Arrhythmias

The Effects of Biphasic Waveform Design on Post-Resuscitation Myocardial Function

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OBJECTIVES	This study examined the effects of biphasic truncated exponential waveform design on survival and post-resuscitation myocardial function after prolonged ventricular fibrillation (VF).
BACKGROUND	Biphasic waveforms are more effective than monophasic waveforms for successful defibrillation, but optimization of energy and current levels to minimize post-resuscitation myocardial dysfunction has been largely unexplored. We examined a low-capacitance waveform typical of low-energy application (low-energy biphasic truncated exponential [BTEL]; 100 μF, ≤200 J) and a high-capacitance waveform typical of high-energy application (high-energy biphasic truncated exponential [BTEH]; 200 μF, ≥200 J).
METHODS	Four groups of anesthetized 40- to 45-kg pigs were investigated. After 7 min of electrically induced VF, a 15-min resuscitation attempt was made using sequences of up to three defibrillation shocks followed by 1 min of cardiopulmonary resuscitation. Animals were randomized to BTEL at 150 J or 200 J or to BTEH at 200 J or 360 J.
RESULTS	Resuscitation was unsuccessful in three of the five animals treated with BTEH at 200 J. All other attempts were successful. Significant therapy effects were observed for survival (p = 0.035), left ventricular ejection fraction (p < 0.001), stroke volume (p < 0.001), fractional area change (p < 0.001), cardiac output (p = 0.044), and mean aortic pressure (p < 0.001). Hemodynamic outcomes were negatively associated with energy and average current but positively associated with peak current. Peak current was the only significant predictor of survival (p < 0.001).
CONCLUSIONS	Maximum survival and minimum myocardial dysfunction were observed with the low-capacitance 150-J waveform, which delivered higher peak current while minimizing energy and average current. (J Am Coll Cardiol 2004;43:1228–35) © 2004 by the American College of Cardiology Foundation

Both experimental (1–3) and in-hospital clinical data (4,5) have demonstrated that biphasic waveform electrical shocks are as effective for initial defibrillation as monophasic waveform shocks of significantly higher energy levels. In addition, in a study of out-of-hospital cardiac arrest, a higher rate of return of spontaneous circulation and better cerebral performance at the time of hospital discharge were achieved with impedance compensating 150-J biphasic shocks than with monophasic shocks (6).

We have previously compared fixed-energy 150-J biphasic waveform shocks with escalating energy monophasic damped sine waveform shocks of 200, 300, and 360 J in a porcine model after either 4, 7, or 10 min of untreated ventricular fibrillation (VF). Fixed low-energy biphasic waveform shocks proved to be as effective as escalating energy monophasic waveform shocks for the restoration of spontaneous circulation, but low-energy biphasic waveform

shocks had the additional advantage of minimizing post-resuscitation myocardial dysfunction (7,8).

It has been previously shown that a biphasic truncated exponential (BTE) waveform may be designed to minimize the defibrillation threshold in terms of either energy or peak current but that these two notions of optimization result in different waveform shapes (3). These waveform variants generally are achieved through the appropriate choice of the defibrillation capacitor (e.g., 100 μF for low-energy biphasic truncated exponential [BTEL] at 150 J vs. 200 μF for high-energy biphasic truncated exponential [BTEH] at 200 to 360 J). Low-energy biphasic truncated exponential waveforms are generally characterized by higher peak current but lower energy and average current than their BTEH counterparts. Although both waveform variants are commonly available in commercial products, the question remains as to which of these approaches might result in better outcome, as characterized by survival and post-resuscitation myocardial function.

The present study was designed to address these issues by examining resuscitation outcome after 7 min of untreated VF. Two biphasic truncated exponential waveforms representative of commercially available products were investigated. The first product used a smaller defibrillator capacitor to deliver lower energy and lower average current for a given

Abbreviations and Acronyms

BTEH = high-energy biphasic truncated exponential
BTEL = low-energy biphasic truncated exponential
CO = cardiac output
CPR = cardiopulmonary resuscitation
EF = ejection fraction
FAC = fractional area change
LV = left ventricle/ventricular
SV = stroke volume
VF = ventricular fibrillation

value of peak current (BTEL, 100 μF). The second product used a larger defibrillator capacitor to deliver higher energy and higher average current for a given value of peak current (BTEH, 200 μF) (Fig. 1). Each waveform was applied at two different doses, as shown in Figures 1 and 2. Outcome variables included the success of initial resuscitation, post-resuscitation myocardial function, and duration of survival. We hypothesized that biphasic waveform defibrillation with a BTEL waveform at 150 J would be as effective as the same waveform at 200 J for the return of spontaneous circulation after 7 min of untreated VF while it would simultaneously minimize post-resuscitation myocardial dysfunction. We also hypothesized that BTEL waveform shocks at 150 J would be as effective as BTEH shocks at 200 and 360 J for the return of spontaneous circulation after 7 min of untreated VF while it would simultaneously minimize post-resuscitation myocardial dysfunction.

METHODS

Protocol approval was obtained from the Institutional Animal Care and Use Committee. The research laboratories of the Institute of Critical Care Medicine are fully accredited by the Association for Assessment and Accreditation of Laboratory Animal Care International.
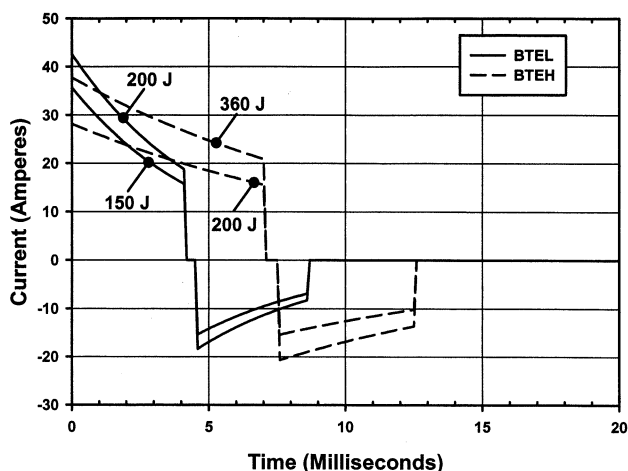
Figure 1. Biphasic waveforms used for the present study. Low-energy biphasic truncated exponential (BTEL) uses a 100-μF defibrillation capacitor, whereas high-energy biphasic truncated exponential (BTEH) uses 200-μF.
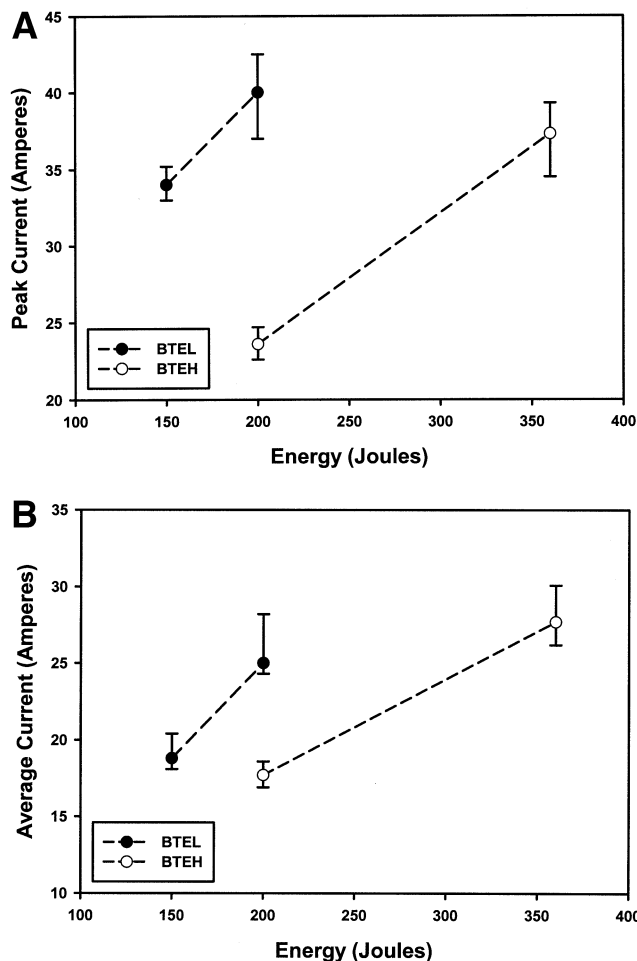
Figure 2. Energy versus electrical current relationships for the therapies used in the study (**circles** and **error bars** indicate median and interquartile range): **(A)** peak current versus energy; **(B)** average current versus energy. BTEH = high-energy biphasic truncated exponential; BTEL = low-energy biphasic truncated exponential.

Animal preparation. Male domestic pigs weighing between 40 and 45 kg were fasted overnight except for free access to water. Anesthesia was initiated by intramuscular injection of ketamine (20 mg/kg) and completed by ear vein injection of sodium pentobarbital (30 mg/kg). Additional doses of sodium pentobarbital (8 mg/kg) were injected to maintain anesthesia at intervals of 1 h. Animals were mechanically ventilated with a volume-controlled ventilator (Model MA-1, Puritan-Bennett, Carlsbad, California). End-tidal PCO_2 was monitored with an infrared analyzer (Model 01R-7101A, Nihon Kohden Corp., Tokyo, Japan). Respiratory frequency was adjusted to maintain $P_{ET}CO_2$ between 35 and 40 mm Hg.

For the measurement of left ventricular (LV) functions, we used a Doppler transesophageal echocardiographic transducer (Model 21363A, Hewlett-Packard Corp., Andover, Massachusetts). For the measurement of aortic pressure, a fluid-filled catheter was advanced from the left femoral artery into the thoracic aorta. For the measurements of right atrial, pulmonary arterial pressures, and blood

Table 1. Waveform Characteristics

	BTEL 150 J	BTEL 200 J	BTEH 200 J	BTEH 360 J
Defibrillation capacitor	100 μF	100 μF	200 μF	200 μF
Capacitor charge voltage	1,790 V	2,130 V	1,580 V	2,110 V
Phase 1 to phase 2 duration ratio	50/50	50/50	60/40	60/40
Phase 1 to phase 2 strength-duration ratio	2.3	2.3	2.3	2.3
Waveform duration (50 ohms)	8.4 ms	8.4 ms	12.5 ms	12.5 ms

BTEH = high-energy biphasic truncated exponential; BTEL = low-energy biphasic truncated exponential.

temperature, a thermodilation-tip catheter was advanced from the left femoral vein and flow directed into the pulmonary artery. For inducing VF, a pacing catheter (EP Technologies Inc., Mountain View, California) was advanced from the right cephalic vein into the right ventricle.

Waveforms. Two impedance compensating biphasic truncated exponential waveforms were investigated. The defibrillation capacitor, capacitor charge voltage (and therefore peak current), waveform duration (and therefore average current), and phase duration ratio were chosen to be similar to commercially available products. For both waveforms, the trailing-edge voltage of the first phase was equal to the leading-edge voltage of the second phase, with a 0.4-millisecond inter-phase delay. Impedance compensation was achieved by modulation of the waveform duration, while maintaining phase duration ratio, in order to deliver the selected energy—although the limited impedance range of this animal model (53 ± 7 ohms) resulted in little variation in waveform duration. Further details of these waveforms are itemized in Table 1.

Experimental procedures. Ten minutes before inducing cardiac arrest, the animals were randomized to a treatment group. Delivering an AC current to the right ventricle induced VF. Mechanical ventilation was discontinued after VF appeared. After 7 min of untreated VF, defibrillation was attempted by delivering up to three shocks of the randomized energy. Fixed energy shocks were used for all waveforms to better examine the effects of the waveform variables. Philips Medical Systems (Seattle, Washington)

provided the research defibrillators and test equipment for confirmation of delivered energy. The shocks were delivered between the positive right infraclavicular electrode and the negative cardiac apical electrode. If VF was not reversed after three shocks, precordial compression was started for 60 s (Thumper, Model 1000, Michigan Instruments, Grand Rapids, Michigan). Coincident with the start of precordial compression, the animal was mechanically ventilated with tidal volume of 15 ml/kg and FiO_2 of 1.0. Precordial compression was programmed to provide 100 compressions per min and synchronized to provide a compression/ventilation ratio of 5:1 with equal compression-relaxation intervals. The compression force was adjusted to decrease the anterior posterior diameter of the chest by 25% so as to maintain the coronary perfusion pressure above 10 mm Hg. After 1 min of precordial compression, another sequence of up to three shocks was delivered if needed. This sequence was repeated until the animal was either successfully resuscitated or pronounced dead after a total of 15 min of cardiopulmonary resuscitation (CPR).

If an organized cardiac rhythm with mean aortic pressure of more than 60 mm Hg persisted for an interval of 5 min or more, the animal was regarded as successfully resuscitated. Animals were then monitored for an additional 4 h. After the panel of 4-h post-resuscitation measurements had been completed, the animals were observed for an additional 68 h. At the end of the 72-h post-resuscitation observation interval, measurements of echo-Doppler myocardial functions were repeated. The animals were then euthanized by intravenous injection of 150 mg/kg pentobarbital, and an autopsy was performed for documentation of significant injuries.

Measurements. The primary dependent variables were return of spontaneous circulation; duration of post-resuscitation survival; and post-resuscitation myocardial function characterized by mean aortic pressure, cardiac output (CO), LV ejection fraction (EF), stroke volume (SV), and fractional area change (FAC).

Myocardial function was measured by a transesophageal echo-Doppler technique developed by us for this porcine model. Cardiac output was calculated as the product of transmitral flow time velocity integral, mitral valve diameter, and heart rate. Left ventricular function was estimated

Table 2. Baseline Hemodynamic Characteristics for Study Groups

	Group				p Value
	A	B	C	D	
Body mass (kg)	41 [40–44]	40 [40–41]	41 [40–43]	44 [43–46]	0.28
Mean aortic pressure (mm Hg)	115 [108–133]	125 [111–133]	119 [104–133]	123 [116–127]	1.00
Ejection fraction (%)	60 [56–63]	60 [59–61]	58 [54–62]	59 [57–59]	0.86
Stroke volume (ml)	34 [29–35]	40 [35–42]	35 [32–37]	37 [31–38]	0.30
Fractional area change (%)	43 [40–44]	43 [37–44]	40 [32–41]	39 [37–45]	0.99
Cardiac output (l/min)	5.9 [5.9–6.9]	5.3 [3.6–7.2]	5.9 [5.7–6.3]	5.5 [5.2–6.2]	0.66

Continuous variables presented as median and [interquartile range].
Group A = BTEL 150 J; Group B = BTEL 200 J; Group C = BTEH 200 J; Group D = BTEH 360 J. Abbreviations as in Table 1.

Table 3. Therapy Characteristics and Outcome Variables

	Group				p Values						
	A	B	C	D	Overall	A vs. B	A vs. C	A vs. D	B vs. C	B vs. D	C vs. D
Waveform	BTEL	BTEL	BTEH	BTEH							
Energy, J	150	200	200	360							
Peak current, amperes	34.0 [33.0–35.2]	40.0 [37.0–42.5]	23.6 [22.6–24.7]	37.3 [34.5–39.3]	< 0.001*	< 0.001†	< 0.001†	< 0.001†	< 0.001†	0.002†	< 0.001†
Average current, amperes	18.8 [18.1–20.4]	25.0 [24.3–28.2]	17.1 [16.9–18.6]	26.7 [26.2–30.1]	< 0.001*	< 0.008†	0.063	0.036	0.016	0.786	0.057
Return of spontaneous circulation	5/5 (100%)	5/5 (100%)	2/5 (40%)	5/5 (100%)	0.035*	1.000	0.083	1.000	0.083	1.000	0.083
72-h survival	5/5 (100%)	5/5 (100%)	2/5 (40%)	5/5 (100%)	0.035*	1.000	0.083	1.000	0.083	1.000	0.083
Shocks to resuscitate	1 [1–2]	3 [2–5]	5 [4–10]	4 [3–9]	0.074						
Total delivered energy, J	155 [146–304]	563 [405–1,000]	994 [797–2,016]	1440 [1,080–3,282]	0.008*	0.057	0.016	0.016	0.310	0.151	0.548
Duration of CPR, s	106 [94–133]	83 [80–90]	909 [260–972]	218 [90–425]	0.025*	0.548	0.016	0.500	0.032	0.143	0.143
Mean aortic pressure (% of baseline)	94% [79%–101%]	74% [73%–80%]	98%‡ [88%–109%]	75% [67%–75%]	< 0.001*	< 0.001†	0.164	0.004†	< 0.001†	0.757	0.002†
Ejection fraction (% of baseline)	95% [95%–97%]	75% [67%–75%]	62%‡ [50%–74%]	53% [42%–53%]	< 0.001*	< 0.001†	< 0.001†	< 0.001†	0.819	0.033	0.090
Stroke volume (% of baseline)	110% [104%–118%]	69% [69%–74%]	64%‡ [41%–86%]	48% [46%–69%]	< 0.001*	< 0.001†	< 0.001†	< 0.001†	0.935	0.577	0.672
Fractional area change (% of baseline)	104% [87%–112%]	71% [66%–72%]	65%‡ [48%–82%]	58% [56%–68%]	< 0.001*	0.011	0.011	< 0.001†	0.059	0.029	0.762
Cardiac output (% of baseline)	113% [81%–121%]	97% [95%–97%]	118%‡ [115%–121%]	67% [54%–107%]	0.044*	0.052	0.531	0.383	0.005†	0.386	0.138

Continuous variables presented as median and [interquartile range]. Hemodynamic descriptive statistics are reported at 30 minutes post-resuscitation. *Statistically significant overall effect with $p < 0.05$. †Statistically significant between-group effect with $p < 0.008$. ‡Hemodynamic descriptive statistics for therapy C exclude three of five animals that did not survive and are therefore biased high. **Boldface** indicates values that are statistically significant.

BTEH = high-energy biphasic truncated exponential; BTEL = low-energy biphasic truncated exponential; CPR = cardiopulmonary resuscitation. Group descriptions as in Table 2.

by measurements of LV end systolic and end diastolic volume, based on FAC and resulting SV and EF.

Other measurements were recorded to test for population bias and to assist in consistent performance of the resuscitation protocol. Measurements of aortic and right atrial pressure allowed for estimation of coronary perfusion pressure. End-tidal CO_2 was measured continuously so as to provide indication of appropriate ventilation and as a quantitative indicator of relative pulmonary blood flow during precordial compression.

Pulmonary artery and pulmonary occlusive pressures, end-tidal PCO_2, and the lead II electrocardiogram, together with measurements of LV function, were continuously measured and recorded as previously described (7–9). A quantitative neurological alertness score (9) was used for evaluating neurological recovery at 12-h intervals for a total of 72 h. Aortic and mixed venous blood gases, hemoglobin, and oxyhemoglobin were measured with a blood gas analyzer and Co-Oximeter (Models 1306 and 482, Instrumentation Laboratory, Lexington, Massachusetts) adapted for porcine blood. Arterial blood lactate was measured (Model 23L, Yellow Springs Instruments, Yellow Springs, Ohio). These measurements were obtained 30 min before cardiac arrest and at hourly intervals after resuscitation for a total of 4 h.

Analysis. All outcome variables were tested for significance of overall therapy effect (i.e., waveform/dose) using exact non-parametric methods. The Fisher-Freeman-Halton test was used for tables of counts (e.g., success vs. failure), whereas the Kruskal-Wallis analysis of variance was used for continuous variables. If a significant overall therapy effect was identified ($p < 0.05$), additional between-group comparisons were performed using the Fisher exact test for the counts and exact Wilcoxon-Mann-Whitney tests for the continuous data, with time stratification used for repeated measures. Adjustment for multiple comparisons in the between-group tests was made via downward adjustment of the alpha level (Bonferroni method); thus, results were considered significant only if $p < 0.008$. Exact nonparametric statistics were calculated using StatXact Version 5.0.3 (Cytel Software, Cambridge, Massachusetts).

Statistically significant overall waveform effects were further examined to identify relationships between outcome variables and waveform design parameters: energy, peak current, and average current. Statistically significant relationships were identified via multiple regression analysis using Statistica version 6.1, Statsoft (Tulsa, Oklahoma) or logistical regression analysis using LogXact Version 4 (Cytel Software).

RESULTS

Twenty-two experiments were completed. Two experiments were excluded because one animal had echocardiographically and pathologically documented myocardial hypertrophy and another animal had a marked elevated airway
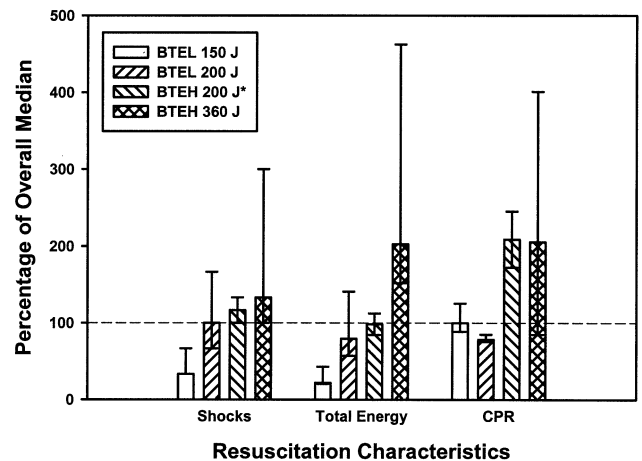

Figure 3. Resuscitation characteristics (median and interquartile range) versus therapy as a percentage of the overall median for each characteristic. Overall median values: shocks = 3.5, total energy = 770 J, cardiopulmonary resuscitation (CPR) = 158 s. *BTEH 200 J data exclude three of five animals that failed resuscitation and are therefore biased low. BTEH = high-energy biphasic truncated exponential; BTEL = low-energy biphasic truncated exponential.

pressure during both baseline and CPR. There were no differences in weight, transthoracic impedance, or baseline measurements of hemoglobin and oxyhemoglobin, blood gas, arterial lactate, end-tidal CO_2, pulmonary arterial pressure, right atrial pressure, heart rate, the calculated coronary perfusion pressure, or neurological alertness score among the four groups. Similarly, there were no differences in baseline hemodynamic characteristics (Table 2).

Table 3 contains a summary of all outcome observations. A significant overall effect was detected for survival as a function of waveform ($p = 0.035$), with all animals being successfully resuscitated after delivery of BTEL 150-J or 200-J shocks as well as BTEH 360-J shocks. However, only two of five animals were successfully resuscitated after BTEH 200-J shocks. Between-group survival comparisons did not reach significance. All resuscitated animals survived for more than 72 h, with no differences in neurological alertness score among the four groups.

Duration of CPR and total energy required for resuscitation both exhibited significant therapy effects ($p = 0.025$ and 0.008, respectively). Animals treated with BTEL shocks required fewer shocks, less CPR, and required less total energy to resuscitate than animals treated with BTEH (Fig. 3, Table 3).

Myocardial function, as evidenced by hemodynamic performance, was reduced in all animals after successful resuscitation. Significant waveform/dose effects were exhibited for all measures ($p < 0.001$ for all but CO, for which $p = 0.044$). Descriptive statistics for hemodynamic performance 30 min post resuscitation are itemized in Table 3, as well as p values for between-group comparisons (time stratified over the 4-h observation period). All measures are normalized to baseline and are thus expressed as percentages of pre-arrest values. Hemodynamic performance both 30 min
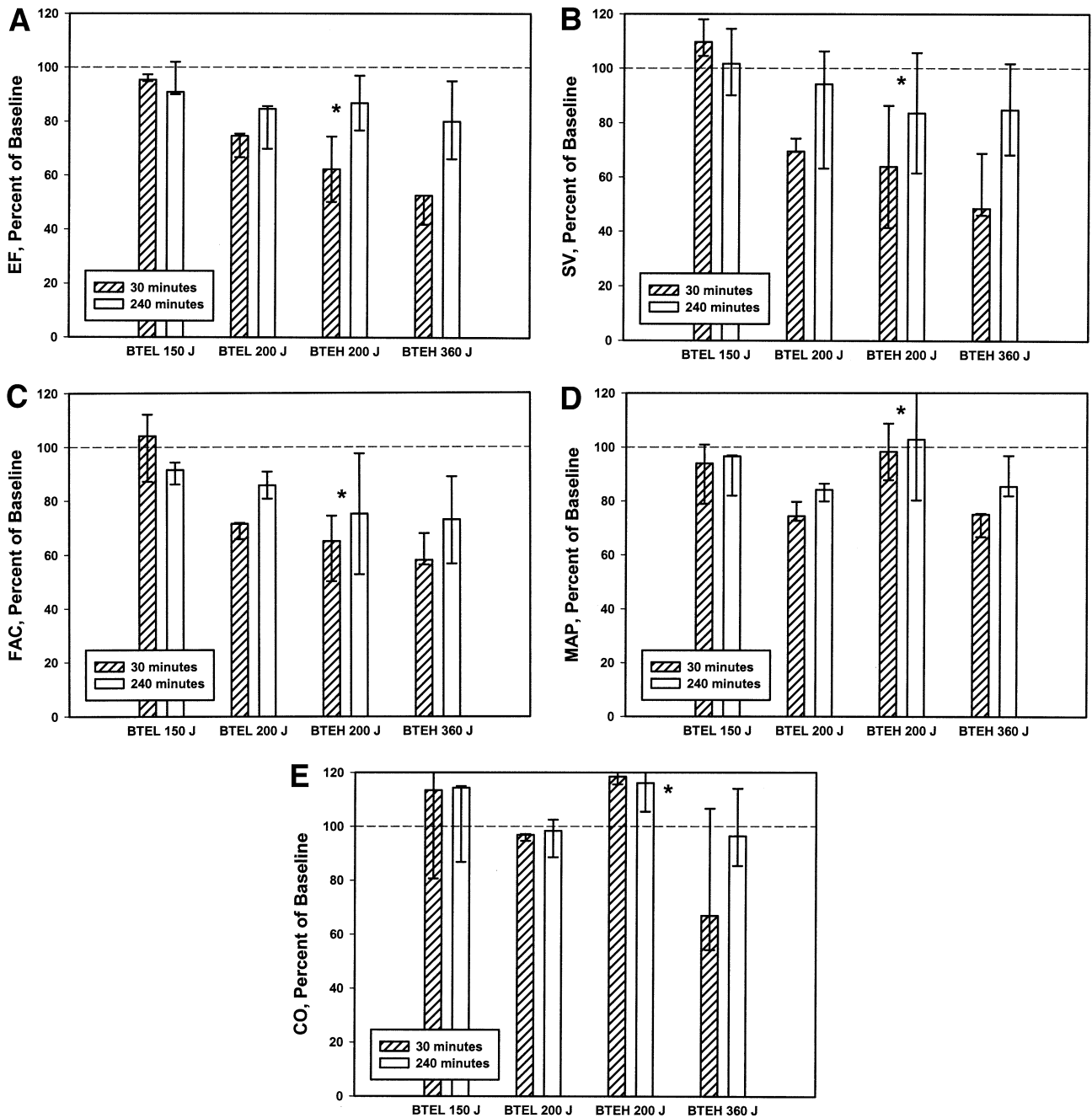
Figure 4. Hemodynamic outcome variables versus therapy at 30 and 240 min post resuscitation shown as median and interquartile range: **(A)** left ventricular ejection fraction (EF); **(B)** stroke volume (SV); **(C)** fractional area change (FAC); **(D)** mean aortic pressure (MAP); and **(E)** cardiac output (CO). *BTEH 200 J data exclude three of five animals that failed resuscitation and are therefore biased high. BTEH = high-energy biphasic truncated exponential; BTEL = low-energy biphasic truncated exponential.

post resuscitation and at the end of the 4-h observation period are plotted for the various waveform/dose combinations in Figure 4. Although post-resuscitation hemodynamics continuously improved over time, substantial deficits were still apparent in animals treated with higher energy shocks at the conclusion of the 4-h observation period.

Multiple regression revealed that resuscitation interventions (duration of CPR, total energy) and all post-

resuscitation hemodynamic metrics (EF, SV, FAC, CO, mean aortic pressure) were consistently degraded by increased energy and increased average current, with the sole exception of mean aortic pressure, for which energy was a neutral contributor (Fig. 5). Conversely, higher peak currents improved the same metrics. Significant regressions were obtained for CPR (p = 0.02, R^2 = 0.54), EF (p < 0.01, R^2 = 0.75), and SV (p = 0.03, R^2 = 0.57). Finally,
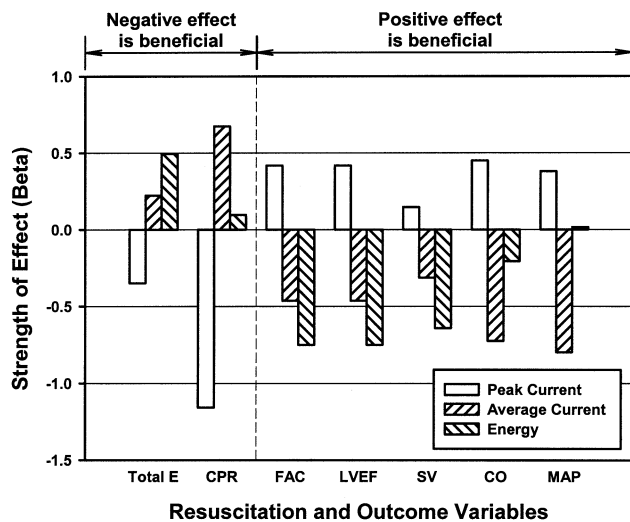
Figure 5. Effect of energy, peak current, and average current on outcome variables expressed as the normalized multiple regression coefficients (beta). CO = cardiac output; CPR = duration of cardiopulmonary resuscitation; E = energy; FAC = fractional area change; LVEF = left ventricular ejection fraction; MAP = mean aortic pressure; SV = stroke volume.

logistic regression indicated that peak current was the only predictor of increased survival ($p < 0.001$, likelihood ratio = 17.6).

DISCUSSION

This study confirmed the hypothesis that biphasic waveform defibrillation with a BTEL waveform at 150 J is as effective as the same waveform at 200 J for successful return of spontaneous circulation while it simultaneously minimizes post-resuscitation myocardial dysfunction. We also confirmed that BTEL waveform shocks at 150 J are as effective as BTEH shocks at 200 and 360 J for successful return of spontaneous circulation while they simultaneously minimize post-resuscitation myocardial dysfunction.

We further demonstrated that these effects are attributable to specific characteristics of waveform design. In particular, higher peak current is positively associated with improved survival, whereas higher energy and higher average current are associated with increased post-resuscitation myocardial dysfunction. These observations argue for a damage mechanism related to cumulative, rather than instantaneous, electrical exposure.

Post-resuscitation myocardial dysfunction has been associated with early death after initial successful resuscitation (10,11). In previous studies, the severity of post-resuscitation myocardial dysfunction was closely related to the duration of cardiac arrest, treatment with beta-adrenergic agents, and the severity of hypercarbic myocardial acidosis (12,13). In recent studies, we implicated the total electrical energy delivered during defibrillation attempts as an important correlate with the severity of post-resuscitation myocardial dysfunction and survival in both rat and pig models (7,8,14). This prompted us to call attention to the benefits of minimizing the electrical energy

delivered during defibrillation attempts, so as to preserve maximal post-resuscitation myocardial function and improve survival.

The results of this study contradict the notion that peak current is the primary correlate of myocardial injury/dysfunction. Studies often cited to support this notion, however, have either failed to provide supporting data (15) or have not controlled peak current independent of energy (16), leaving the two variables confounded and thus preventing separation of cause and effect. One study that carefully controlled energy independent of peak current was reported by Stoeckle et al. (17) in 1968. Their data, from experiments with rectangular monophasic defibrillation pulses, indicate that pulses with peak current below a critical level (40 amperes in their canine model) exhibit a rate of post-shock complication (arrhythmia) independent of current but increasing with energy. These waveforms also exhibited low defibrillation thresholds and high efficacy over a broad range of energy. In contrast, pulses with peak current in excess of the critical limit exhibited high rates of complication at all energies, accompanied by high defibrillation thresholds, and high efficacy for only a narrow range of energy. Thus, there appear to be two distinct damage mechanisms: the first is related to instantaneous exposure to peak currents above a critical level and largely independent of energy, and the second is related to exposure over time, as measured by energy and largely independent of peak current. It is interesting to speculate that modern biphasic defibrillation waveforms, by significantly reducing peak current requirements with respect to their monophasic damped sine predecessors, have avoided the former behavior, thereby achieving high efficacy over a broad range of delivered energy and leaving only energy-dependent dysfunction effects—characteristics consistent with our results.

Further recourse to the literature on this subject is made difficult by the fact that study authors have often used the terms "energy," "power," "voltage," and "current" interchangeably or have not measured and reported all variables (18). The significance of this shortcoming becomes apparent when it is realized that a 200-J waveform can deliver higher peak current than a 360-J waveform (e.g., BTEL vs. BTEH) (Fig. 1).

Study limitations. This study did not examine the effects of energy escalation, which may have benefited the high capacitance waveform (BTEH) by increasing the delivered peak current after failed shocks, thereby improving overall survival beyond that obtained with BTEH 200-J shocks alone. The purpose of this study, however, was to examine the effects of energy, peak current, and average current on post-resuscitation myocardial function; thus, a fixed defibrillation therapy protocol was used.

Our study protocol modeled resuscitation that occurred after 7 min of untreated VF. We felt that this duration of ischemic insult was representative of typical emergency medical service response times. If the duration of untreated VF is significantly reduced (e.g., through improved response

time), the magnitude of post-resuscitation myocardial dysfunction is likely to be reduced. Conversely, our findings are likely to be magnified by an arrest duration in excess of 7 min.

We examined only biphasic truncated exponential waveforms generated with 100-μF and 200-μF capacitors, and peak current was limited to approximately 40 amperes. Extrapolation of our findings to extremes beyond those tested should be performed with caution. Applicability of our findings to clinical practice remains to be demonstrated.

Conclusions. In conclusion, we have demonstrated that for biphasic truncated exponential waveforms representative of commercial implementations, the primary correlate to survival is peak electrical current. Conversely, waveform energy and average current are the primary correlates of post-resuscitation myocardial dysfunction as evidenced by hemodynamic compromise persisting for many hours.

With respect to patient outcome, these findings suggest that peak current is a more appropriate measure of defibrillation dose than either energy or average current and that toxicity may be minimized by simultaneously reducing both of the latter. Furthermore, these conclusions suggest that post-resuscitation myocardial dysfunction is related to a cumulative, as opposed to an instantaneous, electrical exposure mechanism.

In this study, survival was maximized and myocardial dysfunction minimized using a biphasic truncated exponential waveform that simultaneously delivered higher peak current while minimizing energy and average current. Such a waveform may be created through the use of smaller defibrillation capacitors to achieve high waveform tilt.

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