Termination of Malignant Ventricular Arrhythmias with an Implanted Automatic Defibrillator in Human Beings


Article

The development of a clinically applicable, automatic, implantable defibrillator has been described previously.1 This electronic device is designed to monitor cardiac electrical activity, to recognize ventricular fibrillation and ventricular tachyarrhythmias with a sinusoidal wave form, and then to deliver corrective defibrillatory discharges. It is intended to protect patients at particularly high risk of sudden death whenever and wherever they are stricken by these lethal arrhythmias.

After extensive preclinical testing, 2 a pilot study of this new technique was recently initiated at The Johns Hopkins Hospital. This article describes the first three patients in whom the automatic defibrillator was implanted to manage recurrent ventricular tachyarrhythmias that were refractory to medical therapy. Our results suggest that the device can successfully identify and reverse these malignant arrhythmias in human beings.

The automatic implantable defibrillator (developed and manufactured under the name of AIDTM defibrillator by Medrad/Intec Systems, Pittsburgh, Penn.) is encased in titanium, weighs 250 g, and occupies a volume of 145 ml. Lithium
batteries provide a projected monitoring life of three years or the capability of delivering approximately 100 discharges. One defibrillating electrode is located on an intravascular catheter placed in the superior vena cava near the right atrial junction.3 The second electrode, in the form of a cup4 or of a flexible rectangular patch, is placed extrapericardially over the cardiac apex.

The sensing system monitors a sampled probability-density function of the ventricular electrical activity; this function reflects the time spent by the input signal between two amplitude limits located near zero potential.5 For all practical purposes, ventricular fibrillation is identified by the striking absence of isoelectric potential segments. The automatic defibrillator will also respond to ventricular flutter and tachycardia characterized by sinusoidal waveform. When a serious arrhythmia is detected, the device delivers a truncated exponential pulse6 of 25 J; it can recycle three times during a single episode if previous discharges are ineffective, with the strength of the third and fourth pulses increased to 30 J.

The operational readiness of the automatic defibrillator can be evaluated before and after implantation7 by an external analyzer (developed and manufactured under the name of AIDCHECKTM by Medrad/Intec Systems, Pittsburgh, Penn.). The capacitor charging cycle is initiated with a magnet, and the charge is delivered into a built-in test-load resistor. Progressive increases in charging time reflect battery depletion, whereas failure to initiate the cycle indicates abnormal operation of the device. Continuous application of the magnet over the pulse generator disables the device completely if necessary.

An external recorder (developed by The Johns Hopkins University Applied Physics Laboratory) is available to monitor the long-term performance of the implanted defibrillator. Triggered by the defibrillatory shock, it stores 22.5 seconds of electrocardiographic recording preceding the discharge and 67.5 seconds following it, together with the number of fibrillating episodes, the total number of pulses applied, the time at which the episode occurred, and the time that elapsed since the last readout.

The superior vena cava electrode catheter is introduced into the left internal jugular vein and positioned under fluoroscopy. The thorax is entered via the fifth intercostal space, and the apical electrode sutured to the pericardium over the cardiac apex. Both electrode leads are passed under the left costal margin. After proper insertion of the leads into the receptacle of the pulse generator, the unit is placed in a subcutaneous abdominal pocket.

A 57-year-old woman had an inferior myocardial infarction complicated by ventricular fibrillation eight years before the most recent admission; intractable angina associated with ventricular arrhythmias then developed. Coronary-artery
bypass improved the angina but the arrhythmias remained refractory to propranolol, digitalis, quinidine, and procainamide. Two months before admission, ventricular fibrillation occurred outside the hospital and required multiple defibrillations. There was no evidence of acute myocardial infarction. The arrhythmias persisted, and during electrophysiologic testing ventricular tachycardia and fibrillation were repeatedly induced despite therapeutic doses of lidocaine, quinidine, and encaïnide. The automatic defibrillator was implanted on February 4, 1980.

Two weeks later, ventricular flutter was induced during an electrophysiologic study and automatically terminated by the implanted defibrillator. The patient was discharged in satisfactory condition and has remained well over a five-month follow-up period.

A 16-year-old boy was resuscitated from ventricular fibrillation four years before the most recent admission. Physical examination was unremarkable. Although the coronary arteries and left ventricular function were normal on cardiac catheterization, the papillary muscles were prominent. Ventricular tachycardia was induced during electrophysiologic testing. A demand pacemaker was implanted, and the patient was treated with quinidine, phenytoin, lidocaine, propranolol, procainamide, disopyramide, tocainide, and aprindine. These drugs, given alone and in various combinations, resulted in ataxia, lethargy, fatigue, and a decline in academic performance. Recurrent hypotensive ventricular tachycardias necessitated multiple readmissions for cardioversion and prompted a therapeutic trial of encaïnide.

When sustained hypotensive ventricular tachycardia requiring external cardioversion was again induced during electrophysiologic testing, the pacemaker pulse generator was removed, its lead capped in situ, and the automatic defibrillator implanted.

During studies performed after implantation while the patient was taking encaïnide, hypotensive ventricular tachycardia was induced at a rate of 171 beats per minute. An internal discharge delivered 50 seconds later did not terminate the arrhythmia but appeared to accelerate the rhythm into a ventricular flutter-fibrillation at 280 beats per minute. External cardioversion restored normal sinus rhythm.

During the next two weeks, the patient had five episodes of spontaneous hypotensive ventricular tachycardia, and all were automatically reverted to sinus rhythm by the implanted device. The conversions occurred before the patient lost consciousness and were not associated with undue discomfort or pain. Because of the frequent recurrence of these episodes, an external magnet was used to
inactivate the device. Efforts to optimize the pharmacologic treatment continue.

A 43-year-old man with a 10-year history of asymmetric cardiomyopathy had two episodes of ventricular fibrillation outside the hospital and was treated with propranolol, septal myectomy, and a pacemaker. Two months after the operation, progressive dyspnea developed and another episode of ventricular fibrillation occurred. Electrophysiologic testing induced hypotensive ventricular tachycardia despite adequate doses of propranolol and quinidine. Treatment with verapamil began, the pacemaker pulse generator was explanted, and the automatic defibrillator implanted. However, because the verapamil appeared to produce high-grade atrioventricular block, a pacemaker was reimplanted. During the procedure, pacing for a short time at a rate of 130 beats per minute triggered an internal discharge that did not evoke any abnormal rhythm and was well tolerated by the conscious patient. The patient was discharged in satisfactory condition and is taking verapamil.

The need to improve survival in patients likely to have malignant ventricular arrhythmias outside the hospital is generally recognized.8 The available strategies have been recently complemented by promising antiarrhythmic medications9, 10 and new surgical techniques.11 12 13 14 An effective implantable defibrillator could be a useful addition to our therapeutic armamentarium. Its potential capabilities — continuous monitoring, timely recognition of the lethal arrhythmia, and effective defibrillation — have the unique advantage of being permanently available to the patient at risk, without requiring the presence of specialized personnel or additional equipment.3, 15

Although our work has extended over more than a decade, 1 2 3 4 5, 16 17 18 19 20 21 22 23 the structural and functional requirements for an automatic defibrillator suitable for implantation in human beings have only recently been satisfied.1 Before this device was used clinically, an extensive testing program was completed, and an independent evaluation of the device carried out by The Johns Hopkins University Applied Physics Laboratory.2

The criteria for entry into the pilot clinical study were stringent. The initial subjects had to have survived at least two episodes of cardiac arrest not associated with acute myocardial infarction, with ventricular fibrillation documented at least once. One such episode had to have occurred despite presumably effective antiarrhythmic treatment. In the absence of such evidence, treatment with two antiarrhythmic agents given simultaneously was required. Patients who had other chronic or acute illness, who were taking drugs that were not antiarrhythmic agents but were known to influence the electrical activity of the heart, or who had psychological disabilities were excluded. The extremely poor prognosis of patients with these features is exemplified by the fact that four patients identified
as candidates for implantation of the device died before transfer to this hospital.

The surgical procedure was well tolerated, and no evidence of increased ventricular irritability was observed after implantation. To date, seven episodes of ventricular tachycardia and flutter-fibrillation have been documented. Five occurred spontaneously, and two were induced during electrophysiologic studies. All these arrhythmias were correctly identified, and six were automatically reverted to sinus rhythm with single 25–J pulses. In addition, no undue discomfort was observed when the discharge was delivered in conscious patients. During one electrophysiologic study, ventricular flutter-fibrillation possibly induced by the preceding unsuccessful pulse delivered during ventricular tachycardia was externally terminated. In another patient, a rapid ventricular-paced rhythm triggered the device but induced no arrhythmias. Although it was primarily designed to treat ventricular fibrillation, the device is thus responsive to ventricular tachyarrhythmias that satisfy the probability-density-function criteria.5

Although these preliminary results support the validity of the assumptions on which the automatic implantable defibrillator is based, we do not intend to suggest that most of the problems related to clinical implementation of this approach are solved. For example, the requirement for a thoracotomy represents a distinct but, we hope, a temporary drawback. Increased ventricular irritability after implantation of the device, although not observed in our patients or in previous animal studies, is a theoretical possibility. It is important to remember, moreover, that the implanted defibrillator, like implanted pacemakers, could malfunction because of failure of a component, battery depletion, improper sensing or pulsing, or case or lead fracture. Its effectiveness is also a function of the implantation technique, electrode type and configuration, and, most importantly, the individual defibrillation threshold. Although the available data18, 19, 24 25 26 suggest that the requirements will probably not exceed 25 to 30 J, in some damaged or large hearts this amount of energy might not restore normal rhythm. In addition, it is possible that a discharge could transform a ventricular tachycardia into ventricular flutter-fibrillation. Finally, the influence of ischemia and other metabolic and pharmacologic factors on the defibrillation threshold has yet to be investigated.

It is important to emphasize that the automatic defibrillator is not a definitive treatment for recurrent malignant arrhythmias and should not be regarded as a substitute for antiarrhythmic medication or other modes of therapy. These approaches should be viewed as complementary rather than exclusive. Even if safe and effective suppressive medication should become available, one could still risk a therapeutic failure due to individual idiosyncrasy or noncompliance, with the automatic defibrillator then providing a critical backup safety mechanism.
It should be kept in mind that the only purpose of this device is to achieve defibrillation automatically, before the victim of a lethal arrhythmia can be reached by a cardiac resuscitation team. In this situation, it does not matter whether the patient is defibrillated externally or internally.

In view of the increasing ability to identify patients at high risk, including many who are resuscitated from ventricular fibrillation in the absence of myocardial infarction, 27 28 29 the demonstrated effectiveness of the implantable defibrillator has important clinical implications. Although there is a clear need for additional information, our preliminary results are encouraging. If further studies provide evidence of long-term safety and reliability, the automatic implantable defibrillator could become useful to patients with a high likelihood of sudden arrhythmic death.

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