

Ventricular Defibrillation Using Biphasic Waveforms: The Importance of Phasic Duration

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Biphasic waveforms can be used to defibrillate the heart with less energy than that used by monophasic waveforms. In 14 anesthetized open chest dogs with large contoured defibrillation electrodes, the effect on defibrillation efficacy of varying the duration of the two phases of biphasic waveforms was studied. All combinations of 0, 1, 3.5, 6 and 8.5 ms duration were used for both the first and the second phase except for the meaningless case in which both durations were 0 ms. The 3.5-2 waveform (3.5 ms first phase and 2 ms second phase) was also tested.

All the hearts were defibrillated with ≤ 5 joules using any of the 25 waveforms. However, biphasic waveforms with the second phase shorter than or equal to the first had significantly lower defibrillation thresholds than did those with the second phase longer than the first or than did monophasic waveforms of approximately the same total duration. A plot of defibrillation threshold current strength versus second phase duration for all biphasic waveforms

with a 3.5 ms first phase did not produce a hyperbolic strength-duration curve as seen with monophasic waveforms. To verify these findings, defibrillation dose-response curves were obtained for the 3.5-2, 6-6 and 3.5-8.5 biphasic waveforms in another six dogs. The 50 and 80% successful voltage doses of the 3.5-8.5 waveforms were significantly higher than those of the other two waveforms, which were not different from one another.

In conclusion: 1) phasic durations of biphasic waveforms are important determinants of defibrillation efficacy and biphasic waveforms with the second phase shorter than the first are more effective than are those with the reverse sequence; 2) the strength-duration relation for the defibrillation threshold is different for biphasic and monophasic waveforms; 3) defibrillation of the canine heart can be achieved with low energy with use of large contoured pericardial electrodes and suitable biphasic waveforms.

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The automatic implantable cardioverter-defibrillator is an effective means of treating patients with potentially fatal ventricular arrhythmias (1-4). Its electrode system typically consists of a spring electrode with a surface area 7 to 10 cm² or a patch electrode with a surface area 13.5 or 27 cm² (2). It delivers monophasic truncated exponential pulses ranging in duration from 3 to 8 ms depending on the interelectrode impedance (3). The device could be improved if the energy, voltage and current required for defibrillation could be decreased. Possible ways to decrease defibrillation shock

strength are by altering the electrode configuration and shock waveforms. Large contoured electrodes applied to the canine epicardium or pericardium decrease the defibrillation threshold substantially compared with standard patch electrodes (5).

Compared with monophasic waveforms, waveforms consisting of two phases of opposite polarity decrease the shock strength needed for defibrillation (6). Jones and Jones (7,8) reported improvement in the "safety factor" for defibrillation using biphasic waveforms as compared with monophasic waveforms. Schuder et al. (9,10) found a significant increase in the percent success of defibrillation using biphasic rectangular waveforms in which both phases were symmetric as well as in which both phases were equal in duration but the first phase was larger than the second. We found that phasic duration also influences the defibrillation efficacy of biphasic waveforms (5). The 6.5-3.5 waveform (first phase lasting 6.5 ms and the second lasting 3.5 ms) had a lower defibrillation threshold than did the 3.5-6.5 waveform, and

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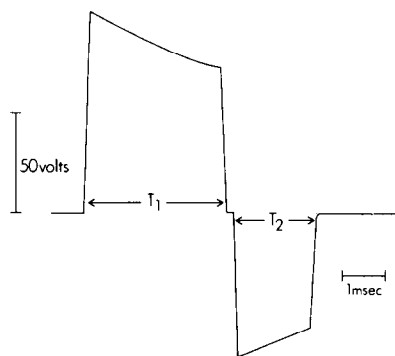


Figure 1. Diagram of a biphasic waveform. Voltage decreases exponentially throughout both phases. T_1 and T_2 denote the duration of the two phases. The time between the trailing edge of the first phase and the leading edge of the second phase is $120 \mu\text{s}$. The trailing edge of the first phase is equal to the leading edge of the second phase.

the defibrillation threshold for the 7.5-2.5 waveform was lower than that for the 2.5-7.5 waveform. Whereas the relation between shock strength and duration for monophasic defibrillation shocks has been well studied (11-13) and found to be hyperbolic for shocks lasting up to 15 to 40 ms, it is not known if biphasic waveforms have a similar strength-duration relation.

The purpose of this study was to investigate the effect of phasic duration on the defibrillation efficacy of biphasic waveforms. The influence of the duration of each of the two phases as well as the duration of the total waveform was studied to determine which biphasic waveforms defibrillate with the least shock strength. Both the defibrillation threshold and the defibrillation percent success curve (14) were measured. The strength-duration relation was also determined for one family of biphasic waveforms.

Methods

Equipment and waveforms. The defibrillator (Intermedics Inc.) delivers truncated exponential waveforms from a $175 \mu\text{F}$ capacitor bank and contains a switch to reverse the polarity at a predetermined time. The leading edge of the second phase begins $120 \mu\text{s}$ after the trailing edge of the first phase and is the same voltage as the trailing edge of the first phase (Fig. 1). The current and voltage of the shocks were measured through 10:1 and 1,000:1 dividers by a waveform analyzer (Data Precision, model DATA 6000) that digitized the signal at a frequency of 200 kHz. Impedance and energy were calculated from the digitized voltage and current by a microprocessor in the waveform analyzer. The lead II electrocardiogram (ECG) and femoral artery pressure were monitored continuously.

Procedure: Part I. Fourteen mongrel dogs (weight 21.5 ± 4.3 kg, mean \pm SD) were anesthetized with intravenous

pentobarbital, 30 to 35 mg/kg body weight, followed by a continuous intravenous infusion of approximately 0.05 mg/kg per min (15). Succinylcholine, 1 mg/kg, was injected intravenously on induction of anesthesia and supplemental doses of 0.25 to 0.5 mg/kg were given when required, but no more than once per hour. The dogs were intubated and ventilated with a Harvard respirator (Harvard Apparatus Co.). Body temperature was continuously monitored and maintained between 36.5 and 38°C with an electric blanket. Arterial blood gases and electrolytes including calcium were determined hourly. Potassium chloride and sodium bicarbonate were given when indicated.

The heart was exposed through a median sternotomy. A pair of large contoured electrodes was sutured to the pericardium (5). One electrode, approximately 39 cm^2 in surface area, was placed over most of the free wall and apex of the left ventricle. A 33 cm^2 electrode was placed to cover most of the outflow tract and base of the right ventricle. Particular attention was made to ensure maximal separation as well as equal spacing between the two electrodes (5).

Ventricular fibrillation was induced with 60 Hz alternating current through a pair of wires sutured to the pericardium between the defibrillation electrodes. The threshold current and voltage for defibrillation was determined by a modification of Bourland's method (12). A shock of 4 joules was used for the initial defibrillation trial of each waveform in the first dog. For subsequent dogs, the initial test shock was the mean threshold of the previous dogs tested for that waveform. The test shock was delivered after 10 s of fibrillation. After unsuccessful shocks, a rescue shock of approximately 1.5 times the mean defibrillation threshold of the previous dogs was used. Before another shock was tested, the dog was allowed to recuperate for ≥ 5 min until hemodynamic stabilization was achieved. The leading edge voltage was decreased by 20 V for the next shock if the previous shock was successful or increased by 20 V if the previous shock was unsuccessful. This procedure was continued until an opposite result was obtained, after which a final shock was given with a leading edge voltage equal to the mean of the last two attempts. The lowest strength successful shock was taken as the defibrillation threshold.

Monophasic and biphasic waveforms that spanned the intervals likely to be of clinical importance for implantable defibrillators were studied. The durations tested for each phase were 1, 3.5, 6 and 8.5 ms. With the use of the Latin square design for sampling (16), all 16 combinations of first and second phase durations were examined. The left ventricular electrode was the cathode for the first phase and the anode for the second phase. The durations of the monophasic waveforms tested were also 1, 3.5, 6 or 8.5 ms. Monophasic waveforms were tested with the left ventricular electrode as the cathode and as the anode. Thus eight monophasic waveforms were tested. It was not possible to determine all 24 defibrillation thresholds in each dog because

about 100 episodes of fibrillation would be required. Therefore a random selection of 12 waveforms was studied in random order in one dog, and the other 12 waveforms were studied in the next dog, also in random order.

In addition, a 3.5-2 waveform (3.5 ms first phase and 2 ms second phase) was tested in all animals. The 3.5-2 waveform was selected because in our pilot studies it had a low defibrillation threshold and it provided more data so that a strength-duration curve could be constructed with a constant first phase duration (3.5 ms) and different second phase durations. The 3.5-2 waveform was always the seventh waveform tested.

Procedure: Part II. Defibrillation success is better represented by a dose-response curve than by a simple threshold value (14,17,18). Because many more shocks are required to determine a dose-response curve than a defibrillation threshold, only a few curves can be found in the same animal. To verify the results found by defibrillation thresholds in Part I of this study, dose-response curves were determined for three of the waveforms in another six dogs (weight 21.0 ± 3.5 kg). Dose-response curves were compared with defibrillation thresholds for the 3.5-2 and 6-6 waveforms, both of which had low defibrillation thresholds in Part I, and for the 3.5-8.5 waveform, which had a high defibrillation threshold in Part I.

Defibrillation thresholds were determined five times for each waveform, and a dose-response curve was calculated from all of the shock attempts used to determine the five defibrillation thresholds. For each of the three waveforms, a defibrillation threshold was determined as described with the mean voltage determined in Part I as the voltage of the initial shock. The other four defibrillation thresholds were determined similarly except that the initial shock voltages were 20 and 30 V above and below the first determined defibrillation threshold. In the process of determining the defibrillation thresholds, 21 ± 5 defibrillation shocks were administered to each dog for each waveform. By noting the success and failure of each attempt, a dose-response relation was calculated for each waveform in each dog.

Statistical analysis. An analysis of variance was used to compare threshold variables among waveforms in Part I. Multiple comparisons between waveforms were made with the Student's *t* test and the Student-Neuman-Keuls test (19). In Part II, the relation between leading edge voltage and percent successful defibrillation for each waveform in each dog was fitted to a dose-response curve with probit regression analysis (19,20). The voltages associated with 50 and 80% predicted success (ED_{50} and ED_{80}) were calculated. For one waveform (3.5-8.5) in one dog (see Dog 6, Table 3) the probit fit to the data was unsatisfactory because the data points were too widely scattered. For this one case, probit regression analysis was performed after grouping data from adjacent voltage levels. Comparisons of ED_{50} and ED_{80} among the waveforms were made using analysis of variance.

and multiple comparisons between waveforms were made with the Student-Neuman-Keuls test. The correlation between the ED_{50} and the mean threshold voltage of all three waveforms was found using least squares linear regression analysis. Results are reported as mean \pm SD, and a $p < 0.05$ was considered statistically significant for all analyses.

Results

Part I

Defibrillation values (Table 1). For the monophasic waveforms, the defibrillation threshold voltage and leading edge current decreased with increasing pulse duration as expected from previous studies (11-13). Reversing polarity of the monophasic waveform did not significantly change the threshold (e.g., 3.5-0 versus 0-3.5). As a group, the biphasic waveforms in which the first phase was longer than the second phase had lower threshold energy, current and voltage than the group in which the first phase was shorter than the second. When each complementary pair (e.g., 3.5-1 versus 1-3.5) was compared individually by the Student's *t* test, the waveform in which the first phase was longer (3.5-1) had a lower threshold than did the complementary waveform (1-3.5). When the same comparisons were made with the more stringent Student-Neuman-Keuls test, similar results were found for most pairs of biphasic waveforms (as indicated by asterisks in Table 1).

Some biphasic waveforms had lower defibrillation thresholds than did monophasic waveforms of similar or even longer total duration. For example, the 3.5-3.5 waveform had a lower defibrillation threshold than did the 8.5 ms monophasic waveform, although the total duration of the 3.5-3.5 waveform was only 7 ms. Similarly, the threshold for the 3.5-1 waveform was lower than for the 6 ms monophasic waveform. However, not all biphasic waveforms had a lower defibrillation threshold than did monophasic waveforms of similar total duration. For example, the 1-8.5 waveform had a higher threshold than did the monophasic 6 ms waveform, even though the total duration of the 1-8.5 waveform was > 6 ms. Thus, not only did biphasic waveforms with the second phase longer than the first have a higher threshold than when the two phasic durations were reversed, they also had a higher threshold than that of monophasic waveforms of similar total duration.

Strength-duration curve (Table 2). The thresholds of all biphasic waveforms with a first phase of 3.5 ms were compared with the 3.5 ms monophasic waveform. For all threshold indexes, i.e., voltage, current and energy, the first three biphasic waveforms (3.5-1, 3.5-2 and 3.5-3.5) were similar. However, the thresholds for these three waveforms were significantly lower than those of the 3.5-6 waveform, which in turn were lower than those of the 3.5-8.5 waveform.

Figure 2 shows a plot of peak current versus duration for these waveforms. With increasing waveform duration, peak

Table 1. Threshold Variables of the Biphasic and Monophasic Waveforms Studied in Part I (14 dogs)

First Phase Duration (ms)			Threshold Energy (joule)				
8.5	1.2 ± 0.6	1.2 ± 0.5*	0.9 ± 0.2*	0.8 ± 0.1*	1.3 ± 0.8		
6	1.3 ± 0.6	0.9 ± 0.3*	0.8 ± 0.3*	0.7 ± 0.2	1.9 ± 1.5*		
3.5	1.2 ± 0.3	0.6 ± 0.2*	1.0 ± 0.4	1.6 ± 0.8*	3.2 ± 0.8*		
1	0.8 ± 0.4	1.0 ± 0.3	1.8 ± 0.4*	1.8 ± 0.6*	2.5 ± 0.9*		
0	—	0.8 ± 0.3	1.1 ± 0.2	1.7 ± 0.7	1.8 ± 0.7		
Second phase duration (ms)			Threshold Voltage (V)				
	0	1	3.5	6	8.5		
8.5	117 ± 30	110 ± 26*	99 ± 10*	89 ± 7*	106 ± 32		
6	127 ± 34	108 ± 19	93 ± 16	81 ± 13	127 ± 43*		
3.5	145 ± 22	99 ± 11*	108 ± 22	128 ± 40	186 ± 29*		
1	198 ± 53	166 ± 25	158 ± 23*	142 ± 26	161 ± 31*		
0	—	202 ± 29	140 ± 16	143 ± 30	139 ± 28		
Second phase duration (ms)			Threshold Current (A)				
	0	1	3.5	6	8.5		
8.5	2.5 ± 0.5	2.7 ± 0.6*	2.0 ± 0.5*	2.0 ± 0.4	2.2 ± 0.7		
6	2.8 ± 0.8	2.2 ± 0.5*	1.9 ± 0.4	1.8 ± 0.3	2.8 ± 1.0		
3.5	3.2 ± 0.5	2.2 ± 0.7*	2.2 ± 0.5	2.7 ± 0.7	4.0 ± 0.5*		
1	4.4 ± 1	3.5 ± 0.6	3.6 ± 0.6*	3.5 ± 0.5*	3.6 ± 1*		
0	—	4.4 ± 0.6	3.1 ± 0.5	3.6 ± 1.1	3.1 ± 1		
Second phase duration (ms)			0	1	3.5	6	8.5

*Complementary pairs are also significantly different by the Student-Neuman-Keuls test. Each biphasic complementary pair is significantly different by the Student's *t* test.

current remained relatively unchanged until the duration of the second phase was longer than that of the first; peak current then increased as duration increased. This plot is not hyperbolic and thus is different from that of monophasic waveforms (11-13).

Impedance. Because the current and voltage of the shocks were digitized at a fast rate by the waveform analyzer, impedance could be calculated throughout the duration of the defibrillation pulse. All biphasic waveforms had similar impedance profiles. As an example, the impedance profile of the 3.5-2 waveform for a 100 V shock is shown in Figure 3. Impedance increased $7.5 \pm 1.8\%$ throughout the first phase. When the polarity was reversed, impedance abruptly decreased. It then increased throughout the second phase by $5.6 \pm 1.3\%$. The impedance of the first phase ($50.4 \pm 8.0 \Omega$) was higher than that of the second phase ($43.6 \pm 6.8 \Omega$). The ratio of the mean impedance of the first phase to that of the second phase was 1.16.

To understand further the cause of the change in impedance throughout the waveform, a 100 V 3.5-2 shock was delivered into a 50 Ω resistor. Impedance remained constant

throughout each phase, 49.4 Ω during the first and 48.3 Ω during the second. The ratio of the impedance of the first phase to that of the second was 1.02. Thus the majority of the impedance drop during the polarity switch did not occur at the defibrillator or the measuring device.

A 100 V 3.5-2 biphasic shock was then discharged into a tank filled with saline solution with an impedance of about 50 Ω . The contoured electrodes were sutured to a sponge submerged in the tank. Impedance increased 6.9% throughout the first phase and 3.7% throughout the second. The mean impedances of these two phases was 54.6 and 48.3 Ω , respectively. The ratio of the two was 1.13, which is similar to the ratio observed when the shock was delivered to the heart. These results suggest that the impedance change was primarily at the electrode-electrolyte (tissue) interface or in the electrolyte (tissue) itself.

Part II

Dose-response defibrillation curves. For each of three waveforms (3.5-2, 6-6 and 3.5-8.5), curves relating percent

Table 2. Threshold Variables of the Monophasic 3.5 ms Waveform and the Five Biphasic Waveforms With a 3.5 ms First Phase

Peak voltage (V)	145 ± 22	99 ± 11	92 ± 20	108 ± 22	128 ± 40	186 ± 29
Peak current (A)	3.2 ± 0.5	2.2 ± 0.7	2.0 ± 0.5	2.2 ± 0.5	2.7 ± 0.7	4.0 ± 0.5
Energy (joules)	1.2 ± 0.3	0.6 ± 0.2	0.6 ± 0.3	1.0 ± 0.4	1.6 ± 0.8	3.2 ± 0.8
Second phase duration (ms)	0	1	2	3.5	6	8.5

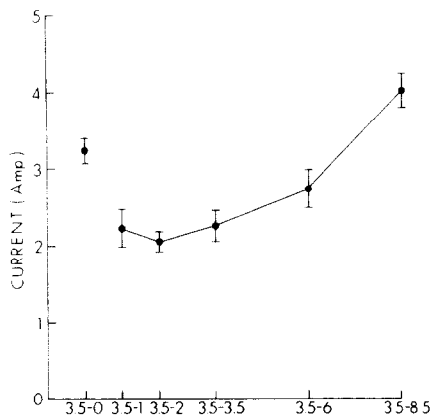


Figure 2. Plot of current strength (mean \pm SD) at defibrillation threshold versus the duration of the second phase of all five biphasic waveforms with a first phase duration of 3.5 ms. As the duration of the second phase increases, threshold current is stable until $T_2 = T_1$, then increases sharply for $T_2 > T_1$. For comparison, the threshold current for the monophasic 3.5 ms waveform (3.5-0) is also shown.

success and defibrillation voltage levels (dose-response defibrillation curves) were constructed for each of six additional dogs (Fig. 4). The curves for the 3.5-2 and 6-6 waveforms were so similar as to be almost superimposable. The curve for the 3.5-8.5 waveform was shifted to the right, which indicated a higher voltage requirement for defibrillation.

For all waveforms in all dogs, the mean defibrillation threshold voltage was highly correlated ($r = 0.99$) with the ED_{50} derived from the dose-response curves (Fig. 5). There were no significant differences for the ED_{50} , ED_{80} or the mean threshold voltage between the 3.5-2 and the 6-6 waveforms (Table 3). However, the ED_{50} , ED_{80} and the mean threshold voltage for the 3.5-8.5 waveform were significantly higher than those for the other two waveforms. Thus, the dose-response method to assess the efficacy of defibrillation

Figure 3. The impedance profile of a biphasic waveform (3.5-2 ms). The impedance was calculated by Ohm's law from the measured voltage and current at each digitized point. Impedance increases during each phase, although at the phase change there is a decrease of impedance.

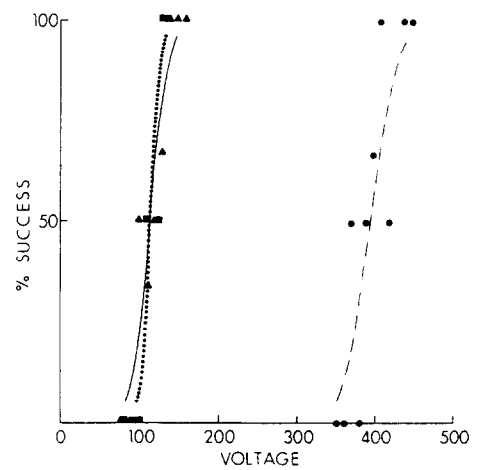
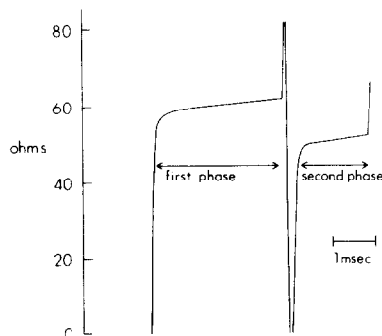


Figure 4. Data points and dose-response defibrillation curves fitted by probit regression analysis for the three waveforms 3.5-2 (triangles, solid line), 6-6 (squares, dotted line), and 3.5-8.5 (circles, dashed line) in a representative dog. The curves for the 3.5-2 and 6-6 waveforms are almost superimposable, but the 3.5-8.5 curve is shifted to the right indicating a higher voltage requirement for defibrillation.

of these three waveforms is in agreement with the defibrillation threshold method.

Discussion

Importance of phasic duration. Biphasic waveforms have been shown to be more effective than monophasic waveforms for ventricular defibrillation in both animals and humans (6,9,10,21,22). In a previous study (5), we suggested that the durations of the two phases influence defibrillation efficacy. This study confirms this finding by a systematic comparison of many complementary pairs of waveforms

Figure 5. Correlation between the 50% predicted success rate (ED_{50}) and the mean defibrillation threshold (DFT) of the three waveforms studied in Part II. There is a close relation with a correlation coefficient of 0.99 and a slope of 1.05.

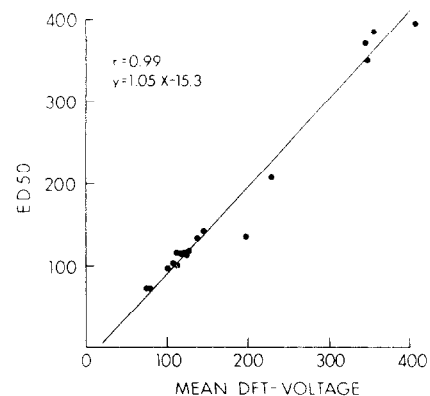


Table 3. Mean Defibrillation Threshold (DFT) Voltages, 50% and 80% Predicted Success (ED₅₀ and ED₈₀) Voltages for the Waveforms 3.5-2, 6-6 and 3.5-8.5 ms From Part II

Dog No.	Mean DFT			ED ₅₀			ED ₈₀		
	3.5-2	6-6	3.5-8.5	3.5-2	6-6	3.5-8.5	3.5-2	6-6	3.5-8.5
1	126	146	348	118	143	352	183	150	548
2	138	124	346	134	115	373	165	136	425
3	108	102	230	103	97	210	110	109	252
4	112	114	352	101	114	383	129	125	452
5	122	118	408	115	116	399	131	125	409
6	78	80	198	73	73	137*	79	93	222*
Mean	114	114	314 [†]	107	110	309 [†]	133	123	388 [†]
SD	21	22	81	21	23	109	37	20	126

*Unable to obtain a good probit fit from the data (grouped data are used); [†]significantly different from waveforms 3.5-2 and 6-6.

spanning a large range of phase durations. The biphasic waveform in which the duration of the second phase was shorter than the first was usually more efficacious than the complementary biphasic waveform of the same total duration in which the phasic durations were reversed. On the basis of inhibition of spontaneous beating in cultured myocardial cells, Jones and Jones (8) suggested that biphasic waveforms with a longer first than second phase may also cause less postshock arrhythmias and myocardial dysfunction.

Because future generation automatic implantable defibrillators will probably employ biphasic waveforms and may allow the durations of the two phases to be programmable, it is important to be aware that some combinations of phasic durations are much better than others. Whether expressed in terms of energy, voltage or current, the 3.5-1, 3.5-2, 6-3.5 and 6-6 waveforms defibrillated with very low thresholds. Because the 6-6 waveform has a total duration of 12 ms and thus delivers almost all of the stored charge, and because it requires a low leading edge voltage, this waveform may be the best of those tested for delivering a single shock with an implantable defibrillator. If two sequential biphasic shocks (23,24) or a triphasic shock (25,26) are to be given through a single capacitor defibrillator, then a shorter shock, e.g., the 3.5-2 waveform may be preferable because more charge is left in the capacitor for the second sequential or third phase shock. The results may not apply to waveforms generated by devices with capacitors markedly different from 175 μ F.

Defibrillation threshold versus probability of success curve. In the Part I study, waveform efficacy was measured by determining the defibrillation threshold (12). By "threshold," we are not suggesting a sharp cutoff point below which all attempts to defibrillate are unsuccessful and above which all attempts are successful. We employed a single value "threshold" to represent the efficacy of a waveform because it could be measured easily with only a few fibrillation-defibrillation episodes. Defining the entire probability of success or dose-response curve is a better way to show the spectrum of responses to different defibrillation strengths

(14). To define the dose-response curve properly, however, many fibrillation-defibrillation episodes must be performed. Thus, it is only possible to evaluate a few waveforms in any one animal. Because we were interested in searching for marked differences in defibrillation efficacy among many different waveforms, we chose to determine the defibrillation threshold instead of the dose-response curve, even though the latter is probably more sensitive for detecting small differences in defibrillation efficacy, particularly away from the midportion of the curve.

To verify the results based on the defibrillation threshold, in Part II we obtained dose-response curves for three waveforms tested in Part I. We chose these three particular waveforms because two of them (3.5-2, 3.5-8.5) had very different thresholds in Part I, whereas two (3.5-2, 6-6) had similar thresholds, both of which were very low. The results based on dose-response curves supported the results on the basis of defibrillation thresholds, showing a marked difference in defibrillation efficacy between the 3.5-2 and 3.5-8.5 waveforms, whereas defibrillation efficacies for the 3.5-2 and 6-6 waveforms were nearly identical.

The defibrillation thresholds found in Part I were near the 50% predicted success (ED₅₀) points on the dose-response curves found in Part II. This point is lower than that reported by Rattes et al. (27) who found that the defibrillation threshold was near the ED₇₅ point. This discrepancy is probably because of differences in the way that the defibrillation threshold was determined (14). We took the mean threshold of the previous experiments as the initial test shock and either increased or decreased shock strength depending on the outcome of the previous shock, whereas they always started from a high value and decreased shock strength until the shock was unsuccessful.

Reason for increased defibrillation efficacy of biphasic shocks. After the switch in polarity of a biphasic waveform, a decrease in impedance was measured across the defibrillation electrodes (Fig. 3). Hence, one possible explanation for the increased defibrillation efficacy of the biphasic waveform could be decreased impedance. During a monophasic

shock and during both phases of a biphasic shock, impedance increases (Fig. 3). One reason for the increase in impedance is the exponential decrease in voltage during the shock because impedance increases as shock voltage decreases (28). Other possible reasons for the increase in impedance are changes in the distribution of ion concentrations, either throughout the extracellular fluid or as polarization at the electrode-electrolyte interface (29). These changes in ion concentration are comparable with charging a capacitor in that the net effect is opposition to an applied voltage that increases with time. Conversely, if the voltage is reversed during the shock, the altered ion concentrations will aid current flow until a uniform distribution of ions is again present. Therefore, the sudden drop in impedance after the reversal of polarity during a biphasic waveform is probably caused by the uneven distribution of ions induced by the first phase of the shock.

However, the decrease of impedance is not sufficient to account for all of the improved efficacy of biphasic compared with monophasic waveforms. The increased efficacy of the biphasic waveforms in terms of voltage and energy is greater than that caused by the decrease in impedance, and increased efficacy was also present when shock strength was expressed in terms of current (Table 1), which should be independent of impedance (30). Decreased impedance also cannot explain why biphasic waveforms with the second phase longer than the first are less effective than monophasic waveforms of the same total duration in spite of the decrease in impedance.

A second explanation for the increased efficacy of biphasic waveforms is that the first phase reactivates sodium channels in the myocardial membrane so that the cells can be excited by the second phase. Jones et al. (31) found that compared with monophasic waveforms, biphasic waveforms reduced the excitation threshold of chick embryo myocardial cells bathed in a high potassium solution. The potential at rest has been reported to be reduced to about -60 mV during reperfusion-induced fibrillation so that sodium channels may be totally or partially inactivated (32). Jones et al. (31) hypothesized that the first phase of the biphasic waveform acts as a "conditioning" pulse, causing hyperpolarization of some portions of the heart. By bringing the transmembrane potential in these portions of the heart closer to the normal potential at rest, the first pulse reactivates the sodium channels. The second phase, which depolarizes these portions of the heart, is then able to excite the cells thus lowering the excitation threshold and hence the defibrillation threshold.

A third possible mechanism for the increased efficacy of biphasic shocks is shortening of the refractory period due to hyperpolarization of the transmembrane potential by the first phase. Cells that are refractory may not be depolarized by the first phase of the shock. Nonetheless, the first phase may still affect the cells by changing the duration of their refrac-

tory period (33). A hyperpolarizing pulse given to the cell during phase two of the action potential shortens the refractory period so that a depolarizing pulse applied immediately afterward can then activate the cell. The biphasic waveform may have the same action.

Strength-duration curve. For monophasic waveforms ≤ 15 to 40 ms long, the strength-duration curve for defibrillation is hyperbolic (11-13). Current and voltage requirements for defibrillation are very high for brief shocks but rapidly decrease as shock duration is increased. This is similar to the strength-duration curve observed for the stimulation threshold of nonfibrillating, fully recovered myocardium (34). As monophasic shocks are extended > 15 to 40 ms, the strength required for defibrillation increases (35). In contrast, we found that the strength-duration curve for biphasic shocks is not hyperbolic. As waveform duration is increased by prolonging the second phase while holding the first phase constant, the shock strength required for defibrillation first decreases as for the monophasic waveform but then begins to increase much earlier than 15 to 40 ms. This is seen most clearly with a first phase duration of 3.5 ms, for which more data points were examined (Fig. 2), but may be true also for biphasic waveforms with other first phase durations, although the nadirs of the curves appear shifted in time (Table 1).

The reason for the shape of the strength-duration curve for biphasic waveforms is not known. One possible explanation is that the trailing edge of the second phase activates fibrillating myocardium by "break" excitation (36,37). Because the trailing edge of the second phase of a truncated exponential waveform becomes smaller with increasing duration, the shock strength required for defibrillation would be expected to increase, as was observed. Other factors, however, besides the trailing edge of the second phase must also be important for defibrillation because two biphasic waveforms of the same total duration and, hence, the same trailing edge voltage of the second phase (e.g., 3.5-8.5 and 8.5-3.5) may have markedly different defibrillation thresholds. These two waveforms also have the same leading edge voltage for the first phase and both deliver the same total amount of charge. The charge delivered during each phase is different, however, as are the trailing edge of the first phase and the leading edge of the second phase. It is not known which, if any, of these variables are responsible for the shape of the strength-duration curve.

Conclusions. Despite the many unknowns about the mechanism of the efficacy of biphasic waveforms, there is little doubt that certain biphasic waveforms are very effective for defibrillation. In this study we were able to defibrillate consistently with large contoured electrodes and biphasic waveforms at low energy, current and voltage. However, we must caution that, for whatever reason, certain biphasic waveforms are not very effective and are even less efficacious than monophasic waveforms.

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