Clinical Efficacy of the Wearable Cardioverter-Defibrillator in Acutely Terminating Episodes of Ventricular Fibrillation

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Although the implantable cardioverter-defibrillator (ICD) is considered a cornerstone of modern cardiology practice for reducing the incidence of sudden cardiac death related to ventricular fibrillation (VF) or ventricular tachycardia (VT),[1,2] several factors limit the prophylactic implantation of the ICD, mainly the inability of invasive and noninvasive laboratory investigations to predict sudden death accurately in a patient population bearing the risk of dying suddenly. As a result, a substantial portion of the at-risk population does not receive adequate preventive therapy and others with lower risk will get a device. A potential solution to this problem could be the use of an external, wearable cardioverter-defibrillator that has defibrillation features similar to those of the ICD (i.e., no operator required to defibrillate), providing protection to the patient until it is determined that the implantation of the ICD is warranted. A device used as a "bridge" to ICD implantation must be capable of reliably terminating episodes of VF/VT, have a highly sensitive and specific algorithm for the detection of VT/VF, and be user-friendly, thereby ensuring patient compliance. This study examines, in a prospective single-center trial, the feasibility and efficacy of transthoracic defibrillation for ventricular tachyarrhythmias by the Wearable Cardioverter-Defibrillator (WCD™ device, LIFECOR, Pittsburgh, Pennsylvania).

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Fifteen survivors of cardiac arrest due to documented VT/VF were prospectively selected as potential study subjects. All patients signed written, informed consent before participating in the study. The study was approved by the investigational review board of the University Hospital Magdeburg, Magdeburg, Germany. Five patients already had an ICD which was temporarily inactivated at the time of WCD™ device testing. The study was performed as part of a routine electrophysiologic study or during ICD testing before patient discharge. All patients were under conscious anesthesia while arterial blood pressure and transcutaneous peripheral oxygen saturation were monitored. Simultaneous sampling of electrocardiographic leads (I to aVF and V_1, V_6) was performed and saved on optical disk for subsequent analysis. For patients with an implanted ICD, an additional telemetric "near-field" intracardiac electrogram (representing the ventricular rate as detected by the ICD) as well as telemetric ICD markers were recorded. Induction of VF in each patient was attempted either by programmed electrical stimulation using a temporary pacing catheter placed under fluoroscopic control at the apex of the right ventricle or by alternating current delivered by the ICD. VF was defined as a polymorphic ventricular rhythm with a cycle length <250 ms. A successful shock delivered by the WCD was defined as one that promptly terminated VF or rapid VT. As a safety precaution, 2 additional self-adhesive, monitor-defibrillator electrode patches were placed on each patient in the right infraclavicular region and close to the apex of the heart. If the shock delivered by the WCD did not successfully terminate the induced VT/VF, a conventional defibrillator was available for use.

The WCD device consists of 2 defibrillation pads, 4 sensing electrodes, and a vibrating active electrode incorporated into a patient-worn garment (Figure 1). The garment is sized to accommodate the chest size and weight of the patient. The electrical hardware is connected to an external device that performs on-line electrocardiographic analysis and charges the capacitors needed to provide a series of shocks with stored energy levels up to 285 Js when the rate of a ventricular arrhythmia is faster than the programmed threshold heart rate. The WCD shock waveform is a monophasic truncated exponential waveform with a fixed tilt but with variable duration according to the actual impedance. The shocking-lead configuration has 2 rectangular pads (total surface area 176 cm^2) located on the middle posterior thorax between the shoulder blades and another rectangular pad (total surface area 88 cm^2) positioned on the anterior chest, approximately at the apex of the heart. Although the WCD is capable of automatically detecting and treating VT/VF, for this initial study, special units were used that were manually armed and discharged by the physician in order to demonstrate the effectiveness of the WCD to terminate a VT/VF episode by a single 230-J stored energy shock. After VT/VF was induced, the physician initiated the device charge cycle. During this charge cycle, the WCD device predicted transthoracic impedance (TTI) by using a "test-pulse" technique similar to that described by Kerber et al.[3] When the WCD indicated the completion of capacitor charging (approximately 20 seconds), a 230-J shock was delivered. The actual TTI, delivered energy, and volt-

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Data contained in this manuscript has been partially presented in abstract form at the XIXth Congress of the European Society of Cardiology, August 24th – 28th, 1997, Stockholm, Sweden.

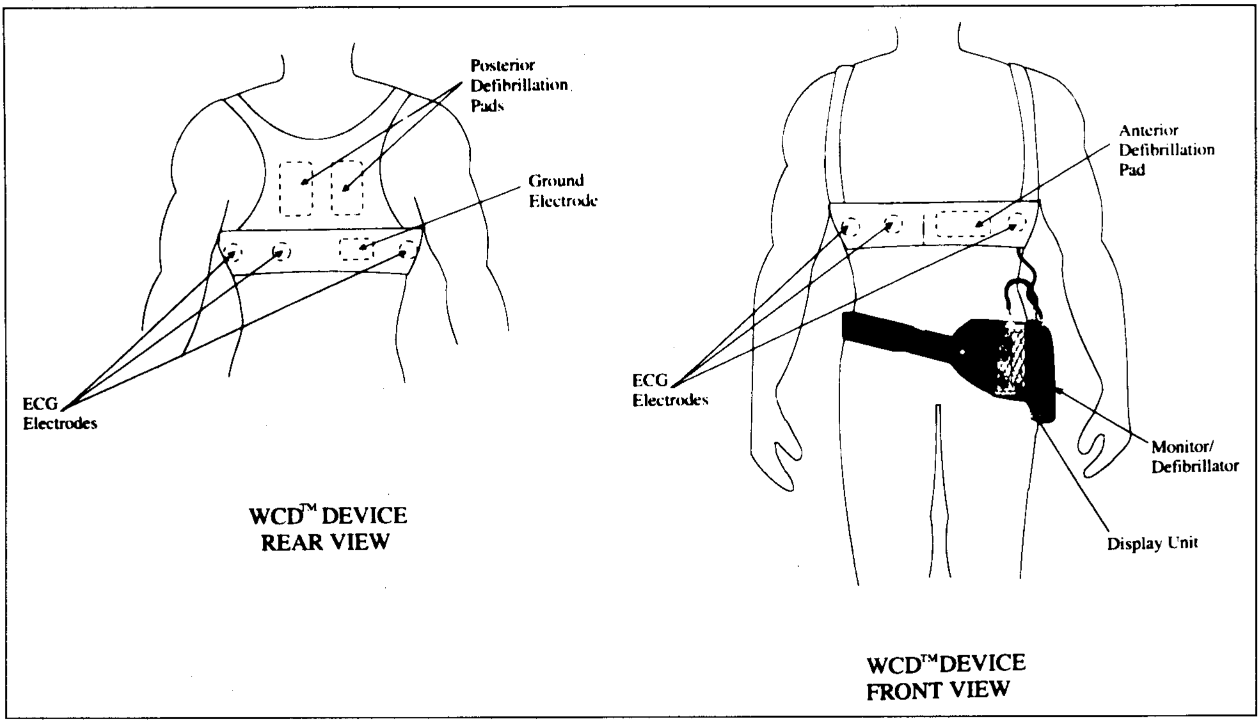

FIGURE 1. Schematic drawing in a front and rear view of the position of the electrocardiography electrodes, defibrillation pads, and device.

age/current versus time profile were recorded for each delivered shock.

All data are reported as mean ± SD. Regression analysis was used for comparing the predicted versus the actual TTI. The correlation coefficient was calculated for the analysis of ventricular ejection fraction and duration of VT/VF with the predicted or actual TTI.

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An episode of VF or rapid VT was induced in 10 of the 15 patients participating in the study. In 3 patients ventricular fibrillation was not inducible, 1 patient developed a "slow" monomorphic VT (heart rate 130 beats/min) and 1 patient had atrial fibrillation with a high ventricular response. Table I lists the clinical data of the study population and their relevant electrophysiologic findings. The mean duration time of the induced VT/VF was 32 ± 15 seconds, with a minimum of 12 and a maximum of 60 seconds. The mean ventricular RR interval was 203 ± 55 ms (range 140 to 333). A single 230-J shock successfully terminated VF in all patients (Figure 2). The mean TTI measured by the test pulse was 45 ± 14 ohms compared with the peak TTI measured at the time of shock delivery of 53 ± 13 ohms ($y = 0.89x + 13.7$; $r = 0.93$). The device correctly identified and classified 9 of 10 induced arrhythmias. In 1 patient the induced episode of VT was not detected by the device because the sensing electrodes were erroneously disconnected at the time of the induction. No postshock supraventricular or ventricular arrhythmias occurred. No relation was found between left ventricular ejection fraction and predicted or actual TTI. Similarly, no correlation existed between duration of VF and measured TTI. Because of the small number of patients, no significant association was found between prescribed antiarrhythmic drugs at the time of testing and TTI.

TABLE I. Clinical and Electrophysiologic Characteristics of the 10 Patients Tested With the Wearable Cardioverter-Defibrillator

Age (yr) (mean ± SD)	63 ± 12
Men/women	7/3
Myocardial infarction (by history)	9
Presenting arrhythmia	
Ventricular tachycardia	4
Ventricular fibrillation	3
Both	3
Left ventricular ejection fraction (%)	21 ± 5
Number of narrowed coronary arteries >50% in diameter (%)	
1	1
2	4
3	5
Antiarrhythmic drug at time of testing	
Amiodarone	7
Sotalol	2
Others	1

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The only consistently effective therapy for terminating an episode of ventricular tachyarrhythmia is the delivery of a timely appropriate defibrillation shock. A therapeutic shock can be accomplished by an external defibrillator transthoracically or by an ICD. With either method, minimizing the interval between the onset of VF and the delivery of the shock is essential for a successful termination of a life-threatening arrhythmia and for avoiding the potential neurologic consequences of a prolonged hypotension or cardiac arrest. Previous studies dealing with transthoracic cardioversion-defibrillation

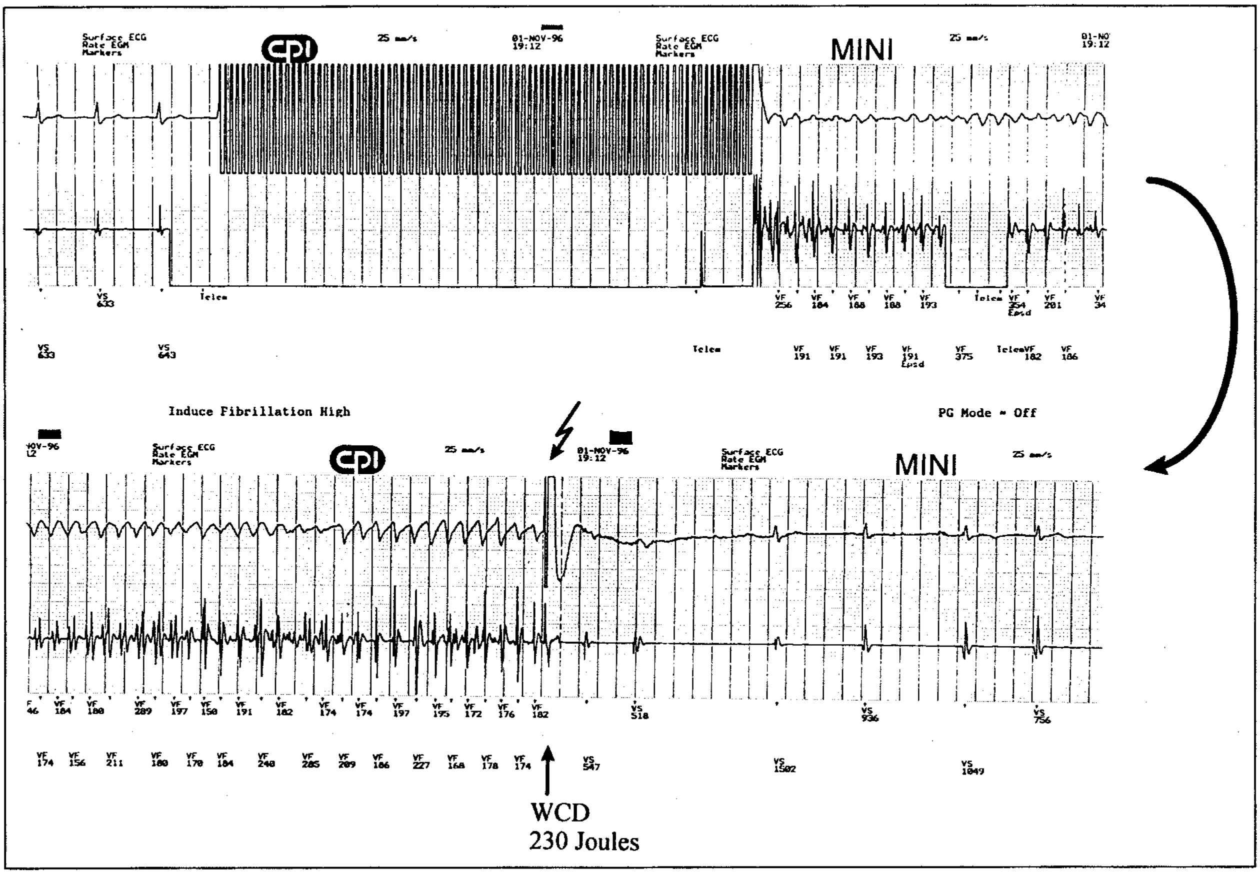

FIGURE 2. Simultaneous electrocardiographic recording of a surface electrocardiogram (L II) and the intracardiac electrogram of an implanted cardioverter-defibrillator (ICD) (Ventak Mini, CPI/Guidant) at the time of the Wearable Cardioverter Defibrillator (WCD) test. The *top strip* demonstrates the induction of a ventricular fibrillation (VF) episode by 30-Hz current, delivered through the ICD (Induce Fibrillation High). The ICD is temporarily deactivated (PG Mode = Off), allowing detection of VF by the WCD. After about 20 seconds (*lower strip*), the WCD terminates VF with a single monophasic shock of 230 J.

have reported acute efficacy of an isolated shock delivered for defibrillation ranging from 50% to 70%.[4] The wide range of reported success and the substantially higher success found in our study reflect the influence of many factors that may affect the defibrillation process. It is well known that the morphologic characteristics of a ventricular arrhythmia, either monomorphic/polymorphic VT or VF, significantly influence the number of necessary shocks and energy requirements.[5] For each patient in our study, the WCD device immediately restored sinus rhythm from an induced VF requiring relatively low energy (delivered energy, 230 J). In contrast, a conventional defibrillation process delivering a similar amount of energy would probably reach a success rate not >70%. The results of this study, however, are consistent with those published by Kerber et al[6] in which the success rate for termination of VF or polymorphic VT was nearly 100% when the first or second shock of 200 to 300 J was delivered to patients having a TTI between 40 and 90 ohms. These findings support the theory that reduced TTI is a determining factor in achieving successful defibrillation. The effect of a larger defibrillation patch and the anteroposterior defibrillation vector generated by the WCD, together with a lower system impedance, may be responsible for the high defibrillation efficacy. The low TTI represents a more homogeneous current flow and distribution through the chest wall and the fibrillating myocardium in a manner similar to that obtained by the TRIAD configuration for endocardial defibrillation by an ICD.[7] Although low TTI and high-energy application may result in excessive current flow and cause prolonged morphologic changes in the myocardium, detectable by electrographic changes and functional impairment, none of these findings were observed in our study. Another reason for the difference between data gathered in this study and previously reported data could be the duration of VF before the defibrillation attempt. In our study the duration of the ventricular arrhythmia was similar to that occurring during ICD implantation, but considerably shorter than that experienced with conventional external defibrillation procedures in an ICU or in an out-of-hospital cardiac arrest. The influence of fibrillation time on transthoracic defibrillation success in humans is still controversial and needs to be determined in further studies.[3,8,9]

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The findings of our initial study demonstrate for the first time the ability to terminate induced VT/VF reliably (100% of all episodes) by a single, monophasic 230-J shock delivered by the WCD. Although limited by sample size, our data suggest

that the WCD could be used as a feasible bridge to definitive implantation of an ICD in patients in whom risk stratification for sudden death is not completed.

1. Moss JA, Hall J, Cannom DS, Daubert JP, Higgins SL, Klein H, Levine JH, Saksena S, Waldo AL, Wilber D, Brown MW, Heo M, for the Multicenter Automatic Defibrillator Implantation trial Investigators. *N Engl J Med* 1996;335: 1933–1940.
2. Cardiovascular News. NHLBI Stops Arrhythmia Study: implantable cardiac defibrillators reduce deaths. *Circulation* 1997;95:2465–2466.
3. Kerber RE, Kouba C, Martins J, Kelly K, Low R, Hoyt R, Ferguson D, Bailey L, Bennett P, Charbonnier F. Advance prediction of transthoracic impedance in human defibrillation and cardioversion: importance of impedance in determining the success of low-energy shocks. *Circulation* 1984;70: 303–308.
4. Dalzell GWN, Adgey AAJ. Determinants of successful transthoracic defibrillation and outcome in ventricular fibrillation. *Br Heart J* 1991;65:311–316.
5. Kerber RE, Kienzle MG, Olshansky B, Waldo AL, Wilber D, Carlson MD, Aschoff AM, Birger S, Fugatt L, Walsh S, Rockwell M, Charbonnier F. Ventricular tachycardia rate and morphology determine energy and current requirements for transthoracic cardioversion. *Circulation* 1992;85:158–163.
6. Kerber RE, Kieso RA, Kienzle MG, Olshansky B, Waldo AL, Carlson MD, Wilber DJ, Schoff AM, Birger S, Charbonnier F. Current-based transthoracic defibrillation. *Am J Cardiol* 1996;78:1113–1118.
7. Klein H, Auricchio A, Huvelle E, Nisam S. Initial experience with a new down-sized implantable cardioverter-defibrillator. *Am J Cardiol* 1996;78:9A–14A.
8. Kerber RE, Sarnat W. Factors influencing the success of ventricular defibrillation in man. *Circulation* 1979;60:226–230.
9. Gascho JA, Crampton RS, Cherwek ML, Sipes JN, Hunter FP, O'Brien W. Determinants of ventricular fibrillation in adults. *Circulation* 1979;60:231–240.