Influence of Phase Duration of Biphasic Waveforms on Defibrillation Energy Requirements With a 70-μF Capacitance

Patrick Schauerte, MD; Friedrich A. Schöndube, MD; Marius Grossmann, MD; Hilmar Dörge, MD; Frank Stein, BSE; Bernd Dohmen, BSE; Abdel Moumen, MD; Konstantin Erena, MD; Bruno J. Messmer, MD; Peter Hanrath, MD; Christoph Stellbrink, MD

Background—Phase duration of biphasic shocks may be an important determinant of defibrillation success. The purpose of this study was to investigate the effect of changing phase duration of biphasic pulses delivered by 70-μF capacitors on defibrillation energy requirements. This may be clinically relevant for the optimization of implantable cardioverter-defibrillator design and programming.

Methods and Results—Defibrillation thresholds (DFTs) were determined for 13 waveforms in 13 pigs by application of a 70-μF capacitance and a transvenous/submuscular lead system. In part I, phase-1 duration varied, preserving a phase-1/phase-2 duration ratio of 60%/40%. The phase-1 durations were 1, 2, 3, 4, 5, and 6 ms. The DFT was lowest (22.5±7 J) for phase 1=3 ms compared with phase 1=1 ms (36.4±7.5 J), 2 ms (25±6.5 J), 4 ms (25±7.6 J), 5 ms (30.7±7.3 J), or 6 ms (32.9±8.1 J) (P<.001). In part II, phase-1 duration was 3 ms but phase-2 duration varied: 0.7, 1.3, 2, 2.7, 3.3, 4, and 6 ms. Significant DFT minima were found at phase 2=2 ms (22.5±4.2 J) and phase 2=4 ms (22.5±4.2 J) compared with phase 2=0.7 ms (31.7±9.3 J), phase 2=3.3 ms (26.7±6.1 J), or phase 2=6 ms (28.3±6.8 J) (P<.05).

Conclusions—The strength-duration curve of biphasic defibrillation shocks demonstrates a single optimum for phase-1 duration. In contrast, two optima with minimal energy requirements were found for phase-2 duration. Optimization of both phases of low-capacitance biphasic shocks may reduce energy requirements for defibrillation. (Circulation. 1998;97:2073-2078.)

Key Words: fibrillation ■ defibrillation ■ death, sudden

Current implantable cardioverter/defibrillators (ICDs) incorporate a 120- to 150-μF capacitor, which accounts for about one third of the total device volume.1 Because ICD volumes may be reduced by incorporation of smaller capacitors, several animal and clinical studies compared ICDs with capacitors of 120 to 140 μF with those with lower capacitors of 60 to 85 μF and revealed an at least equal defibrillation efficacy in terms of stored energy.2–5 However, smaller capacitors must be loaded to a higher voltage for storing the same energy according to the capacitor formula $E = \frac{1}{2} \times C \times U^2$, where $E$ is energy, $C$ is capacitance, and $U$ is voltage. By retaining present maximal ICD voltage outputs, this may decrease the safety margin for defibrillation, assuming an equal defibrillation threshold (DFT) in terms of stored energy. Furthermore, because the capacitor volume is proportional to the maximum capacitor energy, a reduction of the capacitor volume by smaller capacitances is only possible if defibrillation energy requirements are lowered.3 The phase duration of defibrillation shocks is an important factor influencing the DFT.5,7 Because the time constant $\tau$ (time that is required to deliver 68% of the stored energy) is directly related to the capacitor following the equation $\tau = R \times C$ ($R$=resistance), shocks delivered from smaller capacitors need less time to deliver the same amount of energy than shocks from larger capacitors. We hypothesized that DFTs for 70-μF capacitors may have a minimum at other pulse widths and tilts than for larger capacitors and investigated the relation between phase duration of biphasic shocks and the defibrillation energy requirement for 70-μF capacitors.

Methods

Animal Preparation
All studies followed institutional guidelines for animal trials and were undertaken with permission of the competent authorities (Regierungspräsident Köln, April 14, 1992). We studied 13 healthy domestic pigs (German landrace) weighing 70±6 kg. Ketamine (20 mg/kg body wt IM) and atropine (0.14 mg/kg body wt IM) were used to induce anesthesia. The animals were intubated with auffed endotracheal tube and ventilated with NO2/O2 (ratio 3:1) by a...
ventilator (Servo Respirator, Siemens Corp) at 10 to 15 breaths/min. Sodium pentobarbital (1 to 2 mg/kg) was infused to maintain a constant depth of anesthesia. Systemic blood pressure was continuously monitored through a carotid arterial line and blood gas analysis performed every 30 minutes. Metabolic status was further monitored by taking central venous blood samples for electrolytes every 30 minutes. Pulmonary capillary wedge pressure and mixed venous oxygenation were continuously measured by a Swan-Ganz-catheter. Surface ECG was recorded during each fibrillation/defibrillation episode on a 12-channel standard ECG recorder (Mingograph, Siemens) at a paper speed of 25 mm/s. After left jugular venotomy, a transvenous defibrillation electrode (Endotak 072, CPI) was introduced under fluoroscopy in the right ventricular apex. The electrode carries two coils, one positioned in the right ventricular apex (distal coil) and one in the superior vena cava (proximal coil). A submuscular patch (Endotak Sub Q 042, CPI) was then placed at the left thoracic wall opposite to the left ventricle. The electrodes were connected with the distal coil in the right ventricular apex as anode and with the proximal coil and submuscular patch as common cathode during phase-1 of the biphasic shocks. Shocks were delivered by a 70-μF capacitor (model 2394, Medtronic) that allowed separate selection of pulse width, voltage, and intershock delay. Shock voltage values of the leading edge of phase 1 to be charged on the capacitors were calculated according to the equation $E=\frac{1}{2}XCXU^2$. Voltage and current at the leading and trailing edge of phase 1 were recorded on an oscilloscope. Leading edge voltage of phase 2 was adjusted to the same value as the voltage at the trailing edge of phase 1. To estimate the voltage at the trailing edge of phase 1, monophasic shocks of all different phase-1 durations were delivered to each pig heart at each energy level. The leading edge voltage of phase 2 of the biphasic shock was then adjusted to the voltage at the end of the respective monophasic shocks.

**Defibrillation Protocol**

**Part I**

In the first part of the study, six different phase-1 durations were tested in 7 pigs. A constant phase-1/phase-2 duration ratio of 60%/40% was used. The corresponding phase-1 tilt was calculated by $(1-U_2/U_1)\times100 \%$. The phase-1 voltage was kept constant at 0.8 ms. The total shock durations tested were 10.8 ms (phase 1: 6 ms/phase 2: 4 ms/phase 1-tilt: 83%), 9.1 ms (5 ms/3.3 ms/78%), 7.5 ms (4 ms/2.7 ms/68%), 5.8 ms (3 ms/2 ms/60%), 4.1 ms (2 ms/1.3 ms/48%), and 2.5 ms (1 ms/0.7 ms/33%) (Fig 1 top).

**Part II**

In the second part of the study, phase-1 duration was kept constant at 3 ms, which had turned out to be the optimal phase-1 duration in the first part of the study. Phase-2 duration was then systematically varied in another 6 pigs, resulting in different phase-1/phase-2 ratios. The tested durations for phase 2 were 0.7 ms (phase-1 duration/phase-2 duration ratio: 80%/20%), 1.3 ms (75%/25%), 2 ms (60%/40%), 2.7 ms (53%/47%), 3.3 ms (48%/52%), 4 ms (43%/57%), and 6 ms (33%/67%) (Fig 1 bottom).

DFT testing was completely randomized, that is, each shock had a randomly assigned energy level and duration. Each waveform was tested five times at each energy level from 5 to 40 J in 5-J steps. Therefore, in each pig, 240 episodes of ventricular fibrillation were induced during part I and 280 episodes of ventricular fibrillation in part II. The DFT of a waveform was defined as the lowest energy level with at least 80% defibrillation success (ie, four or five of five delivered shocks were successful). Fibrillation was induced through a transvenous defibrillation electrode (Endotak 072, CPI) was introduced under fluoroscopy in the right ventricular apex. The electrode carries two coils, one positioned in the right ventricular apex (distal coil) and one in the superior vena cava (proximal coil). A submuscular patch (Endotak Sub Q 042, CPI) was then placed at the left thoracic wall opposite to the left ventricle. The electrodes were connected with the distal coil in the right ventricular apex as anode and with the proximal coil and submuscular patch as common cathode during phase-1 of the biphasic shocks. Shocks were delivered by a 70-μF capacitor (model 2394, Medtronic) that allowed separate selection of pulse width, voltage, and intershock delay. Shock voltage values of the leading edge of phase 1 to be charged on the capacitors were calculated according to the equation $E=\frac{1}{2}XCXU^2$. Voltage and current at the leading and trailing edge of phase 1 were recorded on an oscilloscope. Leading edge voltage of phase 2 was adjusted to the same value as the voltage at the trailing edge of phase 1. To estimate the voltage at the trailing edge of phase 1, monophasic shocks of all different phase-1 durations were delivered to each pig heart at each energy level. The leading edge voltage of phase 2 of the biphasic shock was then adjusted to the voltage at the end of the respective monophasic shocks.

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To ensure stable conditions of the model, the DFT was determined for the 6 ms/4 ms waveform in part I and for the 3 ms/2 ms waveform in part II at the beginning of the otherwise randomized DFT testing protocol and every 2 hours thereafter. If the DFT energy level differed by ±5 J between these testing episodes, the data obtained for that pig were excluded from analysis.

**Statistical Analysis**

All data are expressed as mean±SD. Overall differences in stored energy at the DFT for the six waveforms in part I and for the seven waveforms in part II were evaluated with ANOVA. When significant differences were present, shock durations were compared with the use of a two-tailed paired t test. Values of $P<.05$ were considered statistically significant.

**Results**

**Part I**

No pig had to be excluded from comparison because of an unstable DFT. The DFT data for each pig and waveform during part I are listed in Table 1. The shock strength–phase-1 duration curve is shown in Fig 2. Lowest defibrillation energy requirements were observed at a phase-1 duration of 3 ms ($22.9±7 \text{ J, } P<.001$, ANOVA). Shocks of shorter phase-1 durations were significantly less successful (1 ms: $36.4±7.5 \text{ J, } P<.001$), as were shocks with a phase-1 duration of 5 ms ($30.7±7.3 \text{ J, } P=.005$) and 6 ms ($32.9±8.1 \text{ J, } P=.003$). The DFT for shocks with a phase-1 duration of 1 ms differed significantly from the DFT for a phase-1 duration of 2 ms ($P=.001$). Similarly, the DFT for phase-1 durations of 4 ms was significantly lower than for phase-1 durations of 5 ms ($P=.02$). There was no significant difference between the...
shocks with a phase-1 duration of 3 ms, 2 ms (25±6.5 J), or 4 ms (25±7.6 J, \(P=\text{NS}\)).

**Part II**

According to the optimal phase-1 duration determined during part I (based on the lowest DFT), a constant phase-1 duration of 3 ms was chosen for part II. Again, no pig had to be excluded from comparison because of an unstable DFT. DFT values for each pig and waveform in part II are provided in Table 2. The phase-2 duration had a significant influence on the stored energy at the DFT (\(P=0.003\), ANOVA). This resulted in two significant DFT minima for varying phase-2 duration. One minimum was observed at 2 ms (22.5±4.2 J) and the other at 4 ms (22.5±4.2 J). Both DFTs were significantly lower than with shocks of intermediate phase-2 duration of 3.3 ms (26.7±6.1 J, \(P=0.04\) each, Fig 3). Shortening of phase-2 duration to 0.7 ms led to a significant increase of the DFT (31.7±9.3 J, \(P=0.03\) compared with a phase-2 duration of 4 ms). The mean impedance at the leading edge of pulse 1 in part I and part II was 34.2±11.6 Ω. Application of a capacitance of 70 μF resulted in a system time constant \(\tau\) of 2.4 ms in this model.

**Discussion**

Internal defibrillation energy requirements can be reduced by using biphasic instead of monophasic waveforms, by use of an active can configuration or by choosing the optimal electrode polarity. The current study focuses on the influence of different phase durations on defibrillation energy requirements with a 70-μF capacitance. Our results indicate that for such a capacitance in this animal model with a tripolar lead system, the optimal phase-1 duration was 3 ms, equaling a phase-1 tilt of 60%. Several animal and human studies have compared the defibrillation efficacy of shock waveforms delivered from currently applied 120- to 140-μF capacitances with smaller capacitances: In animal studies a lower DFT in terms of stored and delivered energy could be demonstrated with an 85-μF capacitor compared with a 140-μF capacitor for both monophasic and biphasic shocks with epicardial defibrillation patches. A dependency of the optimal phase-1 duration on changing electrode impedances has been reported in a clinical study by Swerdlow et al: By comparing shocks of 65% phase-1 tilt delivered from 60- or 120-μF capacitors, it was found that defibrillation efficacy was higher with smaller capacitances for high-impedance pathways, whereas for low-impedance pathways the opposite was true. The authors concluded that for a constant phase-1 tilt, different capacitances and thereby different phase-1 durations might be optimal for changing electrode impedances. Although in a recent clinical study there was a trend toward lower DFTs in terms of energy for 60-μF capacitors as opposed to 120 μF, this did not reach statistical significance. Because most clinically available ICDs apply shock waveforms with a constant phase-1 tilt, all except one of the studies cited above used a 65% tilt of the first phase irrespective of the capacitance applied. However, recent studies raised the issue whether different tilts than 65% would render lower DFTs: Two human studies demonstrated an increased defibrillation success with tilts of 42% to 50% compared with tilts of 65%.

![Figure 2](https://example.com/figure2.png)

**Figure 2.** Mean defibrillation threshold±SD (ordinate) for the 7 pigs of part I plotted against different phase-1 durations (abscissa). The lowest defibrillation thresholds were observed for a phase-1 duration of 3 ms (\(P<0.001\), ANOVA). Probability values given in the figure refer to two-tailed paired \(t\) tests for comparison between individual waveforms.

<table>
<thead>
<tr>
<th>Animal 1</th>
<th>Phase 1: 1 ms</th>
<th>Phase 1: 2 ms</th>
<th>Phase 1: 3 ms</th>
<th>Phase 1: 4 ms</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>40</td>
<td>25</td>
<td>25</td>
<td>25</td>
<td>35</td>
<td>36.4</td>
</tr>
<tr>
<td>Animal 2</td>
<td>20</td>
<td>15</td>
<td>10</td>
<td>15</td>
<td>25</td>
</tr>
<tr>
<td>Animal 3</td>
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<td>25</td>
<td>30</td>
<td>30</td>
<td>35</td>
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<td>15</td>
<td>20</td>
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<tr>
<td>Animal 7</td>
<td>40</td>
<td>20</td>
<td>20</td>
<td>25</td>
<td>25</td>
</tr>
</tbody>
</table>

**TABLE 1.** Defibrillation Threshold Values for the Seven Pigs and Six Different Phase-1 Durations in Part I of the Study

<table>
<thead>
<tr>
<th>Phase 1: 1 ms</th>
<th>Phase 1: 2 ms</th>
<th>Phase 1: 3 ms</th>
<th>Phase 1: 4 ms</th>
<th>Phase 1: 5 ms</th>
<th>Phase 1: 6 ms</th>
</tr>
</thead>
<tbody>
<tr>
<td>36.4</td>
<td>25</td>
<td>22.9</td>
<td>25</td>
<td>30.7</td>
<td>32.9</td>
</tr>
</tbody>
</table>

SD: 7.5 6.5 7 7.6 7.3 8.1

The ratio of phase-1 duration/phase-2 duration was 60%/40%.
to 80% and a 120-μF capacitance. In one study the time constant τ was calculated to be ~8 ms. Therefore, pulse 1 tilts of 65% as used in many clinical devices may be too high, possibly because of refibrillation caused by low-voltage shock tails compared with lower tilts. However, simple replacement of a phase-1 tilt of 65% by a fixed phase-1 tilt of 42% to 50% may not always be advantageous. On the basis of mathematical models, the optimal phase-1 tilt increases with decreasing time constant τ for monophasic as well as for biphasic shocks. We could demonstrate that for a low time constant of 2.4 ms, the optimal phase-1 tilt was 60%. Despite the differences of the models this is higher than the optimal phase-1 tilt for larger capacitances and higher impedances, thus supporting the concept that the optimal phase-1 tilt is dependent on the time constant. Consequently, studies that compare the defibrillation efficacy of shocks delivered from different capacitances should test each capacitance at its waveform with the optimal tilt. In fact, because the volume of the capacitor is proportional to the maximal stored energy, only a reduction of the DFT by low-capacitance shocks with optimized phase duration compared with shocks from currently available 120- to 150-μF capacitances at optimal phase duration would result in lower capacitor volumes.

Several mechanisms have been suggested to contribute to defibrillation with monophasic shocks such as prolongation of action potential duration and refractory period or resynchronization of the repolarization state of the myocardium. Beyond that, depolarization of myocardial cells is considered to be an important mechanism. Experimentally, a hyperbolic stored voltage–shock duration curve at the DFT has been found for monophasic shocks. A similar strength-duration relation has been proposed to sufficiently describe the action of phase-1 of biphasic shocks, which is supported by the findings in part I of the present study.

There are conflicting results on the optimal ratio of phase-1/phase-2 duration. In an animal study, Dixon et al demonstrated lower energy requirements for ventricular defibrillation with biphasic shocks and a second phase of equal or shorter duration than the first phase. Similarly, Gliner et al found lower ventricular defibrillation energy requirements in external defibrillation for a phase-1 duration set at 50% or 60% of the total shock duration. In human atrial fibrillation, Cooper et al most recently observed lowest internal atrial defibrillation thresholds for biphasic shocks with a phase-1 duration longer than phase-2 duration. However, in a human study on internal ventricular defibrillation, different tilts of the second phase of the biphasic shock did not significantly affect defibrillation energy requirements. By contrast, in an animal study on ventricular defibrillation, lower DFTs for shocks with the second phase longer than the first phase were reported. The differences in underlying arrhythmias and models make the comparison between these studies difficult. However, a possible reason for the findings of higher defibrillation success of shocks with phase-2 durations longer than phase-1 duration for lower time constants is provided by Kroll. Because the residual charge left on the cell membrane after delivery of phase 1 is thought to cause postshock arrhythmias, thus diminishing defibrillation success, he assumed that the major function of phase 2 was to unload (“burp”) this residual membrane charge. Thus according to his mathematical calculation, the optimal phase-2 duration would become longer than phase-1 duration at time constants τ shorter than 3 ms as experimentally demonstrated in our study. In the current study, two phase-1/phase-2

### Table 2: Defibrillation Threshold Values for the Six Pigs and Seven Different Phase-2 Durations During Part II of the Study

<table>
<thead>
<tr>
<th>Animal</th>
<th>Phase 2: 0.7 ms</th>
<th>Phase 2: 1.3 ms</th>
<th>Phase 2: 2 ms</th>
<th>Phase 2: 2.7 ms</th>
<th>Phase 2: 3.3 ms</th>
<th>Phase 2: 4 ms</th>
<th>Phase 2: 6 ms</th>
</tr>
</thead>
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<td>Animal 9</td>
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<td>Animal 11</td>
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<td>Animal 13</td>
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<td>20</td>
<td>25</td>
<td>20</td>
<td>20</td>
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</tr>
<tr>
<td>Mean</td>
<td>31.7</td>
<td>26.7</td>
<td>22.5</td>
<td>25</td>
<td>26.7</td>
<td>22.5</td>
<td>28.3</td>
</tr>
<tr>
<td>SD</td>
<td>9.3</td>
<td>9.3</td>
<td>4.2</td>
<td>7.1</td>
<td>6.1</td>
<td>4.2</td>
<td>6.8</td>
</tr>
</tbody>
</table>

*Because of an overall high defibrillation threshold in this animal, 45 J was tested for this waveform.

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**Figure 3.** Mean defibrillation threshold±SD (ordinate) for the 6 pigs of part II plotted against different phase-2 durations (abscissa). Duration of phase-1 was constant at 3 ms. Defibrillation threshold showed two minima, which differed significantly from the neighboring phase-2 durations (P < .003, ANOVA). Probability values given in the figure refer to two-tailed paired t tests for comparison between individual waveforms.
duration ratios with minimal defibrillation energy requirements at the DFT were observed, 60%/40% (phase-2 duration: 2 ms) and 43%/57% (phase-2 duration: 4 ms). Similar findings have been described by Feeser et al. Using a constant phase-1 duration of 3.5 ms, they found two voltage minima at the DFT for phase-2 durations of 4 ms and 7 ms with significantly higher voltages at the DFT for phase-2 durations between these two values. They suggested two possible mechanisms accounting for these observations: (1) Depolarization after hyperpolarization: The role of the second phase is to depolarize that half of the myocardial cell membrane that has been hyperpolarized during phase 1. The effect that hyperpolarization during phase 1 of the biphasic shock reactivates sodium channels that were partially inactivated because of low resting membrane potentials during ventricular fibrillation was originally described by Jones et al. They called this “prepulse conditioning.” (2) Hyperpolarization after depolarization: Excitation on the side of the cell that has been depolarized during phase 1 may be halted by the now-hyperpolarizing phase 2. Halting of myocardial excitation by a hyperpolarizing impulse after the excitation threshold has been reached was described by Weidmann. It was proposed that with increasing phase-2 durations first, the mechanism of depolarization after hyperpolarization might be predominating, thus resulting in lower voltages at the DFT. Then, with longer phase-2 durations the effect of hyperpolarization after depolarization may outweigh the beneficial effect of depolarization after hyperpolarization on the other side of the myocardial cells, thus leading to an increase of the voltage at the DFT. Finally, further prolongation of phase-2 duration makes phase 2 behave like a monophasic shock with a second decrease and increase of the voltage at the DFT. These results on phase-2 durations differ from clinically used shock waveforms that include fixed phase-1/phase-2 duration ratios of 60%/40% or 50%/50%. The latter is based on an animal study of Tang et al that investigated current strength requirements for a constant phase-1 duration of 3.5 ms and changing phase-2 durations. Lowest current at the DFT was demonstrated for a phase-2 duration of 2 ms. However, two optimal phase-2 durations may have been missed in that study because of the large increments of the phase-2 durations in contrast to the results of Feeser et al (1-ms increments) and our own findings (0.6- to 0.7-ms increments).

Study Limitations
In this porcine animal model with a tripolar lead system including a submucosal patch, the time constant τ was short because of the low impedances observed compared with most human ICD implants. However, the finding of more than one optimal phase-2 duration may be extrapolated to other lead configurations and impedances because similar results have been found despite different models. Although the relative heart weight of the applied pigs, which is 0.3% of the body weight of the animal, is comparable to humans, differences of this model due to the lack of organic heart disease and the mode of induction of ventricular fibrillation may have caused differences to spontaneous episodes of ventricular fibrillation in patients.

Conclusions
Similar to monophasic shocks, the shock strength–duration curve of biphasic shocks delivered from 70-μF capacitors demonstrated a single optimum for phase-1 duration at 3 ms, equaling a phase-1 tilt of 60%. In contrast, for an optimized phase-1 duration, two significant minima of defibrillation energy requirements were found for phase-2 duration, thereby supporting the concept that the duration of the two phases is optimized independent of each other. Both may be clinically relevant for future optimization of ICD design and programming.

References


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