Electrical Induction of Ventricular Fibrillation for Resuscitation From Postcountershock Pulseless and Asystolic Cardiac Arrests

Charles T. Leng, MD; Ronald D. Berger, MD, PhD; Hugh Calkins, MD; Albert C. Lardo, PhD; Norman A. Paradis, MD; Henry R. Halperin, MD, MA

Background—There is increasing evidence that defibrillation from prolonged ventricular fibrillation (VF) before CPR decreases survival. It remains unclear, however, whether harmful effects are due primarily to initial countershock of ischemic myocardium or to resultant postdefibrillation rhythms (ie, pulseless electrical activity [PEA] or asystole).

Methods and Results—We induced 15 dogs into 12 minutes of VF and randomized them to 3 groups. Group 1 was defibrillated at 12 minutes and then administered advanced cardiac life support (ACLS); group 2 was allowed to remain in VF and was subsequently defibrillated after 4 minutes of ACLS; group 3 was defibrillated at 12 minutes, electrically refibrillated, and then defibrillated after 4 minutes of ACLS. All group 1 and 3 animals were defibrillated into PEA/asystole at 12 minutes. After 4 minutes of ACLS, group 2 and 3 animals were effectively defibrillated into sinus rhythm. The extension of VF in group 2 and 3 subjects paradoxically resulted in shorter mean resuscitation times (251 ± 15 and 245 ± 7 seconds, respectively, versus 459 ± 66 seconds for group 1; \( P < 0.05 \)) and improved 1-hour survival (10 of 10 group 2 and 3 dogs versus 1 of 5 group 1 dogs; Fisher’s exact, \( P < 0.005 \)) compared with more conservatively managed group 1 subjects.

Conclusions—Precountershock CPR during VF appears more conducive to resuscitation than CPR during postcountershock PEA or asystole. The intentionnal induction of VF may prove useful in the management of PEA and asystolic arrests. (Circulation. 2001;104:723-728.)

Key Words: fibrillation ■ cardiopulmonary resuscitation ■ myocardial stunning

C ountershock termination of prolonged ventricular fibrillation (VF) frequently results in either pulseless electrical activity (PEA) or asystole.1–3 Resuscitation from these postcountershock rhythms rarely proves successful, with short-term mortality rates reportedly >85%.4,5 However, growing evidence suggests that a sustained period of CPR before defibrillation may circumvent the dismal prognosis associated with these nonperfusing rhythms.6–8

Recent efforts to improve survival from out-of-hospital VF arrests have focused primarily on providing the earliest possible access to defibrillation. Although early defibrillation of witnessed arrests has proven benefit,9,10 a disturbing trend toward worsening outcomes appears evident when unsupported VF has been prolonged for >4 to 5 minutes.7,11 In this setting, immediate defibrillation appears to simply convert one nonperfusing rhythm (ie, VF) to another (ie, PEA/asystole).

The primary aim of the present study was to clarify the relative importance of CPR compared with defibrillation after prolonged VF arrest. We predicted that initial efforts at restoring coronary perfusion with CPR would prove more critical to resuscitation than immediate defibrillation as initial definitive therapy for prolonged VF. We also sought to clarify whether the anticipated negative impact of immediate defibrillation would result primarily from countershock-induced injury to ischemic myocardium or to some intrinsic property of the underlying arrest rhythm.

Methods

All experiments were conducted in accordance with the Position of the American Heart Association on Research Animal Use and with the approval of the Animal Care and Use Committee at the Johns Hopkins Hospital.

Animal Preparation

Fifteen consecutive mongrel dogs of similar size and weight (25 to 30 kg) were intubated and mechanically ventilated with an anesthesia ventilator (Narkomed, North American Dräger). General anesthesia was induced with 12.5 to 15.0 mg/kg IV sodium thiopental and maintained with 1% to 3% isoflurane. Then, 7F Millar microma-
nometer catheters were introduced through arterial and venous femoral sheaths and positioned in the right atrium (RA), ascending aorta, and left ventricle (LV) under fluoroscopic guidance. A pacing catheter was placed in the right ventricular apex and connected to an electrophysiological stimulator/recorder (MIDAS 2500, PPG Industries).

Adhesive defibrillation electrodes (R2, Cardiotronics) were applied to the left (anode) and right (cathode) hemithorax and connected to a monophasic defibrillator (Zoll). A pneumatic CPR vest was positioned to perform chest compressions at a fixed rate of 60 cycles/min with peak chest compressions at 225 to 235 mm Hg. Arterial blood gases were monitored, and ventilator adjustments were made to achieve an arterial PCO2 between 35 to 45 mm Hg and a pH between 7.35 to 7.45. A constant infusion of lactated Ringer’s solution was regulated to maintain a mean RA pressure of 2 to 8 mm Hg and an LV end-diastolic pressure (LVEDP) of 8 to 12 mm Hg.

**Experimental Protocol**

Subjects were electrically induced into VF through a right ventricular pacing catheter with a 10-second pulse of alternating current at 60 Hz. General anesthesia was discontinued upon establishment of VF. Three countershocks were sequentially administered as needed to terminate VF with an energy step-up sequence of 200, 300, and 360 J. Vest CPR was initiated in all animals at the 12.5-minute time point and continued until return of spontaneous circulation (ROSC) or 45 minutes elapsed. Assessment of arterial pressure waveforms enabled us to immediately detect ROSC during administered chest compressions. Continuous monitoring of intracardiac electromgrams obviated the need to interrupt chest compressions for surface ECG rhythm diagnosis.

All spontaneous VF recurrences were treated immediately with the last effective countershock energy with step-up to maximum defibrillator output if necessary. Single 360-J countershocks could be administered at 16 minutes and repeated at 3-minute intervals for all dogs proving refractory to earlier attempts at defibrillation. Countershocks for refractory VF were synchronized to follow epinephrine boluses by 30 seconds.

Epinephrine 1 mg IV boluses were administered to all animals at 12.5 minutes and at regular 3-minute intervals regardless of hemodynamic stability. After 45 minutes, animals were observed during a 15-minute period of epinephrine washout and then assessed hemodynamically at the 1-hour study end point. Surviving animals were subsequently euthanized with intravenous KCl. Limited necropsy was performed on all animal subjects.

**Specific Protocols**

Animals were randomized to the following 3 resuscitation protocols. The only differences among them were with respect to the timing of electrical interventions.

**Immediate Defibrillation**

Study animals in the immediate defibrillation (DF) group were promptly defibrillated after 12 minutes of VF (Figure 1). Vest CPR and intravenous boluses of epinephrine were initiated at 12.5 minutes as described above.

**Precountershock CPR**

Animals in the precountershock CPR (CPR) group were subjected to 4 additional minutes of VF (Figure 1). Vest CPR and intravenous boluses of epinephrine were initiated at 12.5 minutes as described above.

**Refibrillation**

Animal subjects in the refibrillation (RF) group were defibrillated after 12 minutes of unsupported VF and then immediately refibrillated at the 12.25-minute time point with a 10-second pulse of alternating current at 60 Hz (Figure 1). Vest CPR and intravenous boluses of epinephrine were initiated at 12.5 minutes. A second round of countershocks was administered at the 16-minute time point.

**Definitions**

Defibrillated animals supporting an LV systolic blood pressure (LVSBP) >50 mm Hg for >1 minute were considered to have achieved ROSC. Resuscitated animals were classified as study survivors if they could maintain a steady-state LVSBP >50 mm Hg after washout of epinephrine at the 1-hour study end point. Resuscitation time was reported as the total time required to achieve ROSC after the 12-minute time point (ROSC time). The time difference between successful defibrillation and documented onset of ROSC was designated as the postarhythmic mechanical arrest (PAMA) interval. For refibrillated subjects, the PAMA interval was based on time of defibrillation after the 16-minute time point.

**Statistical Analysis**

Determination of sample size was based on our previously published data that predicted a >3-minute difference in group resuscitation times and an SD of 75 seconds.8 A power of 80% and P<0.05 were assumed. ANOVA was used to screen for significant variability; then, contrast analyses were performed to isolate differences for our prospective end points. Categorical variables were evaluated by Fisher’s exact analysis. Results were reported as mean±SD. A value of P<0.05 was considered significant.

**Results**

**Baseline Characteristics**

No significant differences in baseline LV hemodynamics or loading conditions were present after randomization to the 3 treatment groups. Mean heart rate, RA pressure, LVSBP, LVEDP, dP/dtmax, and dP/dtmin are presented in Table 1.

**Resuscitation Factors**

By design, the CPR group and RF group were subjected to more cumulative VF than the DF control group (CPR, 963±5.5

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**TABLE 1. Baseline Characteristics**

<table>
<thead>
<tr>
<th></th>
<th>DF (n=5)</th>
<th>CPR (n=5)</th>
<th>RF (n=5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, bpm</td>
<td>168±24</td>
<td>161±31</td>
<td>175±26</td>
</tr>
<tr>
<td>RAM, mm Hg</td>
<td>6.5±2.9</td>
<td>7.2±5.1</td>
<td>6.4±2.6</td>
</tr>
<tr>
<td>LVEDP, mm Hg</td>
<td>17.4±3.5</td>
<td>16.0±5.4</td>
<td>18.5±6.3</td>
</tr>
<tr>
<td>LVSBP, mm Hg</td>
<td>186±22</td>
<td>180±7.5</td>
<td>175±26</td>
</tr>
<tr>
<td>dP/dtmax, mm Hg/s</td>
<td>1980±400</td>
<td>1890±89</td>
<td>2070±280</td>
</tr>
<tr>
<td>dP/dtmin, mm Hg/s</td>
<td>1290±380</td>
<td>1860±170</td>
<td>1980±260</td>
</tr>
</tbody>
</table>

DF indicates immediate defibrillation group; CPR, precountershock CPR group; RF, refibrillation group; HR, heart rate; RAM, mean RA pressure. Values are mean±SD. Differences were not statistically significant.
seconds; RF, 951±12 seconds; DF, 790±41 seconds; P<0.001 versus DF group). These 2 experimental groups also experienced fewer spontaneous recurrences of VF (CPR, 0.4±0.9; RF, 0.4±0.9; DF, 4.6±3.1; P<0.05 versus DF group) and required fewer defibrillatory countershocks (CPR, 1.6±1.3; RF, 2.4±0.9; DF, 6.8±3.6; P<0.05 versus DF group) during attempts at resuscitation. Peak defibrillation thresholds were 220±45 J for the CPR group and 200±0 J for the RF group and were significantly lower than 328±72 J for the DF group (P<0.001), which required countershocks of progressively higher energy over time to terminate VF recurrences. Differences in serum pH and peak coronary perfusion pressures did not prove to be statistically significant. These resuscitation factors are presented in Table 2.

Defibrillation Outcomes

All subjects were successfully defibrillated within the first round of stacked countershocks allowed in their respective treatment groups and with all subsequent countershocks administered for both spontaneous and induced recurrences of VF. All DF group and RF group animals were defibrillated into either PEA (6 of 10) or asystole (4 of 10) after 12 minutes of VF. After 16 minutes of VF, all CPR group and RF group animals were effectively defibrillated into a perfusing sinus rhythm (10 of 10).

Resuscitation Times

CPR group and RF group subjects were more promptly resuscitated than DF group controls (Figure 2) despite significantly longer durations of VF. All animals experienced ROSC except for 2 in the immediate defibrillation group, which were excluded from subsequent analyses. Mean resuscitation times for the CPR group (251±15 seconds) and RF group (245±7.1 seconds) proved significantly shorter than for the DF group (459±66 seconds; P<0.05). These differences were attributable to remarkably shorter periods of postdefibrillation contractile arrest when successful countershocks were preceded by effective CPR during VF (Figure 3). Mean PAMA intervals were 11.2±14 and 5.0±7.1 seconds for the CPR and RF groups, respectively. These were markedly shorter than the mean for the DF group (459±66 seconds; P<0.01).

Survival Outcomes

Shorter resuscitation times translated predictably into overall improved survival (Table 3). All 10 animals in the CPR and RF groups proved hemodynamically stable after epinephrine washout at the 1-hour study end point compared with only 1 animal in the DF control group (P<0.005). The remaining 4 DF group animals represented the only deaths in the study. No deaths could be attributed to mechanical trauma on the basis of necropsy.

Postresuscitation Myocardial Dysfunction

Although there were no significant differences in resuscitation times or arrest survival, mean LVSBP, dP/dtmax, and dP/dtmin trended lower for RF group survivors than for CPR group survivors (P=NS) at the 1-hour study end point (Table 4). Calculations of percent change versus baseline in LV dP/dtmax (CPR, −30±23%; RF, −59±26%; P=0.09) and LV dP/dtmin (CPR, −28±26%; RF, −58±28%; P=0.12) suggested a trend toward more severe postresuscitation myocardial dysfunction for the RF group. Concurrent assessments of heart rate, RA pressure, and LVEDP failed to identify differences in LV loading conditions that could have affected postresuscitation hemodynamics.

Discussion

To the best of our knowledge, the present study is the first to report the feasibility of efforts to intentionally refibrillate.

### Table 2. Resuscitation Factors

<table>
<thead>
<tr>
<th></th>
<th>DF (n=5)</th>
<th>CPR (n=5)</th>
<th>RF (n=5)</th>
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<tr>
<td>Cumulative VF, s</td>
<td>790±41</td>
<td>963±5.5*</td>
<td>951±12*</td>
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<tr>
<td>VF recurrences, n</td>
<td>4.6±3.1</td>
<td>0.4±0.9†</td>
<td>0.4±0.9†</td>
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<tr>
<td>Countershocks, n</td>
<td>6.8±3.6</td>
<td>1.6±1.3†</td>
<td>2.4±0.9†</td>
</tr>
<tr>
<td>Peak DFT, J</td>
<td>328±72</td>
<td>220±45†</td>
<td>200±0.0†</td>
</tr>
<tr>
<td>Peak CPP, mm Hg</td>
<td>29±12</td>
<td>35±18</td>
<td>43±8.8</td>
</tr>
<tr>
<td>pH</td>
<td>7.37±0.06</td>
<td>7.37±0.05</td>
<td>7.36±0.04</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 1, plus DFT indicates defibrillation threshold; and CPP, coronary perfusion pressure. Values are mean±SD.

*p<0.001, †p<0.05 vs DF group.

### Table 3. Resuscitation Outcomes

<table>
<thead>
<tr>
<th></th>
<th>DF (n=5)</th>
<th>CPR (n=5)</th>
<th>RF (n=5)</th>
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<tbody>
<tr>
<td>ROSC, n</td>
<td>3</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>ROSC time, s</td>
<td>459±66</td>
<td>251±15*</td>
<td>245±7.0*</td>
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<tr>
<td>Survivors, n</td>
<td>1†</td>
<td>5</td>
<td>5</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 1. Values are mean±SD.

*P<0.05 vs DF group; †P<0.005 vs CPR and RF subjects.
TABLE 4. Postresuscitation Characteristics

<table>
<thead>
<tr>
<th></th>
<th>CPR (n=5)</th>
<th>RF (n=5)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, bpm</td>
<td>193±26</td>
<td>184±16</td>
<td>0.54</td>
</tr>
<tr>
<td>RAM, mm Hg</td>
<td>8.7±4.2</td>
<td>10±3.7</td>
<td>0.59</td>
</tr>
<tr>
<td>LVEDP, mm Hg</td>
<td>26±7.4</td>
<td>28±6.3</td>
<td>0.67</td>
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<tr>
<td>LVSP, mm Hg</td>
<td>131±32</td>
<td>92±31</td>
<td>0.09</td>
</tr>
<tr>
<td>dP/dtmin,m mm Hg</td>
<td>1315±385</td>
<td>834±495</td>
<td>0.12</td>
</tr>
<tr>
<td>dP/dtmax,m mm Hg</td>
<td>1309±369</td>
<td>828±527</td>
<td>0.14</td>
</tr>
</tbody>
</table>

Abbreviations as in Table 1. Values are mean±SD. Differences were not statistically significant.

Immediate Versus Delayed Defibrillation

The apparent success of hospital critical care units and public access defibrillation programs has served to underscore the importance of early defibrillation.10,13 Reported improvements in survival, however, have been limited largely to witnessed VF arrests of relatively short duration. Although epidemiological data would predict that arrest survival decreases by ~7% to 10% with each passing minute in VF,14 the present work suggests that immediate defibrillation of prolonged VF may reasonably be deferred in favor of initial efforts to restore myocardial perfusion. Current ACLS guidelines have assigned the highest possible priority to immediate defibrillation in the treatment of cardiac arrests due to VF.15 Although there is little doubt that early termination of VF promotes survival, the immediate defibrillation of prolonged VF typically results in a nonperfusing rhythm that proves refractory to conventional therapy.1,3 In our model of out-of-hospital cardiac arrest, defibrillation of VF into PEA or asystole appeared to significantly delay or preclude ROSC. This finding suggests that prolonged VF poses a substantially more complex challenge than rescue from brief VF and that a resuscitation strategy placing unconditional priority on defibrillation may unwittingly compromise survival after prolonged arrests.

A few studies involving VF of prolonged or uncertain duration have suggested that effective CPR may initially prove more critical to resuscitation than immediate defibrillation.6-8 Niemann et al6 were the first to report improvements in resuscitation after 7.5 minutes of unsupported VF when experimental dogs were pretreated with ~5 minutes of manual CPR and high-dose epinephrine before defibrillation. The study, however, had systematic differences in epinephrine dosing between experimental and control animals. Cobb et al7 demonstrated similar improvements in resuscitation outcomes when 90 seconds of precouthernshock CPR was compared with immediate defibrillation for out-of-hospital VF arrests. However, the study was limited by its retrospective design and reliance on historical patient controls. Recently, in our own laboratory, a subgroup analysis of animals initially proving refractory to defibrillation demonstrated a surprising trend toward improved resuscitation rates and less severe myocardial stunning despite the probable selection of a more compromised population of subjects.9 Immediately defibrillated subjects appeared to fare no better than subjects in the delayed defibrillation subgroup despite significantly shorter durations of VF, fewer countershocks, and equivalent coronary perfusion pressures during CPR. The available evidence would therefore suggest that modest delays to defibrillation do not necessarily predict poorer cardiac resuscitation outcomes when ongoing VF is counterbalanced by effective precouthernshock CPR.

The present study demonstrates a substantial benefit of precouthernshock CPR over immediate defibrillation in the late resuscitation of VF arrest. Animals immediately defibrillated after 12 minutes of unsupported VF were uniformly converted to either PEA or asystole (10 DF group and RF group subjects), whereas experimental animals receiving precouthernshock CPR were all defibrillated directly into stable perfusing rhythms after 16 minutes of VF (10 CPR group and RF group subjects). Of the 5 dogs randomized to the immediate defibrillation control group, 2 proved entirely unresponsive to conventional efforts to restore spontaneous circulation, and 2 more proved incapable of maintaining a stable perfusing rhythm by the 1-hour study end point. In addition, mean arrest times were >3 minutes longer for resuscitated control (DF group) animals despite the fact that they were defibrillated 4 minutes earlier than our experimental (CPR group and RF group) animals. This was attributed to protracted intervals of postdefibrillation contractile arrest that were notably absent when concerted efforts were made to restore some degree of coronary perfusion before defibrillation. Such a marked difference in recovery of contractile function between well-controlled study arms would appear to reflect a critical order dependence of treatment rather than a qualitative difference in CPR or epinephrine administration. An alternative approach to cardiac resuscitation may prove more efficacious than current ACLS protocols for most out-of-hospital VF arrests.

Refibrillation

Recurrent episodes of VF are frequently encountered clinically and have typically been regarded as major setbacks to cardiac resuscitation. Spontaneous refibrillation, perhaps inappropriately, diverts attention from other pressing resuscitative concerns toward remedial rhythm analysis and repeated efforts at defibrillation. Although VF recurrences exact a high energetic cost and effectively forestall ROSC, the present work suggests that refibrillation may prove not to be entirely detrimental, particularly when nonperfusing rhythms persist after initial defibrillation. A more permissive attitude toward VF recurrences may ultimately improve survival after prolonged cardiac arrest.
In our study, intentional refibrillation conferred several advantages to experimental animals randomized to the RF group. Compared with DF control animals, RF subjects appeared to have either less tendency or simply less opportunity to spontaneously refibrillate during attempted resuscitation and were therefore less likely to require repeated and potentially harmful high-energy countershocks. The additional cycle of electrical refibrillation and delayed defibrillation resulted in significantly shorter resuscitation times than for our control subjects despite identical administration of CPR, epinephrine, and immediate countershocks to profoundly ischemic myocardium after 12 minutes of unsupported VF. Intentional refibrillation also allowed a simpler and more predictable resuscitation course, with immediate ROSC providing a consistent end point after delayed defibrillation at the 16-minute time point. However, the most provocative finding of our study was that survival from postcountershock PEA and asystole could effectively be transformed in our experimental model from 1 of 5 (immediate defibrillation group) to 5 of 5 (refibrillation group) through the intentional use of electrical refibrillation. These findings challenge the widely held perception of postcountershock PEA and asystole as terminal rhythms and suggest that electrical refibrillation may prove capable of favorably modifying the prognosis of these nonperfusing rhythms.

Potential Mechanisms
The precise mechanism by which immediate defibrillation of prolonged VF compromises arrest survival remains unclear but may have as its basis a more profound degree of postarrhythmic myocardial stunning. Our work demonstrates that early defibrillation followed by CPR during PEA or asystole is less effective in restoring perfusing rhythms than initial CPR during VF and delayed defibrillation. We speculate that a previously uncharacterized interaction between prolonged VF, no-flow myocardial ischemia, and electrical defibrillation may severely limit survival by mediating a more profound degree of excitation-contraction uncoupling after immediate defibrillation. It may prove critically important to restore sufficient metabolic support for myocardial contractility (ie, to reverse tissue acidosis, intracellular calcium overload, and substrate depletion) with effective CPR before attempts at defibrillation.

Conventional wisdom would maintain that fibrillating myocardium suffers prohibitive deterioration over time; however, the administration of CPR and epinephrine in our experimental model appeared to compensate adequately for the metabolic costs of briefly extending VF. Classic descriptions of early fibrillatory contractions by Garrey et al suggest the possibility that VF might itself prove capable of ameliorating postarrhythmic myocardial dysfunction through augmentation of coronary perfusion during CPR. Although irregular, high-frequency membrane potential oscillations unique to VF could conceivably have resulted in favorable increases in intracellular calcium and postextrasystolic potentiation of fibrillatory contractions, we could not demonstrate significantly higher coronary perfusion pressures in VF than during PEA or asystole in our present model.

The experimental arms of our study were specifically designed to isolate the effects of immediate countershocks from the resultant postdefibrillation rhythms. However, an unanticipated need for more frequent countershocks in our defibrillation control group limited our ability to resolve this issue. Although we conjectured that profoundly ischemic myocardium might prove particularly susceptible to injury from immediate defibrillatory countershocks, perhaps as a result of instantaneous generation of free radical species and associated mitochondrial injury, the consideration of refibrillation and defibrillation group end points argued against the supposition that initial countershocks alone accounted for subsequent compromise of resuscitation. A revealing trend toward more severe myocardial stunning, however, became apparent when resuscitated RF group animals were compared with CPR group animals that were not subjected to an additional cycle of immediate defibrillation and refibrillation. By extension, repeated high-energy countershocks administered in a systematic attempt to suppress VF may have served to potentiate typically minor degrees of electrical myocardial injury and may be primarily responsible for producing the phenotype of crippling hemodynamic consequences apparent in our immediate defibrillation group. Thus, frequent defibrillatory countershocks to maintain essentially pulseless rhythms would appear not merely unproductive but ultimately deleterious to resuscitation from VF arrest.

Clinical Implications
The prompt restoration of a spontaneous perfusing rhythm remains the primary determinant of survival after cardiac arrest. Conventional resuscitation strategies often prove insufficient in achieving this goal for the vast majority of out-of-hospital arrests. Our work suggests that a critical order dependence of treatment exists for resuscitation from prolonged VF, with initial restoration of myocardial perfusion constituting a higher priority than immediate defibrillation. This study furthermore suggests that the intentional induction of VF may prove useful in the future management of postcountershock PEA and asystole. Our conclusions represent a fundamental revision of prevailing concepts regarding resuscitation from prolonged cardiac arrests. If these findings can be reproduced in studies of human resuscitation, a substantive change in current ACLS guidelines may be warranted.

Acknowledgment
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References


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