Measurement of cardioversion/defibrillation thresholds in man by a truncated exponential waveform and an apical patch–superior vena caval spring electrode configuration

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ABSTRACT Defibrillation/cardioversion thresholds were measured in 33 patients undergoing defibrillator implants. Each patient had a 12 cm² patch placed near the left ventricular apex via a thoracotomy and a 10 cm² spring lead placed peryvenously at the right atrial–superior vena caval junction. Ventricular tachycardia of stable morphology, polymorphic ventricular tachycardia, or ventricular fibrillation was induced four times in each patient and 1, 5, 10, and 25 J truncated exponential shocks with 60% tilt were given in a random sequence. The conversion rate was constant (77%, 86%, 87%, 85%) with increasing energy for ventricular tachycardia but progressively increased for polymorphic ventricular tachycardia and ventricular fibrillation (8%, 33%, 58%, 92%). The ventricular tachycardia acceleration rates for 1, 5, 10, and 25 J were 23%, 14%, 10%, and 15%. Patients not reliably converted with 25 J may require repositioning of leads or two patches. We conclude that for the spring-patch electrodes, increasing energy from 1 to 25 J improves the conversion rate for polymorphic ventricular tachycardia and ventricular fibrillation; the ventricular tachycardia conversion rate is constant. Acceleration of ventricular tachycardia occurs at all energies. Defibrillator implantation requires extensive intraoperative electrophysiologic testing to ensure safe and reliable termination of ventricular tachycardia and fibrillation.


ALTHOUGH the ability of electrical current to terminate ventricular fibrillation has been known for many years, the extensive investigation and widespread use of this method for the termination of serious sustained cardiac tachyarrhythmias did not occur until the 1960s and 1970s. Most of the experimental work with these techniques has evaluated them for the termination of ventricular fibrillation. The critical concept underlying defibrillation is that adequate current must be applied to the myocardium for sufficient time to terminate the arrhythmia.1–2 A number of waveforms have been evaluated for current delivery, including alternating current, damped sine waves, capacitor discharge, rectangular pulses, and truncated exponentials.2 The defibrillation threshold in animals and man is often a function of the waveform used, heart and body size, the species studied, and the electrode size and configuration.2–5 During the past decade, implantable devices have been developed that permit automatic internal cardioversion of ventricular tachycardia and defibrillation of ventricular fibrillation in man.5–13 There are problems in extrapolating data from previous investigations and prior clinical studies to these new implantable devices. Many prior studies were performed in species other than man and were carried out in animal preparations free of cardiovascular disease. Much of the reported human experience has been with transthoracic rather than direct defibrillation. Those studies evaluating direct defibrillation in man have done so in patients undergoing routine cardiac surgery and who do not have a history of sustained ventricular tachyarrhythmias. Finally, there has been no study of human cardioversion thresholds with electrodes similar to the spring-patch leads currently being implanted.

The availability of these implantable devices has made it both possible and desirable to evaluate the efficacy of arrhythmia termination in patients with his-
tories of episodes of life-threatening ventricular arrhythmias. Programmed stimulation can induce ventricular tachycardia in many of these patients, allowing evaluation of both cardioversion and defibrillation thresholds. We performed the present study of cardioversion and defibrillation thresholds with a superior vena caval spring–apical patch lead system and a truncated exponential waveform in a group of patients undergoing implantation of an automatic cardioverter/defibrillator for treatment of life-threatening ventricular arrhythmias.

Methods

Thirty-three subjects (27 men and six women) with histories of sustained ventricular tachycardia or fibrillation were studied. Each signed written informed consent for the operative placement of the automatic defibrillator, and the defibrillation threshold measurements were made as part of the implant procedure. Before the implant each patient provided a full history, and underwent physical examination and assessment of underlying cardiovascular disease, including coronary and left ventricular angiographic examinations. Each patient underwent a baseline electrophysiologic study, and many underwent serial electrophysiologic studies to evaluate efficacy of antiarrhythmic drugs. All antiarrhythmic drugs were withheld before the implant procedure.

Surgical procedure. The defibrillator leads and generator were implanted with patients under general anesthesia. A small incision was made below the left clavicle and the Seldinger technique was used to place a No. 14F sheath in the left subclavian vein. A titanium spring lead with approximately 10 cm² surface area was placed through this sheath and positioned with approximately half the electrode above the superior vena caval–right atrial junction and approximately half below this junction. In this configuration, the tip of the spring electrode was at or just above the middle right atrial level. A limited thoracotomy was performed in the sixth intercostal space and the cardiac apex was exposed. A 12 cm² titanium mesh patch was sutured in place near the cardiac apex. Two unipolar Medtronic Model 6917A-53T spring-in-sensing leads were inserted approximately 1 cm apart into the left ventricle in an area of healthy-appearing myocardium. The generator end of the superior vena caval lead was inserted into the thoracic cavity and all leads were tunneled to a subcutaneous pocket in the middle left abdomen. Temporary pacing wires were sutured to the epicardial surface of the heart for induction of arrhythmia. Exceptions to this operative procedure included eight patients who underwent a median sternotomy for simultaneous coronary artery bypass grafting and two patients who had right ventricular transvenous bipolar sensing electrodes rather than the epicardial screw-in leads.

Induction of arrhythmia. Ventricular tachycardia was induced with temporary pacing wires sutured to the left ventricle and programmed stimulation with up to three extrastimuli. Stimuli pulse widths varied from 2 to 6 msec with currents up to 5 mA. In patients whose arrhythmias were not easily induced with programmed stimulation or in whom ventricular fibrillation was the specific rhythm to be induced, induction of arrhythmia was accomplished with a rectified 60 Hz sine wave, which provided a 120 Hz signal of 20 V amplitude. This was applied across the temporary pacing electrodes for up to several seconds. Electrograms from surface leads I, II, III, aVL, aVF, and V₁, as well as instantaneous aortic pressure and the signal across the apical patch–superior vena caval spring leads and the bipolar sensing leads, were recorded with an Electronics for Medicine VR16 recorder at 25 to 100 mm/sec paper speed.

Cardioversion/defibrillation. The cardioversion/defibrillation pulses were applied to the heart directly across the apical patch–superior vena caval spring leads, with the patch being the cathode and the spring the anode. The waveform used was a truncated exponential with 60% tilt; initial voltage was varied to maintain a 6.5 msec pulse duration into a 50 Ω load for all energies. This pulse was delivered from a battery-operated device with energy variable from 1 to 40 J (figure 1). Shocks were synchronized with local activation at the site of the bipolar sensing electrodes.

For each patient a sustained ventricular tachyarrhythmia was induced four times, and after the rhythm was well established a cardioversion/defibrillation shock was given. For each patient the data used for defibrillation threshold measurements were the outcome of the first shock delivered for each of the four episodes of arrhythmia. The energy of the first shocks were 1, 5, 10, or 25 J, with the sequence randomly selected from a list of all permutations of these four numbers. If the initial shock failed to convert the induced rhythm, a 25 J rescue shock was then given. If one or two 25 J rescue shocks were unsuccessful, generally a third 40 J rescue shock was given. If the rescue shocks were unsuccessful, arrhythmias were externally converted with standard external anteroposterior paddles. Each episode of induced ventricular tachyarrhythmia was classified as ventricular tachycardia of stable morphology, polymorphic ventricular tachycardia, or ventricular fibrillation on the basis of the surface electrocardiograms. For each episode we recorded the time from onset of the arrhythmia to delivery of the first shock. For ventricular tachycardia and polymorphic ventricular tachycardia, the occurrence of acceleration by the shock was also noted. For each energy level we calculated the percent success rate for converting each type of rhythm as well as the percentage of episodes accelerated by the shocks.

Results

Patients. Thirty-three patients participated in this study. Their average age was 52.4 ± 12.0 (range 29 to 71). The underlying diagnosis was coronary artery disease in 25 patients, apparently normal heart in two patients, mitral valve prolapse in two patients, congestive cardiomyopathy in two patients, and valvular disease and long QT syndrome in one patient each. The average ejection fraction was 33.2 ± 16.7%, average
left ventricular end diastolic pressure was 18.7 ± 10.5 mm Hg, and the average cardiothoracic ratio was 49.3 ± 10.9. Patients had experienced 3.4 ± 1.8 episodes of sustained ventricular tachyarrhythmia. The primary clinical arrhythmia was ventricular tachycardia in 11 patients, ventricular fibrillation in 16 patients, and ventricular tachycardia/ventricular fibrillation in six patients. Twenty-six patients had one or more cardiac arrests requiring cardiopulmonary resuscitation and 24 of these had one or more out-of-hospital arrests. The average number of antiarrhythmic drugs that failed clinically or at electrophysiologic study was 4.0 ± 2.4.

Arrhythmias induced. For the purpose of the cardioversion/defibrillation threshold determinations, only the outcome of the initial shock given for the first four sustained episodes of a ventricular tachyarrhythmia induced for each patient was considered. Thus a total of 132 episodes of ventricular tachyarrhythmia were induced. Fifty-five episodes (42%) were ventricular tachycardia of stable morphology, 28 episodes (21%) were polymorphic ventricular tachycardias, and 49 (37%) were ventricular fibrillation. Arrhythmias were induced by programmed stimulation in 21 patients and rectified alternating current in 12 patients.

Cardioversion/defibrillation thresholds. Table 1 and figure 2 summarize the cardioversion/defibrillation thresholds in these patients. For ventricular tachycardia of stable morphology, 77% of episodes were converted successfully to sinus rhythm by 1 J and the success rate did not improve significantly as energy was increased to 25 J. In contrast, for both polymorphic ventricular tachycardia and ventricular fibrillation the successful conversion rate increased with increasing energy. For polymorphic ventricular tachycardia, 25% of episodes were converted successfully with 1 J and the success rate increased progressively to 71% for 25 J. For ventricular fibrillation only 8.3% of episodes were successfully converted with 1 J and the success rate increased progressively to 92% for 25 J. Overall, 39% of the induced ventricular tachyarrhythmias were converted successfully with 1 J and 85% were converted successfully with 25 J (figure 3).

The average time from onset of arrhythmia to delivery of the cardioversion/defibrillation shock for 1, 5, 10, and 25 J was 6.5 ± 5.0, 6.1 ± 3.0, 6.3 ± 3.1, and 7.3 ± 3.2 sec, respectively.

Acceleration. Acceleration was considered to have occurred when either ventricular tachycardia of stable morphology or polymorphic ventricular tachycardia were converted by the delivered shock to a more rapid or less organized rhythm (figure 4). For 1, 5, 10, and 25 J the acceleration rates were 23%, 14%, 10%, and 15%, respectively (figure 5).

Rescue shocks and additional defibrillation threshold testing. For the purposes of measurement of the cardioversion/defibrillation thresholds, we considered only the first four episodes of arrhythmia induced. However, most subjects received additional shocks delivered

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**TABLE 1**

Percentage of successful conversions and accelerations for each energy level

<table>
<thead>
<tr>
<th>Energy Level</th>
<th>1 J</th>
<th>5 J</th>
<th>10 J</th>
<th>25 J</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stable VT</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Polymorphic VT</td>
<td>77% (13)</td>
<td>86% (14)</td>
<td>87% (15)</td>
<td>85% (13)</td>
</tr>
<tr>
<td>Ventricular fibrillation</td>
<td>25% (8)</td>
<td>57% (7)</td>
<td>67% (6)</td>
<td>71% (7)</td>
</tr>
<tr>
<td>Acceleration of VT</td>
<td>8.3% (12)</td>
<td>33% (12)</td>
<td>58% (12)</td>
<td>92% (13)</td>
</tr>
<tr>
<td></td>
<td>23% (21)</td>
<td>14% (21)</td>
<td>10% (21)</td>
<td>15% (20)</td>
</tr>
</tbody>
</table>

**FIGURE 2.** Percentage of successful conversions for the 1, 5, 10, and 25 J shocks for termination of ventricular tachycardia of stable morphology (top), polymorphic ventricular tachycardia (middle), and ventricular fibrillation (bottom). Note that increasing energy from 1 to 25 J does not significantly affect the conversion rate for ventricular tachycardia of stable morphology but does result in an increase in success rate for both polymorphic ventricular tachycardia and ventricular fibrillation.
and had more than four episodes of arrhythmia. All patients in whom one or more of the initial 1, 5, 10, or 25 J shocks failed to terminate the ventricular tachyarrhythmia received additional 25 or 40 J rescue shocks after approximately 5 to 10 sec. Since the implanted device delivers approximately 25 J, for many patients several additional episodes of tachyarrhythmia were induced after the formal defibrillation threshold measurements were completed, and a 25 J shock was given to test the reliability of this energy for termination of arrhythmias. With both the formal defibrillation threshold testing and subsequent testing, the 33 patients underwent an average of 6.5 ± 4.0 (range 4 to 22) inductions of arrhythmia. Including primary and rescue shocks they received an average of 9.6 ± 8.3 (range 4 to 42) cardioversion and/or defibrillation shocks. Eleven external cardioversions were required in three patients. Only four patients received more than 15 shocks. Total creatine kinase and the MB fraction were measured in all patients on the morning of the first postoperative day (approximately 12 to 24 hr after the implant). Although total creatine kinase was elevated in all patients, only eight patients had MB fractions above 3% and three of these eight had undergone concomitant coronary artery bypass grafting. No patient showed a clinical worsening of left ventricular function after surgery.

As our clinical experience with measuring cardioversion/defibrillation thresholds accumulated, the safety of our technique became apparent as well as the possibility of acceleration by 25 J. Therefore, later in the series all patients in whom the rhythm induced during the defibrillation/cardioversion threshold testing was stable-morphology or polymorphic ventricular tachycardia also underwent induction of ventricular fibrillation with the rectified alternating current generator to be certain the device to be implanted could terminate acceleration rhythms or spontaneously occurring episodes of ventricular fibrillation. The value of inducing ventricular fibrillation in addition to ventricular tachycardia was clearly demonstrated in one patient who recently had ventricular tachycardia terminated with all energies tested, including 1 J. However, when a more disorganized rhythm was induced, 25 J was not reliably effective.

Solutions to inadequate energy. Intraoperative testing indicated that a 25 J shock delivered by the 12 cm² apical patch and the 10 cm² superior vena caval spring was unreliable for termination of ventricular tachyarrhythmias in five (12%) of 42 patients receiving implanted defibrillators. We could not clearly relate in-

FIGURE 3. Percentage of successful conversions for 1, 5, 10, and 25 J shocks for all episodes of ventricular tachycardia (VT) and ventricular fibrillation (VF) combined.

FIGURE 4. Example of acceleration of ventricular tachycardia by 25 J shock. Shown are electrograms from six surface ECG leads, a spring-patch bipolar lead (SP-patch), the local ventricular bipolar signal, and the aortic pressure (Ao). Induction of ventricular tachycardia is achieved by a brief application of alternating current. After an initial change in morphology, ventricular tachycardia settles into a ventricular tachycardia of stable morphology. The 25 J shock is given approximately 6.5 sec after the onset of the arrhythmia and results in acceleration to a faster, less organized rhythm with a fall in arterial pressure. A subsequent rescue shock terminates this rhythm.
ability to defibrillate with 25 J to underlying cardiac diagnosis, myocardial size observed at the time of surgery, preoperative ejection fraction, or cardiothoracic ratio. In fact, among the five patients who were not consistently defibrillated with the spring-patch electrode configuration were two of the five subjects with essentially normal left ventricles. In these five patients, attempts were made to improve the efficiency of defibrillation by repositioning the superior vena caval spring to higher and lower positions and/or by moving the apical patch or using two patches for defibrillation rather than the spring-patch pair. Our limited experience to date suggests that of these options, the most efficient method for dealing with insufficient energy is to use two patches for defibrillation. We prefer the use of two large (24 cm²) patches in these cases, with one positioned posterior to the left ventricle and the other placed anteriorly over the right ventricle. In three of these five patients the use of two patches has permitted consistently successful defibrillation. In one patient, repositioning a 12 cm² patch to nearer the cardiac apex resulted in reliable defibrillation, and in another patient 25 J accelerated ventricular tachycardia to a more disorganized rhythm that could not be reliably terminated by 25 J with a variety of lead configurations. The defibrillator was removed from the latter patient, in whom a suture line from a previous aneurysm resection somewhat limited the optimal positioning of two patches. Thus in only one of 42 patients did we fail to identify an electrode configuration that would permit reliable termination of arrhythmia with 25 J.

Discussion

This study provides a systematic evaluation of the cardioversion and defibrillation energy requirements in a group of patients with life-threatening ventricular arrhythmias, with use of an apical extracardiac patch and a superior vena caval spring electrode system designed for permanent implantation. Since we limited our cardioversion/defibrillation energies to four values (1, 5, 10, and 25 J), to randomize the sequence of energies delivered as well as to minimize the total number of shocks given to each patient, we did not measure a precise threshold for each patient. Nonetheless our data provide a good estimate of the energy requirements for an implanted device to be used in patients with recurrent ventricular tachyarrhythmias and indicate that there are clearly different energy requirements for the termination of organized ventricular tachycardia and polymorphic ventricular tachycardia and ventricular fibrillation. Low energies of 1 J are as efficacious for terminating ventricular tachycardia of stable morphology as higher energies up to 25 J. However, for ventricular fibrillation, low energies are inadequate and energies greater than 10 J are required for reliable termination. This may be attributable to differences in mechanisms proposed for stable ventricular tachycardia and ventricular fibrillation. Many episodes of sustained organized ventricular tachycardia may be caused by reentry that occurs in either a small circuit or a large pathway such as the circumference of a ventricular aneurysm. Termination of such a rhythm should only require transient depolarization of a portion of the reentrant pathway that is excitable at the time of the shock. Ventricular fibrillation, on the other hand, may involve multiple reentrant circuits or other mechanisms and probably requires simultaneous depolarization of considerably larger amounts of myocardial tissue.

Acceleration of ventricular tachycardia by 10% to 23% of shocks regardless of energy confirms the absolute requirement for back-up defibrillation capability for any automatic transvenous cardioverter. The occurrence of acceleration despite local R wave synchronization is in fact not surprising, especially if many ventricular tachycardias are caused by reentry. For reentrant ventricular tachycardia, complete synchronization of a shock is in fact theoretically impossible. For the rhythm to persist there must always be an area of excitable tissue, and there is no point in the cardiac cycle when a shock could be delivered and all cells would be refractory. Depending on the physical location of the local bipolar sensing electrodes used in our study for synchronization, a shock could be given before, during, or after the QRS complex on the surface electrocardiogram. With a fixed placement of the sensing electrodes, synchronization may occur at different times relative to the surface electrocardiogram for different ventricular tachycardia morphologies if the ven-
Tricular activation sequence is different for each morphology. Even synchronization of a shock with the QRS complex means only that the shock is synchronized with the largest muscle mass and other areas of the ventricle may still be excitable.

The waveform tested in this study was a truncated exponential and was chosen because it has a number of practical advantages for use in an implanted device. The waveform duration was approximately 6.5 msec for a 50 Ω lead (actual duration varies slightly depending on each individual patient’s impedance), since in animal preparations this duration maximizes efficacy of defibrillation. Our waveform with a 60% tilt did not cause serious arrhythmias, although occasionally postshock rhythms were accelerated idioventricular rhythms. No hemodynamically important bradyarrhythmias occurred in any patient.

Our study confirms the need for and safety of extensive electrophysiologic testing at the time of automatic defibrillator implantation and suggests that it is inadequate to induce only one or two episodes of ventricular tachycardia and observe proper function of the implanted device. We believe that a limited measurement of cardioversion/defibrillation thresholds similar to those we performed provides useful information. Because the energy delivered by the currently implanted device is approximately 25 J, these measurements demonstrate the margin of safety of the device for arrhythmia termination. In addition, use of 25 J for rescue shock when lower energies fail to terminate the arrhythmias provides further documentation that the device will reliably terminate the arrhythmias on multiple occasions. These multiple inductions and terminations also provide additional opportunities to note the possibility of acceleration of arrhythmias. We believe that the frequency of ventricular tachycardia acceleration we observed requires that ventricular fibrillation be induced and reliably terminated by 25 J in every patient even if this rhythm has not occurred clinically.

In our series, 12% of patients had inadequate arrhythmia termination with 25 J delivered via the spring-patch lead configuration. The observation that two of the five patients with essentially normal myocardial size and function were difficult to defibrillate raises questions about the dose concept of defibrillation in man. Although this factor undoubtedly is important, other facts such as energy distribution for a given electrode configuration may be more important in many subjects. Further work is needed to optimize electrode configuration in the treatment of arrhythmias in man. Our limited experience suggests that the use of two 24 cm² patches rather than the 12 cm² patch and 10 cm² spring improves the efficacy of defibrillation. We believe that large patches should always be available before a proposed defibrillator implant. In our opinion the use of limited thoracotomy rather than the subxiphoid approach facilitates the testing and electrode replacement and movement that are sometimes necessary in these patients.

References
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