houston, TX.

Robert Lutten, MD, Professor, Pediatrics and Emergency Medicine, University of Florida, Jacksonville, Jacksonville, FL.

Ghazala Q. Shariieff, MD, FAAP, FACEP, FAAEM, Associate Clinical Professor, Children's Hospital and Health Center/University of California, San Diego; Director of Pediatric Emergency Medicine, California Emergency Physicians.

Gary R. Strange, MD, MA, FACEP, Professor and Head, Department of Emergency Medicine, University of Illinois, Chicago, IL.

CPR — Cardiopulmonary resuscitation
DC — Direct current
Hz — Hertz (measure of frequency)
I — Current (measured in amperes)
P — Power
PALS — Pediatric advanced life support
R — Resistance (measured in ohms)
TBSA — Total body surface area
V — Volt

Critical Appraisal Of The Literature

The current literature pertaining to electrical and lightning injuries consists largely of class II and III evidence. Since electrical injuries make up only a small percentage of overall admissions to tertiary burn units, it takes many years to generate an adequate study population. Currently, other than the American Burn Association's Advanced Burn Life Support (ABLS) and the American College of Surgeons' Advanced Trauma Life Support® (ATLS®) courses, no national or international recommendations are published on the care of the patient acutely injured by electricity. The majority of evidence pertaining to high-voltage contact is based on studies of young adults. Therefore, the way we currently care for children and adolescents with high-voltage injuries is extrapolated from this data. There are a few articles concentrating on pediatric electrical injuries, but they consist mostly of young children with injuries due to low-voltage contact. From these sources there is good evidence suggesting that, in the absence of a known loss of consciousness, cardiac arrest, prolonged contact, or an abnormal cardiac rhythm, continued cardiac monitoring after low-voltage contact in an asymptomatic child is not required.

Epidemiology, Etiology, And Pathophysiology

Overall, electrical injuries constitute a small minority of total burn patients. Approximately 2-7% of all admissions to specialized burn units are due to electrical injuries.¹⁻¹¹ In the ED, electricity is responsible for approximately 2-3% of all children seen with burns.¹² Death from electricity (electrocution) is a relatively rare event. In the United States electricity is responsible for more than 500 deaths annually.¹² Naturally occurring lightning is responsible for about 75-150 deaths annually.¹³ While the potential for morbidity is greatest in high-voltage and lightning injuries, the potential for death exists in all types of electrical exposures. In-home deaths account for the majority of pediatric mortalities; 69% in one study of pediatric and adolescent electrocutions.¹⁴ Homicide and suicide are rare but reported etiologies of electrocution in adolescents and adults.¹⁴⁻¹⁷

Males experience thermal and electrical burns more often than females.^{3,11,18-20} This gender bias becomes increasingly evident as children grow into adulthood, and the injuries from electricity become work-related. Studies done in burn centers find that the number of female patients is small.^{1,5,10} In terms of age, a bimodal distribu-

tion is observed in pediatric patients admitted to burn units.^{3,6,20} The first and largest of the 2 peaks occurs during early childhood.^{3,11,18,20} In a retrospective study of Canadian electrical injuries, 68% of all electrical injuries were in children less than 4 years old.¹⁸ A second, smaller peak occurs during adolescence.^{3,11,20}

It is important to note that the 2 incidence peaks have very different characteristics, as the mechanisms of injury incurred by younger children differ from those during adolescence. Young children are overwhelmingly injured by contact with low-voltage sources.^{3,18,20,21} The most common mechanisms in this age group are injury due to placing objects (eg, keys, safety pins, hairpins, or metal chopsticks) into an electric socket, or sucking or chewing on an electric cord while the child is at home.^{3,18-20,22,23} Other, less common mechanisms of electrical injury that have been reported in children are kite flying, bathtub-related injuries, and lightning strikes.²⁴⁻²⁶

The second peak that arises during adolescence is due to the emergence of risk-taking behaviors and work-related activities. These injuries commonly involve outdoor high-voltage sources.^{3,11,20} Commonly described mechanisms include climbing trees, electrical poles, and high-tension structures, as well as playing on railroad tracks or riding on the tops of trains.^{27,28} As adolescents move into adulthood, electrical injuries are overwhelmingly found in the work setting. High-risk occupations include linemen, electricians, construction workers, and painters — these comprise the vast majority of patients cared for in adult centers.^{1,29}

The Physics of Electricity

Understanding electrical injuries requires a basic knowledge of general physics. There are 6 variables that contribute to the outcome of any electrical injury: voltage (V), resistance (R), current (I), type of current (alternating or direct — AC or DC), duration of exposure to current (measured in seconds), and the current's pathway in the body.³⁰

Voltage is a measure of electromotor force, or the difference in pressure at 2 points between which current flows. In patients with direct current electrical injuries, one contact point is the electrical source from which the current flows, and the other contact point is the ground source to which the current flows. With the much more common alternating-current injury, voltage reverses with respect to ground 50% of the time, making use of the terms "entrance" and "exit" points misnomers. Hence the deep burns characteristic of electrical contact are called just that — "contact points." Voltage is the only value that is routinely known, since the voltage of a specific source can often be learned by taking a history. As a result, electrical injuries are arbitrarily categorized into 3 separate classifications, based on the voltage of the contacted source; low voltage (less than 1000 volts), high voltage (1000 volts or more), and lightning.

Resistance is a measure of the impedance that a current encounters moving through a specific substance, and the unit of resistance is the ohm.³⁰ Resistance is dependent on the specific chemical and structural properties of an

object and the environment in which that object exists. Resistance also depends upon the area in contact with the source, magnitude of current, duration of current flow, and presence of moisture.³¹ Within the human body, the various tissues possess their own individual resistances. Bone has the highest tissue resistance, while nervous tissue, blood vessels, and muscle tissue have the lowest.³¹

The main resistor to electricity in the human body is the skin. Dry human skin over the palms or soles has a resistance of approximately 100,000 ohms.³¹ However, if the skin surface is wet, its resistance can drop to as low as 2500 ohms. This allows a much larger quantity of current to pass into the body. Hunt found that, once the resistance of skin is overcome, electrical current travels through the body's tissues not as multiple individual resistors, but as a single unit.³² Furthermore, the extent of tissue damage is defined by the total cross-sectional surface area through which the current travels. Smaller cross sections have a smaller area over which to distribute the current, thus incurring more tissue damage. This explains why extremities are significantly more likely than the thorax to sustain more severe injury.³²

Current is a measure of the flow of negatively charged electrons. The unit of current is the ampere (amp), which translates into electrons per second. There are 2 types of current: alternating and direct. Direct current (DC) flows in a single direction, while alternating current (AC) alternates at a specific frequency between points of contact. In the United States, the frequency of household electricity is 60 hertz (Hz), meaning that there are 60 cycles or alterations of current direction per second. Clinically, nearly all high- and low-voltage burns are due to alternating current. It is believed that alternating current is 3 times more dangerous than direct current of the same voltage.³³ The reason for this is that AC causes repeated muscle contraction, or tetany, once a certain threshold is breached. Since the flexor muscles of the hand are stronger than the extensor muscles, once a patient has grasped the current source, they will be unable to let go, and a "locked on" phenomenon occurs. Due to this inability to release the source, the duration of contact is increased, magnifying the amount of electrical energy transferred to the body, thereby producing greater tissue injury. Direct current, on the other hand, causes a single, whole-body contraction, resulting in a "blast effect" that typically propels the patient away from the electrical source. However, this difference is thought to be meaningful only at low voltages, because at high voltages both alternating and direct currents have similar effects.¹² Lightning causes injuries in a different manner from generated electricity, with lightning properties being more similar to DC.

The 3 variables of voltage, current, and resistance are interrelated by Ohm's law, where current is equal to voltage divided by resistance ($I = V/R$). Thus, one can estimate the magnitude of the current if voltage and resistance are known. This is important to recognize, since thermal energy is generated by the transformation of electrical energy into heat, as expressed by Joule's law — $P =$

I^2RT ($P =$ power; $T =$ time, or the duration of contact in seconds). This equation illustrates that, while resistance and length of exposure are important variables, the magnitude of current is exponentially more significant. Thus, according to Joule's law, current is the most important factor in determining the severity of tissue injury.

The path that the current follows within the body has a direct relationship to patient morbidity. In a small, prospective pilot study of adult patients presenting with high-voltage electrical injuries, risk factors for the presence of myocardial injury were assessed.³⁴ Patients were divided into 2 groups — those with evidence of myocardial necrosis and those without. (For this investigation, evidence of myocardial necrosis was defined as elevation of serum creatinine kinase (CK) to more than twice normal levels, and a creatinine kinase MB (CK-MB) fraction greater than 3%.) Current pathways were categorized as either vertical (ie, 1 contact point above the pubic symphysis and 1 contact point below the pubic symphysis) or horizontal (ie, both contact points on the same side of the pubic symphysis). A vertical pathway had a statistically significant difference from a horizontal pathway in predicting the presence of myocardial necrosis and a larger body surface area burn. Chandra et al hypothesized that, since the contact points were further apart, the current had a longer transport time, which resulted in greater tissue injury. Furthermore, since blood vessels and myocardium have lower resistance than other tissues, there would be a greater predisposition for myocardial injury with the vertical pathway.³⁴ These results are somewhat controversial and difficult to interpret, as the investigators defined myocardial damage by an elevation of CK and CK-MB. It is unclear whether this is an appropriate distinction, as other studies have shown that CK-MB is a nonspecific indicator of myocardial damage.³⁵⁻³⁷ The authors acknowledged this controversy and maintained their conclusion, based on the timing and rise of the CK observed in their patients.³⁴ In addition, a retrospective chart review of 66 patient deaths found that mortality due to lightning injuries was more common in patients with leg burns and head burns than in those with injuries localizing to the upper extremity and trunk.³⁸ Despite differences in the characteristics of currents, it appears that the vertical pathway through the body may have some clinical significance.

Lastly, electricity causes death and morbidity via multiple mechanisms. Thermal energy is created from electrical energy, causing both superficial and deep-tissue injury. However, burns can also be produced by arcing of electricity. An "arc" or "flash" occurs when 2 points (either source and patient, or source and nearby object) exchange a current, and the patient is in close proximity to this energy exchange. The resistance of the air between the 2 points can generate temperatures up to 3000 degrees Celsius, causing severe burns in addition to the direct electrical injury.¹² This exchange is extremely intense and may ignite clothing or the surroundings, causing a mixture of both thermal and electrical burns to the same patient. Electricity is also known to have a direct effect on cell membranes

(electroporation), which causes disruption of normal cell membrane physiology.³⁹ Finally, falls and/or severe muscle contractions may cause associated multisystem trauma.

Lightning

Lightning injuries have an approximate 30% mortality, with up to 70% of survivors sustaining significant morbidity.⁴⁰ Lightning-related injuries in children are rare. Most occur while playing sports on an open field — ie, during soccer, football, and baseball.^{41,42} A naturally occurring phenomenon, lightning is generated when static electricity from a positively charged cloud is transferred to a negatively charged cloud or to ground. This transfer of current is extremely large; up to 12,000-200,000 amps, with voltages in the millions.⁴³ Lightning is clearly dangerous, and a single lightning strike has been reported to cause multiple casualties.^{26,41,43}

Distinctly different from generated electricity, lightning causes injury via multiple mechanisms: direct strike, side flash, ground strike, and blunt injury.⁴⁴ A direct strike is the most dangerous mechanism and occurs when lightning directly strikes a person, while a side flash occurs when a nearby object is struck by lightning, and the patient is injured by force from the energy exchange. Ground strikes occur when lightning hits the ground with people nearby. Current travels through the ground and, when it reaches a person, goes up one leg and back down the other, due to the voltage differential between the extremities. Lastly, blunt injury can occur by a single muscular contraction generated by DC current. This will cause the patient to be thrown away from the site of contact.

Lightning injuries are often confused with high-voltage electrical injuries. However, lightning must be distinguished as a separate injury mechanism. Despite the high voltage and large current delivery of lightning, the duration of contact is extremely short. As a result, lightning typically does not cause the deep-tissue injury seen in high-voltage contacts. Furthermore, a “flashover”

effect often occurs with lightning. In a “flashover,” electric current runs along the skin surface into the ground, rather than through the patient’s body. This usually causes no cardiorespiratory effects and little to no cutaneous injury.⁴⁵ The characteristic cutaneous finding is a linear or branching (arborescent) fern-like pattern of very superficial injury (Lichtenberg figure).

Differential Diagnosis

The challenge in the diagnosis and management of electrical injuries is related to the multiorgan system effects that electrical current has on the body. Further difficulty arises when the incident is unobserved and exact details are unclear. The following sections explain specific clinical manifestations of electrical injuries in various organ systems, and they are categorized by voltage source. (Table 1)

High Voltage Cardiovascular

The effects of electricity on the myocardium are most commonly seen in high-voltage contact. Electricity affects the cardiovascular system by a few basic mechanisms; direct effects on myocardial cell membranes by transformation of electrical energy into thermal energy, myocardial ischemia secondary to coronary artery vasospasm as a direct result of the electrical current, or indirect effects from a large release of systemic catecholamines.^{31,46} The most common clinical manifestation of myocardial injury is an arrhythmia. Due to decreased resistance of the myocardium and conducting system, arrhythmias are common and are thought to result from a passage of current through the thorax.^{34,38} Clinically, myocardial injury often manifests more as a cardiac contusion than as an ischemic infarction.

Life-threatening arrhythmia is the most common cause of death in high-voltage contact.^{14,16} In adult studies an abnormal cardiac rhythm is present in 5-33% of patients with high-voltage injuries, and as high as 41% in all categories of voltage contact.^{1,5,8,29,33,37,47,49} A cohort study with a large population of pediatric high-voltage electrical

Table 1. Comparison Of Clinical Findings In Low-Voltage, High-Voltage, And Lightning Injuries.

	Low-Voltage	High-Voltage	Lightning
Young children	++++	+	+
Adolescents	++	++++	+
Cardiac arrest:			
Caused by ventricular fibrillation	++++	+++	+
Caused by asystole	+	+++	++++
Cutaneous burns	+	++++	++
Deep-tissue electrical burns	+	++++	+
Myoglobinuria	+	+++	+
Cataracts	+	++	++
Multisystem trauma	+	+++	++
Need for surgical amputation	+	+++	+
Morbidity	+	++++	+++

+ Rare, ++ Uncommon, +++ Common, ++++ Frequently

burns found that 7% of patients suffered a cardiopulmonary arrest.³ However, this incidence is quite high, and results from this series may be biased, as it was based on a high-risk referral population. Another study of pediatric and adolescent electrical injuries found only an 8% incidence of EKG abnormalities on admission, and no cardiac arrests.¹¹

Common arrhythmias encountered by the emergency physician are; sinus tachycardia, nonspecific ST changes, QT interval prolongation, first- and second-degree heart block, and premature atrial or ventricular contractions.^{33,50,51} In patients who have experienced high-voltage contact and present without a palpable pulse, asystole and ventricular fibrillation are the most common rhythms encountered.^{48,50,52} Arrhythmias are thought to be an instantaneous complication of electrical contact, and if they are not found en route to the hospital or upon evaluation in the ED, it is unlikely that they will subsequently develop.^{11,53-61}

Myocardial infarction (MI) is a rare but thoroughly documented complication of electrical injuries.^{35,36} In most adult series and case reports, a small number of patients are routinely diagnosed with an MI.^{1,62,63} We are unaware of any reports of myocardial infarction in a young child due to high-voltage contact. While this is a theoretical possibility, high-voltage contact is unusual in this population.

Soft-tissue Burns

Electrothermal burns resulting from the transformation of electrical into thermal energy constitute one of the most difficult and challenging aspects of electrical injury care. High-voltage alternating current causes significant muscle damage as it traverses human tissue, due to high current intensity and prolonged muscle tetany. This deep damage fits the ICD-9 codes for "deep burn necrosis." In adult series, injuries most commonly involve the upper extremity, due to work-related contact.^{1,5,8} This predominance of upper extremity involvement was also seen in 2 series consisting of significant numbers of pediatric and adolescent high-voltage burns.^{11,64}

Most patient series make a distinction between flash burns and those deep-tissue electrical burns through which a current has physically passed. The clinical findings of a flash burn do not differ from other thermal burns, as the depth of the injury may range from superficial to full thickness. However, serious, deep electrical burns often have multiple types of burns that contribute to their clinical presentation. Often an electrical arc is produced, generating extreme temperatures prior to contact with the patient's skin. This burns the epidermis and dermis, inciting tissue breakdown. As the current contacts the patient's skin, the greatest resistor to current flow is now easily overcome, resulting in a large flow of current into the contacting body part. Additional burn injury is often inflicted by the ignition of clothing or flammable substances, thereby increasing the extent of thermal burns. If skin resistance is already low (wet skin), then current may bypass the skin almost entirely, producing little to no superficial injury. In these cases the damage to the deep tissues may be significant, despite minimal evidence of a

contact at the skin's surface.

Patients admitted with electrical burns usually have less total body surface area (TBSA) burned than do patients admitted with flash burns.^{1,5} Despite this, overall morbidity and the need for operative intervention is much greater.^{1,5} Furthermore, Xiao and Cai showed that electrical burns with a lower TBSA had a higher incidence of surgical amputation.⁵ These deep electrical burns are often further complicated by the coexistence of fractures, compartment syndrome, and neurovascular injury.

Rhabdomyolysis and Acute Renal Failure

Electrical injury with significant amounts of tissue damage causes an almost instantaneous loss of body fluids from third spacing, predisposing the patient to hypovolemia. Due to injured myocytes, myoglobin is released and enters into the systemic circulation, rising to a peak level at around 18-24 hours.⁶⁵ This rise may continue past 24 hours, if tissue injury is ongoing and exacerbated by a compartment syndrome and/or continued tissue ischemia. Destruction of just 2 cc (2 g) of skeletal muscle tissue can produce a level of creatinine phosphokinase (CK) that is 10 times normal.⁶⁶ In the presence of hypovolemia, free myoglobin is toxic and can precipitate in the renal tubules, resulting in acute tubular necrosis and acute renal failure. However, in a well-hydrated patient, significant myoglobinuria may go almost clinically undetected.

The presence of gross hemochromogenuria, or pigmented urine, was observed in 18-37% of patients in adult series of those with high-voltage contact.^{1,5,62,67} In an attempt to predict which patients would have myoglobinuria, Rosen et al completed a retrospective, multivariate analysis on 162 patients. They found that high-voltage exposure, prehospital cardiac arrest, full thickness burns, and compartment syndrome were associated with the presence of myoglobinuria.⁶⁸ Previous estimates of ATN associated with the presence of myoglobinuria were reported at 14% by DiVincenti et al.⁶⁹ However, with the current clinical standard of aggressive intravenous fluid resuscitation, the development of acute renal failure is rare.

Neurological

Neurological injury is a relatively frequent early and delayed complication of high-voltage contact, with severity ranging from a minor irritation to severe impairment. Pathologic changes are a direct effect of electrical current on nervous tissue, mechanical trauma, and ischemic insult resulting from vascular thrombosis and/or cardiac arrest.^{70,71} Grube reported that 73% of adult patients suffered acute and/or delayed neurological complications as a result of high-voltage contact.⁷² This incidence of neurological complications was higher than previously reported large series.^{1,29,33,62} However, definitions of neurological complications may vary between investigations. This suggests that previous studies are not uniform in their designs, and that in the acute setting, subtle neurological findings may easily be missed. Acute management in the ED is often directed towards other, more pressing issues.

Cherington has suggested a method of categorizing the neurologic sequelae of electrical and lightning injuries. He divided clinical findings into 4 groups; immediate and transient, immediate and prolonged, delayed and progressive, and those related to lightning.⁷³ The immediate and transient category includes: transient loss of consciousness, retrograde amnesia, confusion, seizures, paresthesias, and extremity weakness.⁷³ Grube found 29 patients (45%) with a loss of consciousness in a series of 64 patients contacting high voltage. Loss of consciousness was the most common immediately apparent neurologic symptom. Out of the 29 patients, 20 regained consciousness prior to arriving in the ED. However, 6 of the 20 had persistent or delayed central nervous system neuropathy upon discharge.⁷² A comparatively lower incidence of adolescent patients with a loss of consciousness (14%) was found by Rai.³ Most series state that a persistent coma is an ominous finding and has a poor prognosis.⁷²

Etiologies of immediate and prolonged symptoms include: cerebral infarction, intracranial hemorrhage, cerebral edema, global encephalopathy, cerebellar dysfunction, peripheral neuropathy, and spinal cord injury.⁷³ Global hypoxic ischemic encephalopathy may be evident in patients who have suffered a cardiac arrest. Coexistence of head trauma in the presence of an electrical contact may complicate the diagnostic picture. Chen found an incidence of 3.2% of adult patients with an intracranial hemorrhage associated with high-voltage electrical contact. By history, all of the patients suffered a traumatic brain injury from falling.⁷⁴ A similar incidence of traumatic brain injury (2.3%) was found by Arnoldo et al in their study.¹ Other CNS symptoms that have been reported and may be considered delayed, as they are not often apparent in the ED, are memory loss, behavior changes, and posttraumatic stress.⁷²

Spinal cord injuries have been reported in both acute and delayed settings,^{71,75-78} though the incidence of spinal cord injury is low (3-8.6%, depending on the series).^{69,75,78} Severely ill patients may require chemical sedation and paralysis, which mask spinal cord symptoms in the acute setting. Furthermore, early clinical evidence of injury to the spinal cord by both physical exam and diagnostic imaging may not be present. In a small series of 5 patients with clinical evidence of spinal cord involvement, Varghese found that all 5 patients had no documented neurological symptoms on admission. All 5 showed delayed symptoms of spinal cord injury 1-4 weeks after electrical contact had occurred.⁷⁵ It appears that a common finding in spinal cord electrical trauma is a motor deficit — be it quadriplegia, or paraplegia with preservation of sensation.⁷¹

Grube reported that 34% of patients experienced peripheral neuropathy with high-voltage contact. Of these, 64% improved, and 36% progressed. The median, ulnar, and radial nerves were the most commonly involved — a distribution similar to previous series.^{33,69,72} Lastly, delayed neurological syndromes are common. A delayed peripheral neuropathy was found in 17% of patients with high-

voltage contact, with presentation following injury as long as 2 years, mandating long-term follow-up.⁷² Long-term clinical findings include abnormal nerve conduction, motor weakness, paresthesias, hyperesthesia, and decreased deep tendon reflexes.^{79,80}

Orthopedic

Fractures and dislocations are commonly associated with high-voltage electrical contact. Extreme muscle contraction and blunt trauma, most commonly from falls, contribute to the presence of orthopedic injury. There are associated fractures in 8-11% of adults, with fractures to the skull, humerus, radius, ulna, clavicle, femur, scapula, and vertebral bodies being described.^{1,4,69} Posterior dislocation of the shoulder is an unusual injury that has reportedly occurred with electrical contact. Posterior shoulder dislocations make up only 2-3% of all shoulder dislocations, and electrical contact is one of the main mechanisms.⁶⁵

Compartment syndrome is an ominous complication of high-voltage injury seen in 54% of adult patients in 1 series.⁸¹ Due to tissue swelling and inflammation, compartment syndrome may complicate the clinical picture and requires immediate surgical intervention.

Ischemic injury to the extremity is further complicated by vascular thrombosis. Due to effects of electricity on the media of the arteries, endothelial injury and vascular thrombosis are known problems.^{82,83} Damage is most commonly to the medium- and small-sized arteries, due to their inability to dissipate heat. Progressive ischemia due to macro- and microthrombi may lead to tissue necrosis and the need for limb amputation.⁸¹ Arterial thrombosis may be immediate or delayed in its onset, and the severity may not be related to the appearance of the injury to the overlying skin.⁸³

Eyes

Eye injury is not uncommon in patients with high-voltage contact. Acute physical findings associated with high-voltage contact are mydriasis and anisocoria, which are usually transient and thought to be to the result of transient autonomic dysfunction.⁸⁴ The most common late eye injury is cataracts. These have been reported to occur in up to 6% of victims of electrical contact.⁸⁵ While cataracts can manifest in the acute period, they are more commonly delayed. Patients usually present with a complaint of decreasing vision on the affected side that appears 1 month to 2 years after the electrical injury.^{85,86} In high-voltage contact, cataracts are often unilateral and found on the side of source contact.⁸⁶ Other, less common — but nonetheless reported — eye pathologies include iritis, uveitis, and macular changes.⁸⁴

Abdominal and Thoracic Injury

Although rare, electrical injuries to the internal organs of the thorax and abdomen have been reported. Lung parenchyma and abdominal viscera are not resistant to the effects of high-voltage electricity. However, these injuries generally occur only when the thorax or abdomen is a contact point. A direct electrical burn to the left lower lobe was reported by Masanes et al.⁸⁷ Kumar analyzed 8 report-

ed abdominal injuries and found that the small bowel and gall bladder were the most commonly involved viscera.⁸⁸

Low Voltage Cardiovascular

In the few pediatric cohort studies, the incidence of cardiac arrhythmias with low-voltage contact is small — 0-4.8%.^{56-58,60} In most adult series, the incidence is only slightly higher — 1.7-8.9% of patients with low-voltage contact.^{5,49} However, one series of adult patients by Hussmann et al reported a very high incidence (40%) of cardiac arrhythmias in patients with low-voltage contact.⁸ It was unclear why the incidence was so high in their series, since they did not comment on this specific finding. However, we suspect that, similar to previously stated variations in neurological diagnosis, the determination of an abnormality on EKG also varies between studies.

Although it appears that cardiopulmonary arrest is more common in high-voltage contact, low-voltage contact has also been reported to cause life-threatening arrhythmias. Ventricular fibrillation is the most common arrhythmia found in patients with low-voltage contact presenting without a palpable pulse.⁶⁹ Myocardial infarctions have also been reported with low-voltage electrical contact in healthy teenagers.^{90,91} Both cases showed patent coronary arteries on angiography. One patient had Q waves on EKG, associated with normal echocardiography, while the other had evidence of ventricular hypokinesis on echocardiography.^{90,91} These findings suggest that coronary artery vasospasm was the cause of ischemia, rather than thrombosis due to preexisting disease. We are unaware of any reports of a young child with evidence of a myocardial infarction due to low-voltage exposure.

Soft-tissue Burns

Low-voltage contact occurs in all age groups, with in-home exposures being most common. The most common low-voltage contact injury involves the hands and mouth of young children. These burns can range from superficial to full thickness and are usually found at points of contact. In most pediatric series, low-voltage electrical burns involve less than 3% TBSA.^{21,56,57}

Mouth burns are specific to younger children and can result in significant morbidity. These are most common in children less than 2 years old, but may be seen in children up to 6 years of age.^{28,92-94} Burns are usually a direct result of sucking on the female end of an electric cord or a cord connector, or biting through the insulation of a cord, where the patient's saliva acts as a conductor and completes the circuit.⁹⁵ Oral burns were the most common electrical burns found in children exposed to low-voltage sources in some studies.^{19,28} However, other studies indicate that burns to the hands are more common.^{6,18,21,56,57} These burns are created by a combination of arc and current contact burn. In one report, a 5% incidence of cardiac arrest has been associated with oral burns to the mouth.⁹⁶ However, this was not found in other series of low-voltage contact burns.^{56,57} Labial artery bleeding is a potential complication of an oral electric burn. The hemorrhage

can occur anytime from the acute burn period up to 21 days after the primary event, but usually occurs when the eschar from the burn falls off at 7-14 days.⁹³

Neurological

Neurological complications are rare in low-voltage electrical contact. Bailey et al found 1.3% of patients in a series of children with low-voltage contact had a history of loss of consciousness.⁵⁶ In adults, Arnoldo et al found a higher incidence — 26% of patients with low-voltage contact.¹ The reason for this difference in incidence is unclear. One can only hypothesize that, since the injury mechanisms are different between adults and children, loss of consciousness may be more common in work-related activities, as they may involve a higher incidence of blunt trauma. Peripheral neuropathy and reflex sympathetic dystrophy have been reported with low-voltage contact.^{97,98} However, spinal cord injuries, already rare even with high-voltage contact, do not appear to occur with low-voltage contact.

Other Complications

With low-voltage contact, myoglobinuria and associated acute renal failure are extremely rare. Fractures and osteonecrosis can occur with prolonged muscle contraction from low-voltage AC.⁹⁹⁻¹⁰¹ Vascular thrombosis has been reported as a delayed complication of low-voltage contact.⁸³ Finally, cataracts are rare and do not normally occur in children who are exposed to household voltages. However, the development of cataracts has been reported in adult patients with low-voltage contact.⁸⁵

Lightning Injuries

Cardiovascular

Immediate cardiac arrest is the primary cause of death following lightning strike.^{38,45} The effects of lightning on the heart have been described as unsynchronized “cosmic defibrillation,” due to the large direct current applied to the body.³¹ This produces depolarization of the entire myocardium, resulting in ventricular standstill and/or asystole.³⁸ In theory, due to the automaticity of the heart, a normal sinus rhythm will spontaneously resume along with a return of circulation. However, a simultaneous respiratory arrest is often present, due to lightning's effect on the respiratory center within the medulla. This manifests as apnea and may be confounded by transient diaphragm and chest wall paralysis.⁴⁵ Furthermore, it is not unusual for the respiratory arrest to persist for a prolonged period of time. If appropriate CPR is not initiated, then a secondary arrhythmia, such as asystole or ventricular fibrillation, will develop, due to tissue hypoxia.^{31,38}

From the largest series of patients injured by lightning, Cooper analyzed 66 patients — 58 by literature review and 8 treated personally by the author. This article is one of the main sources quoted from the literature on lightning. However, the author herself acknowledges that her data were less than complete, and that the main goal had been to search for prognostic signs of death.³⁸ Cooper found that 30% of patients injured by lightning have had a cardiopulmonary arrest.³⁸ Lichtenberg et al found that a direct lightning strike carries a high risk for myocardial

damage. In his study, out of 4 direct strikes, 1 patient died from a cardiac arrest, and 2 patients had evidence of myocardial injury by elevated serum cardiac enzyme levels and abnormal findings on echocardiography in the first 4 hours of hospitalization.¹⁰² In those patients who do not suffer an immediate cardiac arrest, QT interval prolongation and ST changes may be apparent on EKG in the ED.

These changes are often transient and usually result in no significant sequelae.¹⁰²

Similar to high-voltage contact, myocardial infarction is a rare but documented complication of lightning strikes. In a study of 47 children involved in a single lightning strike, a 14-year-old female suffered an immediate cardiac arrest. Upon presentation to the ED, she had EKG evi-

Ten Pitfalls To Avoid

1. "He was not complaining of any pain to his scalp, so I did not examine it."

The scalp must be thoroughly searched for contact points, as if you were looking for head lice. These lesions are often painless and can easily be missed. A meticulous physical exam is essential. Bear in mind that other common areas of contact are the hands and feet.

2. "The extent of the burns on the skin determines the extent of the injury."

High-voltage electrical burns can involve significant deep-tissue injury, despite the benign appearance of the skin. It is a mistake to estimate severity of electrical burns based on the size of cutaneous burns.

3. "Household voltage contact does not kill."

Death can occur due to a wide range of voltages. In low-voltage contact, most commonly ventricular fibrillation results in a cardiac arrest. There are many variables — not just voltage — that determine the outcome of the incident.

4. "This is an electrical injury, so we can remove the cervical collar without imaging."

Very often, contact with high-voltage electricity will occur above ground, and an associated fall can occur. The emergency practitioner must consider that the patient has experienced multisystem trauma and treat the patient accordingly. Furthermore, it is important to consider intracranial injury in patients with altered mental status that is not improving.

5. "The skin burns on his arm cover less than 5% TBSA. I don't think that he should require much fluid resuscitation."

Similar to estimating the severity of the injury, fluid resuscitation cannot be based on total body surface area burned. An electrical burn can require a greater volume of fluid resuscitation than the same-sized thermal burn. Underestimation of fluid resuscitation can have devastating results, such as acute renal failure and the need for amputation.

6. "He was in pain, so I decided to perform a brachial plexus nerve block."

Regional and spinal anesthesia should be avoided in patients with electrical injury. Since neurological impairment is a complication of both local anesthesia and electrical

contact, the exact cause of the injury may be questioned later. It's best to provide analgesia in the form of narcotic medications, and steer clear of any controversy, while still relieving your patient's pain.

7. "All children who experience an electric shock require 24-hour cardiac monitoring."

Children exposed to household voltage who are asymptomatic, have small cutaneous burns, have a normal cardiac rhythm, and have no history of a loss of consciousness or prehospital cardiac arrest may be safely discharged from the ED, as long as they have no other injuries to address. Their risk for a delayed arrhythmia is extremely small.

8. "All children with oral electrical burns to the mouth require admission to monitor for bleeding."

While labial artery hemorrhage can occur acutely, it most commonly occurs 7-14 days after the incident, when the eschar falls off. Bleeding is often easily controlled with direct pressure. In rare cases, it may require a single figure-8 suture to attain hemostasis. Children with oral burns do not need to be hospitalized; however, proper instructions should be discussed with the parents prior to discharge from the ED.

9. "The injuries did not appear severe, so I decided not to transfer the patient to a burn center."

Electrical burns are considered major burns that require specialized care. A burn center has the capability to provide multidisciplinary care and long-term follow-up for these patients. Early transfer to a referral burn center is critical to decrease morbidity.

10. "The patient's neurological exam was fine when I discharged her from the ED. I did not think anything would happen later on."

Despite apparently normal clinical findings in the ED, it is important to provide the family with proper discharge instructions and follow-up, due to the possibility of delayed complications. Delayed neurological sequelae, such as cataracts, paresthesias, pain, and reflex sympathetic dystrophy, can develop from a matter of days up to 2 years after the initial contact. Children will often develop psychological manifestations in response to the experienced trauma. ▲

dence of an inferior wall myocardial infarction. She died 7 days after the strike. On autopsy there was evidence of an inferior wall myocardial infarction.²⁶ Lastly, it appears that patients experiencing a ground strike are at a low risk for cardiac arrest, as the current probably does not pass through the thorax with this mechanism of exposure.^{26,38,102}

Soft-tissue Burns

Despite the extremely high voltage found with lightning, significant deep-tissue injury does not routinely occur, since the current is direct, and the duration of contact is for just milliseconds. However, if the patient is in contact with a metal object, a contact-type, deep-tissue burn can occur, similar in character to a burn created by high-voltage contact. Clothes can also ignite and cause associated thermal burns.²⁶ Cooper found that 89% of patients suffered at least 1 burn, and 63% had multiple cutaneous burns in her series of patients injured by lightning.³⁸ In a study of 17 adult and pediatric victims of a single lightning strike, 11 patients had external burns.⁴³ The extent of their burns ranged from less than 1% to 18% TBSA. Investigators found that 10 patients had the characteristic “tiptoe” burns to their toes and soles of their feet.⁴³

A pattern called “feathering” is a pathognomonic feature of a lightning strike. This is thought to be the result of a flashover current traveling on the surface of the skin, rather than passing through the patient’s body. The nonblanching, reddish-brown erythematous pattern of feathering is not a burn, but is thought to be an inflammatory response to the electrical current passing through the skin.⁴² This pattern was seen in only 1 patient in the study by Fahmy et al.⁴³

Neurological

Neurological complications of lightning injuries are similar to those seen with high-voltage electrical contact. The most devastating complications are hypoxic-ischemic encephalopathy, cerebral or cerebellar infarction, intracranial hemorrhage, spinal cord injury, and myelopathy.^{44,103} Confusion and amnesia are almost universal.³⁸ As with high-voltage electrical injuries, a prolonged state of coma indicates a poor prognosis and may suggest the presence of hypoxic-ischemic injury to the brain.¹⁰⁴

There is a transient paralysis — called keraunoparalysis — specific for lightning injury. Immediately after a direct or ground strike, the lightning renders the patient paralyzed, with extreme vasoconstriction to 1 or more of the extremities. This phenomenon most commonly affects the lower extremities and will usually resolve within minutes to 1 hour.¹⁰⁵ The finding of paraplegia in this setting may help distinguish clinically between keraunoparalysis and cerebral injury or infarct that more commonly present as hemiplegia. Typically, keraunoparalysis resolves by the time emergency care providers assess the patient and will not be clinically observed. In children who have experienced a lightning strike, neuropsychiatric manifestations have been reported. These symptoms include fear, nightmares, and mild headaches beginning days to months after the experience.²⁶

Other Complications

Other common clinical findings in patients injured by lightning are cataracts and perforation of the tympanic membrane. In a series of 18 adult patients, 12 had evidence of rupture of the tympanic membrane. In the ED these patients often complain of tinnitus and hearing impairment.¹⁰⁶ Cataracts are more common than in high-voltage electrical contact and are usually bilateral.⁸⁶ Myoglobinuria can occur, but not to the degree found in high-voltage injuries.

Prehospital Care

At the injury scene, the emergency care providers should first assess the area and ensure their own safety. The injured patient may still be in contact with the power source, which should be turned off prior to any rescue attempts. Victims in water must be approached with extreme caution. Early and aggressive cardiopulmonary resuscitation (CPR) and use of an automatic external defibrillator (AED) is recommended, as case reports suggest the possibility of survival, despite an out-of-hospital cardiac arrest.^{40,107-109} This rule holds true especially for lightning strikes, as cardiac function may return quickly.

Prehospital care providers and EMS should be prepared to care for multiple patients at the scene of a high-voltage or lightning-strike incident.^{26,43,73} Due to the possibility of a good prognosis with early CPR, standard traumatic triage protocols may not apply.¹¹⁰ Victims without signs of life should have first and immediate care. The triage priorities in cases of electrical and lightning exposure are different from standard traumatic scenarios.^{40,45} At the scene, patients with clear signs of life typically do well, and can wait a short period before receiving aid.³⁸ Furthermore, clinical findings, such as fixed and dilated pupils, absent pulses, and apnea, are unreliable in this setting and should not be used clinically to stop resuscitation in the field. (See first section under ED Evaluation — Acute Stabilization and Cardiopulmonary Resuscitation.)

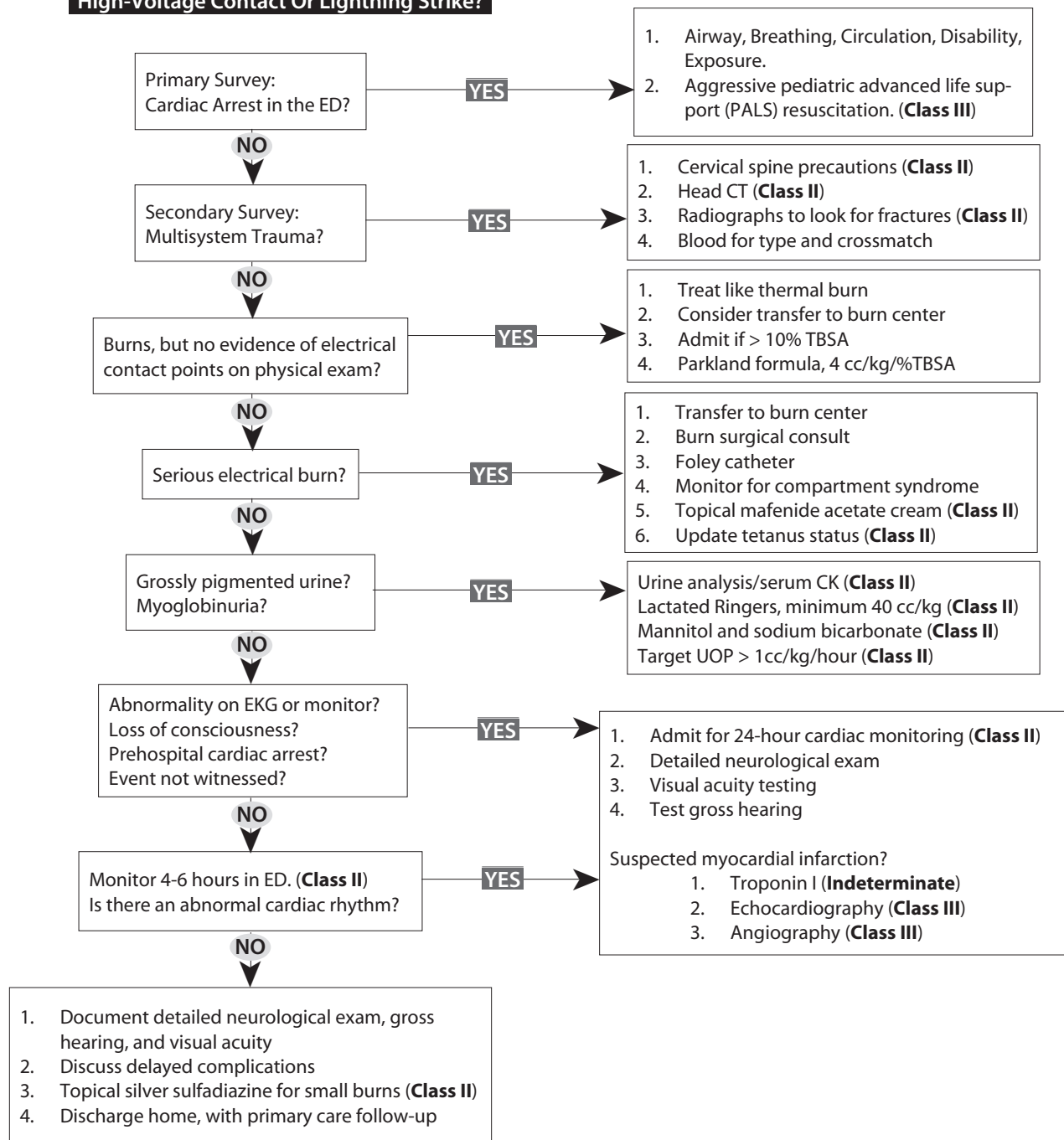
Falls and subsequent blunt trauma are routinely associated with high-voltage contact and lightning strikes. These victims should be assessed and treated, assuming the presence of multisystem trauma. Proper cervical spine immobilization should be implemented. All immediate, life-threatening traumatic injuries are sought and managed during transport to the ED.

Finally, when emergency care providers are called to assess a person injured by electricity, they should take all complaints seriously. One study in the United Kingdom found that 25% of patients who were asymptomatic at the time of EMS call were admitted to the hospital for burn care, an associated traumatic injury, or cardiac monitoring.¹¹¹ Furthermore, as some clinical symptoms may be delayed (neuropathy, cataracts, pain, etc), even clinically asymptomatic patients should be strongly encouraged to seek appropriate medical care. All electrical burns are classified as major burns optimally cared for in qualified burn centers.

Continued on page 12

Clinical Pathway: ED Management Of High-Voltage Electrical Contacts And Lightning Strikes

High-Voltage Contact Or Lightning Strike?



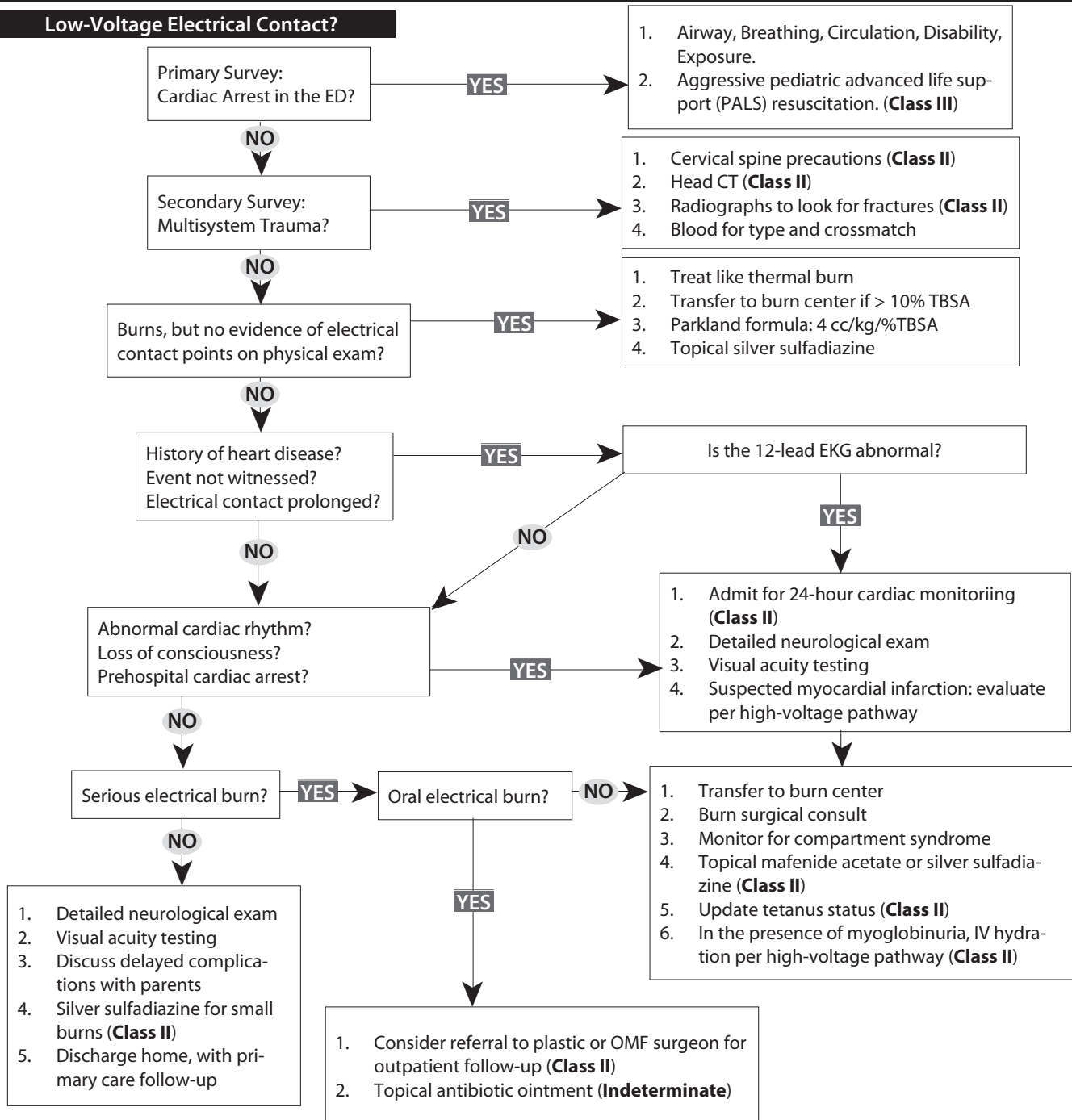
The **evidence for recommendations** is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II:** Acceptable and useful. Good evidence provides support. **Class III:** May be acceptable, possibly useful. Fair-to-good evidence provides support. **Indeterminate:** Continuing area of research.

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

Copyright ©2005 EB Practice, LLC. 1-800-249-5770. No part of this publication may be reproduced in any format without written consent of EB Practice, LLC.

Clinical Pathway: ED Management Of Low-Voltage Electrical Contacts

Low-Voltage Electrical Contact?



The **evidence for recommendations** is graded using the following scale. For complete definitions, see back page. **Class I:** Definitely recommended. Definitive, excellent evidence provides support. **Class II:** Acceptable and useful. Good evidence provides support. **Class III:** May be acceptable, possibly useful. Fair-to-good evidence provides support. **Indeterminate:** Continuing area of research.

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

Copyright ©2005 EB Practice, LLC. 1-800-249-5770. No part of this publication may be reproduced in any format without written consent of EB Practice, LLC.

ED Evaluation

Acute Stabilization and Cardiopulmonary Resuscitation

A good generalized approach to the child critically injured by electricity and/or lightning is for the resuscitation team to care for them as if dealing with a patient with blunt trauma and a potential crush injury. Electrical injury patients can be complex, often requiring a combination of advanced cardiac life support and trauma care. Patients with high-voltage contact or lightning strike should initially be assumed to have multisystem trauma. Follow cervical spine precautions until the patient has been properly assessed and evidence of injury excluded. Establishing an airway can be difficult, since tissue swelling may develop rapidly. Elective intubation should be achieved prior to signs of upper airway obstruction in patients with severe injuries to the face, mouth, and anterior neck.⁴⁰ Acute stabilization should follow standard trauma assessment protocols consisting of primary, secondary, and tertiary physical surveys. The focus should be on the so-called ABCDEs of trauma care (airway, breathing, circulation, disability, and exposure).

In patients without a perfusing cardiac rhythm, the institution of early and aggressive CPR and the use of an AED following pediatric advanced life support (PALS) protocols should be performed. Patients may appear dead. Due to intense vasospasm a pulse may not be palpated. Apnea and dilated and nonreactive pupils shortly after an electrical shock or lightning strike are transient phenomena and must not be mistakenly used as criteria to end resuscitation early.^{84,112,113} Oxygen by face mask should be implemented and intravenous access attained with the institution of aggressive hydration, when clinically indicated. The Glasgow Coma Scale (GCS) score can be used as an assessment tool, but to the authors' knowledge, there is no publication that analyzes its prognostic accuracy. Furthermore, unlike the GCS score for blunt trauma, it does not appear to correlate well with eventual neurological prognosis. Lastly, all obvious fractures should be stabilized to decrease pain and the chance of neurovascular compromise.

History

Once it is clear that acute stabilization has been accomplished, a more detailed history and physical exam can be completed. A good history of the preceding events may be taken from the patient, bystanders, or prehospital personnel. It is important to remember that burn patients may not always remember exactly what happened, and emotional witnesses may provide inaccurate information.¹¹⁰ Nevertheless, it is helpful to first determine the source of the electrical shock, so that the contact voltage may be estimated. This may help to stratify risk and guide clinical evaluation, as in general a greater number of complications arise from high-voltage contact and lightning strikes. Familiarity with the voltage of common electrical sources is enormously useful. The voltage in high-tension power

lines exceeds 100,000 volts, decreasing to 7200-14,400 volts in distribution lines. Outlet voltage within residential homes is 110-220 volts (North America) and 220 volts (Europe).

Knowing exactly where the patient was injured is also key. Was the child or adolescent injured at home, at work, or at play? What were they doing at the time of the electrical contact? What was the surrounding environment like? Was water in the vicinity? Obtain a clear picture of the type of burn mechanism. Was the contact with the source prolonged or brief? Was there an associated thermal burn, due either to a flash burn or to the ignition of a chemical, the patient's clothing, and/or the surroundings (a fallen wire starting a grass fire, for instance)? The answers to these questions may help guide your physical exam in search of the contact points, in order to identify the path the current took through the body. By performing a good clinical assessment, you may also be able to determine that no current passed through the body at all.

Since other traumatic injuries often coexist with an electrical injury, it is important to inquire about the possibility of blunt trauma. Was there an associated fall or blunt force from a side strike? Was the patient ambulatory at the scene? Did the patient have any loss of consciousness or a period of unresponsiveness that would suggest a cardiac arrhythmia or central nervous system injury? Are there any associated symptoms, such as chest pain, palpitations, confusion, paresthesias, or weakness?

Past medical history using the AMPLE mnemonic (allergies, medications, procedures, last meal, and events leading to the incident) should be asked. This information should include any previous neurological deficits and preexisting cardiac conditions.

Physical Examination

A thorough physical exam with meticulous medical documentation should be performed in all patients with electrical injuries. Points of contact should be identified. In low-voltage injuries, the contact points are commonly found on the hands and mouth. Soles of the feet must always be examined, as they are usually the ground contact. The scalp should be meticulously checked for hidden lesions, as though you were looking for lice. These lesions are usually painless, which means that even significant injury is often overlooked. Typically in low-voltage contact, arcing is not a significant factor, and thus firmness and duration of contact will often dictate the shape and severity of the burn.¹¹⁰ If the contact was not very firm and was of short duration, then small blisters or grayish-yellow marks might appear on the skin. Contact burns due to low voltage often appear as small black marks, similar to a small cigarette burn on a tablecloth. The burns may be partial thickness to full thickness, depending on the duration of contact and amount of heat generated by the current. If a high-voltage source is involved, then often there is a component of arc and flash burn, combined. The presence of charring becomes more common.¹¹⁰ If firm contact has been made, then the current may generate enough heat to split the epidermis-dermis junction and form a blister, which may be surrounded

by an area of blanched skin, representing local arteriole spasm from the electrical current. Some erythema may also be present around the wound edges. Lastly, if firm contact with a high-voltage source or prolonged contact with a low-voltage source occurs, charring, blistering, and deep muscle damage may be present, with an occasional blowout-type injury having deep tissues visible. Arc burns may be present across flexed joints for the axillae, antecubital fossa, and groin. These superficial or deep burns appear as "kissing" lesions, where the electrical current tried to shortcut across the joint.

Electrical burns to the mouth often present as small oval, grey-yellow ulcers with a depressed center. Characteristically, the pain from the lesion is not severe.¹¹⁶ These burns most commonly involve the commissures of the lips, but have been known to involve the tongue, buccal sulcus, alveolus, and palate.¹¹⁶

All extremities and the spine should be examined for evidence of fractures and dislocations. Point tenderness, a visible deformity, or unexplained swelling should prompt further evaluation. Physical evidence of compartment syndrome should be sought: firmness of the tissue compartments and pain on passive extension, along with an assessment of neurovascular status to the distal extremity. Loss of or changes to the pulse are usually a very late sign of compartment syndrome. The assessment may require more objective means, such as measuring compartment pressures. Although invasive, this may clarify the need for surgical intervention, as injury to nerves and blood vessels may mimic symptoms of compartment syndrome in the acute setting. A thorough neurological exam should be performed and documented after acute stabilization has been accomplished. This exam includes muscle strength, cranial nerve function, cerebellar function, deep tendon reflexes, 2-point sensation, clonus, mental status, and visual acuity, evaluating for spastic dysfunction as well as flaccidity.

Laboratory Studies

Asymptomatic children with brief low-voltage contact, with or without superficial burns, do not require any laboratory evaluation.^{56,57,60} However, patients with myalgias, prolonged low-voltage contact, or any high-voltage contact should have at minimum a screening urine analysis to look for evidence of hemochromogens or hematuria from traumatic injury. If the urine analysis is negative for blood, then additional serum studies are not indicated. However, when a urine analysis is positive for blood — but on microscopy there are no red blood cells — one must consider myoglobinuria in the setting of the patient injured by electricity. In these cases baseline complete blood cell count, serum electrolytes, blood urea nitrogen, and creatinine should be evaluated. Creatinine kinase levels do not add much towards clinical care, other than to warn of the potential presence of deep injury. It appears that the peak CK level within the first 48 hours of hospitalization helps predict severity of muscle injury, risk of amputation, and length of hospital stay. However, this relationship does not appear to be linear.⁶³

In patients with chest pain, palpitations, and abnormal EKG findings, it is unclear if serum studies are beneficial in the diagnosis of a myocardial injury. Troponin I may be the most specific test to screen for myocardial ischemia; however, there are no published studies that evaluate its utility specifically with patients injured by electricity. However, evidence does suggest that serum creatinine kinase MB fraction (CK-MB) is a poor laboratory test to determine myocardial injury in patients who have had contact with electricity, and this test should not be used alone to make the diagnosis of myocardial ischemia.³⁵⁻³⁷ CK-MB can be increased through damage to skeletal muscle, such that its rise is not solely related to injury to the myocardium. One study has shown that skeletal muscle damaged by electricity may contain and release as much as 25% CK-MB fraction, as opposed to the usual 2-3%.³⁷ Lastly, sending blood for type and crossmatching should be considered, if patients are going to require emergent surgical interventions.

Diagnostic Imaging

Regardless of the associated voltage, imaging may be required on certain patients who have experienced contact with electricity. Radiographs of the cervical spine and any bony area of concern for a fracture should be performed, when appropriate. Computed tomography of the head to look for intracranial hemorrhage and cerebral edema should be performed in patients with suspected head trauma or a loss of consciousness without immediate recovery or improving mental status.⁷⁴ Similarly, any neurological deficit that is not improving should be evaluated by magnetic resonance imaging to identify an injury to the spinal cord.

Cardiac Monitoring

Prior to the 1980s, standard medical care dictated that all patients with electrical contact be admitted to the hospital for 24-hour cardiac monitoring, due to a concern for a late-onset arrhythmia. Two publications reported clinically significant, delayed arrhythmias in patients injured by electricity.^{52,67} However, retrospective studies and audits of prospective protocols have shown that routine, prolonged cardiac monitoring is unnecessary in patients with a normal EKG on presentation to the ED. Purdue and Hunt first reported a retrospective cohort of 48 patients (mean age 25.6 years) admitted for 24-hour cardiac monitoring after high-voltage contact. They found that no patients with a normal EKG in the ED subsequently developed a significant arrhythmia. Furthermore, 2 patients were diagnosed with myocardial infarctions, and both had abnormal EKG findings in the ED. The authors concluded that only patients with a history of a loss of consciousness, a documented abnormal EKG or cardiac rhythm, known cardiac arrest in the field, or significant electrical burn required admission and cardiac monitoring.⁵³ Subsequent retrospective studies verified these results in the setting of both high- and low-voltage contact in patients of all ages, indicating that patients with normal EKG in the ED are at very low risk for a delayed arrhythmia.^{11,54-61}

Furthermore, it appears that even a screening EKG may not be required on every patient seen in the ED. In a retrospective study, Bailey reported that a screening EKG should not be performed on all children who come in contact with low-voltage electrical sources. They felt that routine EKGs in these cases are unnecessary, and only those children with a loss of consciousness, prolonged muscle contraction, a transthoracic current pathway, a history of cardiac problems, or contact with an electrical source greater than 240 volts required a screening EKG and possible cardiac monitoring.⁵⁶

Most recently, an audit of a prospective protocol in adults was published.⁶¹ This study found that if the EKG is normal on admission and if the patient has no other injuries to medically or surgically attend to, then these patients can be safely discharged from the ED to follow up with their primary care providers. If the EKG has an abnormality, an observation period of 4-6 hours is required, and a repeat EKG should be performed, since many of the EKG changes may be transient and clinically insignificant. If the EKG changes resolve, then these patients may be safely discharged.⁶¹ Otherwise, they are admitted for continuous monitoring. Furthermore, a similar study was performed over a 4-year period in 224 children. This study had no mortalities, and under the prospective protocol, only 13% of the patients (29/224) required admission for continuous monitoring. The researchers found an impressive decrease in the total hours of pediatric intensive care cardiac monitoring utilization; from 1238 down to 421 hours when compared to the previous 4 years, prior to implementation of the studied guidelines.⁶⁰

Currently there is little information on lightning injuries; however, it would appear that these patients can be treated much the same as those with a high-voltage injury. One study found that if the lightning did not cause an immediate cardiac arrest at the time of the incident, then there was a very low risk of a death.³⁸

Fluid Resuscitation

Large-bore intravenous access should be immediately attained with the initiation of aggressive fluid resuscitation in the presence of clinical hypotension, deep-tissue injury, or grossly visible hemochromogens in the urine. A minimum of 40 cc/kg of Ringers lactate is a good starting point for fluid replacement. Unfortunately, with deep-tissue electrical burns, using formulas traditionally applied to the thermal burn may grossly underestimate the true volume of necessary fluid replacement. While the cutaneous burns in electrical injuries are usually smaller than standard thermal burns, fluid requirements are larger, because large amounts of fluid are rapidly lost by third spacing into the damaged deep tissue. One study found that patients with a true high-voltage electrical injury required about 1.7 times the volume of intravenous fluids in the first 24 hours than did those patients with flash burns of the same body surface area.⁴⁷ A Foley catheter should be placed early, to quickly assess the quality and color of the patient's urine. For practical purposes, if the pigmentation cannot be readily observed, there is little risk of renal in-

jury and treatment is directed toward continued adequate urine output.

A true guide to fluid management should be the patient's urine output. Intravenous fluids should be continued at a rate that maintains a urine output that is greater than 1-1.5 cc/kg/hr in children, and greater than 1.5- 2 cc/kg/hr in infants. This rate should provide clear urine on gross observation. If there is grossly pigmented urine or no urine is produced in immediate response to the initial fluid bolus, then treatment with a single dose of mannitol 0.5-1 g/kg intravenous push and sodium bicarbonate 1-2 mEq/kg intravenous push is instituted. Aggressive fluid hydration and systemic alkalization are thought to decrease the precipitation of myoglobin in the renal tubules. There is some evidence that the treatment with mannitol and sodium bicarbonate decreases the incidence of acute tubular necrosis and acute renal failure in patients with myoglobinuria.¹¹⁴ The benefit of these medications is not completely clear, as aggressive volume resuscitation

Cost-Effective Strategies: Preventing Electrical Injuries

The best strategy is always prevention of electrical injuries. Rai et al found that from 1967 to 1997, there was a decline in the number of patients admitted to their institution for injuries due to low-voltage contact.³ The authors felt that certain preventative measures had contributed to this decline. They pointed out that specific interventions, such as the addition of ground fault circuit interrupters (GFCIs) to residential homes and the use of thicker insulation on appliance wires, may have helped.³ A GFCI continuously monitors and measures a specific electrical circuit. When it senses that there is a drain of current from the circuit, the power is automatically turned off within 1/40 of a second, preventing serious injury. In 1987 the United States Consumer Product Safety Commission (CPSC) successfully incorporated the utilization of GFCIs into the National Electric Code (NEC). This made it mandatory for all newly built homes to have GFCIs installed in bathrooms, kitchens, basements, and near hot tubs or spas.²⁰ However, not all rooms are required to have GFCIs, and older homes may not have them installed at all. As for electrical cords, no federal safety standards exist, and all the current industry standards are voluntary. Perhaps modification of their penetrability has reduced the number of cord-biting injuries.²⁰ Finally, prevention is not as simple as covering up wall outlets with plastic protectors. As one study has determined, depending on the wall outlet protector chosen, a 2-year-old child could remove them from the wall on average within 9.5 seconds, and a 4-year-old child within 2.6 seconds.¹²³ Unfortunately, all attempts at injury prevention have been limited to young children. Rai found that the incidence of high-voltage injuries to adolescent patients had not changed over the 35-year period reviewed.³ Thus, for this group, electricity remains a very serious and deadly problem. ▲

alone, without diuretics and alkalinization, may prevent acute renal failure.⁴⁷ However, there is currently no class I evidence that answers this question. In the Arnoldo study, over a 20-year period, these medications and aggressive fluid hydration were used as routine care for electrical-injury patients with gross hemochromogenuria — during this time not a single patient developed acute renal failure as a result of myoglobinuria.¹ Others have found similar success in preventing acute renal failure with aggressive intravenous hydration.^{47,67} Large, though not excessive, intravenous fluid volumes should not be a problem, as most children and adolescents do not have other comorbidities to complicate management, and their kidneys should be able to tolerate the large fluid volumes until the pigment is cleared from their urine.

For brief, low-voltage contacts and flashover lightning strikes, in the absence of pigmented urine and with no strong evidence of deep-tissue injury, intravenous fluid at a maintenance rate with burn resuscitation in the standard fashion is acceptable, until further clinical evaluation is completed. In these clinical scenarios aggressive fluid hydration is not justified, as myoglobinuria is unlikely.

Wound Care

Patients with small, superficial electrical burns to the hands and feet are treated with topical silver sulfadiazine (Silvadene[®]) cream, with daily dressing changes. Small electrical contact points of the mouth are treated conservatively. The wound is gently washed and covered with a thin layer of neomycin/bacitracin ointment. Parents are instructed to compress the commissure area if bleeding occurs (usually 7-14 days post-burn) and bring the child to the ED. Repair is performed with a single 4-0 chromic figure-of-8 suture, if required.

Due to its ability to penetrate burn tissue, mafenide acetate (Sulfamylon[®]) cream is used in deep-tissue electrical burns. However, Sulfamylon[®] is a carbonic anhydrase

inhibitor and can cause a metabolic acidosis when applied to large surface areas and with prolonged use. Pain is a limiting characteristic when applied to more superficial wounds.

Regional nerve blocks and spinal anesthesia are avoided in these patients, because controversy may arise concerning the exact cause of any later neurological deficit. Systemic narcotics, rather than regional anesthesia, will help you steer clear of any debate. Intravenous morphine sulfate (0.1 mg/kg) or fentanyl (1 mcg/kg) should provide adequate analgesia.

All patients with burn injury should have their tetanus status updated. Empiric prophylactic antibiotics are not effective in the prevention of wound infection, and may even be harmful, by assisting in the selection of drug-resistant organisms.

Surgical Intervention

Surgery is seldom needed for low-voltage oral commissure burns, while mid-lip injuries require surgical consultation. Most commissure burns heal spontaneously, requiring only mouth stretching exercises to achieve optimal results. Most surgeons take a delayed approach to surgical intervention, depending on the extent and severity of the injury; however, there is some controversy.^{92,95,115-118} A delay will allow swelling to subside and viable tissue to become more clearly identified. Currently, immediate postinjury splinting helps to preserve oral symmetry, and surgery may be performed at a later date, as needed.⁹⁵ However, in the ED, a consultation and referral to a dentist for possible oral splinting, and to a plastic or oral surgeon familiar with oral electric burn care, is necessary at a minimum, if the patient is not admitted. If this cannot be accomplished, prompt transfer to a tertiary burn care center where this condition can be addressed appropriately should be initiated.

Patients with high-voltage deep-tissue injuries should

Key Points For Electrical Injuries And Lightning Strikes

1. Electrical burns are considered major burns optimally cared for at a burn center. Early surgical intervention may decrease mortality and the development of further complications.
2. Death can result from all types of voltage contact. There are many variables, not only voltage, that determine the outcome of the incident.
3. Patients who have experienced electrical contact should be treated like multisystem trauma patients with possible crush injuries, until these are ruled out.
4. Patients without signs of life should have early and aggressive CPR, as their outcome is uncertain. Good outcomes have been reported, despite out-of-hospital cardiac arrest, in patients injured by lightning and electricity.
5. Patients with electrical contact require a meticulous physical exam to locate points of contact.
6. Clinicians should not attempt to determine the severity of the electrical injury or estimate fluid resuscitation from

the appearance of the cutaneous burns.

7. In the presence of pigmented urine, aggressive intravenous hydration is essential. The addition of mannitol and sodium bicarbonate may also help decrease the incidence of acute renal failure.
8. Children exposed to household voltage who are asymptomatic, experienced a short duration of contact, have small cutaneous burns, have a normal cardiac rhythm, and have no history of a loss of consciousness or cardiac arrest may be safely discharged from the ED, as long as they have no other injuries to address.
9. Labial artery bleeding can occur 7-14 days after initial contact in children with oral electrical burns. This is regularly controlled with direct pressure, and rarely requires suturing.
10. Despite apparently normal clinical findings in the ED, it is important to provide the family with proper discharge instructions and follow-up, due to the potential for delayed complications. ▲

be transferred to a burn center with specialized team care. Early and aggressive tissue debridement can be life-saving and may help to prevent future complications, such as myoglobinuric renal failure, sepsis, and the need for amputation.^{5,8} Serial operations may be required, as the full extent of the injury may not be completely clear after the first surgical procedure. An escharotomy and/or fasciotomy may need to be performed, due to tissue swelling and compartment syndrome. The possibility of multiple procedures should be explained to the parents in the ED, as this may assist them in coping with the difficult hospital course.

Approximately 18-42% of patients with high-voltage injuries seen at adult burn centers will require at least one operation to amputate a body part.^{1,4,5,10,29,67,81} In similar studies of adolescent high-voltage contact, the incidence of amputation is 26-38%.^{3,11,64} The possibility of amputation does exist with low-voltage contact; however, the incidence is much lower than that of high-voltage contact.^{1,8,81}

Special Circumstances

One population that is rare in the pediatric ED is pregnant teenagers. However, the pediatric emergency practitioner must be prepared to care for the pregnant female who has experienced an electrical shock. Previous case reports have documented ill effects of electrical contact, citing first trimester fetal demise and stillborn fetuses.^{119,120} However, more often than not, case reports draw attention to poor outcomes, and it is difficult to make clear determinations. A prospective study of 31 women who were followed from the time of electrical contact until delivery showed that the majority (28) had been in contact with household sources. Out of the total 31, 28 women delivered normal infants, and 1 child was born with a ventricular septal defect. Only 2 women subsequently had spontaneous abortions, and both cases were not thought to be associated with the electrical contact. In a similar control group, the incidence of spontaneous abortion was comparable. The investigators concluded that simple exposure to household current does not pose a major risk to the fetus.¹²¹ Nevertheless, the emergency practitioner should always consult obstetrical services to perform fetal monitoring and perform an ultrasound to document fetal well-being, no matter how trivial the contact may appear. One situation in which emergency physicians may be called upon to actually deliver an electrical shock to a pregnant woman is when cardioversion or defibrillation is indicated. Pregnancy is not a contraindication to cardioversion or defibrillation and can be safely performed. It is thought that the delivery of electrical current to the chest with standard cardioversion or defibrillation does not result in clinically important current delivery to the pelvis or fetus.¹²² Cardioversion or defibrillation should not be withheld or delayed in the treatment of pregnant young women.¹²²

Controversies/Cutting Edge

Early initiation of PALS and use of an AED should be

promoted in all patients with injury due to electricity and no signs of life. Aggressive cardiopulmonary resuscitation is currently recommended, as case reports suggest the possibility of good outcomes, despite an out-of-hospital cardiac arrest.^{40,107-109} This rule holds true especially for lightning strikes, as normal cardiac function may return quickly. These patients may have apnea and only require respiratory support. In these cases actual blood loss or tissue damage may be limited, and a patient who suffered a cardiac arrest due to electricity may still do well with early treatment.¹¹⁰ However, this is controversial, and one must be aware that aggressive CPR does not necessarily equal prolonged CPR. The currently accepted cardiopulmonary resuscitation standard is based on isolated case reports of miraculous recoveries despite out-of-hospital cardiac arrests, and it is difficult to make a strong recommendation for prolonged heroic attempts on all patients.

Disposition

Serious electrical burns are major burns optimally cared for at a burn center. Rapid transfer for early surgical intervention may decrease mortality and the development of further complications. Multidisciplinary resources available at these centers are readily capable of caring for acute problems, as well as providing the long-term follow-up these patients require.

In the majority of young children with low-voltage contact, a cardiac evaluation is unnecessary if they are asymptomatic, have no cardiac history, the electrical contact is known to be brief, and there was no loss of consciousness or prehospital cardiac arrest. If there are no other injuries to address, then these patients can be safely discharged from the ED to follow-up with their primary care physicians. Their risk of developing a subsequent arrhythmia is very low to nonexistent. Oral electrical burns may require referral to a dentist, plastic surgeon, or oral-maxillofacial surgeon.

All patients with high-voltage contact and lightning injuries should have a screening EKG on presentation and cardiac monitoring for a minimum of 4-6 hours. If the patient has any history of a documented arrhythmia, loss of consciousness, and/or cardiac arrest, then admission and monitoring for at least 24 hours should be strongly considered. Patients with severe electrical burns, multisystem trauma, and central nervous system injuries should be admitted for medical and/or surgical care.

Finally, patients and their families should be informed about the late (up to 2 years) development of cataracts and other neurological and/or psychological complications that may stem from the event. With this knowledge, proper referrals can be made with the help of the child's primary care physician, if delayed problems do arise. ▲

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The find-

ings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study, such as the type of study and the number of patients in the study, will be included in bold type following the reference, where available. In addition, the most informative references cited in the paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.

- 1.* Arnoldo BD, Purdue GF, Kowalske K, et al. Electrical Injuries; a 20-year review. *J Burn Care Rehab* 2004;25:479-484. **(Retrospective cohort; 700 patients)**
2. Kumar P, Chirayil PT, Chittoria R. Ten years epidemiological study of paediatric burns in Manipal India. *Burns* 2000;26:261-264. **(Retrospective cohort; 309 patients)**
- 3.* Rai J, Jeschke MG, Barrow RE, et al. Electrical injuries: a 30 year review. *J Trauma* 1999;46:933-936. **(Retrospective cohort; 185 patients)**
4. Babik J, Sandor, Sopko. Electrical burn injuries. *Ann Burn Fire Disast* 1998;11:153-155. Available at: http://www.medic.com/annals/review/vol_11/num_3/text/vol11n3p153.htm. Accessed August 20, 2005. **(Retrospective cohort; 96 patients)**
- 5.* Xiao J, Cai BR. A clinical study of electrical injuries. *Burns* 1994;20:340-346. **(Retrospective cohort; 173 patients)**
6. Wallace BH, Cone JB, Vanderpool RD, et al. Retrospective evaluation of admission criteria for paediatric electrical injuries. *Burns* 1995;21:590-539. **(Retrospective cohort; 35 patients)**
7. Gang RK, Bajec J. Electrical burns in Kuwait: a review and analysis of 64 cases. *Burns* 1992;18:497-499. **(Retrospective cohort; 64 patients)**
8. Hussmann J, Kucan JO, Russell RC, et al. Electrical injuries-morbidity, outcomes, and treatment rational. *Burns* 1995;22:530-535. **(Retrospective cohort; 129 patients)**
9. Mericer C, Blond MH. Epidemiological survey of childhood burn injuries in France. *Burns* 1996;22:29-34. **(Retrospective cohort; 937 patients)**
10. Tredget EE, Shankowsky HA, Tilley WA. Electrical injuries in a Canadian burn care. *Ann N Y Acad Sci* 1999;888:75-87. **(Retrospective cohort; 74 patients)**
- 11.* Celik A, Ergun O, Ozok G. Pediatric electrical injuries: a review of 38 consecutive patients. *J Pediatr Surg* 2004;39:1233-1237. **(Retrospective cohort; 38 patients)**
12. Koumbourlis AC. Electrical injuries. *Crit Care Med* 2002;30:S424-S430. **(Review)**
- 13.* Cooper MA. Emergent care of lightning and electrical injuries. *Semin Neurol* 1995;15:268-278. **(Review)**
14. Byard RW, Hanson KA, Gilbert JD, et al. Death due to electrocution in childhood and early adolescence. *J Paediatr Child Health* 2003;39:46-48. **(Retrospective cohort; 16 deaths)**
15. Wright RK, Davis JH. The investigation of electrical deaths: a report of 220 fatalities. *J Forensic Sci* 1980;25:514-521. **(Retrospective cohort; 220 deaths)**
16. Brokenshire B, Cairns FJ, Koelmeyer TD, et al. Deaths from electricity. *N Z Med J* 1984;97:139-142. **(Retrospective cohort; 95 deaths)**
17. Bligh-Glover WZ, Miller FP, Balraj EK. Two cases of suicide electrocution. *Am J Forensic Med Pathol* 2004;25:255-258. **(Case report; 2 deaths)**
18. Nguyen BH, Mackay M, Bailey B, et al. Epidemiology of electrical and lightning related death and injuries among Canadian children and youth. *Inj Prev* 2004;10:122-124. **(Retrospective cohort; 606 patients and 21 deaths)**
19. Zubair M, Besner GE. Pediatric electrical burns: management strategies. *Burns* 1997; 23:413-420. **(Retrospective cohort; 127 patients)**
20. Rabban JT, Blair JA, Rosen CL, et al. Mechanisms of pediatric electrical injury. *Arch Pediatr Adolesc Med* 1997;151:696-700. **(Retrospective cohort; 144 patients)**
21. Baker MD, Chiaviello C. Household electrical injuries in children. *Am J Dis Child* 1989;143:59-62. **(Retrospective cohort; 144 patients)**
22. Robinson M, Sewerd P. Electrical and lightning injuries in children. *Pediatr Emerg Care* 1986;2:186-190. **(Review)**
23. Lee JW, Jang YC, Oh SJ. Paediatric electrical burn: outlet injury caused by steel chopstick misuse. *Burns* 2004;30:244-247. **(Retrospective cohort; 57 patients)**
24. Tiwari VK, Sharma D. Kite flying: a unique but dangerous mode of electrical injury in children. *Burns* 1999;25:537-539. **(Retrospective cohort; 6 patients)**
25. Budnick LD. Bathtub-related electrocutions in the United States, 1979-1982. *JAMA* 1984;252:918-920. **(Retrospective cohort; 95 deaths)**
26. Myers GJ, Colgan MT, VanDyke DH. Lightning Strike disaster among children. *JAMA* 1977;238:1045-1046. **(Retrospective cohort; 47 patients)**
27. Sternick I, Gomes RD, Serra MC, et al. "Train surfers": analysis of 23 cases of electrical burns caused by high tension overhead railway cables. *Burns* 2000;26:470-473. **(Retrospective cohort; 23 patients)**
28. Rabban JT Alder J, Rosen C, et al. Electrical injury from subway third-rail: a serious injury associated with intermediate voltage. *Burns* 1997;23:515-518. **(Retrospective cohort; 16 patients)**
29. Hammond JS, Ward CG. High voltage electrical injuries: management and outcome of 60 cases. *South Med J* 1988;81:1351-1352. **(Retrospective cohort; 60 patients)**
30. Kouwenhoven WB. The effects of electricity on the human body. *Bull Johns Hopkins Hosp* 1964;115:425-446. **(Review)**
31. Jain S, Bandi V. Electrical and lightning injuries. *Crit Care Clin* 1999;15:319-330. **(Review)**
32. Hunt JL, Mason AD, Masterson TS, et al. The pathophysiology of acute electrical injuries. *J Trauma* 1976;16:335-340. **(Animal model research)**
33. Solem I, Fischer RP, Strate RG. The natural history of electrical injury. *J Trauma* 1977;17:487-492. **(Retrospective cohort; 64 patients)**
34. Chandra NC, Siu CO, Munster AM. Clinical predictors of myocardial damage after high voltage electrical injury. *Crit Care Med* 1990;18:293-297. **(Prospective cohort; 24 patients)**
35. Housinger TA, Green L, Shahangian S, et al. A prospective study of myocardial damage in electrical injuries. *J Trauma* 1985;25:122-124. **(Prospective cohort; 16 patients)**
36. McBride JW, Labrosse KR, McCoy HG. Is serum Creatinine Kinase-MB in electrically injured patients predictive of myocardial injury? *JAMA* 1986;255:764-768. **(Retrospective cohort; 36 patients)**
37. Hammond J, Ward CG. Myocardial damage and electrical injuries; significance of early elevation of CPK-MB isoenzyme. *South Med J* 1986;79:414-416. **(Retrospective cohort; 36 patients)**
- 38.* Cooper MA. Lightning injuries: prognostic signs for death. *Ann Emerg Med* 1980;9:134-138. **(Retrospective cohort; 66 patients)**
39. Lee RC, Gaylor DC, Bhatt D, et al. Role of cell membrane rupture in the pathogenesis of electrical trauma. *J Surg Res* 1988;44:709-719. **(Animal model research)**
40. American Heart Association. Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Part 8: advanced challenges in resuscitation: section 3: Special Challenges in ECC: 3G: Electric Shock and Lightning Strikes. *Resuscitation* 2000;46:297-299. **(Review)**
41. Cherington M, Martorano FJ, Siebuhr LV, et al. Childhood lightning injuries on the playing field. *J Emerg Med* 1994;12:39-41. **(Case report)**
42. Steinbaum S, Harviel JD, Jaffin JH, et al. Lightning strike to the head: case report. *J Trauma* 1994;36:113-116. **(Case report)**
43. Fahmy FS, Brinsden MD, Smith J, et al. Lightning: the multisystem group injury. *J Trauma* 1999;46:937-940. **(Retrospective cohort; 17 patients)**
44. Nield LS, Kamat D. Long term sequelae of lightning strike in a child: a case report and review. *Clin Pediatr* 2004;43:653-657. **(Case report)**
- 45.* Fontanarosa PB. Electrical shock and lightning strike. *Ann Emerg Med* 1993;22:378-387. **(Review)**
46. Robinson NMK, Chamberlin DA. Electrical injury to the heart may cause long term damage to conducting tissue: a hypothesis and review of the literature. *Int J Cardiol* 1996;53:273-277. **(Review)**
47. Luce EA, Gottlieb SE. "True" high tension electrical injuries. *Ann Plast Surg* 1984;12:321-326. **(Retrospective cohort; 48 patients)**
48. Diamond TH, Twomey A, Myburgh D. High voltage electrical injury. *S Afr Med J* 1982;27:318-321. **(Case report)**
49. Arrowsmith J, Usgaocar RP, Dickson WA. Electrical injury and the frequency of cardiac complications. *Burns* 1997;23:576-578. **(Retrospective cohort; 145 patients)**
50. Kyriacou DN, Zigman A, Sapien R, et al. Eleven year old male with high voltage electrical injury and premature ventricular contractions. *J Emerg Med* 1996;14:591-597. **(Case report)**
51. Das KM. Electrocardiographic changes following electric shock. *Indian J Pediatr* 1974;41:192-193. **(Case report)**
52. Jensen PJ, Thomsen PE, Bagger JP. Electrical injury causing ventricular arrhythmias. *Br Heart J* 1987;57:279-283. **(Case report)**
- 53.* Purdue GF, Hunt JL. Electrocardiographic monitoring after electrical injury: necessity or luxury. *J Trauma* 1986;26:166-167. **(Retrospective cohort; 48 patients)**
54. Fatovich DM, Lee KY. Household electric shocks: who should be monitored? *Med J Austr* 1991;155:301-303. **(Retrospective cohort; 20 patients)**
55. Cunningham PA. The need for cardiac monitoring after electrical injury. *Med J Austr* 1991;154:765-766. **(Retrospective cohort; 70 patients)**
- 56.* Bailey B, Gaudreault P, Thivierge RL, et al. Cardiac monitoring of children with household electrical injuries. *Ann Emerg Med* 1995;25:612-617. **(Retrospective cohort; 141 patients)**
- 57.* Garcia CT, Smith GA, Cohen DM. Electrical injuries in a pediatric emergency department. *Ann Emerg Med* 1995;26:604-608. **(Retrospective cohort; 54 patients)**
58. Wilson CM, Fatovich DM. Do children need to be monitored after electric shocks? *J Paediatr Child Health* 1998;34:474-476. **(Retrospective cohort; 44 patients)**
59. Norquist C, Rosen CL, Adler JN, et al. The risk of delayed dysrhythmias after electrical injury. *Acad Emerg Med* 1999;6:393-a. **(Abstract)**
- 60.* Bailey B, Gaudreault P, Thivierge RL. Experience with guidelines for cardiac monitoring after electrical injury in children. *Am J Emerg Med* 2000;18:671-675. **(Prospective cohort; 224 patients)**
- 61.* Blackwell N, Hayllar J. A three year prospective audit of 212 presentations to the emergency department after electrical injury with a management protocol. *Postgrad Med J* 2002;78:283-285. **(Prospective cohort; 212 patients)**

62. Wilkinson C, Wood M. High voltage electric injury. *Am J Surg* 1978;136:693-696. **(Retrospective cohort; 28 patients)**
63. Ahrenholz DH, Schubert W, Solem LD. Creatinine kinase as a prognostic indicator in electrical injury. *Surgery* 1988;104:741-747. **(Retrospective cohort; 116 patients)**
64. Burke JF, Quinby WC, Bondoc C, et al. Patterns of high tension electrical injury in children and adolescents and their management. *Am J Surg* 1976;133:492-497. **(Retrospective cohort; 29 patients)**
65. Fish RM. Electrical injury, part II: specific injuries. *J Emerg Med* 1999;18:27-34. **(Review)**
66. Brumback RA. Rhabdomyolysis following electrical injury. *Semin Neurol* 1995;15:329-334. **(Review)**
67. Hunt JL, Sato RM, Baxter CR. Acute electrical burns. *Ach Surg* 1980;115:434-438. **(Retrospective cohort; 102 patients)**
68. Rosen CL, Adler JN, Rabban JT, et al. Early predictors of myoglobinuria and acute renal failure following electrical injury. *J Emerg Med* 1999;17:783-789. **(Retrospective cohort; 162 patients)**
69. DiVincenti FC, Moncrief JA, Pruitt BA Jr. Electrical injuries: a review of 65 cases. *J Trauma* 1969;9:497-507. **(Retrospective cohort; 65 patients)**
70. Abramov GS, Bier M, Capelli-Schellpfeffer M, et al. Alteration of sensory nerve function following electrical shock. *Burns* 1996;22:602-606. **(Animal research)**
71. Ko SH, Chun W, Kim HC. Delayed spinal cord injury following electrical burns: a 7-year experience. *Burns* 2004;30:691-695. **(Retrospective cohort; 13 patients)**
- 72.* Grube BJ, Heimbach DM, Engrav LH, et al. Neurologic consequences of electrical burns. *J Trauma* 1990;30:254-258. **(Retrospective cohort; 90 patients)**
73. Cherington M. Central nervous system complications of lightning and electrical injuries. *Semin Neurol* 1995;15:233-240. **(Review)**
74. Chen CT, Yang JY. Electrical burns associated with head injuries. *J Trauma* 1994;37:195-199. **(Retrospective cohort; 249 patients)**
75. Varghese G, Mani MM, Redford JB. Spinal cord injuries following electrical accidents. *Paraplegia* 1986;24:159-166. **(Retrospective cohort; 5 patients)**
76. Lochaitis A, Parker J, Stavropoulou J, et al. Neurological disorders following electrical burn injuries. *Ann MBC* 1991;84-90. **(Case report)**
77. Ratnayake B, Emmanuel ER, Walker CC. Neurological sequelae following a high voltage electrical burn. *Burns* 1996;22:574-577. **(Case report)**
78. Arevalo JM, Lorente JA, Balseiro-Gomez J. Spinal cord injury after electrical trauma in a burn unit. *Burns* 1999;25:449-452. **(Retrospective cohort; 57 patients)**
79. Haberal MA, Gurer S, Akman N, et al. Persistent peripheral nerve pathology in patients with electrical burns. *J Burn Care Rehabil* 1996;17:147-149. **(Retrospective cohort; 25 patients)**
80. Farrell DF, Starr A. Delayed neurological sequelae of electrical injuries. *Neurology* 1968;18:601-606. **(Case report)**
81. Garcia Sanchez V, Morell PG. Electric burns: high and low tension injuries. *Burns* 1999;25:357-360. **(Retrospective cohort; 179 patients)**
82. Hunt JL, McManus WE, Haney WP et al. Vascular lesions in acute electrical injuries. *J Trauma* 1974;14:461-473. **(Retrospective cohort; 11 patients)**
83. Bongard O, Fagrell B. Delayed arterial thrombosis following an apparently trivial low voltage electric injury. *Vasa* 1989;18:162-166. **(Case report)**
84. Miller BK, Goldstein MH, Monshizadeh R, et al. Ocular manifestations of electrical injury: a case report and review of the literature. *CLAO J* 2002;28:224-227. **(Case report)**
85. Saffle JR, Crandall A, Warden G. Cataracts: a long term complication of electrical injury. *J Trauma* 1985;25:17-21. **(Retrospective cohort; 13 patients)**
86. Reddy SC. Electric cataract: a case report and review of the literature. *Eur J Ophthalmol* 1999;9:134-138. **(Case report)**
87. Masanes MJ, Gourbiere E, Prudent J, et al. A high voltage electrical burn of lung parenchyma. *Burns* 2000;26:659-663. **(Case report)**
88. Kumar S, Thomas S, Lehri S. Abdominal wall and stomach perforation following accidental electrocution with high tension wire: a unique case. *J Emerg Med* 1993;11:141-145. **(Case report)**
89. Sharma BC, Patial RK, Pal LS, et al. Electrocardiographic manifestations following household electrical current. *J Assoc Physicians India* 1990;38:938-939. **(Case report)**
90. Walton AS, Harper RW, Coggins GL. Myocardial infarction after electrocution. *Med J Austr* 1988;148:365-367. **(Case report)**
91. Xenopoulos N, Movahed A, Hudson P et al. Myocardial injury in electrocution. *Am Heart J* 1991;122:1481-1484. **(Case report)**
92. Orgel MG, Brown HC, Woolhouse FM. Electrical burns of the mouth in children: a method for assessing results. *J Trauma* 1978;15:285-289. **(Retrospective cohort; 51 patients)**
93. Nichter LS, Morgan RE, Bryant CA, et al. Oral cavity electric burns. *Compr Ther* 1985;4:65-71. **(Review)**
94. Milano M. Oral electrical and thermal burns in children. *ASDC J Dent Child* 1999;66:116-119, 185. **(Review)**
95. Linebaugh ML, Koka S. Oral electrical burns: etiology, histopathology, prosthodontic treatment. *J Prosthodont* 1993;2:136-141. **(Review)**
96. Oeconomopoulos CT. Electrical burns in infancy and early childhood. *Am J Dis Child* 1962;103:35-38. **(Review)**
97. Parano E, Uncini, Incorpora G, et al. Delayed bilateral median nerve injury due to low tension electrical current. *Neuropediatrics* 1995;27:105-107. **(Case report)**
98. Demun EM, Redd JL, Buchanan KA, et al. Reflex sympathetic dystrophy after minor electric shock. *J Emerg Med* 1993;11:393-396. **(Case report)**
99. Stueland DT, Stamas P, Welter TM, et al. Bilateral humeral fractures from electrically induced muscular spasm. *J Emerg Med* 1989;7:457-459. **(Case report)**
100. Govoni M, Orzincolo C, Bigoni M, et al. Humeral head osteonecrosis by electrical injury. *J Emerg Med* 1993;11:17-21. **(Case report)**
101. Hostetler MA, Davis CO. Galeazzi fracture resulting from electrical injury. *Pediatr Emerg Care* 2000;16:258-259. **(Case report)**
- 102.* Lichtenberg M, Dries D, Ward K, et al. Cardiovascular effects of lightning strikes. *J Am Coll Cardiol* 1993;21:531-536. **(Retrospective cohort; 19 patients)**
103. Aslan S, Yilmaz S, Karcioğlu O. Lightning: an unusual cause of cerebellar infarction. *Emerg Med J* 2004;21:750-751. **(Case report)**
104. Kravitz H, Wasserman MJ, Valaitis J, et al. Lightning injury. *Am J Dis Child* 1977;131:413-415. **(Case report)**
105. ten Duis HJ, Klasein HJ, Reenalda PE. Keraunoparalysis, a 'specific' lightning injury. *Burns Incl Therm Inj* 1985 Oct;12(1):54-57. **(Case report)**
106. Gluncic I, Roje Z, Gluncic V, et al. Ear injuries caused by lightning: report of 18 cases. *J Laryngol Otol* 2001;115:4-8. **(Retrospective cohort; 18 patients)**
107. Ravitch MM, Lane R, Safar P, et al. Report of a case with recovery after cardiac massage and prolonged artificial respiration. *N Engl J Med* 1961;264:36-39. **(Case report)**
108. Taussig HB. "Death" from lightning and the possibility of living again. *Ann Intern Med* 1968 68:1345-1353. **(Case report)**
109. Moran KT, Thupari JN, Munster AM, et al. Electric and lightning induced cardiac arrest reversed by prompt cardiopulmonary resuscitation. *JAMA* 1986;255:2157. **(Letter)**
110. Fish RA. Electric injury, Part I: Treatment priorities subtle diagnostic factors, and burns. *J Emerg Med* 1999;17:977-983. **(Review)**
111. Morrison M, Woollard M. Outcome of asymptomatic electric shock victims requesting an emergent ambulance. *Prehosp Emerg Care* 2004;8:400-404. **(Retrospective cohort; 52 patients)**
112. Abt JL. The pupillary response after being struck by lightning. *JAMA* 1985;254:3312. **(Letter)**
113. Norman ME, Albertson D, Young BR. Ophthalmic manifestations of lightning strike. *Surv Ophthalmol* 2001;46:19-24. **(Review)**
114. Eneas JF, Schoenfeld PY, Humphreys MH. The effect of infusion of mannitol-sodium bicarbonate on the clinical course of myoglobinuria. *Arch Intern Med* 1979;139:801-805. **(Retrospective cohort; 20 patients)**
115. Hirschfeld JJ, Assael LA. Conservative management of electric burns of the lips of children. *J Oral Maxillofac Surg* 1984;42:197-202. **(Case reports)**
116. Palin WE Jr, Sadove AM, Jones JE, et al. Oral electrical burns in a pediatric population. *J Oral Med* 1987;42:17-21. **(Retrospective cohort; 45 patients)**
117. Ortiz-Monasterio F, Factor R. Early Definitive treatment of electrical burns of the mouth. *Plast Reconstruct Surg* 1980;65:169-176. **(Case reports)**
118. Thomas SS. Electrical burns of the mouth: still searching for an answer. *Burns* 1996;22:137-140. **(Retrospective cohort; 5 patients)**
119. Steer RG. Delayed fetal death following electrical injury in the first trimester. *Aust N Z J Obstet Gynecol* 1992;32:377-378. **(Case report)**
120. Leiberman JR, Mazor M, Molcho J, et al. Electrical accidents during pregnancy. *Obstet Gynecol* 1986;67:861-863. **(Case reports)**
121. Einaron A, Bailey B, Inocencio G, et al. Accidental shock in pregnancy: a prospective cohort study. *Am J Obstet Gynecol* 1997;176:678-681. **(Prospective cohort; 31 patients)**
122. Fatovich DM. Electric shock in pregnancy. *J Emerg Med* 1993;11:175-177. **(Review)**
123. Ridenour MV. Age appropriateness and safety of electric outlet protectors for children. *Percept Mot Skills* 1997;84:387-392. **(Prospective; 37 patients)**

Physician CME Questions

17. When a lightning injury occurs, which of the following mechanisms of injury is most dangerous to the patient?
- blunt injury
 - direct strike
 - ground strike
 - halo impact
 - side flash
18. With regard to acute, high-voltage eye injuries, which of the following eye findings are the most commonly seen?

- a. cataracts and tearing
 - b. hyphema and conjunctivitis
 - c. lid swelling and cortical blindness
 - d. miosis and disconjugate gaze
 - e. mydriasis and anisocoria
19. Which of the following skin findings is pathognomonic for lightning strikes?
- a. "dolphin fins"
 - b. "feathering"
 - c. "mowed lawn"
 - d. "rats' teeth"
 - e. "whirlpool"
20. For a patient who has sustained a serious electrical injury, which of the following is the best indicator that fluid management is adequate?
- a. adequate urine output
 - b. normal mental status
 - c. quick capillary refill
 - d. swollen lips and eyelids
 - e. the onset of edema
21. Of all admissions to specialized burn units, approximately what percentage is due to electrical injuries?
- a. 5%
 - b. 25%
 - c. 50%
 - d. 75%
 - e. 95%
22. A child who has just sustained a high-voltage injury exhibits apnea and pupils that are fixed and dilated. Based on this information, which of the following is true?
- a. No further resuscitation should be attempted, as it is futile.
 - b. Resuscitation should proceed per standardized protocols.
 - c. The child should not be touched, as the rescuer might be endangered.
 - d. The child was already dead before the electrical injury.
 - e. This situation does not occur, as the pupils constrict in this setting.
23. Which of the following is the most appropriate treatment for small, superficial electrical burns to the hands and feet?
- a. dry povidone-iodine dressings
 - b. peroxide washings twice daily
 - c. spring water soaks every day
 - d. topical silver sulfadiazine cream
 - e. wet-to-dry dressings twice daily
24. Of the following, which is the most common cause of low-voltage electrical injuries in young children?
- a. electrical objects falling into the bathtub
 - b. flying a kite into overhead electrical lines
 - c. ingestion of disc batteries from their toys
 - d. lightning strike while playing outdoors
 - e. placing an object in an electric socket
25. Which of the following laboratory findings is most consistent with the diagnosis of rhabdomyolysis?
- a. cerebrospinal fluid with marked pleocytosis and an elevated protein level
 - b. dip urinalysis positive for blood without red blood cells on microscopy
 - c. electrocardiogram with marked ST depressions and a short PR interval
 - d. head computerized tomographic (CT) scan with enlarged ventricles
 - e. isolated elevation of serum chloride on a set of serum electrolytes
26. In the setting of an electrical injury, which of the following is true regarding rhabdomyolysis?
- a. Rhabdomyolysis does not occur in children.
 - b. Rhabdomyolysis leads to immediate renal failure.
 - c. Prehospital cardiac arrest is a risk factor.
 - d. Hydration status does not affect the risk.
 - e. Myoglobin plays no role in these injuries.
27. With regard to high-voltage, alternating-current injuries, which of the following body locations is most commonly involved?
- a. lower back
 - b. genitals
 - c. back of head
 - d. umbilicus
 - e. upper extremity
28. According to the retrospective study by Bailey et al, for children who come in contact with low-voltage electrical sources, which of the following is NOT an indication for an electrocardiogram (EKG)?
- a. bilateral oral commissure burns
 - b. electrical source greater than 240 volts
 - c. history of cardiac problems
 - d. loss of consciousness
 - e. prolonged muscle contraction
29. Which of the following occupations is most commonly associated with electrical injuries?
- a. golfer
 - b. model
 - c. painter
 - d. photographer
 - e. waitress
30. Which of the following is the correct unit of measure for resistance?
- a. Ampere
 - b. Joule
 - c. Hertz

Physician CME questions conclude on back page

- d. Ohm
- e. Volt

31. When someone accidentally grabs onto an electrical source and can't let go, which of the following is true?

- a. The current was most likely alternating current.
- b. The electrical source was highly magnetic.
- c. The person has excessive metal in their pockets.
- d. The person is truly suicidal and refusing to let go.
- e. This situation doesn't happen, due to the "blast effect."

32. Which of the following tests is indicated for the ED evaluation of asymptomatic children with a brief, low-voltage contact?

- a. CT scanning of the head
- b. electrocardiogram
- c. no tests are indicated
- d. troponin I
- e. urinalysis

Class Of Evidence Definitions

Each action in the clinical pathways section of *Pediatric Emergency Medicine Practice* receives a score based on the following definitions.

Class I

- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:

- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II

- Safe, acceptable
- Probably useful

Level of Evidence:

- Generally higher levels of evidence
- Non-randomized or retrospective studies: historic, cohort, or case-control studies
- Less robust RCTs
- Results consistently positive

Class III

- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:

- Generally lower or intermediate

levels of evidence

- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate

- Continuing area of research
- No recommendations until further research

Level of Evidence:

- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

Significantly modified from: The Emergency Cardiovascular Care Committees of the American Heart Association and representatives from the resuscitation councils of ILCOR: How to Develop Evidence-Based Guidelines for Emergency Cardiac Care: Quality of Evidence and Classes of Recommendations; also: Anonymous. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part IX. Ensuring effectiveness of community-wide emergency cardiac care. *JAMA* 1992;268(16):2289-2295.

Physician CME Information

This CME enduring material is sponsored by Mount Sinai School of Medicine and has been planned and implemented in accordance with the Essentials and Standards of the Accreditation Council for Continuing Medical Education. Credit may be obtained by reading each issue and completing the printed post-tests administered in December and June or online single-issue post-tests administered at EBMedPractice.net.

Target Audience: This enduring material is designed for emergency medicine physicians.

Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

Date of Original Release: This issue of *Pediatric Emergency Medicine Practice* was published August 29, 2005. **This activity is eligible for CME credit through August 1, 2008.** The latest review of this material was August 3, 2005.

Discussion of Investigational Information: As part of the newsletter, faculty may be presenting investigational information about pharmaceutical products that is outside Food and Drug Administration approved labeling. Information presented as part of this activity is intended solely as continuing medical education and is not intended to promote off-label use of any pharmaceutical product. *Disclosure of Off-Label Usage:* This issue of *Pediatric Emergency Medicine Practice* discusses no off-label use of any pharmaceutical product.

Faculty Disclosure: In compliance with all ACCME Essentials, Standards, and Guidelines, all faculty for this CME activity were asked to complete a full disclosure statement. The information received is as follows: Dr. Ota, Dr. Purdue, Dr. Kwon, and Dr. Schultz report no significant financial interest or other relationship with the manufacturer(s) of any commercial product(s) discussed in this educational presentation.

Accreditation: Mount Sinai School of Medicine is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

Credit Designation: Mount Sinai School of Medicine designates this educational activity for up to 4 hours of Category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit actually spent in the educational activity. *Pediatric Emergency Medicine Practice* is approved by the American College of Emergency Physicians for 48 hours of ACEP Category 1 credit (per annual subscription). This continuing medical education activity has been reviewed by the American Academy of Pediatrics and is acceptable for up to 48 AAP Credits. These credits can be applied toward the AAP CME/CPD Award available to Fellows and Candidate Fellows of the American Academy of Pediatrics.

Earning Credit: Two Convenient Methods

- **Print Subscription Semester Program:** Paid subscribers with current and valid licenses in the United States who read all CME articles during each *Pediatric Emergency Medicine Practice* six-month testing period, complete the post-test and the CME Evaluation Form distributed with the December and June issues, and return it according to the published instructions are eligible for up to 4 hours of Category 1 credit toward the AMA Physician's Recognition Award (PRA) for each issue. You must complete both the post-test and CME Evaluation Form to receive credit. Results will be kept confidential. CME certificates will be delivered to each participant scoring higher than 70%.
- **Online Single-Issue Program:** Paid subscribers with current and valid licenses in the United States who read this *Pediatric Emergency Medicine Practice* CME article and complete the online post-test and CME Evaluation Form at EMPractice.net are eligible for up to 4 hours of Category 1 credit toward the AMA Physician's Recognition Award (PRA). You must complete both the post-test and CME Evaluation Form to receive credit. Results will be kept confidential. CME certificates may be printed directly from the Web site to each participant scoring higher than 70%.

***Pediatric Emergency Medicine Practice* is not affiliated with any pharmaceutical firm or medical device manufacturer.**

CEO & Publisher: Robert Williford. **President & General Manager:** Stephanie Williford. **Executive Editor:** Cheryl Strauss.

Direct all editorial or subscription-related questions to EB Practice, LLC: **1-800-249-5770** • Fax: 1-770-500-1316 • Non-U.S. subscribers, call: 1-678-366-7933

EB Practice, LLC • 305 Windlake Court • Alpharetta, GA 30022

E-mail: emp@empractice.net • Web Site: <http://EBMedPractice.net>

Pediatric Emergency Medicine Practice (ISSN 1549-9650) is published monthly (12 times per year) by EB Practice, LLC, 305 Windlake Court, Alpharetta, GA 30022. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. This publication is intended as a general guide and is intended to supplement, rather than substitute, professional judgment. It covers a highly technical and complex subject and should not be used for making specific medical decisions. The materials contained herein are not intended to establish policy, procedure, or standard of care. *Pediatric Emergency Medicine Practice* is a trademark of EB Practice, LLC. Copyright ©2005 EB Practice, LLC. All rights reserved. No part of this publication may be reproduced in any format without written consent of EB Practice, LLC. Subscription price: \$299, U.S. funds. (Call for international shipping prices.)