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Management of Arrhythmias in the Emergency Department

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Little raises the anxiety level of a physician more than a call to the emergency department about a patient who has an arrhythmia. Despite devoting significant time in training to the mechanisms and treatment of cardiac rhythm disturbances, most physicians are uncomfortable dealing with them. This article strives to provide practical and concise advice on initial diagnosis and management of arrhythmias that present to the emergency department. There has been no attempt to be exhaustive in the descriptions. Common pitfalls and concerns are addressed. The article is divided into a discussion of tachyarrhythmias, both wide and narrow complex, bradyarrhythmias, and management of arrhythmia devices—pacemakers and defibrillators. Finally, no account of emergency department arrhythmias can be complete without some mention of syncope.

The acute treatment of cardiac rhythm disturbances is quite simple if one follows a few basic rules (Box 1). First, if the presenting arrhythmia is fast, and the patient is hemodynamically unstable, regardless of the arrhythmia mechanism, perform a direct current transthoracic cardioversion to restore normal sinus rhythm. The patient should either be unconscious or sedated before delivery of the energy. Second, if the patient presents with a bradycardia that is hemodynamically unstable, pace the heart either transvenously or transthoracically. The former is a more reliable method. For all other arrhythmias in which the patient is hemodynamically stable, obtain a 12-lead ECG; there is time to think before acting. By following these two simple rules, all acute arrhythmic emergencies can be handled appropriately.

Tachycardias

Tachycardias can be divided into two categories based on the width of the QRS complexes, narrow complex (QRS duration $\leq$ 120 milliseconds) or wide complex (QRS duration $>$ 120 milliseconds) tachycardias. When determining the QRS width, it is important to use at least two orthogonally placed lead systems. A single-lead rhythm strip is often inadequate. In any given single-lead recording, a wide complex tachycardia may appear narrow (Fig. 1). If the arrhythmia has a narrow QRS complex, it is by definition a supraventricular tachycardia (SVT). These arrhythmias are usually benign, and often the patient can be treated and discharged from the emergency department to complete the evaluation as an outpatient. If the tachycardia has a wide QRS complex, it is either a ventricular tachycardia (VT) or, extraordinarily rarely, a SVT with aberrant conduction or pre-excitation. In contrast to the narrow complex tachycardias, these tachycardias are usually malignant and require hospitalization for further treatment.

The differential diagnosis between a SVT with aberration and VT has fascinated physicians for years. Several algorithms have been proposed to differentiate between these arrhythmias based on the 12-lead ECG pattern [1–3]. It seems as though physicians try to demonstrate their diagnostic acumen by diagnosing SVT with aberration in patients who present with wide complex tachycardias. The reality, however, is that in attempting to prove their diagnostic skills, physicians who

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diagnose a wide complex tachycardia as SVT with aberration are wrong most of the time [4]. The correct diagnosis is important, because medication given for the treatment of SVT may be harmful or fatal to a patient in VT [5–7].

In addition to characterizing the tachycardia by its QRS morphology, other important clinical and ECG information can be helpful in diagnosing the arrhythmia and treating the patient. Dissociation between the atrium and ventricle is diagnostic of VT but is recognized only about 25% of tracings [4]. The converse, however, is not true, because ventriculo-atrial association is common during VT. Other morphologic features that suggest VT include a QRS width greater than 160 milliseconds, the QRS axis in the frontal plane (far left or right), concordance of the QRS complexes across the precordium, and a time from the onset of the R wave to the nadir of the S wave in any precordial lead of more than 100 milliseconds [1,2,8,9]. Clinical data are also useful for establishing the correct diagnosis. A history of a previous myocardial infarction or structural heart disease is highly suggestive that the wide complex tachycardia is VT [8,10,11]. A careful history and physical examination often will be sufficient to establish the presence or absence of structural heart disease in the patient. If this information is insufficient, a transthoracic echocardiogram will define the patient's cardiac structure and function. Lastly, hemodynamic stability during the wide complex tachycardia is often assumed to be evidence that the tachycardia is caused by SVT with aberration or pre-excitation. This assumption is clearly incorrect. Steinman and colleagues [12] demonstrated that VT is the correct diagnosis in 85% of patients who present with a wide complex tachycardia without significant hemodynamic compromise.

Box 1. Rules for the acute management of arrhythmias

If heart rhythm is fast and the patient is hemodynamically unstable, shock the patient as quickly as possible and keep shocking until normal sinus rhythm is restored.

If the heart rhythm is slow and the patient is symptomatic, pace the heart with either a transthoracic or transvenous pacemaker.

Supraventricular tachycardias

Many articles and reviews have been written about the diagnosis and management of SVT. The reader is referred to the American College of Cardiology/American Heart Association/European Society of Cardiology guidelines for a complete review [13]. This article focuses on a simplified, practical approach to diagnosing and treating these common arrhythmias.

When approaching a patient who has SVT, it is helpful to have in mind the different arrhythmias that cause narrow complex tachycardias. An easy framework on which to base a differential diagnosis is to think anatomically, beginning where the heartbeat starts at the sinus node and ending at the atrioventricular (AV) node. SVT can be caused by sinus tachycardias (appropriate, inappropriate, or reentrant), atrial tachycardias, atrial flutter, atrial fibrillation, junctional tachycardia, AV nodal reentry, or AV reciprocating tachycardia using a bypass tract. It is important to diagnose the arrhythmia mechanism, because treatment is specific for individual arrhythmias. For example, appropriate sinus tachycardia is treated by identifying the cause for the arrhythmia, such as fever, hypovolemia, or hyperadrenergic states rather than simply slowing the heart rhythm with beta-blockers.

Patients who have SVT usually present with palpitations. They notice a rapid heart rate and seek medical attention if it persists. They usually give a history of paroxysmal symptoms that have been present for years. Initial evaluation in the emergency department includes an ECG, history, and complete physical examination. If symptoms persist, a Valsalva maneuver or carotid sinus massage, provided there is no carotid disease, might be useful in either terminating the arrhythmia or causing the loss of ventricular capture for a single beat to allow diagnosis of the arrhythmia mechanism [14]. If the arrhythmia is not terminated by these bedside maneuvers, adenosine (6 or 12 mg intravenously) will usually cause AV nodal block, either terminating the arrhythmia or allowing the visualization of the underlying mechanism. This drug has a rapid onset and short duration of action with a half-life of less than 10 seconds. It is important to administer the adenosine rapidly and to follow the infusion with a flush of saline to affect a result. The usual side effects of flushing, dyspnea, and chest discomfort are usually short lived [15,16]. Severe complications are extraordinarily rare, but case reports of prolonged asystole,

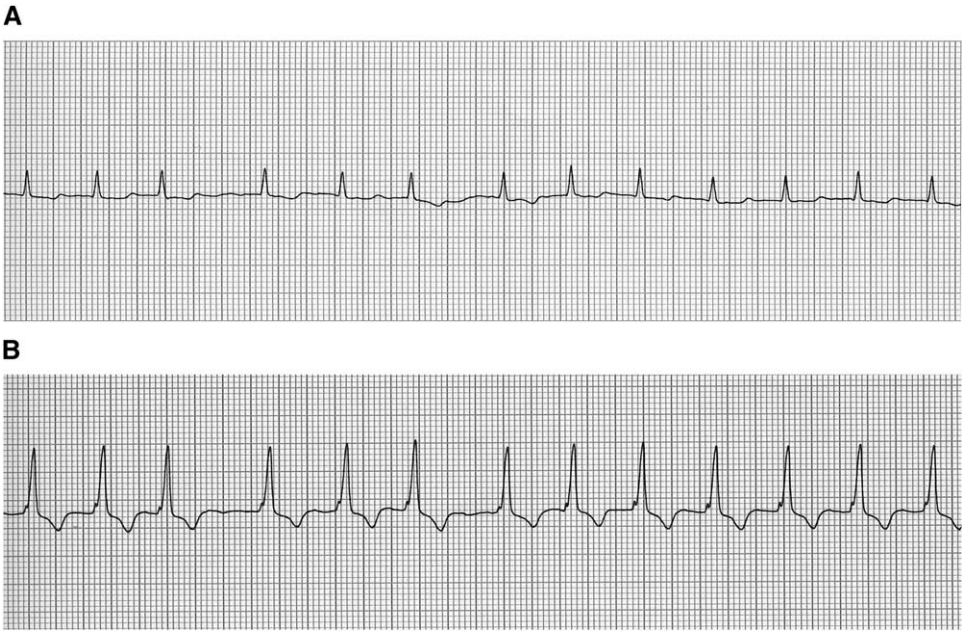

Fig. 1. Rhythm strips from a patient. (*A*) A rhythm strip from lead 2, which has a QRS duration of 70 milliseconds. (*B*) The simultaneous rhythm strip from lead V1. The QRS duration in this strip is 140 milliseconds.

VT and fibrillation, and bronchospasm have been published. Resuscitation capability must be immediately available. There are additional considerations to remember about adenosine. The methylxanthines, such as theophylline and caffeine, are competitive antagonists. Dipyridamole may potentiate adenosine's effects. The denervated, transplanted heart is supersensitive to adenosine [17].

Other intravenous AV nodal blocking agents such as beta-blockers or calcium-channel blockers (verapamil or diltiazem) have been used to treat SVT acutely. These drugs, however, have longer half-lives than adenosine and cause other problems such as hypotension (verapamil) or severe bradycardia (beta-blockers). Because of these potential complications, they have been largely replaced by adenosine [18].

Most SVTs are benign and can be treated in the emergency department, and the patient can be discharged home. The exception is atrial fibrillation in a patient who has Wolff-Parkinson-White syndrome who has rapid conduction in the bypass tract from the atrium to the ventricle. This arrhythmia presents as an irregularly irregular arrhythmia with QRS complexes of variable duration (Fig. 2). This arrhythmia may present with hemodynamic compromise if the ventricular rate is sufficiently fast. As mentioned earlier, any SVT with hemodynamic compromise should be treated by electrical cardioversion. This presentation is also the one case when calcium-channel blocking drugs and adenosine should be avoided. These drugs often increase conduction through the bypass tract while decreasing it through the AV node, thus increasing the ventricular response rate and causing hemodynamic compromise [5]. The ideal drug for this condition is intravenous procainamide, which slows the ventricular rate by blocking conduction through the bypass tract.

Most SVTs are caused by a reentrant circuit involving the AV node or use the AV node to conduct to the ventricle. Therefore, long-term treatment is directed at decreasing conduction through the AV node. Beta-blockers and calcium-channel blockers are particularly useful for treating these arrhythmias. Once patients have been stabilized in the emergency department, they can usually be discharged on either a long-acting beta-blocker or calcium-channel blocker to complete their evaluation in the outpatient clinic. If patients have recurrent episodes despite these medications, a referral to the electrophysiologist for consideration of an ablation is warranted. Most SVTs can be cured with an ablation [18].

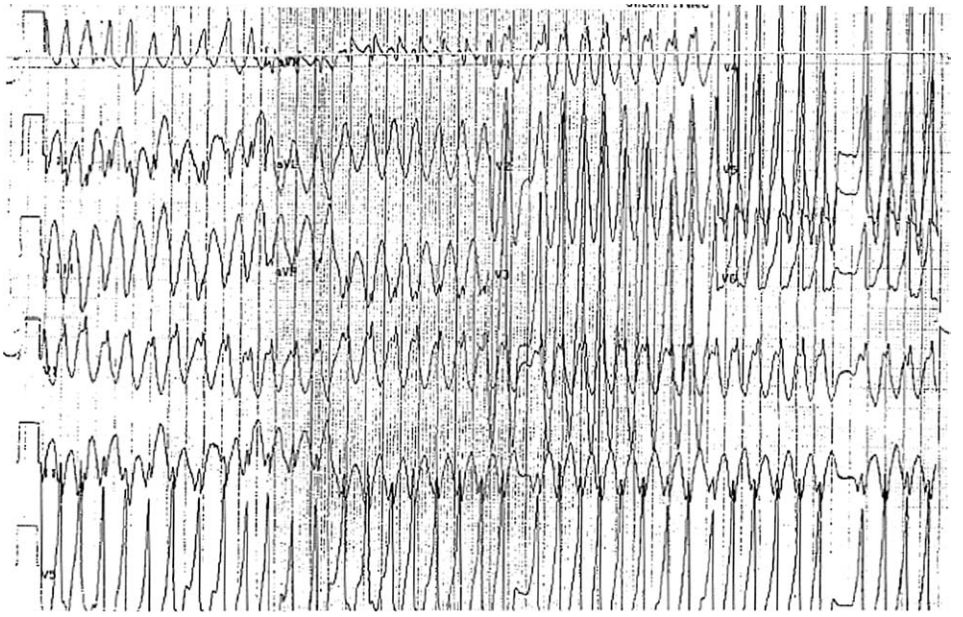

Fig. 2. Pre-excited atrial fibrillation.

Atrial flutter and atrial fibrillation

Atrial flutter and atrial fibrillation represent a special challenge to the emergency department physician. Both arrhythmias may present as incidental findings in a patient who presents to the emergency department for other complaints. These arrhythmias are common in the general population, with the incidence increasing with advancing age [19,20]. In the emergency department, the same rules apply as for other supraventricular arrhythmias. Cardioversion is the treatment of choice if the patient is hemodynamically compromised. If the patient is hemodynamically stable, control the ventricular response rate with AV nodal blocking medications such as beta-blockers and calcium-channel blockers. Digoxin is a third-line drug, which may be useful in patients who have left ventricular dysfunction. The AV nodal blocking properties of digoxin are caused by an increase in vagal tone and thus are not useful for a physically active patient. It is important to begin anticoagulation in patients who present with atrial fibrillation or atrial flutter because these arrhythmias predispose the patient to thromboembolic events [21–25]. Anticoagulation recommendations are well summarized and have been recently updated [26]. Recently, a consensus agreement has been proposed on a treatment scheme for these arrhythmias in both acute and chronic settings [27].

Of special consideration is the patient who presents with atrial fibrillation and an acute myocardial infarction. Although the diagnosis is commonly considered, few patients who present with new-onset atrial fibrillation are having an acute myocardial infarction. In fact, when atrial fibrillation occurs during an acute myocardial infarction, it is an indication of significant myocardial damage and pulmonary congestion. The treatment of choice is diuresis and hemodynamic support rather than rate control with AV nodal blocking agents [28,29].

Ibutilide, an intravenous class III anti-arrhythmic, has been used successfully for conversion of atrial fibrillation and atrial flutter, particularly when they are of short duration, and can be considered for acute cardioversion in the emergency department. Because of its potential side effects, it has been recommended that ibutilide be limited to patients who have an ejection fraction greater than 30%. There is an increased risk of torsades de pointes with lower ejection fractions, which may be minimized by pretreatment with magnesium [30–32].

Ventricular tachycardia

Again, the management of VT should not be frightening to the emergency department

physician. Most patients who present with these arrhythmias have underlying heart disease. If the patient is hemodynamically unstable, perform an electrical cardioversion as quickly as possible. The rate of survival depends on the speed of cardioversion [33]. Support the patient hemodynamically following advanced cardiac life support protocols. In the acute phase of treatment, the intravenous drug of choice is amiodarone. When given to patients in the ambulance, amiodarone improves survival to admission to the hospital when compared with lidocaine [34]. Amiodarone should be administered as a 150-mg bolus, followed by an infusion at 1 mg/minute for 6 hours, decreasing to 0.5 mg/minute subsequently. One complication that is specific to intravenous amiodarone is hypotension, secondary to the detergent, polysorbate 80, used to dissolve the drug [35]. Other medications that can be used if more readily available are lidocaine and procainamide, the latter being useful when atrial fibrillation with pre-excitation is suspected. Polymorphic VT or torsades de pointes responds to intravenous magnesium (1–5 g), and VT storm often responds to intravenous beta-blockers and often with amiodarone supplementation [36–39]. Once stabilized, all patients who have VT should be admitted to the hospital for further evaluation and treatment.

Cardioversion

Cardioversion refers to the process of terminating arrhythmias either by pharmacologic agents, direct current, or a combination of both. Cardioversion is used to restore normal sinus rhythm to relieve symptoms and reduce the heart rate. Stroke risk must be assessed before every cardioversion. Hemodynamic stability provides opportunity to choose therapies, drug versus electrical. Pharmacologic cardioversion has the advantage of not requiring sedation, which may a particular concern in patients who have severe respiratory disease. Drugs commonly used for cardioversion are those already mentioned for the treatment of VT and SVT. Direct current cardioversion, however, is the most efficacious means for restoring normal rhythm. In the past, investigative efforts directed toward the optimal vector, pad/paddle size, and energy requirement have been the subject of much discussion. The one substantial improvement has been the introduction of biphasic shocks. There continues to be controversy over the ideal waveform, but from a practical perspective, the current, commercially available external defibrillators are all highly effective. Skin erythema is common. Tachyarrhythmias, bradyarrhythmias, and pulmonary edema have been rarely reported complications. If cardioversion is not successful, repeating with increased power, changing the vector, use of paddles with manual compression, or some combination often is successful.

Syncope

Syncope is the sudden, transient loss of consciousness and postural tone with spontaneous recovery, most often caused by generalized cerebral hypoperfusion [40]. Loss of consciousness must involve either both cerebral hemispheres or the reticular activating system of the brainstem. It is the result of decrease in cardiac output or loss of vascular tone or both.

The differential diagnoses of altered mental status include syncope, seizure, obtundation, delirium, dementia, coma, change in postural tone, and drop attacks. It is not regional hypoperfusion, which may present as transient ischemic attack or stroke. It may be confused with dizziness, vertigo, disequilibrium, or lightheadedness. Syncope is rarely caused by acute coronary syndrome or myocardial infarction with the possible exception of the very elderly or when an arrhythmia occurs [41,42]. Syncope is not caused by hypoglycemia.

Causes for cardiovascular syncope include (1) circulatory obstruction such as that occurring with critical aortic stenosis, hypertrophic obstructive cardiomyopathy, and pulmonary embolism; (2) arrhythmia such as VT, atrial flutter with 1:1 conduction, or occasionally bradycardia; (3) orthostatic intolerance associated with the autonomic nervous system including neurocardiogenic syncope, postural orthostatic tachycardia syndrome, and primary and secondary autonomic failure [43].

The patient's history provides the best means of elucidating the origin of a syncopal spell [44]. A description of a prodrome such as palpitations, blurred vision, nausea, warmth, diaphoresis, or lightheadedness is important, as are postevent symptoms such as nausea, warmth, diaphoresis, and fatigue. Activity, position, recurrence, family history of syncope, sudden death, unexplained accidents including drowning and motor vehicle accidents, and epilepsy are all relevant [44]. The physical examination should be directed to orthostatic vital signs, murmurs, prominent P2, gallops, and other evidence of compromised ventricular function. The 12-lead ECG should be examined

for evidence of heart block, ectopy, prior myocardial infarction, and the QT interval. Very rarely, classic findings of a delta wave, epsilon wave, or Brugada syndrome will suggest a cause. The selective use of cardiac diagnostic tests should be considered. These include echocardiography, tilt table testing, ambulatory monitoring, or electrophysiologic studies. Without specific neurologic findings, extensive neurologic testing has a low yield and generally is not warranted. Another pressing question is when to hospitalize a patient who has syncope. The Task Force on Syncope from the European Society of Cardiology recommended hospitalization of those with (1) known or suspected significant heart disease, (2) ECG abnormalities suspected of arrhythmic syncope, (3) syncope during exercise, (4) syncope causing severe injury, and (5) a family history of sudden death and recommends that occasionally others be admitted to the hospital for further evaluation [45].

The outcome of patients who have syncope is largely dependent on the cause. Those with a cardiac cause fare the worst. Patients who have a noncardiac cause have an intermediate risk, and those with neurocardiogenic syncope have a mortality rate of nearly nil. Ambulatory outcome data from the Framingham study were collected on 2336 men and 2873 women whose ages were between 30 and 62 years at entry. A 26-year surveillance revealed that 3.0% of men and 3.5% of women had at least one syncopal episode In 79% of men and 88% of women who experienced isolated syncope, there was no excess risk of stroke or myocardial infarction, and there was no excess all-cause or cardiovascular mortality [46]. Quinn and colleagues [47] described 684 visits to San Francisco emergency departments for syncope. Predictors of serious outcome (defined as death, myocardial infarction, arrhythmia, pulmonary embolism, stroke, subarachnoid hemorrhage, or repeat emergency department visit with hospitalization at day 7) include abnormal ECG, dyspnea, hematocrit below 30%, systolic blood pressure below 90 mm Hg, and history of congestive heart failure.

In certain populations, syncope can also be harbinger of increased mortality. Kapoor and colleagues [48] reported on 204 patients who presented with syncope to the emergency departments, hospitals, or clinics. Causes of mortality at 1 year were 30% cardiovascular, 12% noncardiovascular, and 6.4% of unknown categories. He further reported on mortality at 24 months in a 2 × 3 matrix of elderly (60–90 years) and young (15–59 years) persons. Mortality from cardiovascular syncope was 38.1% and 32.5%, respectively. Noncardiovascular syncope had a mortality of 21.6% and 4.7%, respectively, and unknown causes had 20.4% and 2.5%, respectively [49]. Using a risk score which assigned one point each for age above 65 years, history of cardiovascular disease, syncope without prodrome, and abnormal ECG, Colivicchi and colleagues [50] reported that the mortality of 270 patients who presented to the emergency department was 0.8%, 20%, 35%, and 57% for patients assigned 1 through 4 points, respectively [50].

Implanted cardiac devices

Besides patients who have active arrhythmias, the other large group that presents to the emergency department is those who have implanted cardiac devices. They may have unique complaints related to their hardware. Three types of devices are discussed here: pacemaker, implantable cardioverter defibrillators (ICDs), and insertable loop recorders. Devices are most commonly found in the left pectoral fossa. The right pectoral fossa and abdomen are alternate sites. Very rarely inframammary or inguinal placement is used. At the time of implantation, the device manufacturer sends the patient an identification card that provides basic information such as manufacturer, model numbers, serial numbers, and telephone numbers for contact. All patients are strongly encouraged to carry this card with them at all times.

Device-related implant problems that might appear in the acute setting include pocket hematoma, wound dehiscence, upper extremity deep venous thrombosis, pneumothorax, and lead dislodgement. Chronic device complications include battery exhaustion and catastrophic, random component failure. Time-independent problems include oversensing which results in underpacing or inappropriate shocks, undersensing producing overpacing and failure of ICD therapy, infection, and erosion.

Chief complaints that are possibly device related include syncope, near syncope, palpitations, and infections. Of course there are a plethora of chief complaints that are not device related but still impact care. For instance, patient who has an implanted cardiac device cannot undergo a MRI scan at present. CT scans are safe, but the metal may produce some scatter in chest compromising image quality. If emergent surgical procedures are contemplated, the device may require

reprogramming or magnet placement to use electrocautery safely. An American Heart Association Scientific Advisory was published in 2004 on ICD management for the non-electrophysiologist [51].

Permanent pacemakers at a minimum pace at least one chamber although typically they have many more functions. Pacemakers are conventionally described by the NBG code. (The acronym NBG stands for the North American Society for Pacing and Electrophysiology [NASPE] and British Pacing and Physiology Group [BPEG] Generic.) An abbreviated description of the code identifies the chamber(s) paced (first letter), chamber(s) sensed (second letter), pacemaker response (third letter), and rate responsiveness (fourth letter). A is for atrium; V is ventricle; D is dual chamber or dual function that is both triggered and inhibited; I equals inhibited, and R is for rate responsiveness. Thus a VVIR pacemaker paces the ventricle and senses in the ventricle; its output is inhibited by a sensed event, and it is rate responsive, that is, the motion sensor is active. The most common designations seen are DDD, DDDR, VVI, and VVIR.

When a patient who has a pacemaker presents to the emergency department, the first concern is whether pacemaker malfunction could account for the presenting symptoms. If the patient has the device checked regularly, and the ECG does not demonstrate a problem, the likelihood of a pacemaker malfunction is quite small. The reliability of pacemakers is such that, if the above conditions are met, the device is almost certainly functioning as programmed. It is certainly reasonable to interrogate the pacemaker, but one must prepare for alternative causes.

All ICDs have defibrillation capability (shock therapy) as well as ventricular anti-bradycardia pacing. They often have additional features such as dual-chamber pacing, biventricular pacing, and atrial defibrillation capabilities as well a variety of diagnostic recording capabilities. If a patient presents to the emergency department with a cardiac arrest, advanced cardiac life support protocols should be followed as if the patient does not have a defibrillator. If the device has not terminated a ventricular arrhythmia, external defibrillation should be performed. The only caveat is to avoid placing the pads or paddles directly over the pulse generator. Contact with the patient as an internal shock is delivered by the ICD will not cause harm the emergency department personnel.

The most disconcerting situation for both patient and physician is that of a patient who presents with multiple ICD shocks. The physician needs to assess whether the shocks were appropriate or spurious. Repetitive, appropriate shocks indicate electrical storm. One must search for a cause such as active ischemia, electrolyte abnormalities, or deterioration of left ventricular function. This determination must be concurrent with management as described above for ventricular storm. Spurious shocks could occur as the result of rapid, nonmalignant rhythms such as sinus tachycardia or rapid atrial fibrillation or a malfunctioning device (noise on the rate sensing lead). In this situation, it is important to deactivate or inhibit ICD therapy. Deactivation is done with a magnet placed over the generator or by reprogramming the device. In addition to the cardiac issues, the psychologic stress of multiple shocks should be addressed.

The insertable loop recorder is a leadless device implanted subcutaneously in the left pectoral fossa. The purpose of this device is to record spontaneous arrhythmias and triggered events. It has service life in excess of 1 year and no therapeutic capability. Except for recognizing that it is neither a pacemaker nor a defibrillator, it is not discussed here.

Magnets and devices

Each manufacturer produces a doughnut-shaped magnet for use with devices. Pacemakers and defibrillators have different responses to a magnetic field. The typical response of a pacemaker is asynchronous pacing, that is, VOO or DOO. It does not sense and does not track or inhibit; it simply paces. Defibrillators' anti-tachycardia therapies (shocks and anti-tachycardia pacing) are inhibited by a magnet. The defibrillator's anti-bradycardia pacing function is not affected by a magnet.

Making the situation more confusing, some models of pacemakers and defibrillators can have their typical magnet responses disabled, in which case the effects described above will not occur. It is uncommon to program a device with the magnet function disabled. Recently, however, one manufacturer has recommended that the magnet function on several models of defibrillator be disabled as a means for dealing with a manufacturing defect.

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