Optimization of Biphasic Waveforms for Human Nonthoracotomy Defibrillation

John F. Swartz, MD; Ross D. Fletcher, MD; Pamela E. Karasik, MD

Background. Biphasic waveforms reduce defibrillation threshold (DFT) in a wide variety of models. Although there are several human studies of long-duration, high-tilt biphasic waveform defibrillation, the specific biphasic waveform shape required to achieve optimal DFT reduction is unknown.

Methods and Results. This study tested the effect of single capacitor biphasic waveform tilt modification on DFT using a paired study design in 18 patients undergoing nonthoracotomy defibrillator implantation. Baseline DFT was obtained using a 65% tilt, simultaneous pulse, bidirectional monophasic shock from a right ventricular cathode to a coronary sinus or superior vena cava lead and a subscapular patch. The single-capacitor biphasic waveform shocks, delivered over the same pathways, consisted of either both phases at 65% tilt (65/65 biphasic waveform) to produce an overall tilt of 88% and a delivered energy 11% greater than monophasic shock or both phases at 42% tilt (42/42 biphasic waveform) to produce an overall tilt of 66% and delivered energy equal to monophasic shock. The 65/65 biphasic waveform reduced stored energy DFT 28%, from 16.2±4.4 J with monophasic shock to 12.1±5.3 J (P<.02); however, it did not significantly reduce the delivered energy DFT. In contrast, the 42/42 biphasic waveform required 49% less stored energy (16.2±4.4 J, monophasic shock, vs 8.3±3.3 J, biphasic waveform; P<.001) and 49% less delivered energy (14.2±3.8 J, monophasic shock, vs 7.3±2.9 J, biphasic waveform; P<.001) than monophasic shock for successful defibrillation. The 42/42 biphasic waveform delivered energy DFT was 4.6±5.2 J (39%) less than 65/65 biphasic waveform DFT (P<.002).

Conclusions. DFT reduction is an inherent electrophysiological property of biphasic waveforms that is independent of delivered energy. Overall biphasic waveform tilt and the relative amplitudes of the waveform phases are important factors in defibrillation efficacy. Defibrillation with a 42/42 biphasic waveform is more efficacious than 65/65 biphasic waveform defibrillation; however, the optimal biphasic waveform remains unknown. (Circulation. 1993;88:2646-2654.)

Key Words: defibrillation • death, sudden • defibrillators • waveforms

Although biphasic waveform defibrillation is generally regarded as more efficacious than monophasic waveform defibrillation, the optimal biphasic waveform shape has not been identified. Very limited information is available regarding application of biphasic waveforms for human defibrillation. In each of the human defibrillation studies to date, monophasic waveforms were compared with longer-duration, higher-tilt biphasic waveforms having a greater delivered energy for any given leading edge voltage. Results of these studies were reported only in terms of stored energy defibrillation threshold values, which fail to account for waveform-dependent differences in the efficiency of stored energy delivery. Therefore, it is unknown whether the mechanism of biphasic waveform defibrillation threshold reduction in these studies was due to additional energy delivery by the biphasic waveform or due to inherent electrophysiological effects of the biphasic waveform.

Clinical trials of implantable biphasic waveform defibrillators are underway even though there are no data available regarding the optimal biphasic waveform for humans. The purpose of this study, therefore, was to investigate the separate effects of additional energy delivered during the second phase of a long-duration, high-tilt biphasic waveform from the intrinsic effects of the biphasic waveform itself. We hypothesized that a biphasic waveform reduces defibrillation energy requirements when compared with the monophasic waveform of equivalent delivered energy. We further hypothesized that biphasic waveform tilt influences defibrillation efficacy, with high waveform tilts adversely affecting defibrillation efficacy. To test these hypotheses, we evaluated defibrillation efficacy for three different waveforms using a paired study design in 22 consecutive patients undergoing nonthoracotomy defibrillator implantation. The waveforms tested were a standard 65% tilt monophasic, a biphasic with 65% tilt for each phase (88% overall tilt), and a biphasic with 42% tilt for each phase (66% overall tilt).

Methods

Patient Population

Patients included in this study were referred to the Veterans Affairs Medical Center, Washington, DC, for
therapy of medically refractory sustained monomorphic ventricular tachycardia or aborted sudden cardiac death. All subjects gave written informed consent for the device implantation protocols (Medtronic PCD NTL or Cardiac Pacemakers, Inc, PRx-ENDOTAK), which were approved by the institutional review board and committee for the protection of human subjects. The study population comprised 20 men and 2 women. Mean patient age was 56.9±16.0 years. Patient height was 70.6±1.7 in. and weight was 187±34 lb. The mean left ventricular ejection fraction was 31.1±12.5%, with 8 patients having New York Heart Association class III functional status, 8 class II, and 6 class I functional status. The medical history was remarkable for clinically significant atherosclerotic coronary artery disease in 15 patients, 14 of whom also had a history of remote myocardial infarction. Five patients had undergone one or more coronary artery bypass grafting procedures. One patient had multivascular heart disease and was status post–mitral and aortic valve replacement. Clinical arrhythmia presentation was recurrent sustained monomorphic ventricular tachycardia in 15 patients and aborted sudden cardiac death in 9 patients. Four patients were taking amiodarone at the time of device implant, 3 were taking quinidine, and 1 was on sotalol.

**Device Implantation**

Defibrillation system implantation was accomplished in the cardiac catheterization laboratory in all patients. Patients were brought to the catheterization laboratory in a postabsorptive, mildly sedated state. Additional preprocedure sedation was achieved using a combination of midazolam and fentanyl. Continuous arterial blood pressure monitoring was established via percutaneous right radial or right femoral arterial access. A 6F combination pacing and monophasic action potential recording catheter was positioned at the right ventricular outflow tract via a femoral venous approach. The left precordial chest wall, left axilla, and abdomen were then prepped and draped in the usual sterile fashion. After local anesthesia with 1% lidocaine, the left subclavian vein was percutaneously entered. Two separate subclavian vein entry sites were established in those patients undergoing PCD (model 7217B, Medtronic, n=15 patients) implantation. The medial entry site was used to place a unipolar superior vena cava or coronary sinus lead (model 6963, Medtronic). Because of its larger diameter, the tripolar right ventricular lead (model 6966, Medtronic) was positioned at the right ventricular apex through the more lateral subclavian entry site. Only one subclavian entry site was required for patients undergoing VENTAK PRx (model 1705, Cardiac Pacemakers, Inc, n=7 patients) with ENDOTAK-C (model 0074, Cardiac Pacemakers, Inc) implantation. PACing, R wave amplitude, and slew rates were used to document adequate right ventricular lead positioning and performance.

A subscapular chest wall patch position was used in all patients. The patch (model 6999, Medtronic, or model 0063, Cardiac Pacemakers, Inc) was placed via a 4- to 6-cm incision made parallel to the fifth left intercostal space and extending from the anterior axillary line to the midaxillary line. The subscapular space was accessed by gentle separation of the serratus anterior digitations just posterior to their fourth and fifth costal origins. The patch was placed deep to the serratus anterior at the level of the inferior angle of the scapula. This patch placement resulted in right ventricular defibrillation lead superimposition on the patch silhouette in a 30° right anterior oblique fluoroscopic projection with 25° caudal angulation. Fig 1 shows posteroanterior and lateral radiographic images of the lead and patch positions used in this study. After patch placement and before any defibrillation threshold testing, individual pathway integrity and impedance (right ventricle to superior vena cava or coronary sinus and right ventricle to subscapular patch) were assessed by delivering a synchronous 0.6-J shock during sinus rhythm. Defibrillation waveform voltage, current, and impedance of all shocks were oscillographically monitored (model 5113, Tektronix, Beaverton, Ore) and recorded on film for analysis.

**Defibrillation Threshold Testing**

Single-shot defibrillation thresholds (DFT) were determined after 10 seconds of electrically induced ventricular fibrillation. An initial energy value of 18 J was used to initiate DFT testing. If 18 J failed to defibrillate, subsequent episodes were attempted at 21, 24, and 28 J until defibrillation was successful. Patients received 360-J transthoracic rescue shocks immediately after any unsuccessful defibrillation attempt. If the initial 18-J shock successfully defibrillated, subsequent episodes were performed using 15, 10, 5, and 2.5 J until defibrillation failed. All fibrillation-defibrillation episodes were separated by at least 5 minutes. The DFT was defined as the lowest energy that resulted in successful defibrillation. Monophasic waveform DFT was determined first in all patients in order to establish successful device implantation criteria (DFT ≤25 J for PRx-ENDOTAK or DFT ≤18 J for PCD/Transvene) before biphasic waveform testing. The order in which the two biphasic waveform DFTs were tested was randomized to prevent the possibility of outcome bias due to time dependent changes of DFT. The initial biphasic waveform energy tested was established at the previously determined monophasic waveform defibrillation threshold. A protocol identical to that described for monophasic waveform testing was used for both biphasic waveforms. Important features of the three tested waveforms are shown in Fig 2. The monophasic waveform (panel A) had an overall tilt of 65%. This waveform was directly compared with the 65/65 (panel B) and 42/42 (panel C) biphasic waveforms. The 65/65 biphasic waveform delivered 11% more energy than the 65 monophasic for any given leading edge voltage or stored energy setting, whereas the 42/42 biphasic and 65 monophasic waveforms delivered equivalent energies for any given leading edge voltage or stored energy value. Defibrillation pulses were derived from the asynchronous discharge of a 120-μF capacitor (model 2394, Medtronic).

**Statistical Analysis**

Defibrillation threshold data are reported as mean±1 SD. Because of the nonparametric distribution of data, waveform-dependent differences in defibrillation threshold were sought using Friedman’s repeated measures ANOVA on ranks. Student-Newman-Keuls pairwise multiple comparisons of the 65 monophasic, 65/65
Fig 1. Comparative views of the defibrillation lead systems used in this study. A, Posteroanterior (left) and lateral (right) radiographs of a PRx/ENDOTAK system with subscapular chest wall patch. This was the lead system tested in 7 study patients. This patient also has a dual-chamber pacemaker with leads positioned in the right atrial appendage and right ventricular outflow tract. B, Posteroanterior (left) and lateral (right) radiographs of the Transvene PCD system with a subscapular patch. The superior vena cava lead is positioned to approximate the proximal coil position of the PRx/ENDOTAK system (A). Three patients were studied with this lead configuration. C, Posteroanterior (left) and lateral (right) radiographs of a Transvene PCD system using a coronary sinus lead and subscapular patch. The Transvene PCD system with a coronary sinus lead position was present in 8 of the study patients.
biphasic, and 42/42 biphasic waveform defibrillation thresholds were then used to quantify the specific waveform effects. Lead system effects on the relative performance of each waveform tested were investigated using Kruskal-Wallis one-way ANOVA on ranks. Differences were considered statistically significant at the .05 significance level.

Results

Implant Procedure

Each of the 22 patients had successful device implantation. The right ventricular lead served as a common cathode for simultaneous bidirectional pulsing to the anodal subscapular patch and anodal transvenous lead. The transvenous anode was positioned in the coronary sinus in 7 patients and in the superior vena cava in 15 patients for the final implant configuration. However, waveform testing reported in this study was performed using a configuration using a coronary sinus lead in 11 patients and a superior vena cava position in 11 patients. Patients underwent 14.6±4.3 ventricular fibrillation-defibrillation episodes to complete the study protocol. Complete data sets were not obtained in 4 patients. Equipment-related technical problems during biphasic waveform testing prevented protocol completion in 3 patients. Protocol testing was prematurely terminated because of concerns for patient clinical status in one case. Eighteen patients (10 with a superior vena cava lead position and 8 with a coronary sinus lead position) had adequate defibrillation threshold data sets for all three waveforms. The 42/42 biphasic waveform DFT was determined before the 65/65 biphasic waveform DFT in 10 of the patients, while the 65/65 biphasic was tested before the 42/42 biphasic in 8 patients. Evaluation of waveform efficacy differences was based only on paired data set analyses.

Defibrillation Threshold Testing Results

The overall monophasic waveform defibrillation threshold for the implanted lead system configuration in the 22 patients was 15.4±4.3 J. Since 4 patients did not complete all waveform testing, this value is different than the 16.2±4.4 J DFT reported for paired data comparisons of the monophasic and biphasic waveforms in the 18 study patients. Both biphasic waveforms produced a significant decrease in the stored energy defibrillation threshold. The 65/65 biphasic reduced stored energy defibrillation threshold 25%, from 16.2±4.4 J 65 monophasic to 12.1±5.3 J 65/65 biphasic; n=18, P<.02 (Fig 3). Defibrillation threshold was reduced by the 65/65 biphasic in 12 of 18 patients, whereas no effect was noted in 5 patients and threshold was higher in one. The 42/42 biphasic waveform reduced defibrillation threshold 49%, from 16.2±4.4 J 65 monophasic to 8.3±3.3 J 42/42 biphasic; n=18, P<.001 (Fig 4). The 42/42 biphasic waveform reduced defibrillation threshold in 17 of 18 patients, but threshold remained unchanged in one.

Paired comparison of the 42/42 and 65/65 biphasic waveform revealed that the short-duration biphasic waveform reduced stored energy defibrillation threshold 31%, from 12.1±5.3 J 65/65 biphasic to 8.3±3.3 J 42/42 biphasic; n=18, P<.05 (Fig 5). Defibrillation threshold was lower for the 42/42 biphasic in 10 of the 18 patients, whereas equivalent results were obtained for the 42/42 and 65/65 biphasic waveforms in 7 patients. Only one patient had a greater 42/42 biphasic than 65/65 biphasic stored energy defibrillation threshold.

Delivered Energy Analysis

Since the 65/65 biphasic waveform delivered energy was 11% greater than for either the 65 monophasic or 42/42 biphasic at any given stored energy level, defibrillation threshold data were also analyzed with respect to
the actual delivered energy. Both the 65 monophasic and the 42/42 biphasic waveforms deliver 87.5% of the total stored capacitor energy during a defibrillation pulse, whereas the 65/65 biphasic waveform delivers 98.5% of the stored energy. Equivalent overall tilt and energy delivery of the 65 monophasic and 42/42 biphasic waveforms occurred at pulse widths of 6.7 ± 1.0 milliseconds for the 65 monophasic and 6.9 ± 0.8 milliseconds for the 42/42 biphasic waveform. The 0.2-millisecond longer 42/42 biphasic duration was due to a switching delay between the biphasic pulse phase reversal as shown in Fig 1. Additional energy delivery by the 65/65 biphasic waveform produced a longer duration of 13.8 ± 1.6 milliseconds. This additional delivered energy decreased the defibrillation threshold difference between 65 monophasic and 65/65 biphasic waveforms. The 65 monophasic delivered energy defibrillation threshold of 14.2 ± 3.8 J was not significantly different than the 11.9 ± 5.2 J 65/65 biphasic delivered energy threshold; n=18, P=.29 (Fig 6). The delivered energy 65/65 biphasic defibrillation threshold was less than the 65 monophasic threshold in 12 patients and greater in 6 patients.

Analysis of delivered energy data confirmed the lower defibrillation threshold of a 42/42 biphasic waveform with respect to both the 65 monophasic and 65/65 biphasic waveforms. The 42/42 biphasic delivered energy defibrillation threshold of 7.3 ± 2.9 J was 49% lower than the 14.2 ± 3.8 J 65 monophasic threshold; n=18, P<.001 (Fig 7), and 39% lower than the 11.9 ± 5.2 J 65/65 biphasic threshold; n=18, P<.002 (Fig 8). The 42/42 biphasic delivered energy defibrillation threshold was lower than the 65 monophasic threshold in 17 of 18 patients and equivalent in one. Similarly, the 42/42 biphasic delivered energy threshold was lower than the 65/65 biphasic threshold in 17 of 18 patients and higher than the 65/65 biphasic in one patient.

Effect of Lead System Configuration

Kruskal-Wallis one-way ANOVA of the nonrepeated measures of defibrillation threshold demonstrated equivalent outcomes for each of the three lead systems evaluated in this study regardless of the defibrillating waveform used. Relative defibrillation efficacy of the 65 monophasic, 65/65 biphasic, and 42/42 biphasic was not altered by the lead system variations included in this study.

Discussion

In this study we prospectively tested the hypothesis that biphasic waveform reduction of defibrillation energy requirements in humans is dependent on inherent electrophysiological effects of the biphasic waveform and not due to additional energy delivery associated with the biphasic waveform second phase. Our results show that a single-capacitor biphasic waveform with a 42% tilt of each phase (66% overall tilt) reduces defibrillation threshold when compared with a monophasic waveform of equivalent delivered energy, therefore confirming that biphasic waveform reduction of defibrillation energy requirements in humans is an inherent electrophysiological property of the biphasic waveform. In this study, 49% less stored energy was required for defibrillation with the 42/42 biphasic waveform than compared with a 65% tilt monophasic waveform. Improvement of defibrillation energy requirements with the 42/42 biphasic waveform was quantitatively identical when analyzed in terms of delivered energy. In addition, this study demonstrated a significant effect of overall waveform tilt on biphasic waveform efficacy. The efficacy of biphasic waveform
defibrillation, whether measured in terms of stored or delivered energy, was compromised by a high overall waveform tilt. The 42/42 biphasic waveform stored energy defibrillation threshold was 31% lower than the 65/65 biphasic threshold in this patient series. When analyzed with respect to delivered energy, the 42/42 biphasic defibrillation threshold was 39% lower than the 65/65 biphasic threshold.

Although less effective than the 42/42 biphasic waveform, the long-duration, high-tilt 65/65 biphasic waveform still required 25% less stored energy than the 65 monophasic waveform for successful defibrillation. However, when examined in terms of actual energy delivered, there was not a statistically significant difference in the 65/65 biphasic and 65 monophasic waveform defibrillation thresholds. The stored energy defibrillation threshold lowering effect of a long-duration, high-tilt biphasic waveform found in this study parallels the findings of other investigators. Bardy et al. showed a 26% reduction of stored energy epicardial defibrillation requirements, from 8.5 ± 1.6 J to 6.3 ± 5.2 J, using a high-tilt (65% tilt each phase, 88% overall tilt), single-capacitor biphasic waveform. In a separate study, Bardy and coworkers found no advantage of the single-capacitor, high-tilt biphasic waveform as compared with monophasic waveform defibrillation in a single-pathway nonthoracotomy system using the coronary sinus as the cathode and the right ventricle as the anode. However, the same biphasic waveform improved stored energy thresholds 25% for an epicardial patch system and 32% for right ventricular lead to chest wall patch nonthoracotomy defibrillation. In a study of nonthoracotomy defibrillation in 38 patients, Saksena et al. found no threshold-lowering effect of biphasic waveforms for initial shock voltages at or below 400 V. However, for initial shock voltages between 401 and 580 V, high-tilt (65% tilt each phase, 88% tilt overall), single-capacitor biphasic waveform defibrillation was more efficacious than monophasic defibrillation. A later, paired study of monophasic and biphasic waveform nonthoracotomy defibrillation in 9 patients by Saksena et al. revealed a 40% stored energy defibrillation threshold decrease, from 16.3 ± 4.4 to 11.3 ± 6.2 J, with the same high-tilt, long-duration biphasic waveform.

The lack of a demonstrable delivered energy defibrillation threshold–lowering effect with the 65/65 biphasic
waveform in this study is also consistent with the results of other investigations using the 65/65 biphasic waveform.\textsuperscript{2-5} Analysis of actual delivered energy for the defibrillation thresholds reported in each of these studies failed to confirm a statistically significant 65/65 biphasic waveform reduction in the energy required for defibrillation. Inability of the high-tilt biphasic waveform to reduce delivered energy requirements for defibrillation suggests that any benefit derived from the use of a biphasic waveform is countered by the long pulse duration and high overall tilt of a 65/65 biphasic waveform. Long defibrillation pulses with very low truncation voltages or no truncation are known to promote refibrillation.\textsuperscript{6} The effects of waveform tilt and pulse duration on defibrillation threshold have been specifically investigated in a number of studies. For trapezoidal waveform pulse widths between 2 and 20 milliseconds, Bourland et al\textsuperscript{7} were unable to demonstrate any effect of tilt variation between 15\% and 90\% in a study of transthoracic defibrillation of dogs and ponies. In contrast, a direct correlation was noted between pulse width and defibrillation threshold, with a nearly linear increase of the delivered energy defibrillation threshold for pulse width durations increasing from 2 to 20 milliseconds. The study of Bourland et al is the only investigation to evaluate the independent effects of monophasic waveform tilt and pulse duration on defibrillation energy requirements. Chapman et al\textsuperscript{8} noted optimal monophasic waveform nonthoracotomy defibrillation in canines at pulse widths between 7.5 milliseconds (52\% tilt) and 15 milliseconds (77\% tilt). In a study of epicardial canine defibrillation, Feeseer et al\textsuperscript{9} demonstrated significant increases in defibrillation energy requirements as single capacitor biphasic waveforms were prolonged beyond one time constant in duration, producing overall tilts greater than 63\%. This increase in defibrillation energy requirements was reversed with low-tilt, equiduration biphasic waveforms. A marked decay in biphasic waveform defibrillation efficacy was also noted with greater biphasic waveform tilt in the studies of Tang et al.\textsuperscript{10} In those studies, defibrillation energy requirements increased as waveform duration was increased from one to two time constants. Although the effect of biphasic waveform defibrillation energy reduction was present for waveforms as short as 0.25 time constants, increases in peak voltage and current requirements were noted for pulses less than 0.87 time constants in duration.

Waveform features other than pulse duration and tilt may also affect defibrillation energy requirements. One such feature is the ratio of leading edge amplitudes for the first and second phases of the biphasic waveform. Although there are no human data to compare with our results, several animal studies have demonstrated a pronounced effect of phase amplitude ratio on biphasic waveform defibrillation efficacy. In a study of defibrillation in 100-kg calves, Schuder et al\textsuperscript{11} noted a 30\% increase in defibrillation efficacy when the phase amplitude ratio was increased from 0.25 (phase 2 leading edge amplitude equals one fourth of phase 1 leading edge amplitude) to 0.5 for biphasic waveforms between 4 and 16 milliseconds in total duration. More recently, Kavanagh et al\textsuperscript{12} reported lower voltage and energy defibrillation thresholds for single capacitor biphasic waveforms (phase amplitude ratio <1) as compared with a two-capacitor waveform with equal first- and second-phase amplitudes. Dixon et al\textsuperscript{13} also found significantly higher defibrillation energy requirements for a two-capacitor biphasic waveform having a second-phase to first-phase ratio of 2 when compared with a single-capacitor exponential biphasic waveform of equal duration. The effect of second-phase amplitude variation from 0.2 to 1.4 times the first-phase, leading edge amplitude was extensively explored by Feeseer et al using an epicardial delivery system in canines. A bimodal effect of phase amplitude ratio variation was identified, with optimal defibrillation occurring when the second-phase, leading edge amplitude was 60\% of the first-phase leading edge. Although only limited conclusions may be drawn from the present study regarding the effect of biphasic waveform phase amplitude ratio, the differential efficacy of 65/65 and 42/42 biphasic waveforms correlates well with animal models. The 42/42 biphasic, with a 0.58 second-phase amplitude to first-phase amplitude ratio, reduced defibrillation energy requirements 39\% as compared with the 65/65 biphasic waveform with a 0.35 phase amplitude ratio.
Further investigation of the effect of biphasic waveform phase amplitude ratio in humans is required.

Design of the optimal biphasic waveform for defibrillation is a complex process based on many factors. The rationale for choosing a specific biphasic waveform to optimize human defibrillation must be based on a firm knowledge of defibrillation mechanisms. Early theory suggested that defibrillation is based on the excitation and depolarization of a critical mass of tissue.14 In accordance with this hypothesis, specifically shaped biphasic waveforms known to reduce defibrillation energy requirements also reduce energy requirements for refractory period cellular stimulation and for cellular stimulation in conditions of high potassium and rapid pacing.15-17 However, recent studies of short-duration monophasic and biphasic waveforms demonstrated a dissociation of biphasic waveform defibrillation threshold reduction and refractory period response generation.18 The ability to dissociate these two phenomena suggests that additional factors must influence defibrillation outcome. Factors such as fibrillation reinduction, transient conduction block within regions of high-voltage gradient, and prolongation of repolarization have been studied recently as important phenomena affecting defibrillation.19-24 Although these and other studies have provided some insight, until the mechanisms of biphasic waveform defibrillation are completely understood, identification of the optimal biphasic waveform for human defibrillation will remain largely a trial and error process.

Study Limitations

Conclusions of this study are based on single-shock defibrillation threshold values. Although use of the defibrillation threshold as a measure of defibrillation efficacy implies a distinct energy cutoff for defibrillation success, defibrillation is a probabilistic phenomenon. The limitations and relation of defibrillation threshold as a measure of defibrillation efficacy to the more correct method of dose-response curve generation were studied and reported by McDaniel et al.25 Generation of defibrillation dose-response curves requires numerous defibrillation-defibrillation sequences at each energy level tested. When multiple waveforms are being compared using a paired study design, the number of sequences required to construct dose-response curves for each waveform becomes prohibitive and may compromise patient safety. Despite the inherent limitations, use of a defibrillation threshold to assess waveform effects on defibrillation efficacy has been found to be a useful clinical tool. Davy et al26 found defibrillation threshold to correlate with the energy required to achieve a 71±26% defibrillation success rate in a study of open-chest dogs. Similar results were reported by Rattes et al.27 who found defibrillation threshold equivalent to the energy required to achieve a 76±7% defibrillation success rate in a study of open-chest pigs.

Although 42/42 biphasic waveform defibrillation was more efficacious than 65/65 biphasic defibrillation in this study, the 42/42 biphasic is not necessarily the optimal biphasic waveform for human defibrillation. Identification of a single optimal waveform for defibrillation may not be possible. The highly variable cardiac structure and function encountered in clinical practice make it unlikely that a single and specific defibrillation waveform will be most efficacious in all patients under all circumstances. Further, greater 42/42 biphasic waveform defibrillation efficacy, as found in this study, may not be a universal finding for all defibrillator lead systems. Additional investigation of short-duration biphasic waveform defibrillation is required to confirm the findings of this study and to fully define the effects of lead system configuration on biphasic waveform defibrillation.

Clinical Implications

The results of this study suggest that biphasic waveform defibrillators currently under investigation may not provide the optimal waveform. Until the optimal biphasic waveform features are identified, waveform parameters such as tilt, phase amplitude ratio, and phase durations should be programmable to permit defibrillation optimization in individual patients.

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References


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