

2

Acute Emergencies in the Dermatology Office

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The dermatologic office emergency is often dismissed as medical oxymoron. The medications administered or procedures performed rarely precipitate a crisis. Nevertheless, certain medical questions and emergencies are relevant to the dermatologic physician, regardless of their low frequency. Based upon the limited literature available and clinical experience, this chapter is designed to offer a practical approach to selected emergency issues that are potentially encountered in the practice of medical and surgical dermatology.

The Emergency Plan

Emergency Medical Services

Ischemic heart disease and sudden cardiac death remain the leading cause of death in the United States and most developed nations.¹ Approximately two thirds of those deaths (about 1000 cases a day) occur outside the hospital setting.² Most of these deaths are reversible if acted upon promptly and systematically, according to the American Heart Association's Cardiac Care Committee. The American Red Cross's health and safety committee further avers that roughly one third to one half of sudden cardiac arrests are preventable if an organized chain of survival resuscitation plan and defibrillation were initiated within the first 5 minutes of arrest.³⁻⁶ While the vast majority of cardiac arrests occur at home, outdoors, or in cars, some inevitably occur in a physician's office.

While ventricular fibrillation is no longer thought to be the most common presenting rhythm in patients who suffered sudden cardiac death, it is the most common initial rhythm in survivors of cardiac arrest.⁷ Terminating ventricular fibrillation can be achieved by electrical defibrillation, and the time from arrest to defibrillation is the most important factor predicting survival. Defibrillation delivered within 1 minute of arrest is associated with a >70% survival rate, dropping by 10% for each minute defibrillation is delayed.⁷⁻⁹

Emergency medical services (EMS) have emerged over the past few decades to increase the chances of survival for victims of cardiac arrest outside the hospital setting. The American Heart Association's four-step chain of survival system includes (1) early access to an emergency medical system; (2) early initiation of cardiopulmonary resuscitation (CPR); (3) early defibrillation; and (4) an early advanced life support system.¹⁰ The highest hospital discharge rate for cardiac arrest has occurred in victims treated with basic life support (BLS) within 4 minutes of arrest and advanced cardiac life support (ACLS), including defibrillation, within 8 minutes.¹¹

Level of Training

For the dermatologist formulating an emergency plan, the primary considerations are: What kind of EMS system exists in the area? How fast can it be mobilized? These questions directly relate to the level of emergency training and equipment necessary for the office. A dermatology unit located within an academic medical center may lie yards away from a level one trauma emergency room. Similarly, a hospital code team may be available by page within minutes. Alternatively, an office may be situated in a city such as Seattle, Washington, with a well-established and pioneering EMS system with an average response time of 4 minutes.^{3,12} In such settings, extensive emergency equipment and ACLS training may be redundant. More practical approaches would then include making sure the patient is placed on the surgical table in the Trendelenberg position. Alternatively, the patient could be placed supine on the floor with arms alongside the body. Loosening or removing clothing and providing materials for obtaining intravenous access facilitate the efforts of the EMS team. If necessary, CPR should be initiated early and continue until the code team is ready to take over. A dermatologist located in a more rural setting, where an EMS system is not able to respond

within minutes of an arrest, should have a more self-sufficient emergency plan. In this scenario, ACLS certification and more emergency equipment would be appropriate (see below).

Anesthesia Considerations

Class I facilities are those in which cutaneous surgical procedures are performed under topical, local, or regional anesthesia (nerve blocks). Anesthesia may be supplemented with oral or intramuscular analgesics (e.g., meperidine) or sedatives (e.g., benzodiazepines). Class II facilities offer the intravenous administration of sedatives or analgesics in addition to Class I anesthesia. Class III facilities offer general anesthesia with the external support of vital body functions.¹³ BLS certification is recommended for class I facilities, whereas at least one staff member in a class II or III facility should be ACLS trained.¹⁴ General dermatology and most surgical dermatology suites fall under the class I designation. Therefore, ACLS training is not routinely indicated for these facilities. It is possible, however, that some hospitals or health care centers may require ACLS certification regardless of office category to confer privileges.

Should more than 8 minutes transpire from the time EMS is called until they can provide defibrillation, if necessary, then the office should be prepared to initiate ACLS. If EMS can be mobilized more quickly, then the facility classification should determine ACLS certification needs. Nevertheless, contacting your local EMS for training and equipment and training recommendations appears prudent.

Against the background of these guidelines, it is useful to review several surveys regarding the preparedness and certification of family practitioners and pediatricians. In a survey of several dozen Michigan physicians, half of whom were family practitioners, Kobernick found that over 70% of the respondents had seen at least one case of chest pain, dyspnea, and seizures in their office.¹⁵ Over half had witnessed anaphylaxis and syncope. However, only 11% of the group had adequate equipment to manage all of these emergencies. Of all practitioners surveyed, approximately 70% were BLS certified and 35% were ACLS certified.

Fuchs and colleagues studied 280 pediatricians and family practitioners in the Chicago area.¹⁶ Certification in BLS was achieved by 88% and in ACLS by 16%. Fewer than one third of the offices in which physicians reported seeing a child each week with asthma, anaphylaxis, sickle-cell crisis, status epilepticus, and sepsis were fully equipped to treat these emergencies. Finally, Altieri and colleagues analyzed responses from 175 pediatricians in the Washington, DC, metropolitan area. A total of 77% of office practices included a member with BLS training, 25% included a member with ACLS. Less than half of the

offices felt adequately equipped for life-threatening emergencies.¹⁷ These surveys were in predominantly urban settings where the EMS system was readily accessible. The initial task of improving such statistics would seem to belong to the primary care providers whose medical purview includes these emergencies on a more regular basis. Dermatologists should at least concentrate on increasing BLS certification and developing an emergency plan.

The Office Team

Each staff member should have a prearranged clearly defined role for an effective office emergency plan. The ultimate goal is to stabilize the patient until the EMS support arrives. The patient is assessed by the physician and nurse. CPR is initiated if indicated, while an assigned staff member activates the EMS system. Another staff member can serve as the designated recorder of event times, serial vital sign measurements, and administered medications. Further roles can include obtaining intravenous access, greeting and escorting the arriving EMS team to the patient, and communicating with family. The crash cart inventory should be periodically reviewed and renewed as necessary. Mock codes can be helpful to further refine the office team emergency plan. Such a plan should be documented and distributed to the staff. Figure 2-1 outlines a sample office plan during such a code. For a more detailed review of BLS and ACLS implementation, the reader is advised to consult other sources, including American Heart Association texts, for appropriate guidelines.¹⁸⁻²⁰

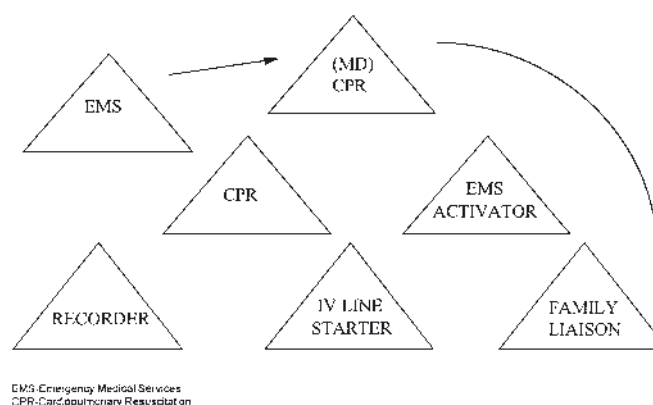


FIGURE 2-1. By clearly defining each staff member's role during a medical emergency, the physician can stabilize a patient until emergency medical services (EMS) support arrives. Various roles are illustrated above, with the physician as the main coordinator. When EMS arrives, the physician can assume the role of communicating with the patient's family.



FIGURE 2-2. An office crash cart should be readily accessible, with clearly labeled and updated contents.

Equipment

The telephone remains the most important piece of emergency equipment in the dermatologist's office, as it provides the first link in the chain of initiating the EMS process. The use of 911 systems has simplified the process, although it is not universally available in the United States. Enhanced 911 systems provide dispatchers with the address and phone number of the caller automatically.^{20,21} An office crash cart should be readily available. Sometimes pictures of each drawer or compartment in a crash cart can be displayed across the top of the cart to minimize last-minute frenetic searching for supplies. Equipment and medications are described elsewhere.^{22,23} Reasonable basic equipment would include a portable oxygen tank with mask or nasal cannula, oral or nasopharyngeal airway, intravenous catheters, tubing, fluids, and suction. Medications include epinephrine 1:1000, lorazepam (Ativan), diazepam (Valium), diphenylhydramine (Benadryl), dextrose 50%, nitroglycerin tablets, and baby aspirin (Fig. 2-2).

Automatic External Defibrillators

To enhance the survival rate of sudden cardiac arrest in the community, the American Heart Association began a more concerted effort in the past decade to promote the use of the automated external defibrillator (AED).²⁴⁻²⁶ The goal is to provide critically important prompt defibrillation from first responders with minimal training (police officers, firefighters, security guards, flight attendants, some laypeople) until further help arrives. While such devices were first introduced for clinical use in the late 1970s, technological advances in the past two decades have greatly increased the safety and efficacy of AEDs by nonmedical personnel.^{27,28,33-36}

Automatic external defibrillators are computerized devices consisting of adhesive electrodes which recognize cardiac rhythms and deliver an electrical shock across the chest wall to terminate ventricular fibrillation. If ventricular fibrillation or rapid, pulseless ventricular tachycardia is detected by the electrode/defibrillator pads placed over the cardiac apex and upper right chest, then the operator is advised by audible and/or visual prompts to deliver a shock by pressing a button. The operator does not need to know how many joules to use because it is preprogrammed. Because an operator is required to activate the shock switch, these devices are considered "automated" rather than "automatic."^{27,33-36}

Following shock delivery, the AED will reanalyze the rhythm and advise additional shocks if fibrillation persists. Following three unsuccessful shocks, the AED will recommend resuming CPR. The AED will cease shocks if it detects that ventricular fibrillation has terminated. Trained first responders can often deliver the first defibrillation shock within 30 seconds of turning on the device. Current AED models are portable, lightweight (under 10 lbs.), and cost approximately \$2500 to \$3000 (Fig. 2-3). A variety of studies show that the ability of AEDs to detect ventricular fibrillation approaches 100% sensitivity and specificity.²⁷⁻³⁶

It should be stated that AEDs are only one part of an effective EMS system. Concomitant bystander CPR and the rapid mobilization of an advanced cardiac life support team are also important factors in patient survival. Given the emerging presence of AEDs in non-hospital settings, it seems reasonable for dermatologists practicing in more remote communities with limited or no EMS service to consider including an AED among their emergency equipment.



FIGURE 2-3. Automatic external defibrillators have been made increasingly more portable and user friendly. Adhesive electrodes are placed over the patient's cardiac apex and upper right chest to detect the cardiac rhythm. With audible and visual prompting, the device advises the operator to deliver a defibrillating shock by pressing a button.

Anaphylaxis

Anaphylaxis is a generalized multiorgan allergic reaction characterized by rapid evolution. Reactions may begin with a prodrome of cutaneous features that include diffuse erythema, pruritus, or urticaria, followed by inspiratory stridor, laryngedema, bronchospasm, hypotension, cardiac arrhythmias, or hyperperistalsis. Systemic reactions can be mild, moderate, or severe because any combination of the above signs and symptoms can occur.^{37,38} Anaphylaxis is a potentially life-threatening event, accounting for approximately 1500 deaths annually in the United States.³⁹

In classic anaphylaxis, the offending antigen binds to immunoglobulin E (IgE) on mast cells and basophils, initiating the release of preformed mediators such as histamine and newly formed mediators such as prostaglandins and leukotrienes. Vascular permeability is increased and vascular smooth muscle is relaxed, leading to hypotension. Pulmonary smooth muscle constriction results in the characteristic bronchospasm.^{40,41} Onset of symptoms is usually immediate, occurring within seconds to minutes of exposure to the offending antigen. Peak severity then progresses over the next 5 to 30 minutes. Some reactions,

however, may present up to 4 hours after antigen ingestion (e.g., medication or food).^{39,41}

Etiologies include a variety of microorganisms, medications, foods, physical factors, and exercise. For many, no cause can be identified.⁴² Implicated foods in adults include peanuts, shellfish, tree nuts (e.g., almonds, hazelnuts, walnuts, pecans), and other fish.^{41–44} In children, eggs, peanuts, milk, soy, tree nuts, shellfish, and wheat are most commonly responsible.^{41,44} The most common antibiotics causing anaphylaxis are penicillins, cephalosporins, sulfonamides, and vancomycin.^{44–46} Penicillin in particular accounts for 75% of anaphylactic mortalities in the United States, with fatalities occurring in 1 in 50,000 to 1 in 100,000.^{39,46} Insect stings, particularly from the fire ant or Hymenoptera (bee, wasp, hornet, yellow jacket, sawfly), result in approximately 40 to 100 anaphylactic deaths per year in the United States.^{39,43,47} A subset of anaphylactic reactions are exercise induced. These reactions can occur after ingestion of a specific food (exercising within 4–6 hours after eating a particular food) or several hours following ingestion of any food. Alternatively, reaction to exercise alone without any temporal relationship to meals can occur.⁴¹

Anaphylactoid reactions are clinically similar, but are not IgE mediated. Therefore, they are not allergic reactions. They occur by directly stimulating mast cells and basophils, provoking the release of the same histamines, leukotrienes, and prostaglandins implicated in the clinical presentation of anaphylaxis. Anaphylaxis and anaphylactoid reactions are often referred to collectively as *anaphylaxis*. An important distinction is that the latter can often be prevented by pretreatment with steroids and antihistamines. Anaphylactoid reactions are most commonly caused by radiocontrast media, aspirin, non-steroidal anti-inflammatory agents, opioids, and muscle relaxants.^{39,41,43}

The incidence of anaphylactic reactions in the dermatology office is likely very low because the most common etiologies are not often directly relevant to the typical office encounter. Nevertheless, etiologies could include preoperative antibiotic prophylaxis with a penicillin or cephalosporin, local anesthesia infiltration with an ester anesthetic or lidocaine with methylparaben (see Lidocaine “Allergy” below), an insect sting, or an ill-fated sampling of displayed waiting room food. Bacitracin, neomycin, and topical nitrogen mustard–induced anaphylaxis have been reported.^{48–54} Many of these cases exhibited concomitant stasis dermatitis and/or ulceration, presumably providing more rapid systemic absorption of the offending agent. Similarly, the medical disinfectant chlorhexidine has been shown to rarely cause anaphylactic symptoms during or shortly following surgical procedures, with specific IgE and positive skin test reactions demonstrated.^{45,55–57} Avoiding mucous membrane appli-

cation and using lower concentrations of chlorhexidine (0.05%) may minimize these reactions.

Similarly, while natural rubber latex allergies are becoming increasingly more recognized, a small percentage of patients can manifest anaphylactic reactions. Such patients usually have undergone multiple surgeries, require long-term bladder care, or are health workers more habitually exposed to such materials than the average patient. Alternatively, atopic individuals have an enhanced risk of anaphylaxis to natural rubber latex.⁴⁵ In the acutely bronchospastic, hypotensive patient, the dermatologist should therefore consider latex or topical medication-induced anaphylaxis.

Prompt recognition is the key to anaphylaxis management. While the history and clinical presentation are often diagnostic, sometimes laboratory evaluation can be helpful, consisting of elevated plasma or urinary histamine levels or plasma tryptase levels drawn after the onset of symptoms.^{39,41,57} The patient should be placed supine on the examining table, preferably in the Trendelenberg position, and tight clothing should be loosened. Vital signs can be assessed while low-flow oxygen (1–2 L/min) by face mask is initiated. Efforts to eliminate or minimize antigen exposure include wiping off the antigen (e.g., bacitracin) or applying a tourniquet proximal to the injection site (e.g., anesthetic or insect sting), which is loosened every 5 minutes for at least 3 minutes.⁵⁷ The designated staff member can then activate the EMS system according to office protocol. Intravenous access may be needed for fluid resuscitation in cases of persistent hypotension.

The essential medication for the initial treatment of anaphylaxis is epinephrine. Its α 1-agonist properties increase peripheral vascular resistance and decrease urticaria, its β 1-agonist activities increase cardiac rate and contractility, and the β 2-agonist component relaxes bronchial smooth muscles. As soon as anaphylaxis is suspected, 0.3 to 0.5 mL of epinephrine 1:1000 is administered subcutaneously or intramuscularly, usually into the upper arm. Gentle massage of the injection site facilitates absorption. A patient taking a β -blocker should be started on a lower dose (0.2 mL). The pediatric dose is 0.01 mL/kg, up to a maximum of 0.3 to 0.5 mL.^{37,39–41,58} Rapid inactivation of epinephrine may require repeat dosing two or three times at 5 to 10 minute intervals. Severe hypotension may require intravenous epinephrine (1:10,000 at 1 mcg/min, increased to 10 mcg/min as needed).³⁹ This would be best managed by the summoned EMS team because more thorough cardiac monitoring is required.

Nebulized β -adrenergic agents such as albuterol with or without intravenous aminophylline may be considered in patients with severe bronchoconstriction. Simultaneous H1 and H2 blockade may be used early on (within

the first 20 minutes) to prevent reactions (e.g., a maximum of Benadryl 50 mg IM and ranitidine 1 mg/kg IV), while steroids may prevent a delayed reaction that could occur 3 to 6 hours later.^{39–41,58}

While waiting for epinephrine to take effect, the physician should ensure that an open airway is maintained by appropriate head tilt or jaw thrust maneuvers. The office team should be prepared to initiate BLS if respiratory failure and shock develop.⁵⁸ The summoned EMS team can then arrive prepared for possible ACLS initiation prior to transport to a hospital. All patients with anaphylaxis should be monitored for up to 8 to 24 hours following the initial attack, depending on episode severity, due to the possibility of recurrent symptoms after initial resolution.^{37,39–41} Ultimately, a referral to an allergist-immunologist for further evaluation and management appears prudent.

Vasovagal Syncope

Vasovagal syncope is the most common cause of acute brief unconsciousness. It is far more prevalent than anaphylaxis. There are often no associated cardiac or neurological abnormalities. Emotional stress, acute pain, or fear are precipitating factors, although frequently no cause is identified.⁵⁹

The characteristic prodrome of a vasovagal reaction includes anxiety, diaphoresis, nausea, tachypnea, tachycardia, and/or confusion. The skin becomes pale and cool. Vagal-induced bradycardia in the setting of decreased systemic vascular resistance can initiate collapse. Pseudo-seizure activity can occur, characterized by brief clonic activity. Blood pressure may initially decrease but is restored with recumbency. Conversely, anaphylaxis features warm, erythematous, dry, pruritic skin with tachycardia and recumbent hypertension.^{37,58}

Vasovagal events can be minimized by performing procedures such as injections and biopsies with the patient placed in a supine position. Occasionally, the simple act of a prior explanation of the procedure may allay anxiety. For patients with a known history of vasovagal syncope, it may be wise to place a towel over their eyes to avoid sights of blood, surgical trays, and biopsy specimens. A distracting conversation (“talkesthesia”) can be similarly beneficial. Following the procedure, the patient should slowly sit up and be watched for several minutes while postoperative instructions are discussed. Should a vasovagal reaction develop, the patient is promptly restored to recumbency, preferably in the Trendelenberg position. A cool water wash cloth placed on the forehead with a low power fan directed toward the face is often helpful. Patients should be educated as to the difference between this benign condition and true allergy. In

summary, early recognition, restoration of recumbency, and reassurance constitute the management approach to vasovagal events.^{58,59}

Lidocaine “Allergy”

Lidocaine is extensively used in the dermatology office. Occasionally, a patient will claim an allergy to local anesthetics, including lidocaine. The first report of allergy to a local anesthetic was published in the early 20th century, involving a case of a contact dermatitis on the provider’s (dentist) hand.⁶⁰ Subsequent similar reports all concerned reactions to the ester types of anesthetics. This group of agents, which include procaine, tetracaine, and benzocaine, are derivatives of para-aminobenzoic acid (PABA), an established allergen.⁶¹ Occasionally, more serious clinical presentations, including the urticaria and dyspnea seen in anaphylaxis, may be elicited when obtaining a patient’s medical history. Evaluating this history and managing such patients can be a very frustrating and cumbersome endeavor for the clinician and patient because alternatives, such as general anesthesia in an operating room, are disproportionately expensive and morbid for simple office procedures.

Toxic reactions to lidocaine resulting from overdosage (central nervous system or myocardial depression or excitation, perioral numbness, nausea, seizures, or coma) or vasovagal reactions should be distinguished to avoid confusion with allergy. True allergic reactions to pure lidocaine are extremely rare, although there is a case report literature that supports this phenomenon.^{62–64} Lidocaine belongs to the amide class of anesthetics, which do not cross-react with ester anesthetics. It is estimated that less than 1% of reported “allergic” reactions to local anesthetics are immune mediated, and that amide anesthetics comprise of very small percentage of those reactions.^{62,65,66} A growing case report literature of type IV (delayed type) sensitivity to lidocaine, in which a pruritic erythematous eruption develops 2 days following exposure, has emerged, indicating that such reactions may occur more frequently than previously thought.^{61,67–72}

Methyl- and propylparaben are preservatives added to a variety of lotions, cosmetics, foodstuffs, and lidocaine bottles to extend their shelf life. There is some cross-reactivity with PABA. Similarly, sulphite preservatives added to lidocaine and ester anesthetics are structural analogs of PABA. Sulpha-allergic patients should therefore avoid use of anesthetics containing methylparaben and sulfite preservatives.^{62,73}

Anxiety regarding the use of needles, and/or the effects of the frequently added epinephrine in lidocaine vials, can lead to palpitations, “panic attacks,” and vasovagal events that the patient may long remember as an allergic reaction.

Finally, there are a couple of other issues that should be considered in the differential diagnosis of a lidocaine allergy. Concurrent drug exposures, such as nonsteroidal anti-inflammatory drugs and antibiotics, may be the real culprits. Latex allergies should also be included as a possibility.⁴⁵

Skin prick and intradermal tests with various concentrations of lidocaine with methylparaben and appropriate controls have been advocated in the workup of a lidocaine allergy. Patch testing and intradermal challenge assist in the evaluation of type IV sensitivity.^{66,74,75} Such investigations are best performed by allergists. Negative results with the above screening tests may lead to a challenge with subcutaneous injections of lidocaine. Skin testing results are often equivocal, however. In vitro testing is recommended for those patients with a history of anaphylaxis.^{62,74,75}

If the surgical procedure is minor, one can consider intralesional antihistamines (diphenylhydramine or chlorpheniramine) with the appropriate postoperative sedation precautions. Temporary anesthesia may also be achieved by the intradermal injection of normal saline or a topical cold spray (Frigiderm). Even nondrug approaches such as acupuncture or hypnosis have been offered.⁷⁶ Ultimately, patients may require referral to a surgeon for general operating room excision and closure or other analgesics/sedatives, such as nitrous oxide, meperidine, or ketamine.⁷⁶

It should be recalled that the intravenous administration of lidocaine for ventricular arrhythmias or the induction of anesthesia for surgery (with propofol) is routinely and countlessly performed. Allergic reactions to lidocaine in those settings is extremely rare.⁷³ Consequently, a patient claim of lidocaine allergy should be met with a thorough history evaluation and possible allergy consultation to more fully establish its validity. Adverse drug reactions to local anesthetics, lidocaine in particular, have an established differential diagnosis with “allergy” appearing low on the list.

Acute Stroke

Stroke is the third leading cause of death in the United States (after coronary heart disease and cancer), and the leading cause of adult brain injury. Each year approximately 500,000 Americans suffer a stroke, nearly 25% of which are fatal.⁷⁷ A stroke occurs when the blood supply to a portion of the brain is disrupted, resulting in a sudden neurologic deficit from inadequate oxygen delivery. Roughly 85% of strokes are ischemic, in which a blood clot arising within a cerebral vessel or traveling from elsewhere (an embolism) completely occludes a cerebral artery. The remainder are hemorrhagic strokes, where a cerebral artery ruptures, causing bleeding into the surface

of the brain (e.g., from an aneurysm) or within the brain substance (e.g., from hypertension).^{77–80}

While historically little treatment was available to alter the course of the stroke, the potential role of early surgery for hemorrhagic strokes and the established role of fibrinolytic therapy for ischemic strokes has now justified the rapid evaluation and management of the acute stroke victim.^{81–85}

The American Heart Association and American Stroke Association have established stroke management guidelines for physicians, EMS personnel, and the general public. A seven D's mnemonic had been offered: detection, dispatch, delivery, door, data, decision, and drug.⁸⁶ The first three D's are the purview of the BLS providers in the community, including the dermatology office and EMS responders. **Detection** occurs when the signs and symptoms of a stroke are recognized and the EMS system is activated. The EMS team is **dispatched** and the victim is **delivered** to a hospital appropriate for acute stroke care. Once at the hospital, the patient is quickly evaluated and triaged at the **door** (emergency department), where **data** (a head computerized tomography scan) is obtained and the decision to proceed with fibrinolysis (**drug**) is made.

The dermatologist's role chiefly concerns the detection phase of a sudden neurologic deficit. This may include an alteration in consciousness (confusion, coma, seizures), aphasia, dysarthria, facial weakness or asymmetry, extremity weakness, paralysis, sensory loss, ataxia, visual loss (particularly in one eye), or vertigo. A severe headache, nausea, or vomiting generally suggests hemorrhage rather than ischemia. Signs and symptoms can develop in clusters or in isolation and may wax and wane or advance rapidly.^{87,88}

A quick and practical exam tool to evaluate a stroke is the Cincinnati Prehospital Stroke Scale. It focuses on three major physical findings: facial droop, arm drift, and abnormal speech. The patient is asked to show teeth or smile. Asymmetry may be abnormal. The patient is asked to close eyes and extend both arms straight out front for 10 seconds. If one arm does not move at all or one arm drifts down, this is considered abnormal. The patient is then asked to repeat a phrase. Slurred words, using wrong words, or the failure to speak are abnormal results. An abnormality in any one of these exercises is strongly suggestive of a stroke.⁸⁹

The primary function of the dermatologist who suspects a patient is having a stroke is to immediately activate the EMS system. In most cases, definitive hospital-based intervention (i.e., fibrinolysis) must occur within 3 hours of the onset of stroke symptoms to be beneficial.⁸⁴ The time-critical nature of stroke management means that the patient should be transported rapidly. In the interim, the medical office should attend to the ABCs (airway, breathing, circulation) of basic life support as needed until the EMS responders arrive.

Status Epilepticus

Dermatologists encounter patients whose cutaneous disease has potential epileptic manifestations, most notably those with tuberous sclerosis, neurofibromatosis, Sturge–Weber syndrome, and lupus erythematosus. The theoretical risk of office seizures, therefore, merits brief mention of a responsible approach.

Of the million Americans who suffer from recurrent seizures, approximately 3% to 8% of them will manifest at least one episode of status epilepticus, characterized by general tonic-clonic seizures lasting longer than 20 minutes, or a series of recurrent seizures lasting more than 30 minutes with a loss of consciousness. There may be no previous history of seizures. Status epilepticus is associated with a 6% to 18% mortality and higher morbidity.⁹⁰

The dermatologist's first priority is the maintenance of a patent airway while the EMS is notified. An oral or nasopharyngeal airway may be useful. If available, oxygen can be delivered by face mask. Vital signs should be monitored. The ideal area to place the patient is a soft, flat surface away from sharp objects.^{90,91} Objects and fingers should not be placed in the victim's mouth. If seizure activity has abated and the patient is breathing and has a pulse, placing him/her in a recovery position (a modified lateral position) will allow the clearance of any pooled airway secretions.^{20,92,93} In all likelihood, EMS personnel should have arrived by this point to continue management.

If EMS has not arrived within 5 minutes, a peripheral intravenous line should be placed and blood sampled for serum glucose, calcium, electrolytes, and relevant anticonvulsant levels. Subsequently, a bolus of 50 cc 50% glucose is injected prior to a maintenance infusion of normal saline if available.⁹¹ If EMS is still absent at 10 minutes, then seizure control is necessary. The benzodiazepine lorazepam (Ativan) administered at a dose of 1 to 2 mg intravenously will stop seizures within minutes in the vast majority of patients.⁹⁰ Lorazepam has emerged as the seizure drug of choice with a longer action time, 10- to 12-hour half-life, and lower risk of respiratory depression and hypotension than 5 to 10 mg of diazepam (Valium).^{90,94} Regardless of whether or not seizure activity has ceased, patients should be evaluated in an emergency room for seizure etiology and management options.

Electrosurgery and Pacemakers/Defibrillators

The increasing prevalence of implantable pacemakers and defibrillators has raised questions regarding the safety during electrosurgical procedures. Standard dermatologic

electrosurgery consists of a high-frequency, high-voltage, low-amperage alternating current that generates an intense heat at the electrode, dehydrating and coagulating tissue.⁹⁵ Electrodesiccation (direct tissue contact) and electrofulguration (1–2 mm separation of tip from tissue) are monoterminal techniques for which a dispersive plate is not required. The patient's body acts as its own relative ground. In electrocoagulation, the amperage is greater and voltage lower with a biterminal arrangement. A dispersive plate can enhance its effect. Electrocutting is another high-frequency modality with an undamped sine wave, as opposed to the damped waves of the techniques just described. The net effect is a higher energy current that separates tissue ("cuts"). Electrodesiccation, fulguration, coagulation, and cutting current have been thought to involve a potentially significant transfer of electrical activity to patients with pacemakers.⁹⁶

Fixed-rate pacemakers have no theoretical basis for harmful interaction because they lack a sensing device with which bursts of electrosurgery could interfere. They fire continuously at a programmed rate. A patient's intrinsic heart rate may compete with this arrangement and initiate ventricular tachycardia or fibrillation.^{97,98} Subsequently, more patient-concordant pacemakers were developed.

Demand pacemakers have both a sensing and pacing function. Ventricular-inhibited pacemakers fire when the patient's intrinsic heart rate is slower than the programmed heart rate. A ventricular-triggered type fires at a preset rate when no spontaneous heartbeats are detected. It fires with each spontaneous beat as well, yet has no effect then because cardiac muscle has already been depolarized by the patient's own heartbeat. Many pacemakers have a safety system in which the pacemaker functions in fixed-rate mode in the event of sensory failure. In addition, a switch can be activated via an externally applied magnet to switch from demand to fixed-rate function.^{95,98}

Concerns regarding pacemaker/electrosurgery interactions have evolved from two theoretical scenarios. If a ventricular-inhibited pacemaker misinterprets electrical interference as a spontaneous beat, bradycardia or asystole may ensue if the patient's spontaneous rate is slow or nonexistent. Alternatively, a ventricular-triggered pacer may sense such interference as intrinsic beats and fire inappropriately, provoking tachyarrhythmias.⁹⁵

The practice of electrosurgical restraint with pacemakers originated in the urologic literature of the 1970s, in which very high-energy electrocutting was used during transurethral prostatic surgery. Reports of pacemaker battery depletion featured one device that failed several weeks after electrocutting and another that malfunctioned after 45 minutes of electrosurgery.^{99,100} Others described bradycardia from electrosurgical interference in patients with demand pacemakers.^{101,102} Pacemaker

inhibition with electrocutting but not electrocoagulation has been noted.^{99,103} O'Donoghue reported asystole from 5-second bursts of electrocutting. The pacemaker was converted to fixed-rate mode by applying an external magnet, and the patient fully recovered.¹⁰⁴ By 1975, Krull and colleagues offered dermatologists a set of recommendations based on these experiences. Electrosurgery should be avoided in pacemaker patients if an alternative, equally effective modality existed. Prior consultation with a cardiologist, emergency backup, short bursts of electrosurgery (under 5 seconds), and good grounding away from the heart were all recommended.⁹⁸ Finally, considerations should be given to magnetically converting the pacemaker to fixed-rate mode for the procedure. Several subsequent reports questioned the practice of magnetic conversion, citing cases where electrosurgery caused a program change in certain specific pacemaker models while the magnet was on.^{105,106}

Improvements in pacemaker shielding and filtering systems were reflected in Schultz's series of 33 patients who endured transurethral electrosurgery without incident.¹⁰⁷ Yet despite technological advances in cardiac pacer protective circuitry and reprogrammability, electrosurgery remains cited as a common external cause of transient pacemaker malfunction, even in the 1990s.^{108,109} Levine and colleagues described a pacemaker patient who developed two episodes of electrosurgically induced ventricular fibrillation during a coronary revascularization. During chest closure following the procedure, electrosurgery was used for hemostasis without incident.¹⁰⁸ Kellow recalled a patient in whom a transurethral resection of the prostate was performed. His pacemaker failed to capture due to an altered threshold after at least 5 to 10 minutes of electrocutting.¹¹⁰ Goodman urged fellow anesthesiologists to transcend anecdotal discussions and identify the overall incidence of pacemaker failure during electrosurgery.¹¹¹

Implantable cardioverter-defibrillators (ICDs) are implantable electronic devices that sense cardiac electrical activity and terminate ventricular fibrillation and ventricular tachyarrhythmias. Electromagnetic interference could potentially damage/deactivate the ICD device or trigger the device to deliver a defibrillatory discharge. There are some reports of interference with ICDs, but not in a dermatologic surgical context.^{112–115}

It is therefore important to realize that guidelines for the dermatology patient with a pacemaker or ICD have evolved from a case report literature largely irrelevant in surgical procedure, pacemaker era, electrosurgical modality, procedure duration, and/or energy level. Even more recent reviews in the dermatologic literature persist in referencing nondermatologic literature to justify very cautious management guidelines, including detailed preoperative cardiac clearance, programmer availability for deactivation of the ICD or conversion of pacemaker to

fixed-rate mode, continuous cardiac monitoring with electrocardiogram (ECG) and/or pulse oximetry, ACLS equipment availability including resuscitative drugs if necessary, cardiology/hospital access, and a postprocedure plan to coordinate the reactivation or functional assessment of the device by the patient's cardiologist.¹¹⁶

The most recent survey of dermatologic surgeons corroborates a previous survey regarding the heterogeneity in approach to the electrosurgical precautions taken with cardiac devices.^{117,118} While most surgeons responding indicated that they use short bursts of electrocautery (71%), use minimal power (61%), and avoid use directly around the pacemaker/ICD (57%), the use of heat cautery (34%) or bipolar forceps (19%) to avoid any electrical current passing to the patient is less commonly employed. Finally, obtaining cardiology consultation (11%), deactivating the ICD (15%), or changing the pacemaker to fixed-rate mode (1%), and postoperative evaluation of the device by a cardiologist (2%) are very infrequently utilized.¹¹⁸

Of the 166 Mohs surgeons who responded to the survey, there were six reported incidences of pacemaker reprogramming and four incidences of ICD firing during the surgical procedures. There were 18 patients with "adverse effects," including syncope, altered mental status, palpitations, and one case of hemodynamic instability (no further information known). There were no acute resuscitative efforts needed nor long-term morbidities or mortalities related to the use of electrosurgery in the survey. The overall complication rate thought to result from electrosurgery was calculated to be 0.8 patients/100 years of surgical practice, or roughly one case per three surgical careers of at least 30 years each.

It should be noted that surveys are recall based, and a definitive cause-and-effect relationship between electrosurgery and these events is not certain.

Ultimately, definitive recommendations regarding the use of electrosurgery in patients with pacemakers or ICDs awaits studies that prospectively measure adverse events and compare this to the rate of pacemaker and ICD malfunction in those not undergoing the use of electrosurgery. Each dermatologist needs to pursue a strategy that he/she is comfortable with. It appears prudent at this point to recommend some basic guidelines until more enlightening studies emerge¹¹⁶⁻¹²⁰:

1. Consider electrocautery or bipolar instruments that minimize or eliminate the theoretical risk of interference.
2. Carefully place the dispersive electrode ("ground plate") so that the pacemaker or ICD is not in the path of current flow between the electrosurgical instrument and the ground plate.
3. Ensure that electrical equipment is functioning properly with adequate grounding.

4. Maintain current flow to 3- to 5-second bursts, with pauses between bursts to minimize any prolonged potential interference.
5. Use the lowest electrosurgical current necessary for hemostasis.
6. Have an emergency plan, including defibrillation equipment, if necessary.
7. Consider consulting with the patient's cardiologist prior to surgery for perioperative and postoperative management suggestions.

References

1. Guidelines for cardiopulmonary resuscitation and emergency cardiac care. Emergency Cardiac Care Committee and Subcommittees, American Heart Association. Part I Introduction. *JAMA* 1992;268:2171-2183.
2. Heart and stroke facts: 2000 statistical update. American Heart Association; 1999.
3. Weaver WD, Hill D, Fahrenbruch CE, et al. Use of automated external defibrillator in the management of out-of-hospital cardiac arrest. *N Engl J Med* 1988;319:661-666.
4. Bayes de Luna A, Coumel P, Leclercq JF. Ambulatory sudden cardiac death; mechanisms of production of fatal arrhythmia on the basis of data from 157 cases. *Am Heart J* 1989;117:151-159.
5. White RD, Hankins DG, Bugliosi TF. Seven years' experience with early defibrillation by police and paramedics in an emergency medical services system. *Resuscitation* 1998;39:145-151.
6. Kellermann AL, Hackman BB, Somes G, Kreth TK, Nail L, Dobyns P. Impact of first-responder defibrillation in an urban emergency medical services system. *JAMA* 1993;270:1708-1713.
7. Varon J, Marik P. Treatment of cardiac arrest with automatic external defibrillators: impact on outcome. *Am J Cardiovasc Drugs* 2003;3:265-270.
8. Holmberg M, Holmberg S, Herlitz J. The problem of out-of-hospital cardiac arrest prevalence of sudden death in Europe today. *Am J Cardiol* 1999;83:88D-90D.
9. Auble TE, Menegazzi JJ, Paris PM. Effect of out-of-hospital cardiac defibrillation by basic life support providers on cardiac arrest mortality; a metaanalysis. *Ann Emerg Med* 1995;25:642-648.
10. Cummins RO, Ornato JP, Thies WH, Pepe PE. Improving survival from sudden cardiac arrest: the "chain of survival" concept. A statement for health professionals from the Advanced Cardiac Life Support Subcommittee and the Emergency Cardiac Care Committee, American Heart Association. *Circulation* 1991;83:1832-1847.
11. Eisenberg MS, Bergner L, Hallstrom A. Cardiac resuscitation in the community: importance of rapid provision and implications for program planning. *JAMA* 1979;241:1905-1907.
12. Weaver WD, Copass MK, Hill D, Fahrenbruch C, Hallstrom AP, Cobb L. Cardiac arrest treated with a new automatic external defibrillator by out-of-hospital first responders. *Am J Cardiol* 1986;57:1017-1021.

13. Guidelines of care for office surgical facilities: part I. *J Am Acad Dermatol* 1992;26:763–765.
14. Guidelines of care for office surgical facilities: part II. *J Am Acad Dermatol* 1995;33:265–270.
15. Kobernick MS. Management of emergencies in the medical office. *J Emerg Med* 1986;4:71–74.
16. Fuchs S, Jaffe DM, Christoffel KK. Pediatric emergencies in office practices: prevalence and office preparedness. *Pediatrics* 1989;83:931–939.
17. Altieri M, Bellet J, Scott H. Preparedness for pediatric emergencies encountered in the practitioner's office. *Pediatrics* 1990;85:710–714.
18. Emergency Cardiac Care Committee and subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care, II: adult basic life support. *JAMA* 1992;268:2184–2198.
19. Emergency Cardiac Care Committee and subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care, III: adult advanced cardiac life support. *JAMA* 1992;268:2199–2241.
20. BLS for healthcare providers. American Heart Association; 2001.
21. Emergency Cardiac Care Committee and subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care, IX; ensuring effectiveness of communitywide emergency cardiac care. *JAMA* 1992;268:2289–2295.
22. Thomas RM, Amonette RA. Emergencies in skin surgery. In: Roenigk RK, Roenigk HH, eds. *Dermatologic surgery, principles and practice*. 2nd ed. New York: Marcel Dekker; 1996:77–89.
23. Nagi C, Thomas RM. Recognition and management of office medical and surgical emergencies. In: Wheeland RG, ed. *Cutaneous surgery*. Philadelphia: Saunders; 1994:150–158.
24. Cummins RO. From concept to standard-of-care? Review of the clinical experience with automated external defibrillators. *Ann Emerg Med* 1989;18:1269–1275.
25. Cummins RO, Thies W. Encouraging early defibrillation: the American Heart Association and automated external defibrillators. *Ann Emerg Med* 1990;19:1245–1248.
26. Weisfeldt ML, Kerber RE, McGoldrick RP, et al., for the American Heart Association Task Force on automatic external defibrillation. Public access to defibrillation. *Circulation* 1995;92:2763.
27. Marengo JP, Wang PJ, Link MS, et al. Improving survival from sudden cardiac arrest: the role of the automated external defibrillator. *JAMA* 2001;285:1193–1200.
28. Cummins RO, Eisenberg M, Bergner L, Murray JA. Sensitivity, accuracy, and safety of an automatic external defibrillator. *Lancet* 1984;2:318–320.
29. Liner BE, Jorgenson DB, Poole JE, et al., for the LIFE Investigators. Treatment of out-of-hospital cardiac arrest with low-energy impedance-compensating biphasic waveform automatic external defibrillators. *Biomed Instrum Technol* 1998;32:631–644.
30. Stults KR, Brown DD, Cooley F, et al. Self-adhesive monitor/defibrillator pads improve pre-hospital defibrillation success. *Ann Emerg Med* 1987;16:872–877.
31. Carlson MD, Freeman CS, Garan H, Ruskin JN. Sensitivity of an automatic external defibrillator for ventricular tachyarrhythmias in patients undergoing electrophysiologic studies. *Am J Cardiol* 1988;61:787–790.
32. Poole JE, White RD, Kanz KG, et al. Low-energy impedance-compensating biphasic waveforms terminate ventricular fibrillation at high rates in victims of out-of-hospital cardiac arrest. *J Cardiovasc Electrophysiol* 1997;8:1373–1385.
33. Ramaswamy K, Page RL. The automated external defibrillator: critical link in the chain of survival. *Ann Rev Med* 2003;54:235–243.
34. Varon J, Marik PE. Treatment of cardiac arrest with automatic external defibrillators: impact on outcome. *Am J Cardiovasc Drugs* 2003;3:265–270.
35. Das MK, Zipes DP. Sudden cardiac arrest and automated external defibrillators. *Circ J* 2003;67:975–982.
36. Liddle R, Davies CS, Colquhoun M, Handley AJ. ABC of resuscitation: the automated external defibrillator. *BMJ* 2003;327:1216–1218.
37. Soto-Aguilar MC, deShazo RD, Waring NP. Anaphylaxis: why it happens and what to do about it. *Postgrad Med* 1987;82:154–170.
38. McLean-Tooke APC, Bethune CA, Fay AC, Spickett GP. Adrenaline in the treatment of anaphylaxis: what is the evidence? *BMJ* 2003;327:1332–1334.
39. Tang AW. A practical guide to anaphylaxis. *Am Fam Physician* 2003;68:1325–1332.
40. Bochner BS, Lichtenstein LM. Anaphylaxis. *N Engl J Med* 1991;324:1785–1790.
41. Malde B, Ditto AM. Anaphylaxis. *Allergy Asthma Proc* 2004;25:S52–S53.
42. Kemp SF, Lockey RF, Wolf BL, et al. Anaphylaxis — a review of 266 cases. *Arch Intern Med* 1995;155:1749–1754.
43. Lieberman P. Anaphylaxis and anaphylactoid reactions. In: Middleton E, ed. *Allergy: principles and practice*. 5th ed. St. Louis: Mosby; 1998:1079–1089.
44. Ditto AM, Grammer LG. Food allergy. In: Grammer LG, Greenberger PA, eds. *Patterson's allergic diseases*. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2002:260.
45. Thong BY, Chan Y. Anaphylaxis during surgical and interventional procedures. *Ann Allergy Asthma Immunol* 2004;92:619–628.
46. Joint Task Force on Practice Parameters. The diagnosis and management of anaphylaxis. *J Allergy Clin Immunol* 1998;101:S465–S528.
47. Neugut A, Ghatak AT, Miller RL. Anaphylaxis in the United States: an investigation into its epidemiology. *Arch Intern Med* 2001;161:15–21.
48. Phippen R. Anaphylactoid reaction after Chymacort ointment. *BMJ* 1966;1:1168–1172.
49. Comaish JS, Cunliffe WJ. Absorption of drugs from various ulcers: a cause of anaphylaxis. *Br J Clin Pract* 1967;21:97.
50. Roupe G, Strennegard O. Anaphylactic shock elicited by topical administration of bacitracin. *Arch Dermatol* 1969;100:450–452.
51. Daughters D, Zackheim H, Maibach H. Urticaria and anaphylactoid reactions after topical application of mechlorethamine. *Arch Dermatol* 1973;107:429–430.

52. Vale MA, Connolly A, Epstein AM, et al. Bacitracin-induced anaphylaxis. *Arch Dermatol* 1978;114:800.
53. Schechter JF, Wilkison RD, Carpio JD. Anaphylaxis following the use of bacitracin ointment. *Arch Dermatol* 1984;120:909–911.
54. Phillips TJ, Rogers GS, Kanj LF. Bacitracin anaphylaxis. *J Geriatr Dermatol* 1995;3:83–85.
55. Okano M, Nomura M, Hata S, et al. Anaphylactic symptoms due to chlorhexidine gluconate. *Arch Dermatol* 1989;125:50–52.
56. Pharm NH, Weiner JM, Reisner GS, Baldo BA. Anaphylaxis to chlorhexidine: case report: implication of immunoglobulin E antibodies and identification of an allergenic determinant. *Clin Exp Allergy* 2000;30:1001–1007.
57. Garvey LH, Roed-Petersen J, Husum B. Anaphylaxis I anesthetized patients: four cases of chlorhexidine allergy. *Acta Anaesthesiol Scand* 2001;45:1290–1294.
58. Gordon BR. Prevention and management of office allergy emergencies. *Otolaryngol Clin North Am* 1992;25:119–134.
59. Martin JB, Ruskin J. Faintness, syncope, and seizures. In: Wilson JD, et al., eds. *Harrison's principles of internal medicine*. 12th ed. New York: McGraw-Hill; 1991:134–140.
60. Mook WH. Skin reactions to apothesis and quinine in susceptible persons. *Arch Dermatol* 1920;1:651–655.
61. Mackley CL, Marks JG. Lidocaine hydrochloride. *Am J Contact Dermat* 2003;14:221–223.
62. Chiu C, Lin T, Hsia S, et al. Systemic anaphylaxis following local lidocaine administration during a dental procedure. *Ped Emerg Care* 2004;20:178–180.
63. Giovannitti J, Bennett CR. Assessment of allergy to local anesthetics. *J Am Dent Assoc* 1979;98:701–706.
64. Chin TM, Fellner MJ. Allergic hypersensitivity to lidocaine hydrochloride. *Int J Dermatol* 1980;19:147–148.
65. Verrill PJ. Adverse reactions to local anesthetics and vasoconstrictor drugs. *Practitioner* 1975;214:380–387.
66. Amsler E, Flahault A, Mathelier-Fusade P, Aractingi S. Evaluation of re-challenge in patients with suspected lidocaine allergy. *Dermatology* 2004;208:109–111.
67. Curley RK, Macfarlane AW, King CM. Contact sensitivity to the amide anesthetics lidocaine, prilocaine, and mepivacaine: case report and review of the literature. *Arch Dermatol* 1986;122:924–926.
68. Bircher AJ, Messmer SL, Surber C, Ruffi T. Delayed-type hypersensitivity to subcutaneous lidocaine with tolerance to articaine: confirmation by in vivo and in vitro tests. *Contact Dermatitis* 1996;24:387–389.
69. Whalen JD. Delayed-type hypersensitivity after subcutaneous administration of amide anesthetic. *Arch Dermatol* 1996;132:1256–1257.
70. Briet S, Rueff F, Przybilla B. “Deep impact” contact allergy after subcutaneous injection of local anesthetics. *Contact Dermatitis* 2001;45:296–297.
71. Downs AM, Lear JT, Wallington TB, Sansom JE. Contact sensitivity and systemic reaction to pseudoephedrine and lignocaine. *Contact Dermatitis* 1998;39:33.
72. Kaufman JM, Hale EK, Ahinoff RA, Cohen DE. Cutaneous lidocaine allergy confirmed by patch testing. *J Drugs Dermatol* 2002;2:192–194.
73. Finucane BT. Allergies to local anesthetics — the real truth. *Can J Anesth* 2003;50:869–874.
74. Macy E. Local anesthetic adverse reaction evaluations: the role of the allergist. *Ann Allergy Asthma Immunol* 2003;91:319–320.
75. Chandler MJ, Grammer LC. Provocative challenge with local anesthetics in patients with a prior history of reaction. *J Allergy Clin Immunol* 1987;79:883–886.
76. Lu D. Managing patients with local anesthetic complications using alternative methods. *Penn Dent J* 2002;69(3):22–29.
77. Bronner LL, Kanter DS, Manson JE. Primary prevention of stroke. *N Engl J Med* 1995;333:1392–1400.
78. Pepe PE. The chain of recovery from brain attack: access, pre-hospital care, and treatment. In: *Proceedings of the National Symposium on Rapid Identification and Treatment of Acute Stroke*. Bethesda, MD: The National Institute of Neurological Disorders and Stroke; 1996:20–42.
79. Broderick JP, Brott T, Tomsick T, et al. The risk of subarachnoid and intracerebral hemorrhages in blacks as compared with whites. *N Engl J Med* 1992;326:733–736.
80. Brott T, Thalinger K, Hertzberg V. Hypertension as a risk factor for spontaneous intracerebral hemorrhage. *Stroke* 1986;17:1078–1083.
81. Broderick JP, Brott TG, Tomsick T, et al. Ultra-early evaluation of intracerebral hemorrhage. *J Neurosurg* 1990;72:195–199.
82. The NINDS rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med* 1995;333:1581–1587.
83. Hacke W, Kaste M, Fieschi C, et al. Intravenous thrombolysis with recombinant tissue plasminogen activator for acute hemispheric stroke: the European Cooperative Acute Stroke Study (ECASS). *JAMA* 1995;274:1017–1025.
84. Kwiatkowski TG, Libman RB, Frankel M, et al. Effects of tissue plasminogen activator for acute ischemic stroke at one year. *N Engl J Med* 1999;340:1781–1787.
85. Albers GW, Bates VE, Clark WM, et al. Intravenous tissue-type plasminogen activator for treatment of acute stroke: the Standard Treatment with Alteplase to Reverse Stroke (STARS) Study. *JAMA* 2000;283:1145–1150.
86. Hazinki MF. Demystifying recognition and management of stroke. *Curr Emerg Cardiac Care* 1996;7:8.
87. Barsan WG, Brott TG, Broderick JP, et al. Time of hospital presentation in patients with acute stroke. *Arch Intern Med* 1993;153:2558–2561.
88. Feldmann E, Gordon N, Brooks JM, et al. Factors associated with early presentation of acute stroke. *Stroke* 1993;24:1805–1810.
89. Kothari R, Pancioli A, Liu T, et al. Cincinnati Prehospital Stroke Scale: reproducibility and validity. *Ann Emerg Med* 1999;33:373–378.
90. Selbst SM. Office management of status epilepticus. *Pediatr Emerg Care* 1991;7:106–109.
91. Delgado-Escueta AV, Wasterlain C, Treiman DM, et al. Management of status epilepticus. *N Engl J Med* 1982;306:1337–1340.
92. Handley AJ, Becker LB, Allen M, et al. Single-rescuer adult basic life support: an advisory statement from the Basic Life Support Working Group of the International Liaison Committee on Resuscitation. *Resuscitation* 1997;34:101–108.

93. Turner S, Turner I, Chapman D, et al. A comparative study of the 1992 and 1997 recovery positions for use in the UK. *Resuscitation* 1998;39:153–160.
94. Emergency Cardiac Care Committee and subcommittees, American Heart Association. Guidelines for cardiopulmonary resuscitation and emergency cardiac care, IV: special resuscitation situations. *JAMA* 1992;268:2242–2250.
95. Sebben JE. Electrosurgery and cardiac pacemakers. *J Am Acad Dermatol* 1983;9:457–463.
96. Popkin GL. Electrosurgery. In: Epstein E, ed. *Skin surgery*. Springfield, IL: Thomas; 1982:385–404.
97. Escher DJW. Types of pacemakers and their complications. *Circulation* 1973;47:1119–1129.
98. Krull EA, Pickard SD, Hall JC. Effects of electrosurgery on cardiac pacemakers. *J Derm Surg* 1975;1:43–45.
99. Wajszszak WJ, Mowry RM, Dugan WL. Deactivation of a demand pacemaker by transurethral electrocautery. *N Engl J Med* 1969;280:34–35.
100. Schwingshackl H, Maurer R, Amor H. Interfering influence of low frequency alternating currents on asynchronous and controlled pacemaker systems during the use of electrosurgical devices. *Schweiz Med Wochenschr* 1971;101:46–52.
101. Green LF, Merideth J. Transurethral operations employing high frequency electrical currents in patients with demand cardiac pacemakers. *J Urol* 1972;108:446–448.
102. Smith BR, Wise WS. Pacemaker malfunction from urethral electrocautery. *JAMA* 1971;218:256.
103. Batra YK, Bali IM. Effect of coagulation and cutting current on a demand pacemaker during transurethral resection of the prostate. A case report. *Can Anaesth Soc J* 1978;25:65.
104. O'Donoghue JK. Inhibition of a demand pacemaker by electrocautery. *Chest* 1973;64:664–666.
105. Parsonnet V, Furman S, Smyth NPD, et al. Optimal resources for implantable cardiac pacemakers. Intersociety commission for heart disease resources. *Circulation* 1980;68:226A.
106. Domino KB, Smith TC. Electrocautery-induced reprogramming of a pacemaker using a precordial magnet. *Anesth Analg* 1983;62:609–612.
107. Schultz W. Transurethral electro-resection in patients with cardiac pacemakers. *Urologe* 1979;18:247–249.
108. Levine PA, Balady GJ, Lazar HL, et al. Electrocautery and pacemakers: management of the paced patient subject to electrocautery. *Ann Thorac Surg* 1986;41:313–317.
109. Hayes DL, Vlietstra RE. Pacemaker malfunction. *Ann Intern Med* 1993;119:828–835.
110. Kellow NH. Pacemaker failure during transurethral resection of the prostate. *Anaesthesia* 1993;48:136–138.
111. Goodman NW. Diathermy and failure of cardiac pacemakers. *Anaesthesia* 1993;48:824.
112. Furman S. Electrosurgical device interference with implanted pacemakers. *JAMA* 1978;239:1910.
113. Pinski SL. Emergencies related to implantable cardioverter-defibrillators. *Crit Care Med* 2000;28(Suppl):N174–N180.
114. Niehaus M, Tebbenjohanns J. Electromagnetic interference in patients with implanted pacemakers or cardioverter-defibrillators. *Heart* 2001;86:246–248.
115. Madigan JD, Choudhri AF, Chen J, et al. Surgical management of the patient with an implanted cardiac device: implications of electromagnetic interference. *Ann Surg* 1999;230:639–647.
116. LeVasseur JG, Kennard CD, Finly EM, Muse RK. Dermatologic electrosurgery in patients with implantable cardioverter-defibrillators and pacemakers. *Dermatol Surg* 1998;24:233–240.
117. Sebben JE. The status of electrosurgery in dermatologic practice. *J Am Acad Dermatol* 1988;19:542–549.
118. El-Gamal HM, Dufresne RG, Saddler K. Electrosurgery, pacemakers and ICDs: a survey of precautions and complications experienced by cutaneous surgeons. *Dermatol Surg* 2001;27:385–390.
119. Riordan AT, Gamache C, Fosko SW. Electrosurgery and cardiac devices. *J Am Acad Dermatol* 1997;37:250–255.
120. Martinelli PT, Schultze KE, Nelson BR. Mohs micrographic surgery in a patient with a deep brain stimulator: a review of the literature on implantable electrical devices. *Dermatol Surg* 2004;30:1021–1030.



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