Emergencies related to implantable cardioverter-defibrillators

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Implantable cardioverter-defibrillators (ICDs) have become the dominant therapeutic modality for patients with life-threatening ventricular arrhythmias. ICDs are implanted using techniques similar to standard pacemaker implantation. They not only provide high-energy shocks for ventricular fibrillation and rapid ventricular tachycardia, but also provide antitachycardia pacing for monomorphic ventricular tachycardia and antibradycardia pacing. Devices incorporating an atrial lead allow dual-chamber pacing and better discrimination between ventricular and supraventricular tachyarrhythmias. Intensivists are increasingly likely to encounter patients with ICDs. Electrosurgery can be safely performed in ICD patients as long as the device is deactivated before the procedure and reactivated and reassessed immediately afterward. Prompt and skilled intervention can prove to be life-saving in patients presenting with ICD-related emergencies, including lack of response to ventricular tachyarrhythmias, pacing failure, and multiple shocks. Recognition and treatment of tachyarrhythmia can be temporarily disabled by placing a magnet on top of an ICD. The presence of an ICD should not deter standard resuscitation techniques. Multiple ICD discharges in a short period of time constitute a serious situation. Causes include ventricular electrical storm, inefficient defibrillation, nonsustained ventricular tachycardia, and inappropriate shocks caused by supraventricular tachyarrhythmias or oversensing of signals. ICD system infection requires hardware removal and intravenous antibiotic therapy. Deactivation of an ICD with the consent of the patient or relatives is reasonable and ethical in terminally ill patients. (Crit Care Med 2000; 28[Suppl.]:N174–N180)

KEY WORDS: implantable cardioverter-defibrillator; ventricular tachycardia; ventricular fibrillation; ventricular electrical storm; oversensing; atrial fibrillation

The implantable cardioverter-defibrillator (ICD) has become the dominant therapeutic modality for patients with life-threatening ventricular arrhythmias. ICDs are remarkably effective in preventing sudden cardiac death (1), but their use is not free from complications (2). Most ICD recipients are elderly, suffer from chronic cardiovascular disease, require multiple medications, and experience a substantial number of nonarrhythmic problems. Intensivists are increasingly likely to encounter patients with ICDs. Prompt and skilled intervention can prove to be life-saving in patients presenting with ICD-related emergencies (3) (Table 1).

Current Status of ICD Technology

ICDs are multiprogrammable devices capable of delivering high-energy defibrillation shocks, antitachycardia pacing, or low-energy (cardioversion) shocks for ventricular tachycardia, and pacing for bradyarrhythmias (4). ICDs are implanted transvenously via the subclavian or cephalic veins by using techniques similar to those for standard pacemakers. The ICD generator (volume, <60 mL) contains the electronic circuitry, power source, and memory. A microprocessor coordinates the interplay among the various subsections of the system. Programmable and diagnostic data are stored in volatile memory. Battery longevity for current units varies from 5 to 9 yrs, depending mainly on the frequency of shock delivery. To reliably sense low-amplitude signals during ventricular fibrillation and to avoid sensing of T waves or extracardiac noise, the sensing circuit automatically adjusts gain. Devices can be interrogated and reprogrammed noninvasively with proprietary programmers and software, specific for each manufacturer.

A right ventricular lead is used for sensing and pacing. Shocks are delivered between a ventricular coil in this lead and the case of the generator. Many leads include a second shocking coil at the level of the superior vena cava to improve defibrillation efficiency. Dual-chamber ICDs require an additional right atrial lead. Dual-chamber ICDs are particularly useful for patients who require frequent pacing or who have atrial tachyarrhythmias. Leads are attached to the endocardium either passively with tines or actively via a screw mechanism. The generator is implanted in a subcutaneous or submuscular pocket in the pectoral area. Left-sided implantation is preferable because of the smoother venous route to the heart and a more favorable shocking vector. Adequate pacing and sensing thresholds, as well as reliable detection and termination of ventricular fibrillation, are established intraoperatively. The shocking lead configuration is optimized to achieve a safety margin of ≥ 10 J between the maximum output of the ICD (26–38 J according to the model) and the energy required for consistent defibrillation. The operative mortality rate is $<1\%$ (5). Acute complications include pneumothorax, hemothorax, air embolism, cardiac perforation, pericardial tamponade, lead dislodgment, pocket hematoma, and venous thrombosis (6).

The goal of ICD therapy is prevention of sudden death or syncope caused by ventricular tachyarrhythmias with infrequent delivery of high-energy shocks. For most patients, an optimal "electrical pre-

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scription" can be achieved by careful device programming. ICDs recognize ventricular tachyarrhythmias based on programmable rate and duration criteria. As long as the ventricular depolarization signal is correctly sensed, tachycardias faster than the programmed cutoff rate will be detected. However, sensitivity is achieved at the expense of reduced specificity. Failure to discriminate between supraventricular and ventricular tachyarrhythmias may result in spurious ICD interventions (7). Programming additional diagnostic "enhancements" improves the accuracy of arrhythmia diagnosis (8). The "sudden onset" criterion helps discriminate between sinus tachycardia and ventricular tachycardia, whereas "rate stability" criteria are useful to differentiate rapid atrial fibrillation from ventricular tachycardia. Many models can detect the differences in intracardiac electrogram morphology between ventricular and supraventricular beats. In dual-chamber ICDs, discrimination between ventricular and supraventricular arrhythmias is improved by assessing the atrioventricular relationship.

Antitachycardia pacing is most useful for terminating monomorphic ventricular tachycardia with rates <200 beats/min (9), achieving success rates of ~80% with a low risk of tachycardia acceleration. Antitachycardia pacing is preferable to low-energy cardioversion because of superior patient tolerance, lower energy consumption, and minimal risk of induction of atrial fibrillation (10). Ventricular fibrillation is treated with high-energy (>10 J) shocks. Programming of the first-shock energy based on intraoperative testing results in a very high rate of successful conversion of spontaneous arrhythmias (11). If the first shock fails, the device delivers "back-up" shocks. ICD interventions can at times induce or aggravate ventricular arrhythmias (12). In contrast to its drug-induced counterpart, such proarrhythmia is almost never fatal, but increases the morbidity of this therapeutic modality.

Table 1. Emergencies in patients with implantable cardioverter-defibrillators

Diagnostic and therapeutic procedures that could result in electromagnetic interference
Pacing malfunction
Lack of intervention during ventricular tachyarrhythmias
Cardiopulmonary arrest and resuscitation
Multiple shocks
Suspected infection
Terminal care issues

Emergency Identification and Deactivation of ICDs

Rapid identification of the ICD model may be important when it is necessary to deactivate or reprogram the device. Patients should carry identification cards including information regarding manufacturer, generator model, lead system, therapy options, and a 24-hr emergency contact telephone number. If the model is unknown, an overpenetrated radiograph of the generator will show a radiopaque marker allowing identification.

When the specific programmer is not available, a magnet placed on top of all ICD models will temporarily disable tachyarrhythmia intervention. The magnetic field closes a reed switch in the generator circuit, triggering slightly different responses among models (Table 2). In general, as long as the magnet remains close to the generator, tachyarrhythmia recognition and treatment is disabled and there is no effect on pacing functions. It is necessary to secure the magnet to the generator site with tape to maintain the inactivated status.

Diagnostic and Therapeutic Maneuvers in Patients with ICDs

Antibiotic prophylaxis is not recommended for ICD patients during procedures that may cause transient bacteremia (13). Benzodiazepines are useful when mild to moderate sedation is needed. Benzodiazepines have no proarrhythmic effects and may help ameliorate ventricular arrhythmias facilitated by high sympathetic tone (14). Methohexital is a safe short-acting anesthetic agent in patients with ICDs (15). Tricyclic antidepressants and neuroleptics can exacerbate cardiac arrhythmias. They should be used with caution, especially in patients already receiving antiarrhythmic drugs with similar electrophysiologic properties, because additive effects can result in toxicity.

Many diagnostic and therapeutic procedures used in critically ill patients involve sources of electromagnetic radiation that can interfere with ICD function. Careful planning is required to assure patient safety. Possible adverse effects include inhibition of bradycardia pacing, inadvertent delivery of shocks or antitachycardia pacing, reversion to temporary asynchronous pacing, resetting of programmed parameters, and, less commonly, damage to the generator, the tis-

Table 2. Magnet responses of current implantable cardioverter-defibrillators (ICDs)

Manufacturer	Response
Guidant/CPI	Transient inhibition of tachycardia therapy. R-wave synchronous beeping tones if device is active. Continuous tone if device is inactive. (These functions are programmable and are nominally enabled.) No effect on pacing. If the "Change Tachy Mode with Magnet" function is enabled, magnet application for >30 secs deactivates the device (the ICD remains inactive when the magnet is removed). This function is nominally disabled.
Ela Medical	
Dual-chamber (Defender)	Transient inhibition of tachycardia detection and therapy. DDD pacing at 96 beats/min (full battery), rate decreases as battery becomes depleted.
Single-chamber (Lyra)	Transient inhibition of tachycardia detection and therapy. No effect on pacing.
Medtronic	Transient inhibition of tachycardia detection and therapy. No effect on pacing. If patient alert, function(s) enabled; a short-lasting continuous tone signals normal function; a dual (high/low) tone indicates that a high urgency condition has occurred; and an intermittent (on/off) tone indicates that a low urgency condition has been met. No tones are emitted if the patient-alert function(s) are disabled (nominal settings).
Ventritex	Transient inhibition of tachycardia detection and therapy. (This function is programmable and is nominally enabled.) No effect on pacing.

sues interfacing with the electrodes, or both.

Surgical or endoscopic procedures requiring electrocautery can be performed safely in ICD patients (16, 17) (Table 3). Because electrocautery current can be falsely detected as rapid cardiac rhythms (and trigger inappropriate shocks), personnel should be available to deactivate tachyarrhythmia detection before the procedure and reactivate it immediately at the procedure's conclusion. Positioning of the electrocautery ground electrode to minimize current flow through the ICD electrode system reduces the risk of permanent damage. The patient's cardiac rhythm should be continuously monitored and severe arrhythmias treated as necessary. The device should be thoroughly checked after the procedure to confirm satisfactory operation. Electromagnetic interference is more problematic in patients who depend on the ICD for pacing (18). Because of higher sensitivity (required for the ventricular fibrillation detection) and lack of programmable asynchronous pacing modes, ICDs are more prone than standard pacemakers to respond to electromagnetic interference with catastrophic inhibition of pacing. In these patients, procedures involving electromagnetic sources may require a temporary pacing electrode to prevent severe bradycardia or asystole.

An ICD constitutes a strong contraindication to magnetic resonance imaging (MRI). Exposure to a static magnetic field occurs on entry into the MRI suite. In most ICD models, this results in activation of the reed switch and suspension of tachyarrhythmia detection. However, if this static field is perpendicular to the reed switch axis, the device may remain activated. The static magnetic field can also impart rotational and translational forces to a generator containing sufficient ferromagnetic material. Such forces could, in theory, cause physical pain or even damage the generator. Pulsed radiofrequency fields during imaging can induce interference in the device circuitry and result in spurious tachyarrhythmia detection, pacing inhibition, or rapid pacing.

The risk of serious adverse events and the existence of alternative imaging modalities are obstacles to performance of large-scale systematic studies of MRI in ICD patients. MRI of the head or an extremity (19) appear safer than MRI of the thorax or abdomen. MRI should be evaluated on a case-by-case basis, weighing the perceived risks and benefits. The patient must be monitored by using an electrocardiogram, pulse oxymetry, and direct voice contact during the scan. A thorough plan for the management of arrhythmic emergencies should be devised before the procedure. ICD patients who are pacemaker-dependent should not undergo MRI. In nondependent patients, tachyarrhythmia detection and bradycardia pacing should be disabled. The latter requires programming to the nonpacing OOO mode (when available) or to subthreshold pacing output(s). However, "runaway" (very rapid) pacing synchronized with the radiofrequency pulses may still occur (20).

Peripheral nerve stimulators are used to assess the extent of neuromuscular blockade in the intensive care unit. Facial nerve stimulation with the standard train-of-four mode at 2 Hz can inhibit unipolar pacemakers (21). Although not yet reported, similar pacing inhibition could occur with ICDs. The 2-Hz frequency of stimulation (120 beats/min) is unlikely to trigger ICD interventions unless the detection rate has been programmed very low (in patients with slow ventricular tachycardia). Transcutaneous electrical nerve stimulation should be avoided in ICD patients because it can trigger spurious shocks (18).

Table 3. Management of patients with implantable cardioverter-defibrillators (ICDs) undergoing electrosurgery

Preoperative
Ask the surgeon to consider alternative tools (knife and ligatures, ultrasonic or laser scalpel)
Check device (programming, telemetry, thresholds, battery status)
In the operating room
Disable ICD tachyarrhythmia therapies
Deactivate rate-responsive pacing features
If the patient is pacemaker-dependent:
Decrease the maximum sensitivity
Program the noise reversion mode to asynchronous (DOO, VOO) (not available in Medtronic devices)
Preapply external transthoracic pacing system
With Guidant devices, consider programming a temporary asynchronous mode (DOO, VOO); this requires continuous application of the programmer wand throughout the surgery
Consider insertion of separate transvenous pacing wire
Monitor peripheral pulse, oximeter, or intraarterial blood pressure (ECG is obscured by cautery artifact)
Position the ground plate to keep the active-to-dispersive current pathways as far as possible and perpendicular from the pulse generator-to-electrode pathway; the current should flow away from the pulse generator
Use bipolar cautery whenever possible
Limit cutting current to short bursts interrupted by pauses of at least 10 secs
Use the lowest effective cutting or coagulation power output
Do not use cautery near device
Postoperative
Reactivate tachyarrhythmia therapy as soon as possible
Check device (programming, telemetry, thresholds, battery status) and reprogram, if necessary
Replace generator if circuit damage is documented
Replace lead(s) if pacing threshold is too high

ECG, electrocardiogram.

Pacing Malfunction

The advent of ICDs with dual-chamber rate-responsive pacing capabilities has nearly eliminated the need for separate implanted devices in pacemaker-dependent patients, but has highlighted the risks of ICD pacing malfunction (22). The causes and management of pacing malfunction in ICDs and pacemakers are similar. Noninvasive interrogation and troubleshooting via the programmer generally identifies the problem. Early after implant, failure to capture usually results from lead dislodgment or perforation. Lead failure becomes more common late during follow-up. Lead migration is easily identifiable on chest radiography, but lead fracture rarely is (23). Failure to pace caused by premature battery depletion, random component failure, or software error is rare, but can occur any time after implant. "Make-and-break" signals caused by lead fracture, faulty connections, or intracardiac or extracardiac interference usually produce pacing inhibition accompanied by spurious shocks (see below). Temporary pacing should be instituted promptly in patients with symp-

tomatic pauses or bradycardia. Treatment of bradycardia with isoproterenol should be avoided because it can promote ventricular tachyarrhythmias that the malfunctioning ICD may not be able to terminate. When needed, a temporary pacing wire should be inserted under fluoroscopic guidance to avoid entanglement or dislodgment of the ICD lead(s). This is especially important if the leads were recently implanted. External transthoracic pacing is easy to accomplish, but is poorly tolerated in the unsedated patient. The ICD should be deactivated before initiating temporary pacing because double- or triple-counting of pacing artifacts and QRS complexes can also trigger spurious shocks. Magnet application does not terminate pacemaker-mediated (endless loop) tachycardia in dual-chamber ICDs because asynchronous pacing does not occur. Tachycardia termination requires reprogramming or pharmacologic blockade of ventriculoatrial conduction.

Ventricular Tachyarrhythmias Without ICD Intervention

Patients can present with sustained ventricular tachycardia or ventricular fibrillation without ICD intervention. Failure of arrhythmia detection (caused by malfunction or rate below the programmed cutoff) or exhaustion of programmed therapies are the most common causes (Table 4). Management of these emergencies differs according to the hemodynamic impact of the ongoing tachyarrhythmia.

The ICD patient in cardiac arrest should receive standard cardiopulmonary resuscitation (including prompt external defibrillation) (24). In patients with older-generation thoracotomy systems, epicardial patches may increase energy requirements for external defibrillation. Using the defibrillation paddles or patches in an anteroposterior configuration can circumvent energy shunting and shielding (25). Transvenous ICD systems do not interfere with external defibrillation, but direct application of the defibrillation paddles to a pectoral generator should be avoided. "Committed" (after arrhythmia termination) internal or external shocks can occur when using an automatic external defibrillator in an ICD patient (26). Persons administering cardiopulmonary resuscitation may feel a harmless, weak electric shock on the patient's body surface from an ICD discharge. Shocks will not damage external monitoring equipment. It is advisable (when possible) to deactivate the device, as supraventricular tachycardias are common during cardiopulmonary resuscitation and can trigger ICD discharges that may reinduce ventricular tachycardia or ventricular fibrillation (12). The ICD should be deactivated if resuscitative efforts are unsuccessful to avoid inadvertent shocks to providers of postmortem examination or preparation (27).

ICD patients can also present with monomorphic ventricular tachycardia without hemodynamic compromise. Nonrecognition most often occurs because the ventricular tachycardia is below the programmed detection rate. Antiarrhythmic drug therapy (initiated for ventricular or supraventricular tachyarrhythmias) is a frequent cause of slowing of ventricular tachycardia rates. A 12-lead electrocardiogram should always be obtained. It can help to confirm the diagnosis and guide subsequent attempts at catheter ablation. In the truly stable patient, every effort should be made to interrogate the ICD to elucidate the reason for the lack of intervention before attempting other therapies. Simple reprogramming is often all that is needed to allow an ICD response. The programmer also allows commanded (manual) delivery of antitachycardia pacing or shocks. Sedation should always be considered before reprogramming maneuvers that could result in shock delivery (28). Pharmacologic conversion can be attempted if the programmer is not readily available. Lidocaine is largely ineffective. Intravenous procainamide is more effective, but can result in hypotension when administered rapidly (29). Triggering the ICD (by rapid chest wall stimulation with a temporary pacemaker) is not recommended. The resulting shock will not be synchronized to the QRS and could induce ventricular fibrillation. It is crucial to be ready for external DC cardioversion at the earliest sign of hemodynamic deterioration.

External countershocks may (but rarely) damage the ICD system. The generator should be interrogated to confirm that the programmed parameters have not been reset. Pacing and sensing thresholds should be reassessed post-shock. Subsequent testing of the system against an induced arrhythmia may be required to verify proper shock output circuitry function.

Multiple Shocks

Multiple ICD shocks in a short period of time (≥3 discharges in ≤24 hrs) constitute a medical emergency. They may result from recurrent ventricular arrhythmias ("ventricular electrical storm"), supraventricular arrhythmias, or ICD system malfunction. Multiple shocks produce profound psychological morbidity. Most patients describe the experience as very unpleasant. Many become anxious and agitated (30). When multiple shocks result from ICD system failure, rapid identification of the problem could be life-saving. Shocks result in substantial battery consumption. An ICD designed to last years can become nearly depleted within hours by incessant discharges.

The causes of multiple ICD shocks are myriad. Accurate diagnosis is needed for correct management (31) (Table 5). The intensivist should initiate acute treatment, but prompt consultation with the cardiac electrophysiologist is generally required for definitive therapy. The ICD may be firing appropriately to terminate recurrent ventricular tachyarrhythmias. Alternatively, shocks may occur in series (maximum of seven, according to device model) for one episode of tachycardia that is not easily terminated. Programming inappropriately low first-shock energies may compromise defibrillation because energy requirements increase with longer arrhythmia duration (32). Class I antiarrhythmic drugs and amiodarone may increase defibrillation energy requirements (33). Migration or structural breakdown of defibrillation leads, as well as pneumothorax ipsilateral to the ICD generator (34), can also result in failed defibrillation.

Although current ICDs are generally "noncommitted" (they abort shock delivery, if persistent arrhythmia is not reconfirmed after capacitor charge), some

Table 4. Absence of implantable cardioverter-defibrillator (ICD) intervention during ventricular tachyarrhythmias

With ICD system malfunction
Battery depletion
Component failure
Undersensing:
Decreased amplitude of intracardiac electrogram
Lead malfunction
Without ICD system failure
Inappropriately high rate cutoff[a]
Failure to satisfy multiple detection criteria[a]
Completed cycle, exhaustion of therapies[a]
Cross-inhibition by separate pacemaker

[a]Common causes.

Table 5. Management of patients with multiple implantable cardioverter-defibrillator shocks

	Acute Management	Definitive Management
Appropriate shocks		
Frequently recurring VT or VF	Treatment of reversible triggering causes Sedation Intravenous antiarrhythmic drugs Consider temporary device deactivation	Optimization of oral antiarrhythmic drugs Antitachycardia pacing VT ablation
Failure to terminate VT or VF reliably	External cardioversion/defibrillation	Device reprogramming
Programming of inappropriately low shock output	Avoid class I antiarrhythmic drugs	Optimization of oral antiarrhythmic drugs
Antiarrhythmic drug effect		Surgical revision
Shocking lead dislodgment or failure		
Ipsilateral pneumothorax		
Inappropriate (spurious) shocks		
"Committed" shocks for nonsustained ventricular arrhythmias	Intravenous antiarrhythmic drugs Consider temporary device deactivation	Device reprogramming Optimization of oral antiarrhythmic drugs
Supraventricular tachyarrhythmias	Temporary device deactivation AV nodal blocking drugs Pharmacologic or electrical cardioversion	Device reprogramming Optimization of oral antiarrhythmic drugs Catheter ablation of arrhythmia substrate Catheter ablation of AV junction
Oversensing during baseline rhythm	Temporary device deactivation	Device reprogramming
Oversensing of intracardiac signals (T wave)		Surgical revision
Diaphragmatic myopotentials		Avoidance or elimination of source of interference
Sensing lead failure		
External electromagnetic interference		

VT, ventricular tachycardia; VF, ventricular fibrillation; AV, atrioventricular.

models exhibit committed behavior under certain conditions during normal operation. This can result in multiple shocks for runs of nonsustained ventricular tachycardia long enough to satisfy detection criteria. Despite routine programming of detection enhancements, up to 10% of patients receive spurious ICD shocks for supraventricular rhythms, most often atrial fibrillation or sinus tachycardia. The frequency rate is even higher when devices are left programmed at nominal settings (35). Finally, an ICD can discharge inappropriately during sinus rhythm because of oversensing. Causes of ICD discharge during normal rhythms include lead insulation or conductor breakdown, as well as oversensing of T waves, myopotentials, or external electromagnetic interference (electronic antitheft devices).

Initial evaluation of patients experiencing multiple shocks should be performed in a setting with electrocardiographic monitoring where advanced cardiac resuscitation is immediately available. The device should be interrogated as soon as possible. Analysis of retrieved data facilitates elucidation of complex arrhythmic events. Implanting institutions should provide 24-hr response systems with personnel experienced in the management of ICD patients. Delays may be unavoidable at smaller hospitals. ICDs should not be deactivated until a diagnosis is established. Frightened, anxious patients require sedation. Because most patients with ICDs have impaired left ventricular function, their hemodynamic status should be carefully assessed and heart failure treated promptly. Pulmonary artery catheters (if required) should be inserted under fluoroscopic guidance to avoid dislodging ICD leads.

Shocks preceded by chest pain suggest arrhythmias induced by myocardial ischemia. However, nonspecific chest pain often occurs after multiple shocks. Hypokalemia, hypomagnesemia, and drug-induced proarrhythmia (antiarrhythmic agents or miscellaneous drugs that prolong the QT interval, such as phenothiazines) need to be excluded. The 12-lead electrocardiogram should be examined for rhythm, signs of electrolyte abnormality, or drug toxicity (increased QRS and QT duration), and acute myocardial ischemia. Transient ST segment elevation or depression can occur immediately after an internal defibrillation shock and cannot be interpreted as definite signs of myocardial ischemia (36). Multiple consecutive shocks may release the cardiac isoenzyme, CK-MB, or troponins T and I in the absence of myocardial infarction (37).

Patients with ventricular electrical storm require admission to an intensive care unit. Prophylactic self-adhesive defibrillation pads should be applied. Potentially reversible causes should be treated vigorously (38). Reperfusion therapy should be considered for evolving myocardial infarction. Intravenous magnesium and overdrive pacing are the treatments of choice for drug-induced torsade de pointes (39). Intravenous sodium lactate or bicarbonate can be useful to antagonize proarrhythmic effects of class I antiarrhythmic drugs (40). In most patients, a specific trigger for arrhythmia clustering cannot be identified, and intravenous antiarrhythmic drugs become the mainstays of therapy. Activation of antitachycardia pacing (if not previously done) should be considered in patients with monomorphic ventricular tachycardia. An increase in adrenergic tone is often a feature of electrical storm, and β blockers, if tolerated, may be useful (41). Intravenous amiodarone appears to be the most effective agent for electrical storm (42), and may even suppress ventricular tachycardia that recurs despite chronic oral amiodarone. The detection rate often needs to be reprogrammed to a lower value to account for antiarrhythmic drug-induced slowing of ventricular tachycardia. The ICD should be deactivated during electrical storm only if ventricular tachycardia is hemodynamically stable. Deep sedation and mechanical ventilatory support can stabilize otherwise refractory patients. Patients with incessant or frequent ventricular tachycar-

dia recurrence, despite antiarrhythmic drug treatment, may benefit from catheter ablation of the arrhythmogenic focus (43). Even when electrical storm is acutely controlled, patients remain at higher risk for death during the next few months (44). More definitive therapies (cardiac transplantation) should be considered in patients with end-stage cardiomyopathy. A left ventricular assist device generally maintains an acceptable cardiac output during ventricular tachyarrhythmias. ICD deactivation is recommended in patients receiving ventricular assist devices with electronics housed in the intracorporeal device (HeartMate VE-LVAD, Thermo Cardiosystems, Woburn, MA) because they can be damaged by shocks (16).

The ICD should be deactivated if spurious firing is documented. Patients should remain under continuous electrocardiographic monitoring while their ICD is inactive. The device should be reactivated only after the condition that triggered the spurious shocks is under control. Pharmacologic control of the ventricular rate (A-V nodal blockade) is the initial maneuver when shocks are triggered by atrial tachyarrhythmias. Digoxin alone is generally insufficient. Combination therapy including calcium-channel blockers or β blockers is generally required (45). Esmolol (with a half-life of only 8 mins) is especially useful in the acute setting for patients with left ventricular dysfunction. Antiarrhythmic drug therapy should be considered to achieve conversion or suppress recurrences. Shocks delivered during spontaneous or paced rhythms at rates lower than the programmed detection rate are always caused by oversensing of signals. Treatment will require reprogramming or system revision according to the nature of the problem.

ICD System Infection

An ICD system infection is a serious complication occurring in ≤2% of patients (46) and is more common after generator replacement. Early (≤60 days) and late (>60 days) ICD infections differ in pathogenesis, presentation, microbiology, and treatment. Early infections are most commonly caused by *Staphylococci*. They may result from intraoperative wound or device contamination or via hematogenous seeding (from indwelling catheters, drains, or respiratory or urinary tract infections) in the immediate postoperative period. Late infections may result from transient bacteremia (probably very rare), pocket skin erosion, or delayed onset of infection with microorganisms acquired early after surgery. *S. epidermidis* is frequently associated with an indolent course.

Suspicion of infection is raised by local and systemic signs of inflammation. Clinical manifestations depend on the site of involvement and the time elapsed since implantation. Isolated pocket fluctuence early after surgery is not a specific sign of infection. Most ICD system infections involve the pulse generator pocket, and present with local tenderness, swelling, erythema, and warmth. Frank drainage of pus or device erosion may also be present. Fever and leukocytosis may be initially absent. Blood cultures are rarely positive. In some instances, the wound and pocket may look normal when overt system-related septicemia is present. ICD system infections should *never* be considered localized. The electrodes are always simultaneously contaminated, and microorganisms can migrate along them toward the heart. It is generally not recommended to aspirate a pocket suspected of infection. Imaging (CT scan, gallium scan) is of limited value because normal postoperative fluid collections can mimic findings of infection. Indium-111-labeled leukocyte scintigraphy may be more useful in the early postoperative period because of its leukocyte specificity. ICD lead-related endocarditis is rare (47). Transesophageal echocardiography is the method of choice for demonstration of vegetations attached to endocardial leads (48).

Complete system removal and intravenous antibiotic therapy are needed to eradicate infection (49). A conservative removal of the generator alone and pocket debridement is seldom successful. Empirical oral antibiotics for suspected ICD system infection should be avoided because they can partially suppress the infection, making subsequent diagnosis and treatment more difficult. In the absence of culture results, intravenous vancomycin (alone or combined with an agent effective against Gram-negative rods) is the treatment of choice. Vancomycin provides good coverage against coagulase-negative *Staphylococci*, methicillin-resistant *S. aureus*, *Propionibacterium acnes*, and diphtheroids that are common causes of ICD infection. Removal of chronic ICD leads is technically challenging, but can be performed at low risk with the aid of special tools (50). A new system should be reimplanted (at a new site, such as the contralateral pectoral region) only when repeated blood cultures are sterile. In the interim, high-risk patients should remain hospitalized under continuous electrocardiographic surveillance.

Terminal Care Issues

The presence of an ICD in a terminally ill patient raises medical and ethical issues (51). Physicians are often asked to deactivate the device by patients or relatives. Deactivation of an ICD is appropriate when the device is believed to be prolonging patient suffering. In patients with frequent arrhythmias triggering ICD shocks, deactivation will not only hasten, but also permit a peaceful death. In the United States, such action would be considered withdrawal of treatment, which is both legal and ethical, provided that informed consent is obtained. A do-not-resuscitate order does not automatically imply permission for ICD deactivation, and explicit consent should be obtained (52). Likewise, ICD bradycardia pacing could be disabled. If contemplated, this decision should be explored with sensitivity to ensure that everybody involved has a full understanding of the medical, legal, and ethical implications associated with withdrawal of pacemaker function. In patients who otherwise do not use it, disabling this function will prevent agonal pacing. Disabling pacing in patients who require it, is more problematic. In completely pacemaker-dependent patients, this will result in nearly instantaneous death. In other patients, it would result in symptomatic bradycardia with slow relentless failure of major organs and, perhaps, increase the agony of death. There is little precedent on which to base decisions, but it is recommended that the pacing function not be disabled in patients who use it. If pacing is disabled, care should be taken to ensure that such action is not mistaken for physician-assisted suicide or euthanasia.

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