Journal of the American College of Cardiology
© 2001 by the American College of Cardiology
Published by Elsevier Science Inc.
Vol. 37, No. 6, 2001
ISSN 0735-1097/01/$20.00

LETTERS TO THE EDITOR

The Effects of Biphasic and Conventional Monophasic Defibrillation on Postresuscitation Myocardial Function

In a recent study, Niemann et al. (1) compared the effects of 150-J biphasic truncated exponential waveform shocks and conventional 200-, 300- and 360-J monophasic truncated exponential waveform shocks on the success of defibrillation and on postresuscitation myocardial function in pigs. After 5 min of untreated ventricular fibrillation, there was no difference in the number of animals successfully defibrillated. Postresuscitation left ventricular (LV) function was evaluated with measurements of peak LV pressure, the first derivative of LV pressure (LV dP/dt) and cardiac output measured by the thermodilution method. Again, the authors found no differences between the two groups with respect to measurements that the authors regarded as quantitative indicators of postresuscitation myocardial function.

Their observations contrast with earlier reports (2–4) and a recent report from our own laboratory (5), which had demonstrated that equally effective low-energy biphasic waveform shocks produced less postresuscitation myocardial injury. We believe that the differences are best explained by the experimental procedures employed by the authors.

Baseline mean aortic pressure reported by Niemann et al. (1) was only 70%, and dP/dt was approximately 50% of those measurements observed by our group in a comparable porcine model of more mature pigs (5,6). The hemodynamic differences are summarized in Table 1. Most important, experiments were terminated 60 min after successful resuscitation. Our group has observed that more precise measurements of postresuscitation myocardial function, including stroke volume, fractional area change and pressure-volume relationships are progressively impaired over 240 min following resuscitation (5,6). Finally, although cardiac output was reported by the authors, it was not normalized against heart rate. Earlier observations pinpointed that decreases in stroke volumes are compensated for by disproportionate increases in heart rate (5,6). Finally, the isovolumetric phase index of maximal rate of pressure rise (dP/dt max) is preload dependent (7–10). Without accounting for preload, dP/dt measurements are suspect.

We applaud the efforts of the authors for investigating the effects of new defibrillation energies and waveforms. However, the limitations of this study preclude their challenge to the earlier findings that lower-energy biphasic waveforms minimize postresuscitation myocardial function that evolves over the 4-h interval after successful resuscitation.

Table 1. Baseline Hemodynamic Values in Pigs Prior to Cardiac Arrest

Group	No.	Animal BW (kg)	MAP (mm Hg)	CO (L/min)	dP/dt (mm Hg/s^{-1})
Tang et al. (5)	20	42.0	132.0	5.80	—
Gazmuri et al. (6)	13	38.0	128.0	4.90	2,240
Niemann et al. (1)	38	29.2	79.5	2.85	1,180

BW = body weight; CO = cardiac output; dP/dt = rate of pressure rise; MAP = mean aortic pressure.

Shije Sun, MD
Institute of Critical Care Medicine
1695 North Sunrise Way
Building #3
Palm Springs, California 92262-5309
E-mail: weilm@aol.com

Kada Klouche, MD
Wanchun Tang, MD
Max Harry Weil, MD, PhD, FACC

PII S0735-1097(01)01217-7

REFERENCES

1. Niemann JT, Burian D, Garner D, Lewis RJ. Monophasic versus biphasic transthoracic countershock after prolonged ventricular fibrillation in a swine model. J Am Coll Cardiol 2000;36:932–8.
2. Bardy GH, Marchlinski FE, Sharma AD, et al. Multicenter comparison of truncated biphasic shocks and standard damped sine wave monophasic shocks for transthoracic ventricular defibrillation. Circulation 1996;94:2507–14.
3. Mittal S, Ayati S, Stein KM, et al. Comparison of a novel rectilinear biphasic waveform with a damped sine wave monophasic waveform for transthoracic ventricular defibrillation. J Am Coll Cardiol 1999;34:1595–601.
4. Scheatzle MD, Menegazzi JJ, Allen TL, Durham SB. Evaluation of biphasic transthoracic defibrillation in an animal model of prolonged ventricular fibrillation. Acad Emerg Med 1999;6:880–6.
5. Tang W, Weil MH, Sun SJ, et al. The effects of biphasic and conventional monophasic defibrillation on postresuscitation myocardial function. J Am Coll Cardiol 1999;34:815–22.
6. Gazmuri RJ, Weil MH, Bisera J, Tang W, Fukui M, McKee D. Myocardial dysfunction after successful resuscitation from cardiac arrest. Crit Care Med 1996;24:992–1000.
7. Kass DA, Maughan WL, Guo ZM, Kono A, Sunagawa K, Sagawa K. Comparative influence of load versus inotropic states on indexes of ventricular contractility: experimental and theoretical analysis based on pressure-volume relationships. Circulation 1987;76:1422–36.
8. Perlini S, Meyer TE, Foëx P. Effects of preload, afterload and inotropy on dynamics of ischemic segmental wall motion. J Am Coll Cardiol 1997;29:846–55.
9. Yamada H, Oki T, Tabata T, Iuchi A, Ito S. Assessment of left ventricular systolic wall motion velocity with pulsed tissue Doppler imaging: comparison with peak dP/dt of the left ventricular pressure curve. J Am Soc Echocardiogr 1998;11:442–9.
10. Greaves SC. Assessment of left ventricular systolic function in research and in clinical practice. Heart 2000;83:493–4.

REPLY

We appreciate the insightful review of our article (1) by Sun et al. and wish to offer the following comments regarding their concerns.

The baseline mean aortic pressure (MAP) and cardiac output that we reported are typical for swine anesthetized with isoflurane and nitrous oxide and approximate those recorded in awake animals (2). Although not reported in the article, the observed control heart rate in our animals, approximately 100 beats/min, also approximates that observed in conscious swine. We believe that these values are reflective of stable anesthesia with inhaled agents that are preferred by many for cardiovascular research. In our opinion, control values should ideally reflect those observed in

JACC Vol. 37, No. 6, 2001
May 2001:1753–5

conscious animals. The MAP values as well as left ventricular dP/dt observed by Sun et al. using intermittent intravenous pentobarbital anesthesia are, in fact, excessive when compared to values reported in the literature for swine anesthetized with pentobarbital (3). This suggests that the "control" values reported by Sun et al. are supranormal, possibly reflecting enhanced sympathetic tone of uncertain etiology. We would therefore agree with Sun et al. that these differences of concern to them are due to differences in experimental procedures between our laboratories. However, they are not reflective of inadequate technical skills in our laboratory used in the acquisition and interpretation of hemodynamic data.

Our data do, in fact, support the observations of Sun et al. In a prior publication (4), they reported no differences between defibrillation waveform groups with respect to first shock success or clinically important indexes of postresuscitation cardiac function after a 4-min period of ventricular fibrillation (VF). Observed differences appear to resolve rather than evolve during extended observation. Sun et al. have previously acknowledged the effect of prolonged pentobarbital anesthesia on cardiac mechanics (5). We likewise observed no differences during observation after a 5-min VF period. It would appear that the "best" defibrillation waveform for the treatment of VF of 4- to 5-min duration would be the one that is first available.

We have not systematically investigated the differences between defibrillation waveforms in the management of VF of >5-min duration. It is very likely that if we administered monophasic waveform energy doses similar to those used by Sun et al. in their 7-min swine model (4), an average dose approximating 57 J/kg, we would observe results similar to what they have reported. In our hospital's recent six-year clinical experience with out-of-hospital sudden cardiac death, the largest energy dose used in any patient has been approximately 33 J/kg delivered with seven countershocks. Since the energy doses reported by Sun et al. far exceed what is encountered clinically, our laboratory has no intention of pursuing a similar experimental design due to its lack of clinical relevance.

James T. Niemann, MD
Professor of Medicine
Department of Emergency Medicine, Box 21
Harbor-UCLA Medical Center
1000 West Carson Street
Torrance, California 90509
E-mail: jniemann@emedharbor.edu

PII S0735-1097(01)01220-7

REFERENCES

1. Niemann JT, Burian D, Garner D, Lewis RJ. Monophasic versus biphasic transthoracic countershock after prolonged ventricular fibrillation in a swine model. J Am Coll Cardiol 2000;36:932–8.
2. Lundeen G, Manohar M, Parks C. Systemic distribution of blood flow in swine while awake and during 1.0 and 1.5 MAC isoflurane anesthesia with or without 50% nitrous oxide. Anesth Analg 1983;62:499–512.
3. Stanton HC. Development of cardiovascular control and function. In: Swine in Cardiovascular Research. 1st ed. Boca Raton, FL, CRC Press, 1986;2:49–71.
4. Tang W, Weil MH, Sun S, et al. The effects of biphasic and conventional monophasic defibrillation on postresuscitation myocardial function. J Am Coll Cardiol 1999;34:815–22.
5. Gazmuri RJ, Weil MH, Bisera J, Tang W, Fukui M, McKee D. Myocardial dysfunction after successful resuscitation from cardiac arrest. Crit Care Med 1996;24:992–1000.

Effectiveness of BiPAP for Congestive Heart Failure

We were surprised to read the poor results described in the article by Sharon et al. (1) comparing bilevel positive airway pressure (BiPAP) ventilation with intravenous isosorbide-dinitrate in patients with severe pulmonary edema. These findings are in marked contrast to our own research and experience with this modality (2–4). We routinely use BiPAP ventilatory support in those patients with severe pulmonary edema with acute respiratory failure and imminent need of endotracheal intubation (ETI). Our success rate at avoiding ETI is generally >90% in patients more severely ill than those described in the study by Sharon et al. Our patients receive sublingual nitroglycerin (0.25 mg) along with sublingual captopril (25 mg) to supplement their respiratory support. Although intravenous nitrates may be ideal, we find use of the sublingual route can frequently reverse a patient's respiratory distress before intravenous access is even established.

The fact that two dramatically different outcomes are described for the same intervention may be explained by variations in the overall treatment of the two populations. Our research has shown that an independent predictor of BiPAP failure and subsequent ETI is the use of morphine sulfate. Even moderate amounts such as those used in the study of Sharon et al. seem to be enough to interfere with a patient's abilities to successfully use the BiPAP system.

In treating acute pulmonary edema, high expiratory positive airway pressures (EPAPs) are required, and we routinely begin our BiPAP treatments with EPAPs of 8 to 10 cm H_2O. Patients begun on regimens of any lower pressures are titrated up to a level of ≥10 cm within 1 min of placement of the nasal mask. In the study of Sharon et al., patients were begun with EPAPs of 3 cm H_2O and increased by 1 cm every 3 to 4 min to a maximum of 5 cm H_2O. Given these parameters, we are surprised that the authors experienced any success at all. These pressures are far too low and titration is far too slow for patients with acute respiratory distress. When applied at the higher pressures, BiPAP-treated patients demonstrate marked improvements within a few breaths and are clinically out of danger for ETI within 2 to 3 min.

The presence of positive creatine phosphokinase (CK) markers in BiPAP-treated patients is an artifact of the rapid drop in left ventricular wall pressures that occurs when the BiPAP is applied. There is a washout effect that produces a narrow spike in CK that exceeds normal thresholds for acute myocardial infarctions, although the total amount of CK is the same as that which is slowly washed out over an extended period of time with conventional therapy.

In summary, we believe that the poor outcomes described in the study of Sharon et al. reflect more problems with the manner in which the BiPAP was utilized than a failure of the therapy itself.

Alfred Sacchetti, MD, FACEP
Department of Emergency Medicine
1600 Haddon Avenue
Our Lady of Lourdes Medical Center
Camden, New Jersey 08103
E-mail: a.sacchetti@worldnet.att.net

Russell H. Harris, MD, FACEP

PII S0735-1097(01)01219-0