Anesthesia and the Automatic Implantable Cardioverter/Defibrillator

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VENTRICULAR DYSRHYTHMIAS are the cause of sudden death in an estimated 400,000 people per year.[1] Increased involvement of the public in basic cardiopulmonary resuscitation and improved delivery of advanced cardiac life support have led to improved survival of patients suffering from such malignant rhythm disturbances. Earlier studies of patients surviving the initial arrest and being admitted to hospital showed a hospital mortality of 58%. Of the survivors leaving the hospital, 28% died suddenly within 1 year, despite dysrhythmia prophylaxis considered adequate at that time.[2] In the absence of effective prophylactic therapy for their dysrhythmias, long-term survival remains uncertain.

Advances in understanding and treating recurrent dysrhythmias have been made by studying the electrophysiology of the heart with intracardiac ECG recording in the cardiac catheterization laboratory. Induction of a patient's dysrhythmia by application of properly timed electrical stimuli during cardiac catheterization has allowed patients to be treated with intravenous antidysrhythmia drugs to determine which agent and dose can best inhibit dysrhythmia induction.[3-5] For patients with localized myocardial tissue abnormalities (ventricular aneurysm, ideopathic hypertrophic subaortic stenosis), intraoperative electrophysiologic mapping of dysrhythmogenic foci with subsequent surgical excision can be of benefit.[6] Patients susceptible to potentially lethal dysrhythmias who remain unresponsive to pharmacologic suppression, who cannot tolerate certain side effects of antidysrhythmic drugs, or who have diffuse myocardial disease remain at high risk of sudden death.

Until recently, the prompt availability of direct current countershock by trained personnel was the only reliable method for termination of ventricular fibrillation. To treat episodes of ventricular dysrhythmias outside the hospital, automatic implantable cardioverter/defibrillators (AID) have been developed, and such a device, the AID-B®,‡ recently became available for clinical trial.

The first such device was implanted in February 1980,[7] and since then this device has undergone extensive clinical trials in several centers. We shall review the theory and operation of the AID-B® device, and the anesthetic management of patients presenting for implantation of an AID-B® and of those with an AID-B® in situ.

Theory of Countershocks

The success of countershock in terminating cardiac dysrhythmias depends on the simultaneous depolarization of a critical mass of myocardial cells.[8] This is accomplished by the transfer of an electric current through the myocardium, the variable resistance between the defibrillating electrodes determining the exact current flow. The defibrillator discharge is measured as the total energy delivered in joules (or the equivalent unit: watt-seconds). To terminate ventricular fibrillation, a defibrillator using transthoracic electrodes may require up to 400 joules to overcome the large transthoracic resistance, and a small, implantable device could not practically store such energy levels. The AID utilizes a catheter defibrillating electrode in or near the heart iself. The energy required to terminate a ventricular dysrhythmia by such internal defibrillation is dependent upon the dysrhythmia but is less than that required for external defibrillation. In humans, as little as 1 joule delivered internally terminated 77% of episodes of ventricular tachycardia of stable morphology; 8.3% of episodes of ventricular fibrillation were converted by 1 joule, but normal sinus rhythm in 92% of such episodes was restored by an increase in the energy to 25 joules.[9]

Description of Aid-B®

The AID-B® is shaped much like a pacemaker (fig. 1).[10] It weighs 250 g, occupies a volume of 145 ml, and has a hermetically sealed laser welded titanium case. There are two defibrillating electrodes, both made of silicone rubber and titanium. The first electrode is an intravascular catheter that is placed in the superior vena cava near the right atrium by percutaneous cannulation of the subclavian vein (fig. 2); the other end is tunneled subcutaneously to the left upper quadrant of the abdomen. The second electrode is a wire mesh patch sewn to the cardiac apex. Earlier versions of the AID-B® monitored both the morphology and the rate of the

electrocardiographic signal via the intravascular electrode and the apical patch electrode; this was found to have several disadvantages. This lead geometry accentuated P-wave amplitude causing both P waves and the QRS to be counted in rate determination and made recognition of certain dysrhythmias more difficult in some patients. The current AID-B® uses a separate electrode system for rate determination, which may consist of either a right ventricular endocardial-screw electrode (similar to a pacemaker lead) or a pair of screw-in epicardial electrodes placed on normal myocardium (fig. 2).

A critical feature of this device is the sensing and decision logic, which monitors cardiac rhythm and determines activation of the countershock sequence. The AID-B® utilizes a unique algorithm to recognize malignant rhythms.[10] The morphology of the ECG is moni-

FIG. 1. The AID-B® device with associated electrodes. The spring electrode on the left is one of the two defibrillating electrodes and is placed in the superior vena cava. The other defibrillating electrode is the patch on the right of the photograph, which is sutured to the cardiac apex. In the center is one of the endocardial electrodes placed in the right ventricle for rate sensing (photograph courtesy of Intec Systems, Inc., Pittsburgh, Pennsylvania).
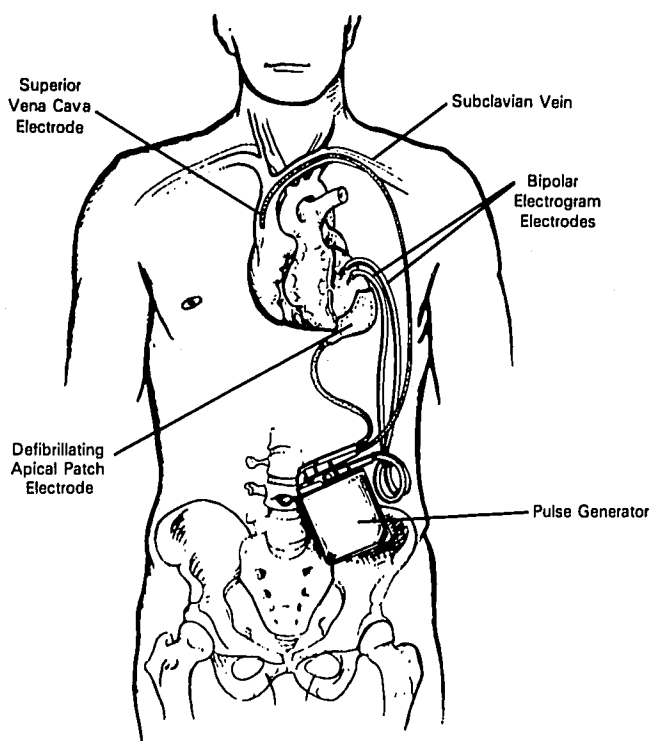

FIG. 2. Placement of the AID-B® and electrodes.

tored by the defibrillating electrodes. This signal is filtered, and the first derivative of the filtered ECG examined to determine the likelihood of finding the signal at any given amplitude (fig. 3). Signals of sinus and other nonmalignant rhythms spend large amounts of time at or near the baseline, and the likelihood of finding the signal near zero amplitude is high. This is not true of ventricular tachycardia and fibrillation signals, which only cross the baseline twice per oscillation. The sensing logic seeks the lack of a "zero peak" in the likelihood distribution. Because certain aberrant but nonmalignant rhythms may satisfy this criterion, a second requirement is that the rhythm must have a rate greater than a threshold minimum to activate the device.[11] This threshold rate is programmable but is usually set at 160 beats per minute. Dysrhythmias can be correctly diagnosed using the rate sensor alone,[12] which minimizes false negatives (lethal rhythms not shocked). This has tended to maximize false positives (inappropriate shocks of benign rhythms) to the extent that 10% of the patients had emotional problems in dealing with frequent, uncomfortable countershocks.[12] The combination of morphology and rate sensing may be optimal.

The AID-B® requires between 5 and 15 s to diagnose the dysrhythmia. When it has determined that a malignant rhythm exists, it initiates a program of countershocks.[13] The output capacitors are first charged, taking 5–15 s, and a 25-joule pulse is delivered. If the malignant
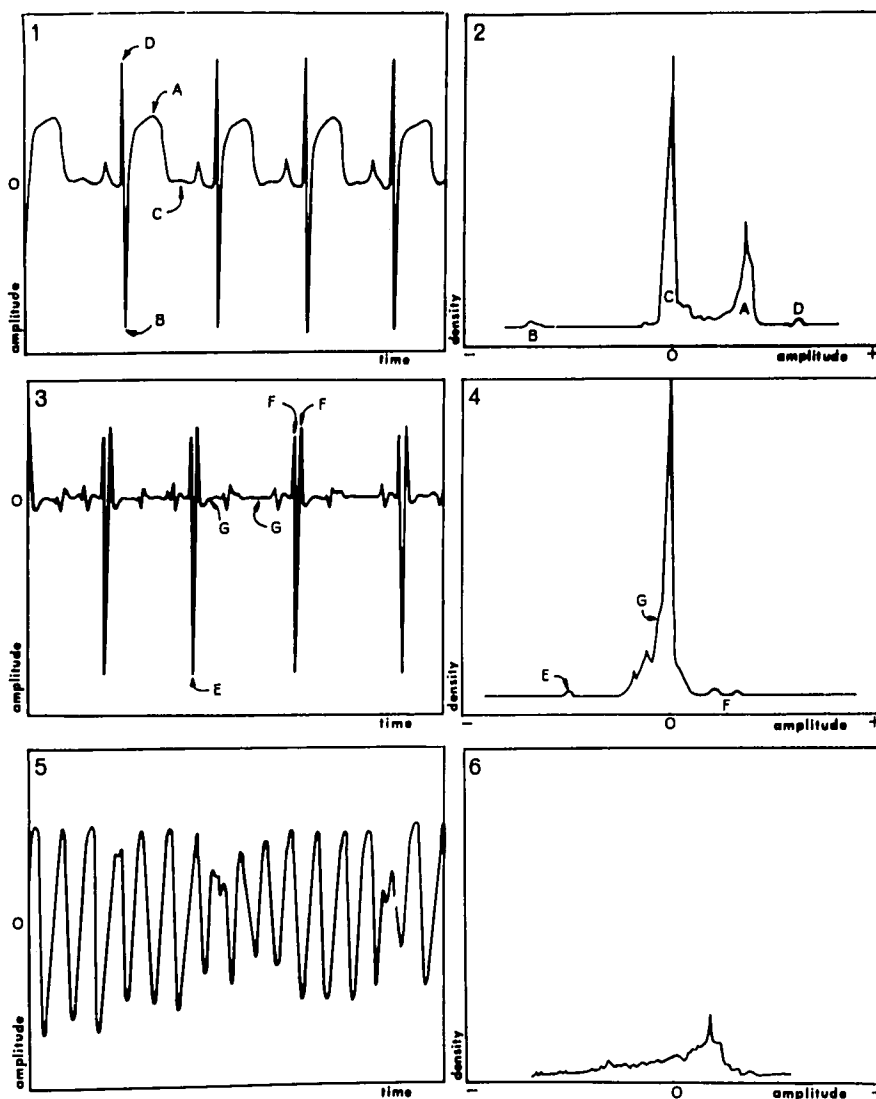
FIG. 3. Morphology criterion for ventricular dysrhythmias from processed ECG signals. Panel 1 shows a filtered ECG tracing of sinus rhythm, while panel 2 shows the probability density function of this waveform with a large peak (C) at zero amplitude and secondary peaks (A and D). Panels 3 and 4 show the first derivative of the above ECG tracing and its corresponding probability density function. The single peak (G) at zero amplitude is more pronounced than in panel 2. Panels 5 and 6 show an ECG tracing of ventricular fibrillation and the corresponding probability density function. Note the signal is rarely at zero amplitude. Reprinted from Mirowski M, Mower M, Reid P, Watkins L, Langer A: The automatic implantable defibrillator. Pace 5:384–401, 1982, with permission of the publisher.

rhythm still is sensed, the device will deliver up to three additional shocks of 30 joules each. Of note is that before each shock, the unit will require time to determine if the dysrhythmia is still present and additional time to charge the capacitors. (This shock sequence is standard in all AID-B® units manufactured after January 1, 1984. A sequence of all 30 joule pulses has been used in certain research protocols.) After completing this four-shock sequence, the device will not discharge a further countershock unless it senses at least 35 s of nonmalignant rhythm. This prevents uncomfortable and wasteful shocks if the dysrhythmia is artifactual or if the rhythm is unresponsive to countershock. Patients who have been observed during such a sequence characteristically might complain of weakness, palpitations, or dizziness, possibly followed by loss of consciousness and cardiovascular collapse. A sudden muscular contraction of the chest caused by defibrillator discharge then would be followed by almost immediate recovery.[14]

The AID-B® is supplied with a noninvasive interro-

gation instrument, which can be used to assure proper operation of the device and to measure the capacitor charge time, a key indicator of the state of the batteries.[13] A magnet is placed over the AID for a few seconds, which initiates a charge cycle. Triggered in this manner, the capacitors will discharge through an internal test load. Patients with an AID-B® in place have the device tested every 2 months, and replacement of the pulse generator is indicated if the capacitor charge time rises greatly over specification.[11] The magnet also can be used to switch the AID-B® from active to inactive state by leaving it over the device for longer than 30 s.[13]

Anesthesia for AID Implantation

The surgical procedure for implanting an AID differs from that used for placing a permanent pacemaker. Exposure of the cardiac apex is necessary for the placement of the patch electrode, and general anesthesia therefore is required for this procedure. The apical

patch electrode may be placed by several surgical approaches including midline sternotomy, lateral thoracotomy, subxiphoid and left parasternal thoracotomy in the supine position.[7] In some cases the AID is placed at the time of other cardiac surgery, and this may determine the surgical approach.

The clinical efficacy of the AID is tested intraoperatively by dysrhythmia induction. The cardiologist may discontinue antidysrhythmic drugs 1–2 days prior to surgery in the hope of easier dysrhythmia induction. It is mandatory to continuously monitor the ECG of all patients subject to recurrent, lethal dysrhythmias so that potentially dangerous dysrhythmias may be detected and treated rapidly. Such monitoring should also include the time of transport to the operating suite.

Monitoring of intraarterial and central venous pressure is established prior to induction of general anesthesia. Procaine may be substituted for lidocaine for local anesthesia during placement of the monitoring lines if lidocaine is thought to interfere with dysrhythmia induction. In patients with markedly depressed cardiac function, severe coronary artery disease, or significant pulmonary hypertension, a pulmonary artery catheter may be indicated with only a slightly increased risk of inducing a persistent rhythm disturbance during insertion[15] but is not used routinely as part of patient management. Subsequent anesthetic technique is determined by the patient's underlying disease, pathophysiologic status, and whether other cardiac surgery will be performed and is essentially the same as for any critically ill patient.

Intraoperative testing of the AID-B® is performed to observe proper function of the device. Electrophysiologic testing demonstrates the ability of the device to consistently terminate dysrhythmias on repeated occasions and to observe the potential for promoting acceleration of ventricular tachycardia to ventricular fibrillation. This intraoperative testing can improve clinical results, since, in a recent report,[9] 12% of patients had inadequate dysrhythmia termination with 25 joules delivered by a catheter-patch system. Substitution of the catheter electrode by a 24 cm² right ventricular patch and enlarging the left ventricular patch to 24 cm² almost always resulted in successful dysrhythmia conversion in these patients.[9]

The pulse generator first is tested in its sterile packaging using the noninvasive interrogation equipment. After electrode implantation and attachment to the AID-B®, the patient's malignant rhythm is induced by administering rapid pacing stimuli. In spite of the history of recurrent dysrhythmias, they are sometimes difficult to induce during anesthesia, and attempts at dysrhythmia induction may be repeated multiple times. Isoproterenol, by infusion, occasionally has been administered to facilitate dysrhythmia induction. Because the rapid pacing stimuli may impede diastolic filling and may depress cardiac output and blood pressure, there may be marked cardiovascular compromise, even without induction of sustained dysrhythmias (fig. 4, *upper panel*). Once dysrhythmia induction is accomplished, the patient may be without cardiac output or blood pressure for up to 30 s while the AID analyzes the signal and charges the output capacitors (fig. 4, *middle panel*). After the AID delivers its shock the arterial tracing should be followed closely, since the ECG tracing is artifactually altered for some time (fig. 4 *lower panel*). Should the device fail, direct current countershock should be applied. If the surgical exposure does not permit internal paddles to be applied to the heart, external countershock must be used. After successful testing the unit is placed in the subcutaneous pocket and the incisions are closed.

Clinical Results

Reports of the clinical experience with the AID-B® to date support the hypothesis that this is an effective lifesaving therapeutic modality in a high-risk group of patients. The results published by Mirowski et al.[14] and by Winkle et al.[12] are summarized in table 1. Because these are continuing studies, these figures change frequently. Life table analysis of the patients from both centers (fig. 5) showed substantially better survival than in other patients with refractory ventricular tachycardia and fibrillation not treated with an AID.[16]

COMPLICATIONS OF THE AID

Failure of the AID-B® unit is possible, although most failures to date have occurred at the electrodes. Complications occurred more frequently with the earlier version of the AID-B®, and the current model incorporates changes in design to reduce the incidence of problems. These complications have included lead fractures or lead insulation breaks, lead migration, rate miscounting leading to spurious shocks, and shock acceleration of ventricular tachycardia to ventricular fibrillation. Infection of the subcutaneous pocket has necessitated removal of the device.[12,14] Significant complications of percutaneous lead insertion, including laceration of major vessels and pneumothorax, also have occurred.

Myocardial damage following both internal and external paddle countershock has been demonstrated in animals[17,18] and occasionally in humans.[19] Clinical studies suggest the probability of damage is small, and limited studies have shown no evidence of myocardial damage following countershock with the AID-B®.[20] However, myocardial damage has been noted adjacent to a defibrillation catheter implanted in the right ventricular endocardium after countershock from a different kind of AID.[21] The significance of this damage for long-term use of the AID remains to be seen.
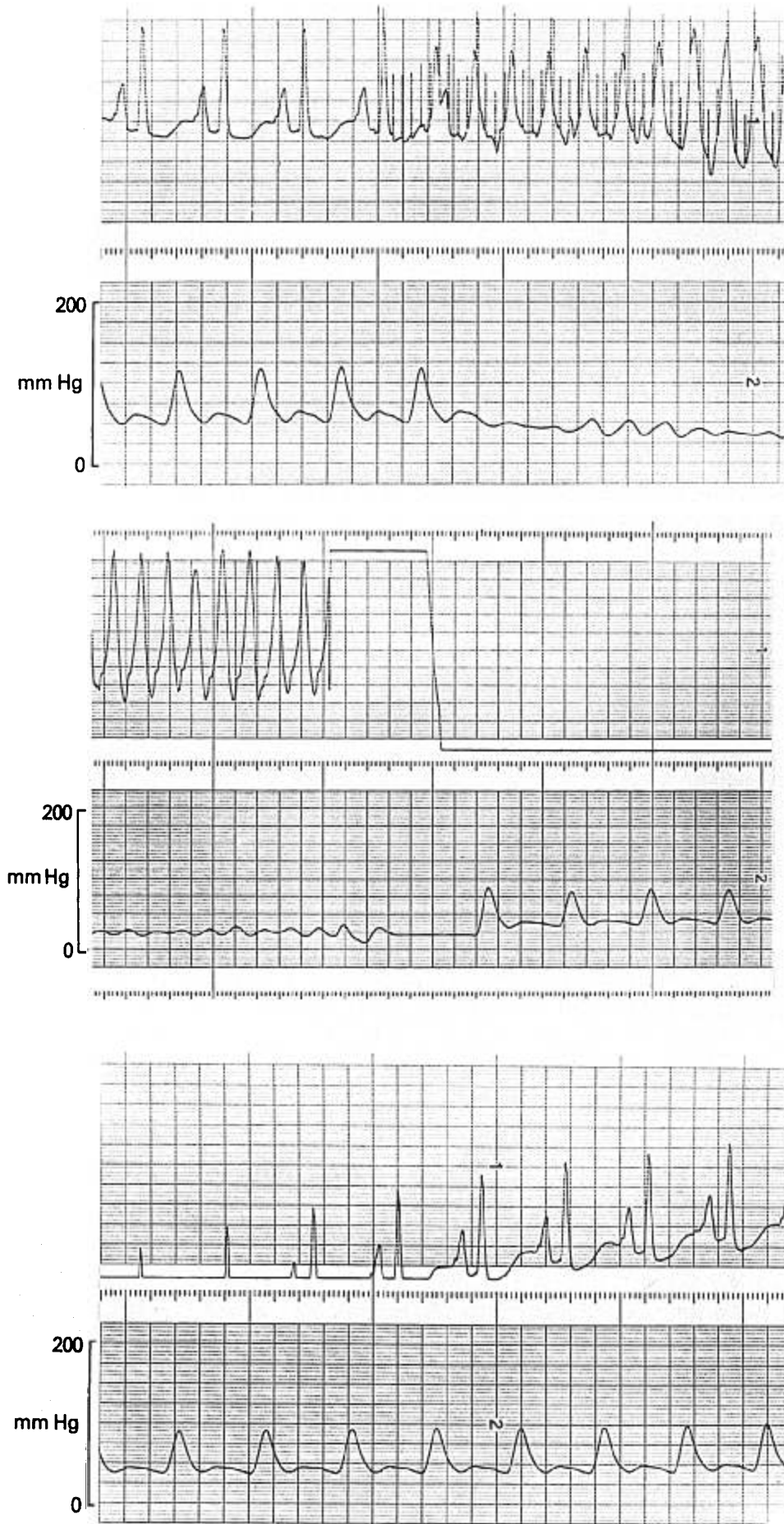
FIG. 4. Physiologic recordings during intraoperative AID testing. In each panel the upper trace is the external electrocardiogram, while the bottom trace is the radial arterial pressure. The top panel shows multiple electrical stimulus spikes applied during sinus rhythm. Note the drop in arterial pressure at this time (see text). The middle panel shows a persistent ventricular tachycardia with virtually no arterial pressure. This is followed by discharge of the AID device, which causes a period of no ECG *trace.* However, the dysrhythmia has been converted to a normal rhythm as evidenced by return of the arterial waveform. The bottom panel illustrates the slow return of the ECG *trace* after automatic defibrillation.

Serious hazards from long-term implantation have not been observed. Early mortality has been associated with the underlying pathology in these high-risk patients; progression of the myocardial disease is the most significant contributor to late mortality.[16]

ANESTHESIA FOR PATIENTS WITH AN AID In Situ

The anesthetic management of patients with an AID in situ is similar to that for patients who have a permanent cardiac pacemaker, although there is little accumulated experience to date. ECG monitoring will be required for all of these patients, but intraarterial pressure monitoring often may not be necessary. In such instances, however, a digital plethysmograph could be employed to indicate the presence of pulsatile flow should complex dysrhythmias arise. Although the AID-B® has been engineered to be immune to outside interference and artifactual signals, this immunity is not absolute. Electrocautery must be used with caution, and the grounding pad should be placed far from the pulse generator and AID leads.[13] A ring magnet must be immediately available should it become necessary to inactivate the unit. We recently have encountered a significant complication due to interaction between the AID and the electrocautery. Prior to removal of the AID generator due to infection of the subcutaneous pocket, it was impossible to deactivate the device using a ring magnet, possibly due to the patient's obesity. While using electrocautery to dissect down to the AID generator, a countershock sequence was initiated that precipitated ventricular tachycardia. The patient promptly was resuscitated by transthoracic cardioversion. We advocate avoiding electrocautery close to the AID generator unless it is certain that the AID has been deactivated. If the device does deliver a countershock, an unpleasant 6-ms, 60-V skin potential may be felt by anyone in contact with the patient.[13]

Problems may occur if internal or external countershocks must be administered to a patient with an AID in situ. Internal shocks can damage an AID[13]; external shocks usually will not damage an AID, but part of the shock current may be shunted through the unit. Interchanging the paddle positions on the chest may help if an initial countershock attempt fails.[22]

AIDs have been successfully implanted in several patients who already had pacemakers in situ. Potentially, pacing spikes could confuse the AID into believing that no dysrhythmia exists when in fact it does. While it has been designed to avoid this problem, there is not yet enough experience with the device to know if this may be a significant limitation of the AID.[21] Because instruments for noninvasive programming of pacemakers may incorporate a permanent magnet that could initiate the AID test mode cycle, programmable pacemakers should be implanted at least 6 inches from an AID.[10]

TABLE 1. Summary of Clinical Results of AID Implantation

	Stanford[12]	Johns Hopkins[14]
Patients	50	65
Disease states		
Coronary artery disease	34	51
Myopathy	7	11
No heart disease	3	1
Others	7	2
Mean follow-up	8 months	15.6 months
Range	(1–33)	(12–34)
Mortality		
Early	1	4
Late	2	13
AID shocks delivered	300	48

Data from referenced series. Some categories have been consolidated for consistency between studies.

FUTURE DEVELOPMENT

The AID-B® has undergone considerable change as a result of the clinical experience and will continue to undergo development. Future models may incorporate pacemaker capabilities. The energy of the shocks and the rate threshold may be noninvasively programmable. Refinements in battery and capacitor technology may allow longer device lifetime (now estimated at about 2 years or 100 shocks)[13]; further miniaturization of the device may be possible.

Other AID devices are under development that may eliminate the need for the apical patch electrode[20] by
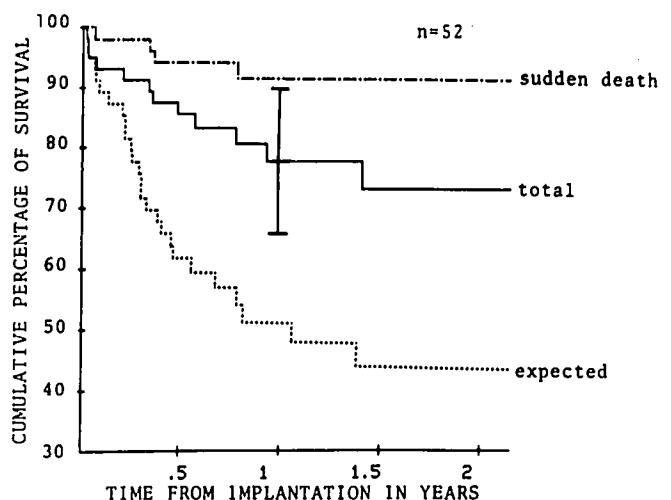

FIG. 5. Life-table analysis of survival following AID implantation. The upper curve represents unwitnessed deaths (assumed to be "sudden death") in patients receiving AIDs. The middle curve represents the total survival experience of these patients. The bottom curve is the estimated survival experience of the AID group had they not received the defibrillator. The error bar shows the 95% confidence limits of the total mortality experience of the AID group at 1 year. The mortality experience of the AID group is clearly superior to the group receiving conventional therapy alone. Received from Mirowski M, Reid PR, Winkle RA: Mortality in patients with implanted automatic defibrillators. Ann Intern Med 98:585–588, 1983, with permission of the publisher.

using only intravascular or intracardiac electrodes. Clinical experience with a "catheter-only" cardioverter has been recently reported.[23] This device is able to convert ventricular tachycardia but lacks the energy capacity to terminate ventricular fibrillation. In one of seven patients receiving the cardioverter, the inability of the dysrhythmia diagnosis system to differentiate atrial fibrillation with rapid ventricular response from ventricular tachycardia led to multiple countershocks, culminating in the production of ventricular fibrillation.[23] This technology is, therefore, not currently a replacement for higher energy AIDs like the AID-B®. However, if further development of such "catheter-only" AIDs provides for better dysrhythmia differentiation and the ability to reliably defibrillate, implantation of AIDs might no longer require general anesthesia.

Conclusions

In the majority of patients with ventricular dysrhythmias, pharmacologic control is effective. For those patients who are unresponsive to drug treatment, the best therapeutic combination for improvement of life expectancy may be electrophysiologic testing, appropriate cardiac surgery, and implantation of an automatic cardioverter/defibrillator. Many new centers are beginning AID implantations,[24] and substantially more anesthesiologists will become involved in the care of these patients. An understanding of the principles of these devices and the special problems associated with these patients as outlined above will make anesthesia safer for this high-risk group.

Addendum. Since acceptance of this article, an updated and more detailed description of the clinical experience with the AID-B® has been published: Echt DS, Armstrong K, Schmidt P, Oyer PE, Stinson EB, Winkle RA: Clinical experience, complications, and survival in 70 patients with the automatic implantable cardioverter/defibrillator. Circulation 71:289–296, 1985.

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