REVIEW ARTICLES

Perioperative Management of the Patient Undergoing Automatic Internal Cardioverter-Defibrillator Implantation

Nicholas Deutsch, MD, Charles B. Hantler, MD, Fred Morady, MD, and Marvin Kirsh, MD

IT HAS BEEN ESTIMATED that 400,000 people die annually from sudden cardiac arrest in the United States.[1] Various treatment modalities have been tried in patients at high risk for sudden death from ventricular fibrillation, but delivery of an electrical countershock of sufficient energy remains the only reliable treatment.[2] The automatic internal cardioverter-defibrillator (AICD) has dramatically changed the treatment of patients at high risk for sudden death. The first clinical trial was by Mirowski et al[3] in 1980. The implantation of AICDs has greatly increased since the Food and Drug Administration approved the device in 1985. There have been more than 3,000 AICDs inserted since 1982, at greater than 200 centers.[4]

The results of conventional treatment are not optimal. The 1-year sudden-cardiac-death rate for survivors of an episode of cardiac arrest receiving empiric drug therapy is 20% to 50%.[5-9] The 1-year sudden-cardiac-death rate for patients with electrophysiologic or Holter-monitor-guided therapy is 2% to 15%.[5,6,9] The results from a number of series of patients with AICDs, however, have been very encouraging. The 1-year sudden-cardiac-death rate of AICD patients has been reported to be about 2%.[10-14]

DEVICE

The AICD device contains lithium batteries, capacitors, a piezoelectric crystal, and connec-

tors for the different electrodes. The internal circuitry is attached to the receptacles for the electrode leads by hermetically sealed feed-throughs, which protect the device from electro-magnetic interference. There are two defibrillating electrodes made of silicone rubber and titanium. The first defibrillating electrode is either an intravascular spring electrode or, alternatively, a patch electrode. The intravascular spring electrode is placed by percutaneous cannulation of the subclavian vein into the superior vena cava near the right atrium. Alternatively, a patch electrode may be placed over the right atrium or ventricle. The other end of the first electrode is tunneled subcutaneously to the left upper quadrant of the abdomen. The second defibrillating electrode is a wire-mesh patch sewn to the cardiac apex. A separate electrode system is used for rate sensing. The rate-sensing electrode is either a transvenous lead placed in the right ventricular apex, or a bipolar epicardial screw-on pair placed on normal myocardium.[15] (See Figs 1 and 2).

The AICD typically requires 5 to 20 seconds to sense ventricular tachycardia or fibrillation. It then takes 5 to 15 seconds to charge its energy-storage capacitors. The first shock delivered is usually 23 to 28 J. After appropriate sensing and charging periods of 10 to 35 seconds, the device will deliver second, third, and fourth shocks, if necessary. The device will not deliver more than four consecutive shocks because this prevents multiple shocks in misdiagnosed rhythms.[15]
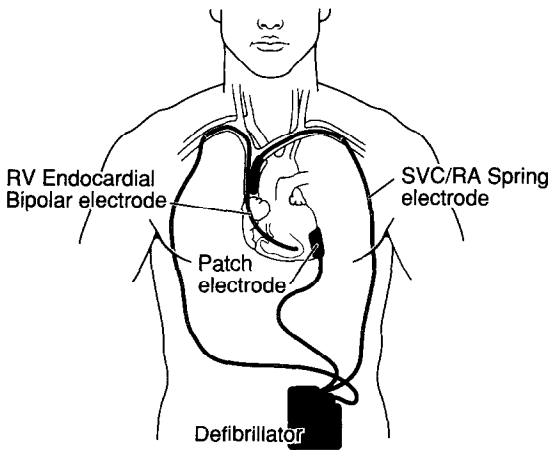
There are two general types of AICDs. One is triggered to deliver a countershock when a heart rate algorithm alone is satisfied, and the other is triggered when both a heart rate and a probability density function (PDF) algorithm are met. The heart rate algorithm is based on the

Fig 1. The electrode configuration with two transvenously placed electrodes, the rate-sensing, bipolar electrode, and the defibrillating, spring electrode, along with a single epicardial defibrillating patch electrode. RV, right ventricle; SVC, superior vena cava; RA, right atrium.

amount of time an average heart rate spends above a preset value, specified by the physician at the time of manufacture. Using only the heart rate algorithm, the device cannot differentiate among sinus, supraventricular, and ventricular tachycardias. The PDF analyzes the amount of time the QRS complex spends away from the isoelectric baseline as sensed by the two defibrillating electrodes. During sinus rhythm or relatively slow supraventricular tachycardia without
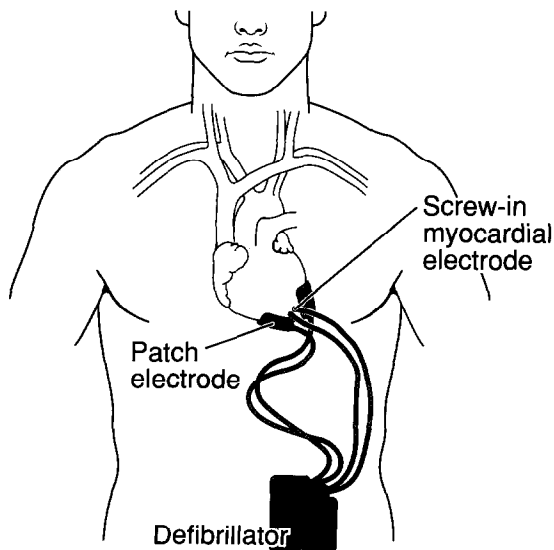
Fig 2. The electrode configuration with all three placed epicardially. The two defibrillating, patch electrodes and the screw-in, rate-sensing electrode are shown.

aberration, the electrocardiographic (ECG) waveform spends most of its time on the isoelectric baseline. Conversely, during ventricular tachycardia or coarse ventricular fibrillation, the ECG waveform deviates from the isoelectric baseline a high percentage of the time and satisfies the PDF.[14,15] The advantage of using a heart rate trigger alone is that a relatively narrow complex ventricular tachycardia, which might not meet the PDF, will be countershocked.[16] In addition, a shock is delivered more rapidly when sensing is based on rate only.

The AICD device has three different modes. They are the active, inactive, and electrophysiologic test modes. Application of a magnet to the AICD for less than 30 seconds will generate a signal that conveys its mode and for more than 30 seconds will change its mode. When a magnet is initially applied to the inactivated AICD, the piezoelectric crystals transmit a constant shrill tone, signifying that the device is deactivated. After 30 seconds, pulsed tones synchronous with the ECG R wave are then emitted. The AICD is now in the electrophysiology test mode, which enables the device to sense a dysrhythmia without discharging. Upon removal of the magnet, the device is activated. Conversely, when a magnet is first applied to the activated device, pulsed tones synchronous with the R wave of the ECG are transmitted, denoting that the device is in the active mode. Thirty seconds later, a continuous shrill is audible, indicating that the device has switched to the inactive mode.[17]

INDICATIONS AND CONTRAINDICATIONS

Criteria for implantation have been liberalized since the AICD was first introduced clinically, and will continue to evolve as more clinical data are obtained. Table 1 lists common indications and contraindications for AICD insertion.[12,15,16,18-21]

PREOPERATIVE CONSIDERATIONS

Candidates for AICD implantation need a thorough preoperative evaluation. This evaluation typically includes cardiac catheterization, including coronary arteriography and left ventriculography, so that decisions about the need for coronary or valvular surgery can be made. These patients will have undergone thorough electrophysiologic testing to determine the inducibility

Table 1. Indications and Contraindications for AICD Insertion

Indications

    History of sudden cardiac arrest not associated with acute myocardial infarction

                and

    ·· `Dysrhythmia that cannot be induced by electro-physiologic testing

                or

    · Pharmacologic failure as assessed by electro-physiologic testing

                or

    · Long QT syndrome

    Life-threatening ventricular tachyarrhythmias and antiarrhythmic drug therapy failure

Contraindications

    Uncontrolled congestive heart failure

    Frequent recurrences of ventricular tachycardia (eg, 1 or more episodes daily) such that the AICD battery would rapidly deplete

Data from Gabry et al,[12] Cannom et al,[15] Winkle et al,[16] Fisher et al,[18] Mirowski et al,[19] Reid et al,[20] and Platia et al.[21]

of ventricular tachycardia and the rates and forms of ventricular tachycardia; they will also have undergone electrophysiologically guided drug therapy. Those able to exercise should be given an exercise-treadmill test preoperatively or just before hospital discharge to assess the need for postoperative β-blockade to prevent the heart rate from reaching a rate that may trigger the AICD.[13,15] Other investigators, in addition, recommend Holter monitoring before surgery.[13,20] Also, it should be known if any intravenous drugs were effective in suppressing the dysrhythmia. Indications for other preoperative tests are the same as with any other major thoracic procedure. Preoperative pulmonary function tests may be useful to rule out chronic obstructive pulmonary disease or interstitial lung disease secondary to amiodarone toxicity.

Patients should be on a stable antiarrhythmic drug regimen at the time of implantation if treating the patient with long-term antiarrhythmics is planned. Antiarrhythmic drugs may alter the defibrillation threshold tested intraoperatively.[22] Not administering the antiarrhythmic drug preoperatively may lead to device failure because the defibrillation threshold can be increased postoperatively when the antiarrhythmic therapy is reinstituted.[22] However, some prefer to discontinue antiarrhythmic drugs 4 to 5 half-lives before surgery so that dysrhythmia induction may be made easier.[23] If the patient is

receiving amiodarone preoperatively and will not be postoperatively, the drug should be discontinued at least 1 month before surgery.[15]

There is a high incidence of congestive heart failure among these patients. This requires the best possible medical control preoperatively. Likewise, any concurrent medical conditions require optimal management. The patients for AICD have been characterized by a number of investigators. Table 2 lists a typical subset of patients for AICD implantation.[24]

ANESTHESIA

The AICD patient must be continuously monitored by ECG even during transport to the operating room. An external cardioverter-defibrillator should be readily available. If the patient has sustained ventricular tachycardia that is hemodynamically stable, a precordial thump may be attempted and then, if unsuccessful, the patient should be sedated and externally cardioverted by synchronous cardioversion starting at 50 J, or at the energy level that was successful in the electrophysiology lab. The energy requirements should then be increased to 100, 200, 300, and 360 J. If the patient has sustained ventricular fibrillation or ventricular tachycardia that is

Table 2. Profile of 70 AICD Patients

Cardiac Disease	
Coronary artery disease	68%
Hypertrophic cardiomyopathy	1%
Mitral valve prolapse	4%
Other valve disease	6%
Long QT syndrome	2%
Myasthenia gravis	2%
No evidence of disease	6%
Cardiac function	
NYHA class	2.2 ± 0.9
Mean CI	2.6 ± 0.7
EF	38% ± 18%
LVEDP	18 ± 10 mm Hg
Spontaneous Dysrhythmia	
Ventricular tachycardia	44%
Ventricular fibrillation	56%
Ventricular tachycardia and ventricular fibrillation	2%
Preoperative	
Mean dysrhythmic episodes	3.3 ± 2.4
No. of cardiac arrests	1.9 ± 1.8
No. of drugs failed	4.0 ± 2.3

Abbreviations: NYHA, New York Heart Association; CI, cardiac index; EF, ejection fraction; LVEDP, left ventricular end-diastolic pressure.

Data from Echt and Winkle.[24]

hemodynamically unstable, then a precordial thump may be tried initially. If that is unsuccessful, immediate defibrillation with the energy level that was successful in the electrophysiologic laboratory or, if unknown, with 200 J, should be attempted and then increased to 300 and 360 J.[25]

General anesthesia is required for AICD placement because the cardiac apex is exposed for patch-electrode placement. Routine monitors, including a five-lead ECG, should be used for anesthesia administration. In addition, these patients need an arterial catheter for beat-to-beat pressure monitoring. Patients for AICD implantation commonly have a central venous catheter inserted. At most centers, a pulmonary artery catheter is used for those patients with markedly depressed cardiac function, severe coronary artery disease, or severe pulmonary hypertension.[15,23] Others, however, use a pulmonary artery catheter as part of their routine management.[13] These monitors are usually inserted before anesthetic induction. Some report using procaine instead of lidocaine for local anesthesia because lidocaine may interfere with the induction of the ventricular dysrhythmia.[23]

Anesthetic management is based on the patient's underlying disease and pathophysiologic status and whether or not other cardiac surgery is being performed. The implantation of an AICD presents a number of challenges to the anesthesiologist. There are high incidences of ischemic heart disease and depressed cardiac function in patients undergoing AICD implantation. Despite this, patients can be managed with the expectation of extubating them on the operating room table or in the immediate postoperative period.

Intraoperatively, a number of tests must be performed. Each individual lead as well as the entire AICD system must be tested. Continuity of the lead system needs to be ensured. Also, proper lead positioning needs to be checked so that the amplitude of the signals is within acceptable limits. The electrode position of the rate-sensing lead should be placed so that the largest ventricular electrogram is obtained. The signal should be no less than 4 mV. This increases the chance of adequate signal detection during ventricular tachycardia. If a spring-patch electrode is used, it should be positioned at the junction of the superior vena cava and right atrium. The

signal from the patch electrode is also recorded. A signal of less than 5 mV implies that the electrode is over previously infarcted myocardium or is making poor contact with the ventricle.

The energy required to convert the patient's malignant dysrhythmia needs to be determined. Intraoperatively, the patient's malignant rhythm is induced by programmed ventricular stimulation or alternating current. In spite of a history of recurrent dysrhythmias and ability to induce these dysrhythmias while awake, they are sometimes difficult to induce under anesthesia. Ventricular tachycardia may not be inducible in as many as 10% to 40% of patients undergoing dysrhythmia surgery who are under anesthesia.[26-28] Multiple attempts at dysrhythmia induction are sometimes necessary. In addition, isoproterenol or epinephrine is sometimes required to facilitate dysrhythmia induction. Some electrophysiologists use alternating current for the induction of ventricular fibrillation.[29] The defibrillation thresholds for both ventricular tachycardia and fibrillation need to be determined when the patient's underlying dysrhythmia is ventricular tachycardia, because ventricular tachycardia may degenerate into ventricular fibrillation before or after cardioversion. The defibrillation threshold is determined by using an external cardioverter-defibrillator attached to the defibrillation leads of the AICD. At least a 10-J margin above the energy required to defibrillate the heart is considered optimal for the energy to be delivered by the AICD.[15] If a defibrillation threshold less than or equal to 15 J is not achieved with the initial lead position, different lead configurations are tested until an adequate threshold is identified. A threshold of less than 25 J is necessary for AICD implantation.[13] Between four and eight cardioversions and defibrillations are fairly typical. As many as 20 to 30 dysrhythmia inductions and countershocks are possible without apparent ill effect to the patient.[16] Intraoperative testing of the lead configuration is thought to improve results because, in one series, 12% of the patients would have had inadequate dysrhythmia termination in the initial configuration.[16] Arterial pressure needs to be monitored continuously during dysrhythmia inductions. Blood pressure should be allowed to return to the baseline level between inductions. If hypotension persists, inotropic sup-

port should be instituted and the number of dysrhythmia inductions kept to a minimum. A minority of patients may exhibit prolonged pauses and severe bradycardia after each shock. These patients may require insertion of a separate, standby ventricular pacemaker.[30]

DRUG INTERACTIONS

Volatile anesthetics suppress the induction of ventricular tachycardia by programmed electrical stimulation in the canine model.[31,32] However, a narcotic-based technique has been shown to have minimal effects on the induction of ventricular tachycardia.[31] The effects of anesthetics commonly used for AICD implantation on defibrillation thresholds are largely unknown. Chloralose and pentobarbital have been reported to have no significant effect.[33,34] Furthermore, there may be significant interactions between anesthetics and antiarrhythmics on the defibrillation threshold. Lidocaine has produced significant, dose-dependent increases in transthoracic and internal defibrillation thresholds in pentobarbital-anesthetized dogs. However, in chloralose-anesthetized dogs, no significant changes were noted with similar lidocaine concentrations.[33,35]

Animal and clinical studies suggest that therapeutic levels of some Vaughan-Williams class-IB and -IC drugs may increase the defibrillation threshold. The effects of class-IA drugs may manifest themselves only at toxic doses.[22] (See Table 3 for a subclassification of class-I antiarrhythmic agents). High doses of quinidine, a class-IA drug, have been reported to cause large increases in the transthoracic defibrillation threshold.[37] Other investigators have found small or no increases in internal defibrillation threshold.[38,39] Flecainide, a class-IC drug, causes a marked increase in defibrillation threshold.[40] Therapeutic doses of propranolol, a class-II agent,

do not alter defibrillation energy requirements.[38] At high doses of propranolol, the internal defibrillation threshold was increased. This is perhaps a result of the membrane-stabilizing effect of high-dose propranolol.[41] Amiodarone, a class-III agent, has been reported to cause a marked increase in the defibrillation threshold.[13,42]

Patients undergoing AICD implantation frequently need concomitant drug therapy. Present devices have a maximum energy output of approximately 30 to 32 J. The safety margin of defibrillation may be inadequate when drugs affecting the electrophysiological characteristics of the diseased myocardium are used. Some authorities recommend testing the efficacy of the AICD in the electrophysiologic laboratory when a drug regimen is changed after AICD implantation.[22]

The work presented has many theoretical implications for anesthesiologists. In a patient with an AICD in situ, it is important to be prepared for AICD failure during anesthesia because anesthetics or adjuncts to anesthesia could alter defibrillation thresholds. Also, if certain anesthetics lower the defibrillation threshold, then patients receiving these anesthetics at the time of AICD implantation would have artificially low threshold measurements, which might put them at risk for subsequent AICD failure. Conversely, if certain anesthetics raise the defibrillation threshold, during AICD implantation they may subject the patient to unnecessary attempts to find a more optimal lead configuration. In practice, there have been no reported cases of adverse anesthetic interactions.

SURGICAL CONSIDERATIONS

There are a number of different surgical approaches used for AICD implantation. If a superior vena caval spring lead or the endocardial rate-detection lead are used, they can be inserted percutaneously through the subclavian vein under local anesthesia before going to the operating room, thereby saving time and the need for fluoroscopy once there. Later, under general anesthesia, the leads are tunnelled subcutaneously to the pulse generator in the abdomen. When either type of lead system inserted from the subclavian vein is used, avoidance of intracardiac monitoring catheters is recommended.

The surgical approaches available for AICD

Table 3. Subclassification of Class-I Antidysrhythmic Agents

A	B	C
Quinidine	Lidocaine	Flecainide
Procainamide	Mexiletine	Lorcainide
Disopyramide	Diphenylhydantoin	Encainide
Tricyclics	Ethmozin	Propafenone
Ajmaline	Aprindine	

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implantation include subxiphoid thoracotomy, median sternotomy, left anterior thoracotomy, and left subcostal thoracotomy in the supine position. The underlying cardiac disease, the presence of a previous cardiac surgical procedure, and the need for myocardial revascularization or endocardial resection determine the approach to be used. The subxiphoid approach is the least traumatic, and operative times are shorter with it. The incision is limited, so postoperative pain is minimal. It is particularly useful for patients with end-stage cardiac disease. However, it requires a pericardium free of surgical scarring. The median sternotomy is the approach of choice for those patients also undergoing open-heart procedures. In addition, it provides the widest cardiac exposure, which is particularly useful when all the leads are epicardial.[28] In patients with a previous median sternotomy, the left anterior thoracotomy is a commonly used alternative.[43] It avoids scar tissue associated with previous cardiac surgery. The left subcostal thoracotomy has been used by some surgeons because it is thought to minimize postoperative pain.[44]

POSTOPERATIVE CONSIDERATIONS

In the immediate postoperative period, AICD patients are susceptible to transient supraventricular and ventricular dysrhythmias. Because these dysrhythmias may trigger unnecessary shocks, the device is turned off in the initial postoperative period, and the patient must be carefully monitored and treated for sustained dysrhythmias. An increased incidence of postoperative ventricular tachycardia has been noted in patients with epicardial rate-counting leads, and atrial dysrhythmias may be more common in those with the patch-patch defibrillation electrode configuration.[13] Thus, transthoracic cardiac defibrillators should be readily available until the unit is activated. External cardioversion has been used successfully without damage to the AICD.[15] The paddles of the external defibrillator should be placed perpendicular to the line between the two implanted defibrillating electrodes in order that the energy pass directly to the myocardium. Adhesive external defibrillation pads, which can be left on the patient while the device is deactivated, allow for ready cardioversion and defibrillation in the proper paddle orientation. The AICD device is activated when the patient is more stable and ready to be transferred to an unmonitored area.[20,24]

During the implantation of the AICD device, patients undergo multiple dysrhythmia inductions. These are associated with periods of low or no blood pressure. It can be expected that the myocardium will be "stunned" postoperatively. Two-dimensional echocardiography was performed in a series of six patients undergoing AICD implantation. The mean ejection fraction before the first induced ventricular dysrhythmia was 28% ± 1.4% versus 27% ± 1.9% after a mean of six ventricular tachycardia/fibrillation episodes. The investigators concluded that electrophysiologic testing during AICD implantation does not have clinically important depressant effects on left ventricular ejection fraction.[45]

At some centers, all patients undergo testing of the device in the electrophysiologic laboratory before discharge. Proper sensing and termination of ventricular tachycardia and fibrillation must be found.[13] After discharge, patients are followed up at 2-month intervals for battery checks. Magnet tests provide a digital readout of the number of shocks delivered and the capacitor charge time. Longevity of the current devices is less than ideal; most units require changes within 24 months.[46] Under sterile conditions in the electrophysiology laboratory, the pulse generator may be replaced under local anesthesia. Yearly chest radiographs should be obtained to check for lead migration or fracture.

COMPLICATIONS

The surgical complications from one recent study[13] are shown in Table 4. The most common

Table 4. Postoperative Complications in 94 Patients

Pulmonary complications	29%
Postoperative ventricular tachycardia	13%
Postoperative atrial fibrillation	9%
Death	3%
Symptomatic pericarditis	3%
Pulmonary edema	2%
Deep-vein thrombus	2%
Infection	2%
Fluid or blood accumulation in pulse-generator pocket	2%
Myocardial infarction	1%

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problems were pulmonary, comprising hypoxemia, pneumonia, left lower lobe collapse, pneumothorax, and pleural effusions. They occurred more frequently in patients who had undergone a left anterior thoracotomy. The majority of these complications resolved with chest physiotherapy.

Later complications included lead migration, lead fracture, pacemaker-AICD interactions, and unnecessary shocks.[15] To minimize unnecessary shocks, treadmill exercise testing should be performed to assess maximal heart rate. β-blockade should then be instituted if necessary.

Inappropriate AICD discharges caused by sensing of noncardiac myopotentials have been reported.[11] By applying a magnet to the AICD, this condition can be diagnosed. Rapid erratic tones that are not synchronous with the QRS complex and are emitted from the piezoelectric crystal when the patient exercises are indicative of this problem.[47]

Patients with pacemakers present special problems for proper AICD function. The pacemaker and AICD may interact in a number of unfavorable ways. During a tachydysrhythmia, the pacer may not sense the ventricular tachycardia or fibrillation and continue to fire at the programmed pacing rate. The AICD may then ignore the tachydysrhythmia in favor of the regular pacing spikes of the pacemaker and, as a result, not fire. Conversely, inappropriate AICD discharge may occur if the AICD double-counts both the pacing spike and the evoked ventricular depolarization. These problems are more common with a unipolar pacemaker because of the larger magnitude of the pacemaker stimulus artifact.[48] Only bipolar pacemakers should be used in conjunction with an AICD.

MANAGEMENT OF PATIENTS WITH AN AUTOMATIC INTERNAL CARDIOVERTER–DEFIBRILLATOR

There has not been a great deal of experience with anesthesia for patients with an AICD in situ. The anesthetic technique should be the same that normally would be used for the given procedure and the given pathophysiological state of the patient. Some suggest a digital plethysmograph as an alternative to an arterial catheter to indicate the presence of pulsatile flow.[23] Monitoring should be guided by the patient's underlying disease and by the surgical procedure. Use of gloves has been advocated because the AICD may discharge at any time and especially during cardiopulmonary resuscitation.[49]

Electromagnetic interference can cause inappropriate AICD inhibition or discharge. Electrocautery is a source of interference that is of particular concern to the anesthesiologist. It is generally recommended that electrocautery be used only when the pulse generator is deactivated.[15] Gaba et al[23] report a patient having a countershock sequence initiated by electrocautery and precipitating ventricular tachycardia in the vicinity of the generator during surgery. Moreover, use of electocautery in proximity to the AICD can cause direct thermal injury to the device, electrode leads, or electrode-myocardial interface. However, the AICD has been designed to resist interference from outside electromagnetic sources, and some think electrocautery can be used with caution while the AICD is activated.[23] If electrocautery is used when the AICD is activated, the electrocautery ground pad should be placed on the patient so that the axis of the ground pad and the surgical site is oriented perpendicular to the axis of the bipolar rate-sensing leads. In addition, the electrocautery unit should be used only in short bursts because the AICD requires 5 to 20 seconds to sense a dysrhythmia. Moreover, a ring magnet must be immediately available to deactivate the AICD device. Whether the AICD is deactivated or activated, an external cardioverter-defibrillator must also be readily available. Nuclear magnetic resonance scanners and shock-wave lithotripsy are other causes of electromagnetic interference important to the anesthesiologist. Patients with AICD devices should not enter the vicinity of a nuclear magnetic resonance scanner, and patients for lithotripsy should have their devices deactivated before treatment.

The pathological effects of the AICD and of multiple internal defibrillations have been examined at autopsy in 25 patients. It was found that pericarditis is a universal finding of little clinical significance. The presence of the superior vena caval lead was associated with thrombi in 17% of the patients. However, no patient in the series died of pulmonary embolism. Multiple

defibrillations led to localized myocardial pathology adjacent to the patch electrode. It was concluded that multiple defibrillations by the AICD cause inconsequential myocardial and pericardial injury.[50] In addition, global and regional left ventricular function have been followed via M-mode echocardiography in 15 patients undergoing AICD implantation. The patients were studied preoperatively and then followed for a period of approximately 6 months after AICD implantation. The investigators inferred that AICD implantation did not significantly alter ventricular function.[51]

FUTURE

A number of improvements will be included in devices of the future. A programmable AICD device was recently released for clinical use that has low-energy cardioversion capabilities, flexible settings for the heart rate which will trigger a countershock, and a choice of including the PDF in the countershock algorithm. Other advances will include backup bradycardia pacing, incorporation of hemodynamic sensors into the device, and antitachycardia pacing. Overdrive pacing can effectively terminate ventricular tachycardia.[52] By combined use of an antitachycardic pacemaker and AICD, the number of discharges from the AICD can be reduced. This would improve patient comfort and improve battery longevity.[53] In addition, the epicardial patch defibrillator may be replaced by a subcutaneous patch defibrillator, thereby simplifying the surgical procedure and perhaps decreasing the rate of morbidity.

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