A Comparison of Biphasic and Monophasic Waveform Defibrillation After Prolonged Ventricular Fibrillation*

Wanchun Tang, MD, FCCP; Max Harry Weil, MD, PhD, Master FCCP; Shijie Sun, MD; Heitor P. Povoas, MD; Kada Klouche, MD; Takashi Kamohara, MD; and Joe Bisera, MSEE

Study objective: To compare the effects of biphasic defibrillation waveforms and conventional monophasic defibrillation waveforms on the success of initial defibrillation, postresuscitation myocardial function, and duration of survival after prolonged duration of untreated ventricular fibrillation (VF), including the effects of epinephrine.

Design: Prospective, randomized, animal study.

Setting: Animal laboratory and university-affiliated research and educational institute.

Participants: Domestic pigs.

Interventions: VF was induced in 20 anesthetized domestic pigs receiving mechanical ventilation. After 10 min of untreated VF, the animals were randomized. Defibrillation was attempted with up to three 150-J biphasic waveform shocks or a conventional sequence of 200-J, 300-J, and 360-J monophasic waveform shocks. When reversal of VF was unsuccessful, precordial compression was performed for 1 min, with or without administration of epinephrine. The protocol was repeated until spontaneous circulation was restored or for a maximum of 15 min.

Measurements and results: No significant differences in the success of initial resuscitation or in the duration of survival were observed. However, significantly less impairment of myocardial function followed biphasic shocks. Administration of epinephrine reduced the total electrical energy required for successful resuscitation with both biphasic and monophasic waveform shocks.

Conclusions: Lower-energy biphasic waveform shocks were as effective as conventional higher-energy monophasic waveform shocks for restoration of spontaneous circulation after 10 min of untreated VF. Significantly better postresuscitation myocardial function was observed after biphasic waveform defibrillation. Administration of epinephrine after prolonged cardiac arrest decreased the total energy required for successful resuscitation.

(CHEST 2001; 120:948–954)

Key words: adrenergic; contractile function; defibrillation; heart failure; ventricular function

Abbreviations: CPP = coronary perfusion pressure; CPR = cardiopulmonary resuscitation; EF = ejection fraction; FAC = fractional area change; LVEDV = left ventricular end-diastolic volume; PETCO₂ = end-tidal PCO₂; RAP = right atrial pressure; SV = stroke volume; VF = ventricular fibrillation

In settings of cardiac arrest and resuscitation, monophasic waveforms, including damped sinusoidal waveforms or truncated exponential waveforms, remain the predominant waveforms utilized for trans-thoracic defibrillation. The American Heart Association guidelines currently advise an initial 200-J shock. If reversal of ventricular fibrillation (VF) is unsuccessful, the energy levels of the shocks are increased to either 200 J or 300 J, and subsequently to 360 J.¹

Both clinical and experimental studies²–⁶ have demonstrated substantial impairment of ventricular function after resuscitation from cardiac arrest. Indeed, postresuscitation myocardial dysfunction has been implicated²³ as a potentially important mechanism, accounting for fatal outcomes after successful resuscitation in 70% of victims within the first 72 h. Studies⁷ from our institute on a murine model implicated the total electrical energy delivered during defibrillation as

*From the Institute of Critical Care Medicine, Palm Springs, CA; and the Keck School of Medicine of the University of Southern California, Los Angeles, CA.

This work was performed at the Institute of Critical Care Medicine, Palm Springs, CA.

This work was supported in part by grant HL54322 from the National Heart, Lung, and Blood Institute, Bethesda, MD; an Established Investigator Research Grant from the Society of Critical Care Medicine, Anaheim, CA; and by a grant-in-aid from Heartstream Operation, Hewlett-Packard Company, Seattle, WA.

Manuscript received September 18, 2000; revision accepted March 14, 2001.

Correspondence to: Max Harry Weil, MD, PhD, Master FCCP, The Institute of Critical Care Medicine, 1695 North Sunrise Way, Building 3, Palm Springs, CA 92262-5309; e-mail: weilm@911research.org
an important correlate with the severity of postresuscitation myocardial dysfunction and postresuscitation survival. This prompted us to investigate the option of utilizing lower-electrical-energy biphasic waveform defibrillation.

We initially compared the effects of low-energy biphasic waveform defibrillation and conventional monophasic waveform defibrillation after a short (4 min) or an intermediate (7 min) interval of untreated VF. Biphasic waveform defibrillation with a fixed energy of 150 J proved to be as effective as conventional monophasic damped sine waveform defibrillation for restoration of spontaneous circulation with significantly lower delivered energy. This was associated with significantly less severe postresuscitation myocardial dysfunction.

In the present study, the effects of low-energy biphasic waveform defibrillation and conventional monophasic waveform defibrillation on the success of initial defibrillation, postresuscitation myocardial function, and duration of survival were compared after a more prolonged duration of untreated cardiac arrest. Since epinephrine is the vasopressor of first choice during cardiopulmonary resuscitation (CPR) and previous reports have demonstrated that epinephrine increases defibrillation thresholds, the effects of the two defibrillation waveforms were therefore also compared following administration of epinephrine. We hypothesized that biphasic waveform defibrillation with a fixed energy of 150 J may be at least as effective as conventional monophasic damped sine waveform defibrillation with increasing energies ranging from 200 to 360 J for restoration of spontaneous circulation after 10 min of untreated VF, with and without administration of epinephrine. We further anticipated significantly reduced severity of postresuscitation myocardial dysfunction with the 150-J biphasic waveform defibrillation.

For the measurement of left ventricular function, a 5-MHz single plane with a 5-MHz continuous-wave Doppler transesophageal echocardiographic transducer with four-way flexure (model 21363A; Hewlett-Packard, Medical Products Group; Andover, MA) was advanced from the incisor teeth into the esophagus for a distance of approximately 35 cm. 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 pressure (RAP), pulmonary arterial pressures, and blood temperature, a 7F, pentalumen, thermodilution-tip catheter was advanced from the left femoral vein and flow-directed into the pulmonary artery. For inducing VF, a 5F pacing catheter (EP Technologies; Mountain View, CA) was advanced from the right cephalic vein into the right ventricle until an ECG current of injury was recorded.

**Experimental Procedures**

Five minutes prior to inducing cardiac arrest, the animals were randomized by the sealed envelope method to either biphasic defibrillation or monophasic defibrillation, with or without administration of epinephrine. VF was induced by progressively increasing alternating-current to the endocardium of the right ventricle from 1 to 2 mA. Mechanical ventilation was discontinued after onset of VF. After 10 min of untreated VF, defibrillation was attempted with either a defibrillator designed to manually deliver up to three biphasic waveform shocks with a nominal energy level of 150 J (Heartstream; Seattle, WA; Fig 1) or by a conventional monophasic waveform defibrillator that provided energy levels of up to 360 J (Codemaster XL Defibrillator; Hewlett Packard; Andover, MA; Fig 1). The shocks were delivered between a (positive) right infracavicular electrode and a (negative) cardiac apical electrode. If VF was not reversed after three shocks, or a pulseless rhythm was encountered after shock(s), precordial compression was begun, utilizing a pneumatic piston-driven chest compressor (Thumper, model 1000; MI Instruments; Grand Rapids, MI). For animals randomized to receive epinephrine, 30 μg/kg of epinephrine was injected into the right atrium and this dose was repeated at 3-min intervals. Coincident with start of precordial compression, the animals received mechanical ventilation with a tidal volume of 15 mL/kg and fraction of inspired oxygen of 1.0. Precordial compression was programmed for 80 compressions per minute and synchronized to provide a compression/ventilation ratio of 5:1 with equal compression-relaxation intervals, ie, a 50% duty cycle. The compression force

**Materials and Methods**

The protocol was approved by the Institutional Animal Care and Use Committee. All animals received humane care in compliance with current principles of care for laboratory animals.

**Animal Preparation**

Male domestic pigs weighing from 40 to 45 kg were fasted overnight except for free access to water. Anesthesia was initiated by IM 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 at intervals of 1 h to maintain anesthesia. A cuffed endotracheal tube was advanced into the trachea. Animals received mechanical ventilation with a volume-controlled ventilator (model MA-1; Puritan-Bennett; Carlsbad, CA). End-tidal PCO₂ (PETCO₂) was monitored with an infrared analyzer (model 01R-7101A; Nihon Kohden; Tokyo, Japan). Respiratory frequency was adjusted to maintain PETCO₂ between 35 mm Hg and 40 mm Hg.

![Image](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21966/ on 06/25/2017)
was adjusted to decrease the anterior to posterior diameter of the chest by 25% such as to maintain the coronary perfusion pressure (CPP) > 12 mm Hg. After each minute of precordial compression, another sequence of up to three shocks was delivered, namely 150 J for each biphasic shock and 200 J, 300 J, and 360 J for monophasic shocks. Defibrillation was repeated at intervals of 1 min until the animal was successfully resuscitated or for a maximum of 15 min. If an organized cardiac rhythm with mean aortic pressure of > 60 mm Hg persisted for an interval of ≥ 5 min, the animal was regarded as successfully resuscitated. The animals were then monitored for an additional 4 h. After a panel of 4-h postresuscitation measurements had been completed, the animals were returned to their cages and observed for an additional 68 h. At the end of the 72-h postresuscitation interval, Doppler echocardiographic measurements of myocardial function were repeated. The animals were then killed by IV injection of 150 mg/kg of pentobarbital. Autopsy was routinely performed to identify any significant injuries to the bony thorax and/or the thoracic and abdominal viscera that occurred during CPR, which would have precluded inclusion of the data on the animal.

Measurements

Dynamic data, including aortic pressure, RAP, pulmonary artery and pulmonary occlusive pressures, PETCO2, and the lead II ECG together with the digital output of the Hewlett-Packard Acoustic Quantification system were continuously measured and recorded on a personal computer-based data acquisition system, supported by CODAS hardware/software (DATAQ, Akron, OH) as previously described.9–11 A total of 16 channels was provided for continuous recording at appropriate sampling frequencies. The CPP was digitally computed from the differences in time-coincident aortic pressure and RAP and displayed in real time. The transthoracic electrical impedance was recorded for each shock.

Myocardial systolic and diastolic functions were measured with transeosophageal Doppler echocardiographic techniques developed by us for this porcine model.9 Echocardiographic measurements were obtained with the aid of the Hewlett-Packard Sonos 2500 echocardiographic system (Hewlett-Packard, Medical Products Group). A technically satisfactory two-chamber view was obtained during each experiment. Left ventricular end-systolic volume and left ventricular end-diastolic volume (LVEDV) were calculated by the method of discs utilizing acoustic quantification technology (Hewlett-Packard; Andover, MA). Ejection fraction, stroke volume, and the rate of change of ventricular volumes were computed. Cardiac output was calculated as the product of transaortic flow time velocity integral, aortic valve diameter, and heart rate. Measurements of LVEDVs and wall thickness served as indicators of diastolic function.

A quantitative neurologic alertness score developed by our group13 was utilized for evaluating neurologic recovery at 12-h intervals for a total of 72 h. The alertness score was based on objective grading of level of consciousness, respiration, posture, and food and water intake. Alertness was scored from 0 (coma) to 100 (fully alert) as shown in Table 1.

Aortic and mixed venous blood gases, hemoglobin, and oxyhemoglobin were measured with an automated blood gas analyzer and a co-oximeter (models 1306 and 482, Instrumentation Laboratory, Lexington, MA) adapted for porcine blood. Arterial lactate level, PETCO2, pulmonary artery pressure, RAP, and neurologic alertness score among the four groups prior to cardiac arrest and after successful resuscitation. The calculated CPP was significantly greater in animals that received epinephrine. There were, however, no differences between biphasic-treated and monophasic-treated animals.

No significant differences in transthoracic impedance were demonstrated among the groups. Greater defibrillation energy was required with monophasic defibrillation, but the differences were statistically insignificant (Table 2). However, significantly greater peak currents and peak voltages were delivered by monophasic defibrillations whether or not the animals were treated with epinephrine (Table 2).

All animals treated with biphasic waveform defibrillation and epinephrine were successfully resuscitated. After monophasic waveform defibrillation with epinephrine, four of five animals were successfully resuscitated. Without epinephrine, only three of five animals were successfully resuscitated after either biphasic or monophasic shocks. No significant differences in 72-h

<table>
<thead>
<tr>
<th>Table 1—Neurologic Alertness Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variables</td>
</tr>
<tr>
<td>--------------------------</td>
</tr>
<tr>
<td>Consciousness</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>Obtunded</td>
</tr>
<tr>
<td>Stupor</td>
</tr>
<tr>
<td>Coma</td>
</tr>
<tr>
<td>Respiration</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>Periodic</td>
</tr>
<tr>
<td>Apnea</td>
</tr>
<tr>
<td>Posture</td>
</tr>
<tr>
<td>Standing</td>
</tr>
<tr>
<td>Sitting</td>
</tr>
<tr>
<td>Lying</td>
</tr>
<tr>
<td>Water intake</td>
</tr>
<tr>
<td>Spontaneous</td>
</tr>
<tr>
<td>None</td>
</tr>
<tr>
<td>Eating</td>
</tr>
<tr>
<td>Spontaneous</td>
</tr>
<tr>
<td>None</td>
</tr>
<tr>
<td>Self care, cleaning</td>
</tr>
<tr>
<td>Normal</td>
</tr>
<tr>
<td>None</td>
</tr>
</tbody>
</table>

A total of 20 experiments were performed and completed. There were no differences in hemoglobin and oxyhemoglobin levels, blood gas measurements, arterial lactate level, PETCO2, pulmonary artery pressure, RAP, and neurologic alertness score between time-based measurements within each group were performed with analysis of variance repeated measurements. When the dependent variable was categorical, including success of resuscitation, and 24-h, 48-h, and 72-h survival, Fisher’s Exact Test was used.
survival were observed (Table 2). After monophasic waveform defibrillation and epinephrine, longer intervals of precordial compression and larger total doses of epinephrine were required prior to successful defibrillation and restoration of spontaneous circulation. The variances were large, however, and the differences were therefore not statistically significant (Table 2).

Myocardial function was reduced in all animals after successful resuscitation. However, significantly lesser impairment followed biphasic defibrillation (Fig 2, 3). Without epinephrine, left ventricular stroke volume (SV), cardiac output, ejection fraction (EF), fractional area change (FAC), and LVEDV were each significantly greater in animals after biphasic defibrillation (Fig 2).

After administration of epinephrine (Fig 3), SV, cardiac output, EF, and FAC were each significantly greater in animals after biphasic defibrillation with significantly lower left ventricular end-systolic volume. These differences demonstrated that biphasic waveform defibrillation produced lesser impairment of left ventricular contractile function. Because both systolic left ventricular posterior wall thickness and diastolic left ventricular posterior wall thickness were significantly less after biphasic defibrillation, lesser

---

**Table 2—Primary Outcome Variables After 10 min of Untreated VF**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Biphasic Without Epinephrine</th>
<th>Biphasic With Epinephrine</th>
<th>Monophasic Without Epinephrine</th>
<th>Monophasic With Epinephrine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight, kg</td>
<td>41 ± 2</td>
<td>42 ± 2</td>
<td>42 ± 2</td>
<td>43 ± 2</td>
</tr>
<tr>
<td>ROSC</td>
<td>3/5</td>
<td>5/5</td>
<td>3/5</td>
<td>4/5</td>
</tr>
<tr>
<td>72-h survival</td>
<td>3/5</td>
<td>5/5</td>
<td>3/5</td>
<td>4/5</td>
</tr>
<tr>
<td>Duration of CPR, min</td>
<td>9 ± 7</td>
<td>2 ± 1</td>
<td>8 ± 7</td>
<td>5 ± 6</td>
</tr>
<tr>
<td>Defibrillation energy, J</td>
<td>2,591 ± 2,272</td>
<td>902 ± 434</td>
<td>3,555 ± 833</td>
<td>1,181 ± 1,179</td>
</tr>
<tr>
<td>Total dose of epinephrine, mg</td>
<td></td>
<td></td>
<td>1.4 ± 0.5</td>
<td>2.2 ± 1.6</td>
</tr>
<tr>
<td>Transthoracic impedance, ohms</td>
<td>57 ± 3</td>
<td>51 ± 5</td>
<td>55 ± 5</td>
<td>48 ± 6</td>
</tr>
<tr>
<td>Peak current, amps</td>
<td>29 ± 2</td>
<td>32 ± 3</td>
<td>47 ± 21</td>
<td>48 ± 41</td>
</tr>
<tr>
<td>Peak voltage, V</td>
<td>1,674 ± 6</td>
<td>1,653 ± 17</td>
<td>2,559 ± 155†</td>
<td>2,277 ± 292†</td>
</tr>
</tbody>
</table>

*Data are presented as No./total pigs or mean ± SD. ROSC = restoration of spontaneous circulation.
†p < 0.001 vs biphasic plus epinephrine.
‡p < 0.001 vs biphasic without epinephrine.
myocardial diastolic dysfunction was observed after biphasic defibrillation (Fig 3).

**Discussion**

The present study demonstrated that biphasic waveform defibrillation with fixed lower delivered energies was as effective as monophasic waveform with escalating delivered energies for successful defibrillation after 10 min of untreated VF. However, myocardial systolic function as measured by SV, EF, and FAC was significantly greater in animals after biphasic defibrillation. In support of earlier investigations, myocardial diastolic function was more optimal after biphasic defibrillation as evidenced by lesser left ventricular wall thickness and greater LVEDV. Since the duration of untreated VF and the CPP produced by chest compression were controlled in the present study, the differences in postresuscitation myocardial function are likely to reflect the difference in either the waveforms or in the total energy delivered. The present study further demonstrated that administration of epinephrine during either biphasic or monophasic defibrillation after prolonged cardiac arrest significantly decreases the energy required for successful defibrillation.

Even though, for approximately 39% of patients (range, 13 to 59%), spontaneous circulation is reestablished following initial successful resuscitation, most victims die within 72 h, primarily of heart failure or recurrent ventricular arrhythmias. CPR itself therefore yields a functional survival rate of only 1.4 to 5%.2,3,14,15 The clinical importance of postresuscitation myocardial dysfunction and its fatal outcome have been documented in large, randomized clinical studies. In the Brain Resuscitation Clinical Trial, > 260 victims were initially resuscitated.15 However, approximately 70% of these resuscitated victims died within the first 72 h. Heart failure, ventricular arrhythmias, and recurrent cardiac arrest were identified as the fatal events. In a high-dose epinephrine study, > 400 victims were initially resuscitated. However, the majority of these resuscitated victims died during the early hours and days; heart failure, ventricular arrhythmias, and recurrent cardiac arrest were again identified as predominant causes.2

The severity of postresuscitation myocardial dysfunction was previously related by us to the duration of cardiac arrest, to treatment with epinephrine, and to hypercarbic myocardial acidosis.1,5 Both of our predecessor studies7,8 and the present study have implicated the defibrillation waveform and/or the total electrical energy delivered during defibrillation attempts as an additional factor. We are therefore prompted to call attention to the potential importance of more optimal defibrillation waveforms and the desirability of minimizing the electrical energy that is delivered during electrical defibrillation attempts such as to favor better postresuscitation myocardial function and thereby improve survival.

After direct-current transthoracic defibrillation was introduced for the treatment of VF by Lown et
both the magnitude and duration of the transthoracic electrical shock utilized for defibrillation from VF were suspect as causes of myocardial injury. In isolated tissue cultures of myocardial cells, irregularities of contraction followed a single shock with a peak intensity of either 80 V/cm or 200 V/cm. Synchronized shocks with an energy of 35 J applied directly to the fibrillating canine ventricle significantly decreased cardiac output. After cardiac arrest in dogs, a transthoracic 400-J countershock produced both histologic and metabolic impairment of myocardial cells. After successful defibrillation of patients who sustained cardiac arrest, postresuscitation hypotension and decreased postresuscitation survival were documented. In a rat model of cardiac arrest and resuscitation, we have previously demonstrated that the severity of postresuscitation myocardial dysfunction was closely related to the energy delivered during electrical defibrillation. The greater the delivered energy, the more severe was the postresuscitation myocardial dysfunction and the shorter was the duration of postresuscitation survival.

During the past 2 decades, biphasic waveform defibrillation has been examined in experimental and clinical settings of VF. In the human electrophysiology laboratory or operating room, impedance-compensating biphasic waveforms with energy levels of 115 J or 130 J were as effective as monophasic waveforms with higher energy levels of 200 J for up to 15 s after VF had been induced. After prolonged VF (6 to 9 min) in out-of-hospital settings, there was a greater number of successful defibrillations with 150-J impedance-compensating biphasic waveform shocks when compared with conventional monophasic waveform.

In a randomized clinical study, the effects of fixed, low-energy biphasic waveform defibrillation on initial success of defibrillation, on the likelihood of restoration of spontaneous circulation, on the incidence of hospital admission, and on the number of victims discharged from the hospital were compared with the effects of conventional escalating monophasic waveform defibrillation. Biphasic waveform defibrillation with a fixed energy of 150 J significantly improved the efficacy of successful defibrillation (98% vs 67%, \( p < 0.0001 \)) when compared with conventional monophasic defibrillation. This was associated with a significantly greater success in the restoration of spontaneous circulation (76% vs 55%, \( p < 0.02 \)). The population of patients was small, and no statistically significant differences in hospital admission and hospital discharge were observed, although neurologic function was better after lower-energy biphasic shocks.

In our previous study in the same porcine model of cardiac arrest and resuscitation, fixed low-energy biphasic waveform defibrillation significantly reduced the severity of postresuscitation myocardial systolic and diastolic dysfunction when compared with escalating high-energy conventional monophasic waveform defibrillation after either short or intermediate duration of cardiac arrest. The present study further demonstrated that these beneficial effects of low-energy biphasic waveform defibrillation were also observed when the duration of cardiac arrest was prolonged. Our results are also in concert with those observed on human patients during implantation of cardioverter-defibrillator. Reddy et al found significantly greater evidence of postshock myocardial ischemic injury with ST-segment elevation after 200-J monophasic waveform defibrillation when compared with 115-J or 130-J biphasic waveform defibrillation.

Following a brief duration of VF, epinephrine administration decreases the threshold of VF and increases the threshold of defibrillation. The duration of action potentials is decreased such that the cycle length is decreased. Accordingly, the number of fibrillation wavelets is increased. When administration of epinephrine follows 10 min of untreated cardiac arrest, as in the present study, there is less energy required for defibrillation because the duration of action potentials is increased and there is a decreased threshold for defibrillation. With increasing duration of untreated VF and therefore the severity of myocardial ischemia, increases in myocardial perfusion that follow the administration of epinephrine may be the explanation for differences in thresholds, contingent on the duration of untreated VF.

The mechanisms that account for the reduced myocardial injury after biphasic waveform defibrillation are not completely understood. Caterine et al demonstrated that the concentration of coronary oxygen free radicals is significantly related to the energy delivered during electrical shocks. These free radicals may explain impaired organellar function with damaged sarcolemma and mitochondria, calcium overload, and impaired cellular oxidative metabolism. In the present study, we compared electrical shocks in which there were differences in both the waveforms and in the total energy delivered. Accordingly, the results leave unanswered whether the differences in postresuscitation myocardial function are related to the waveforms, the total energies, or a combination of the two.

The mechanisms that account for the increased defibrillation efficacy of biphasic defibrillation waveforms also remain incompletely understood. Jones and associates postulated that the first phase of the bipolar waveform acts as a conditioning prepulse that enhances sodium current excitability, thereby reducing the activation threshold for the second phase, which is responsible for defibrillation. Keener and Lewis provided evidence in further support of this.
hypothesis; they explained increased defibrillation efficacy of biphasic waveforms by initial hyperpolarization that abbreviated the refractory period of myocytes, thereby lowering the activation threshold prior to the reversal of electrical polarity.

We conclude that the experimental data herein reported sustain our hypotheses. Biphasic waveform shocks with a fixed energy of 150 J were at least as effective as conventional sequential monophasic waveform shocks with progressive energy levels of 200 J, 300 J, and 360 J for successful defibrillation following prolonged cardiac arrest. In addition, the low-energy biphasic waveform shocks significantly decreased the severity of postresuscitation myocardial dysfunction.

ACKNOWLEDGMENT: The authors thank Mr. David Snyder, Mr. Carl Morgan, and Dr. Dawn Jorgenson of Agilent Technologies, Heartstream Operation for their engineering support.

REFERENCES

1 Guidelines for cardiopulmonary resuscitation and emergency cardiac care: II. Adult basic life support; III. Adult advanced life support. JAMA 1992; 268:2184–2241


