JACC Vol. 6, No. 2
August 1985:461–6

REVIEWS

The Automatic Implantable Cardioverter-Defibrillator: An Overview

M. MIROWSKI, MD, FACC

Baltimore, Maryland

The automatic implantable cardioverter-defibrillator continuously monitors the heart, identifies malignant ventricular tachyarrhythmias and then delivers electrical countershock to restore normal rhythm. There are two defibrillating electrodes which are also used for waveform analysis; one is located in the superior vena cava and the other is placed over the cardiac apex. A third bipolar right ventricular electrode is used for rate counting and R wave synchronization. When ventricular fibrillation occurs, a 25 J pulse is delivered; when ventricular tachycardia faster than the preset rate is detected, the discharge is R wave-synchronized. The clinical evaluation study of this therapeutic method began in February 1980 in patients with recurrent refractory life-threatening ventricular tachyarrhythmias. So far, the device has been implanted in nearly 500 patients with a follow-up period of up to 59 months. The risks and complications associated with this treatment were found to be moderate. Actuarial analysis has demonstrated significant impact on the survival rate of the patients receiving implants with 1 year arrhythmic mortality rate reduced to 2% or less in all groups analyzed.

The available data indicate that the automatic cardioverter-defibrillator can reliably identify and correct potentially lethal ventricular tachyarrhythmias, leading to a substantial improvement in survival in properly selected high risk patients.

(J Am Coll Cardiol 1985;6:461–6)

Management of patients with malignant ventricular arrhythmias occurring outside the hospital continues to represent a formidable challenge. The early hopes placed in prophylactic antiarrhythmic therapy have not yet materialized, and the delivery of a sufficiently strong electrical countershock remains the mainstay of any attempt to terminate ventricular fibrillation and many hemodynamically unstable ventricular tachycardias.

Inasmuch as direct current cardioversion is critically dependent on the rapid availability of medical personnel and equipment, its implementation outside the hospital is rarely successful. To address this problem, the concept of an implantable, fully automatic device capable of identifying and treating life-threatening ventricular arrhythmias was brought forward (1,2). More than 10 years elapsed, however, before such a device could be developed and applied clinically (3). Today, the automatic implantable cardioverter-defibrillator is used with increasing frequency to prevent sudden arrhythmic death in high risk patients. This article will review and summarize the accumulated clinical experience with this new therapeutic method (3–26). The preclinical phase of this work has been reviewed elsewhere (27,28).

From the Departments of Medicine, Sinai Hospital of Baltimore and The Johns Hopkins Medical Institutions, Baltimore, Maryland. Manuscript received January 29, 1985, accepted March 2, 1985.

Address for reprints: M. Mirowski, MD, Sinai Hospital of Baltimore, Baltimore, Maryland 21215.

The Device

The automatic implantable cardioverter-defibrillator is a self-contained automatic system with extensive monitoring, diagnostic and therapeutic capabilities. It is an advanced version of the original automatic implantable defibrillator (AID, Intec Systems, Inc.) introduced into clinical practice in 1980 (3). While the original defibrillator was designed to correct only ventricular fibrillation, the new device also identifies and treats ventricular tachycardias. The addition of cardioverting capabilities has significantly increased the versatility and the clinical impact of the system.

The new device consists of a pulse generator and three electrode leads. The pulse generator weighs 292 g and has a volume of 162 cc. It is housed in a hermetically-sealed titanium case containing the electronic components and power sources; the latter are expected to provide nearly 3 years of monitoring life or the delivery of about 100 discharges. Two transcardiac electrodes deliver the electrical countershock directly to the heart: one catheter electrode is incorporated into an intravascular catheter positioned in the superior vena cava, while the second, a flexible rectangular patch, covers the apex of the heart; these defibrillating electrodes also sense the configuration of the cardiac electrogram. The third lead is a separate catheter containing two closely spaced electrodes on its tip, which is wedged into the right ventricular apex; it provides input signals for heart rate determination and R wave synchronization. Whenever the chest

0735-1097/85/$3.30

JACC Vol. 6, No. 2
August 1985:461–6

is open during the implantation procedure, two epicardial screw-in electrodes can be substituted for this right ventricular rate channel.

The automatic implantable cardioverter-defibrillator continuously monitors and analyzes the patient's heart rate and waveform configuration (29). The latter is expressed in terms of the probability density function, which reflects the time spent by the electrical signal near the isoelectric line. The device makes the diagnosis of "treatment-requiring" arrhythmia when both the heart rate and probability density function exceed critical values, an event that initiates the capacitor charging cycle. When the charge on the capacitors reaches approximately 720 V, a truncated exponential pulse (30) of 25 J is delivered approximately 17 seconds from arrhythmia onset. In the presence of ventricular tachycardia, the pulse delivery is synchronous with the R wave. If the initial discharge does not terminate the abnormal rhythm, the device delivers additional pulses of 30 J each; up to three recyclings may occur during a single arrhythmic episode. Devices with higher energy outputs also are available for use in patients with increased defibrillation threshold. The pulse generator can be deactivated and reactivated at will with the proper use of a magnet.

In addition to the standard automatic implantable cardioverter-defibrillator characterized by the dual detection algorithm, a variant of the device which features a sensing system that relies only on the analysis of heart rate is also available. This "rate only" version of the cardioverter-defibrillator preferred by some investigators (17–20), is more sensitive than the standard unit and theoretically less likely to miss ventricular tachycardias with narrow QRS complexes, but it will also deliver pulses during supraventricular arrhythmias that are faster than the present rate cutoff value of the device.

Testing the device. This outline of the basic structural and functional characteristics of the system would not be complete without mentioning techniques for noninvasively communicating with the cardioverter-defibrillator. By magnetically triggering coded audio signals generated by a built-in piezoelectric transducer and by using a specially designed device, the AIDCHECK (Intec Systems, Inc.), it is possible to interrogate the automatic implantable cardioverter-defibrillator and to obtain information about such clinically important variables as the integrity of the sensing function, the status (active or inactive) of the pulse generator, the degree of battery depletion, capacitor deformation and the cumulative number of pulses that the unit has delivered to the patient. These tests are performed before, during and after implantation.

The Clinical Study

The clinical evaluation of the automatic implantable defibrillator and subsequently of the automatic implantable cardioverter-defibrillator began in February 1980 with the first implantation of the device in a human being (3). Carried out initially on a limited number of patients, first at The Johns Hopkins Hospital and later at Stanford University Hospital, Stanford, California, the study followed rigidly defined criteria and centered on a careful search for weak links in the implanted system. As a direct corollary of this effort, many improvements have been incorporated into the device, better patient selection guidelines have been defined and new procedures and testing techniques have been developed. Encouraging clinical results led to progressive expansion of the study to additional institutions in this and other countries. By the end of 1984, the number of patients who had received an implant was 488; 5,433 pulse generator implant-months had been accumulated; the longest follow-up was 59 months and the mean follow-up was slightly greater than 11 months.

Patient group. *Selection of patients.* Only patients identified as being at extremely high risk of dying from refractory malignant arrhythmias were considered candidates for the procedure. Initially, the patients who were to receive implants were required to have had at least two previous cardiac arrests not associated with acute myocardial infarction; one such episode had to occur despite drug therapy and with the malignant arrhythmia documented at least once. Patients were excluded if their life expectancy was significantly limited by noncardiac disease, if they were receiving drugs (other than antiarrhythmic drugs) known to influence electrical activity of the heart or if psychologic disabilities were present. However, evidence of advanced left ventricular dysfunction was never a contraindication for the procedure.

Currently, the criteria for implantation have become somewhat less stringent. For a patient to be considered for the procedure, only a single episode of ventricular fibrillation or hemodynamically unstable ventricular tachycardia occurring outside the context of acute myocardial infarction is required, provided that there is evidence of incomplete protection by antiarrhythmic drugs, as determined by arrhythmia inducibility during electrophysiologic or stress testing or by the inability to suppress complex ventricular arrhythmias on Holter monitor recordings.

Patient characteristics. This relative relaxation of the entry criteria into the study has done little to change the clinical and demographic characteristics of the patient group. Survivors of recurrent cardiac death still form the great majority of patients receiving implants; for example, the average number of arrhythmic cardiac arrests in 112 patients operated on through September 1984 at The Johns Hopkins and Sinai Hospitals in Baltimore is 3.5 (the two Institutions hereafter will be referred to as Hopkins). The clinical profile of this group seems to be similar to that of patients operated on at other centers. There were 85 men and 27 women whose ages ranged between 16 and 76 years (mean 53). The average ejection fraction was 32%. Eighty-five patients

had coronary artery disease and 23 had nonischemic cardiomyopathy. Prolonged QT interval was present in two and the remaining two patients had primary electrical disease. Before implantation, these patients failed aggressive medical and surgical management and had not responded (on average) to 4.5 antiarrhythmic drugs; 15 patients were treated with coronary bypass grafting, 1 had a myectomy for relief of hypertrophic subaortic stenosis and 10 had had a permanent electronic pacemaker implanted.

Surgical Approaches

Whereas the superior vena cava and the right ventricular electrode catheters were introduced into their intended location using the conventional pervenous technique, proper placement of the apical patch electrode requires a surgical approach. Initially, a median sternotomy or a left lateral thoracotomy was employed for this purpose (14). Subsequently, simpler techniques were developed. The subxiphoid approach, for example, uses a small incision below the xiphoid process to enter the pericardial space anteriorly, allowing the patch electrode to be extended laterally over the apex and sutured proximally to the pericardium (15). More recently, a subcostal approach has also been suggested (21). This technique combines the advantages of a relatively minor surgical procedure with excellent exposure of the left ventricle. Whichever technique is selected, the leads are channeled under the skin and connected to the pulse generator placed in a paraumbilical pocket.

In some centers, a thoracotomy is the preferred surgical approach because it provides the best possible exposure for placement of the patch or even of two patches, which are occasionally necessary to achieve effective defibrillation (17–20,22). At Hopkins, the choice of the implantation technique is determined by clinical circumstances. Thus, median sternotomy is performed when the implantation procedure is associated with corrective open heart surgery, whereas the subxiphoid and subcostal approaches are reserved for patients in whom concomitant cardiac surgery is not indicated. Lateral thoracotomy is used to avoid scar tissue in patients who previously underwent cardiac surgery by sternotomy.

Concomitant cardiovascular surgery. Whenever indicated, implantation of the automatic implantable cardioverter-defibrillator at Hopkins was associated with additional cardiac and particularly antiarrhythmic surgery. In the previously mentioned series of 112 patients receiving implants, 26 underwent mapping-directed endocardial resection, associated in 17 with an aneurysmectomy and in 13 with coronary artery bypass grafting. Another 13 patients had only coronary bypass grafting, and 1 of these had mitral valve replacement. The rationale behind combining implantation of the device with other cardiac procedures is to provide the patient with optimal protection from the lethal arrhythmia. For example, endocardial resection markedly reduces the number of subsequent arrhythmic events but does not eliminate them in all cases because of incomplete ablation of the arrhythmogenic foci or progression of the underlying disease process (16). Approximately 20% of the Hopkins patients who underwent antiarrhythmic surgery had recurrences of malignant arrhythmias, with the automatic implantable cardioverter-defibrillator providing them with a unique backup system to ensure their long-term safety (25).

There is no hard evidence to indicate that coronary artery bypass grafting plays a role in the prevention of sudden arrhythmic death. However, improved vascularization of the heart could be expected, at least on theoretical grounds, to decrease or even eliminate myocardial ischemia capable of triggering a lethal arrhythmia.

Electrophysiologic Evaluation

Before implantation, electrophysiologic testing using programmed electrical stimulation is performed to determine the inducibility of ventricular arrhythmias and the characteristics of those induced. During implantation, output signals from the transcardiac leads and from the rate channel are recorded and analyzed; at Hopkins, lead impedance is also routinely calculated. Malignant arrhythmias are then induced with low level alternating current (13) and the amount of energy required for their termination is measured with an external, nonautomatic pulse generator that uses a waveform identical to that of the automatic inducible cardioverter-defibrillator. The determination of the defibrillation threshold has so far been shown to be a safe procedure, extremely helpful for proper selection of the type of leads to be used, their optimal locations and of the characteristics of the device to be implanted (10,18). After implantation, the patient's malignant arrhythmia is reinduced and the automatic functions of the device are tested. No patient is discharged from the hospital with this device unless its lifesaving capability has been demonstrated.

Functional Variables

The ability of the automatic implantable cardioverter-defibrillator to perform its tasks has been studied in a variety of clinical settings. The monitoring capabilities of the system, the reliability of arrhythmia recognition and the effectiveness and ease of arrhythmia termination have been subjected to particularly close scrutiny. The great majority of the implanted pulse generators achieved their predicted monitoring life in accordance with specifications. However, accelerated battery depletion was observed in some 9% of the devices. The problem was traced to corrosion of the glass insulator in the feedthrough connectors of the battery and has been corrected by application of a protective coating.

The diagnostic accuracy of the device has been found to

JACC Vol. 6, No. 2
August 1985:461–6

be excellent. In the controlled conditions of the electrophysiology laboratory, induced ventricular fibrillation and ventricular tachycardia were correctly identified in 99% of the cases (26). When the proper diagnosis was not made, the cause was easily recognized and corrected. The handful of false negative diagnoses was usually due to lead malposition, 60 cycle interference or interaction with an implanted unipolar pacemaker.

Termination of the malignant arrhythmias was generally accomplished with a single 25 J internal discharge; in rare instances, one or more recyclings were necessary to terminate the abnormal rhythm. The time from the induction of the arrhythmia until its termination ranged between 11 and 36 seconds (average 17). Postdischarge bradycardias were rarely observed.

High threshold. Because of a high defibrillation threshold, standard 25 J discharges delivered through the superior vena cava patch lead configuration were ineffective in restoring normal rhythm in 12% of patients. The increased threshold could not clearly be related to underlying cardiac disease process, size of the heart or extent of left ventricular dysfunction (18). In some patients, when high threshold was associated with hypokalemia or amiodarone therapy, an appropriate electrolyte or pharmacologic adjustment lowered energy requirements (9,24). In others, the problem was remedied by replacing the standard patch electrode with one of a larger size, substituting a second patch electrode for the superior vena cava electrode or searching for more effective lead positions. In particularly difficult cases, implantation of a high energy output pulse generator was required.

Subjective reactions. The internal discharges were generally well tolerated, even when delivered to conscious patients. The subjective reactions ranged from lack of any perceptible sensation to a very painful one, but most of the patients receiving the implants described the discharge as a moderate blow to the chest resulting in a momentary discomfort. No serious emotional problems were observed among the Hopkins patients receiving implants; however, Stanford investigators (20) reported a different experience in patients who had received a large number of discharges within a very short period of time. In the presence of frequent repetitive shocks, it is advisable to temporarily deactivate the unit and stabilize the rhythm in a hospital setting with pharmacologic and other means.

Control of acceleration. Clinical observations also have demonstrated the ability of the device to deal effectively with the phenomenon of acceleration, which can occur whenever ventricular tachycardia is treated electrically with external or transvenous cardioversion (31), antitachycardia pacing (32) or the automatic implantable cardioverter-defibrillator. Under these circumstances the tachycardia, rather than being terminated, may accelerate into a faster, usually less organized rhythm or even degenerate into ventricular fibrillation. This unpredictable development can be observed at all energy levels (18). Although synchronization of the discharge to the R wave decreases the incidence of acceleration, it does not eliminate the phenomenon completely. The cardioverter-defibrillator is the only implantable antitachycardia system capable of controlling acceleration automatically through recycling: the device recognizes the accelerated rhythm de novo and corrects it with one or more subsequent discharges. Several examples of accelerated rhythms so terminated by the device have been reported (6,7).

Complications

The risks and complications observed during the clinical evaluation study of the automatic implantable cardioverter-defibrillator comprise a broad spectrum. As a result of close clinical and engineering interaction, the great majority of problems have been solved and are now of historic interest only. Currently, two potential risk areas should be considered whenever implantation is being contemplated, one related to the methods employed and the other reflecting the design characteristics of the device.

Surgical complications. Among the risks related to methods, surgical complications are the most important. In the Hopkins series of 112 patients receiving implants, 1 operative death occurred as a result of perforation of the subclavian vein by a polyethylene central catheter. Infection occurred in six patients: in four the primary site was the pulse generator pocket, in one it was located at the antecubital cutdown site while in another the origin of infection was unknown. In two cases of infection, complete explanation of the system was required. Postoperative bleeding necessitated transfusions in two patients. Occasional accumulation of sterile fluid in the pulse generator pocket was always uneventfully reabsorbed. Transient pericardial rubs were the rule after implantation. One episode of superior vena cava thrombosis responded well to anticoagulant drugs, but no embolic phenomena were noted. Lead dislodgment requiring repositioning occurred in seven patients; recently, better fixation techniques have reduced the incidence of this complication. No adverse effects related to electrophysiologic testing were observed.

Malfunction. The new technology behind the automatic implantable cardioverter-defibrillator has given rise to a different set of problems. As with any complex system, malfunction may occur. The types of malfunction observed during this study include hermeticity loss, breakdown of the gaseous dielectric, misdirection of the battery testing pulse toward the patient and random component failure. These complications were rare and did not result in permanent harm.

Particularly significant from a clinical viewpoint are false positive discharges. In the early stages of the study, spurious

signals generated by fractured leads or miscounting of the heart rate caused most of the oversensing. With improvements in lead construction and the introduction of a separate rate channel, spurious discharges due to these causes have been virtually eliminated.

Today, oversensing may still occur as a result of interaction of an implanted unipolar pacemaker with the device (23) or particularly fast supraventricular arrhythmias that satisfy the sensing algorithm of the device. Pulse delivery during sinus rhythm can also be caused by nonsustained ventricular tachycardias. Although the sensing function in these instances is entirely appropriate, once the diagnosis is made and the capacitor charging cycle initiated, the device is committed to discharge, even if sinus rhythm has been restored in the interim.

Prevention of unwanted discharges during sinus rhythm. Although these have not induced significant arrhythmias or resulted in any other serious effects, they are clearly undesirable. The incidence of such discharges can be decreased by implementing a few simple clinical measures. The automatic implantable cardioverter-defibrillator to be implanted should have a cutoff rate that is greater than that of the patient's fastest sinus rhythm and less than the rate of his or her ventricular arrhythmia. Moreover, pharmacologic interventions can be used after implantation to modify the patient's rhythm favorably. In patients who require treatment with both this device and a pacemaker, only a bipolar rather than a unipolar pacemaker should be used. Frequent nonsustained ventricular tachycardias should also be controlled with antiarrhythmic medication. During intraoperative electrocautery and in the immediate postoperative period when supraventricular tachyarrhythmias frequently occur, the device should be temporarily deactivated.

Mortality

Because the chief objective of the automatic implantable cardioverter-defibrillator is to prevent sudden arrhythmic death, it is appropriate to examine the impact, if any, this therapeutic intervention has had on the mortality rate of the patients receiving implants. The unique clinical characteristics of the study group and its relatively rapid growth have already made this information available.

The initial data were derived from the analysis of the first 52 patients receiving implants, 42 from Hopkins and 10 from Stanford, the great majority of whom were treated with the original automatic implantable defibrillator device (5). Kaplan-Meier survival curves in this group indicated a total 1 year mortality rate of 22.9% and an arrhythmic 1 year mortality rate of 8.5%. With the increase in the number of patients receiving implants, it was soon possible to compare the respective effectiveness of the original defibrillator with the new one, which treats both ventricular fibrillation and ventricular tachycardia. At 1 year follow-up, the arrhythmic mortality rate of 32 patients who received the original device was 10.6%, and in the 67 patients treated with the new device it was only 2%; the total 1 year mortality rate was 26 and 16.6%, respectively (7). Virtually identical figures were subsequently found in a larger Hopkins series of 112 patients (11).

Survival analysis was also performed in a series of 70 patients treated mainly with the "rate only" type of automatic implantable cardioverter-defibrillator at Stanford University Hospital (20). In this series, Kaplan-Meier curves revealed an arrhythmic 1 year mortality rate of 1.8%. The manufacturer of the device has recently reported to the U.S. Food and Drug Administration data from 323 patients receiving implants (a group that overlaps with the previously described Hopkins and Stanford series) (26). These data were also analyzed in accordance with the type of device the patients received: the 1 year arrhythmic mortality rate was 11.9% in 37 patients treated with an automatic implantable defibrillator, 1.9% in 209 patients treated with the standard automatic implantable cardioverter-defibrillator and 1.3% in 95 patients who received the "rate only" cardioverter-defibrillator.

All of these results are remarkably concordant. While the expected mortality rate of the types of patients included in the study ranged, according to various historical controls, between 27 and 66% (33–39), the model that treated only ventricular fibrillation decreased the 1 year arrhythmic mortality rate to approximately 11%. On the other hand, the improved, currently used automatic implantable cardioverter-defibrillator model reduced this mortality rate to 2% or less, virtually eradicating sudden arrhythmic death during the year after implantation of the device.

Conclusion

During the past 5 years, automatic implantable defibrillating systems have been evaluated for safety and efficacy. The automatic implantable cardioverter-defibrillator, the most recent generation of the device, has been shown capable of reliably monitoring the heart for prolonged periods of time, identifying ventricular fibrillation and the broad spectrum of ventricular tachycardias and terminating them with an internal countershock. The risks and complications associated with the use of the device have been minimal. The available evidence indicates that implantation of this device dramatically decreases the incidence of sudden arrhythmic death in the so treated high risk patients.

References

1. Mirowski M, Mower MM, Staewen WS, Tabatznik B, Mendeloff AI. Standby automatic defibrillator: an approach to prevention of sudden coronary death. Arch Intern Med 1970;126:158–61.

2. Schuder JC, Stoeckle H, Gold JC, et al. Experimental ventricular

defibrillation with an automatic and completely implanted system. Trans Am Soc Artif Intern Organs 1970;16:207–12.

3. Mirowski M, Reid PR, Mower MM, et al. Termination of malignant ventricular arrhythmias with an implanted automatic defibrillator in human beings. N Engl J Med 1980;303:322–4.
4. Mirowski M, Reid PR, Watkins L, et al. Clinical treatment of life-threatening ventricular tachyarrhythmias with the automatic implantable defibrillator. Am Heart J 1981;102:265–70.
5. Mirowski M, Reid PR, Winkle RA, et al. Mortality in patients with implanted automatic defibrillators. Ann Intern Med 1983;98:585–8.
6. Mirowski M, Reid PR, Mower MM, et al. The automatic implantable cardioverter-defibrillator. PACE 1984;7:534–40.
7. Mirowski M, Reid PR, Mower MM, et al. Clinical performance of the implantable cardioverter-defibrillator. PACE 1984;7:1345–50.
8. Reid PR, Mirowski M, Mower MM, et al. Clinical evaluation of the internal automatic cardioverter-defibrillator in survivors of sudden cardiac death. Am J Cardiol 1983;51:1608–13.
9. Reid PR, Mower MM, Mirowski M. Pathophysiology of ventricular tachyarrhythmias amenable to electric control. PACE 1984;7:505–13.
10. Reid PR, Griffith LSC, Mower MM, et al. Implantable cardioverter-defibrillator: patient selection and implantation protocol. PACE 1984;7:1338–44.
11. Reid PR, Mower MM, Griffith LSC, et al. Comparative effects on mortality of the first and second generation implantable defibrillators (abstr). Circulation 1984;70(suppl II):II-401.
12. Mower M, Reid PR, Watkins L Jr, Mirowski M. Use of alternating current during diagnostic electrophysiologic studies. Circulation 1983;67:69–72.
13. Mower M, Reid PR, Watkins L Jr, et al. Automatic implantable cardioverter-defibrillator: structural characteristics. PACE 1984;7:1331–7.
14. Watkins L Jr, Mirowski M, Mower MM, et al. Automatic defibrillation in man: the initial surgical experience. J Thorac Cardiovasc Surg 1981;82:492–500.
15. Watkins L Jr, Mirowski M, Mower MM, et al. Implantation of the automatic defibrillator: the subxiphoidal approach. Ann Thorac Surg 1982;34:515–20.
16. Watkins L Jr, Platia EV, Mirowski M, et al. Treatment of malignant ventricular arrhythmias with combined endocardial resection and implantation of the automatic defibrillator: preliminary report. Ann Thorac Surg 1984;37:60–6.
17. Winkle RA, Bach SM, Echt DS, et al. The automatic implantable defibrillator: local ventricular bipolar sensing to detect ventricular tachycardia and fibrillation. Am J Cardiol 1983;52:265–70.
18. Winkle RA, Stinson EB, Bach SM Jr, et al. Measurement of cardioversion/defibrillation thresholds in man by a truncated exponential waveform and an apical patch-superior vena caval spring electrode configuration. Circulation 1984;69:766–71.
19. Winkle RA, Stinson EB, Echt DS, Mead RH, Schmidt P. Practical aspects of automatic cardioverter/defibrillator implantation. Am Heart J 1984;108:1335–46.
20. 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 1985;71:289–6.
21. Lawrie GM, Griffin JC, Wyndham CRC. Epicardial implantation of the automatic implantable defibrillator by left subcostal thoracotomy. PACE 1984;7:1370–4.
22. Brodman R, Fisher JD, Furman S, et al. Implantation of automatic cardioverter-defibrillators via median sternotomy. PACE 1984;7:1363–9.
23. Bach SM. Aid-B cardioverter-defibrillator: possible interaction with pacemakers. Intec Systems Technical Communication, August 1983.
24. Fogoros RN. Amiodarone-induced refractoriness to cardioversion. Ann Intern Med 1984;100:699–700.
25. Platia EV, Reid PR, Watkins L Jr, et al. Endocardial resection combined with automatic cardioverter-defibrillator implantation in patients with malignant ventricular tachycardias: late followup (abstr). Circulation 1984;70(suppl II):II-413.
26. Intec Systems, Inc. Report of the clinical investigation: automatic implantable cardioverter-defibrillator. November 9, 1984.
27. Mirowski M, Reid PR, Mower MM, et al. The automatic implantable cardioverter-defibrillator. PACE 1984;7:534–40.
28. Winkle RA. The implantable defibrillator in ventricular arrhythmias. Hosp Pract 1983;18:149–65.
29. Langer A, Heilman MS, Mower MM, et al. Considerations in the development of the automatic implantable defibrillator. Med Instrum 1976;10:163–7.
30. Schuder JC, Rahmoeller GA, Stoeckle H. Transthoracic ventricular defibrillation with triangular and trapezoidal waveforms. Circ Res 1966;19:689–94.
31. Zipes DP, Heger JJ, Miles WM, et al. Early experience with an implantable cardioverter. N Engl J Med 1984;311:485–90.
32. Fisher JD, Kim SG, Matos JA, Waspe LE. Pacing for ventricular tachycardia. PACE 1984;7:1278–90.
33. Schulze RA Jr, Strauss HW, Pitt B. Sudden death in the year following myocardial infarction: relation to ventricular premature contractions in the late hospital phase and left ventricular ejection fraction. Am J Med 1977;62:192–9.
34. Swerdlow CD, Winkle RA, Mason JW. Determinants of survival in patients with ventricular tachyarrhythmias. N Engl J Med 1983;308:1436–42.
35. Cobb LA, Baum RS, Alvarez H III, Schaffer WA. Resuscitation from out-of-hospital ventricular fibrillation: 4 years follow-up. Circulation 1975;52(suppl III):III-223–8.
36. Graboys TB, Lown B, Podrid PJ, Desilva R. Long-term survival of patients with malignant ventricular arrhythmias treated with antiarrhythmic drugs. Am J Cardiol 1982;50:437–43.
37. Bigger JT, Weld FM, Rolnitzky LM. Prevalence, characteristics and significance of ventricular tachycardia (three or more complexes) detected with ambulatory electrocardiographic recording in the late hospital phase of acute myocardial infarction. Am J Cardiol 1981;48:815–23.
38. Hamer A, Vohra J, Hunt D, Sloman G. Prediction of sudden death by electrophysiologic studies in high risk patients surviving acute myocardial infarction. Am J Cardiol 1982;50:223–9.
39. Richards DA, Cody DV, Denniss AR, Russell PA, Uther JB, Young AA. Ventricular electrical instability during the first year following myocardial infarction (abstr). Am J Cardiol 1982;49:929.