In a Swine Model, Chest Compressions Cause Ventricular Capture and, By Means of a Long-Short Sequence, Ventricular Fibrillation

Jose Osorio, MD; Derek J. Dosdall, PhD; Robert P. Robichaux, Jr, MD, MPH; Paul B. Tabereaux, MD, MPH; Raymond E. Ideker, MD, PhD

**Background**—During resuscitation, fibrillation often recurs. In swine, we studied refibrillation after long-duration ventricular fibrillation, investigating an association with chest compressions (CCs).

**Methods and Results**—In protocol A, 47 episodes of long-duration ventricular fibrillation lasting at least 2.5 minutes were induced in 8 animals. After defibrillation, CCs were required for 35 episodes and delivered with a pneumatic device (Lucas cardiopulmonary resuscitation). In 9 episodes, refibrillation occurred within 2 seconds of CC initiation (group 1) and in 26 episodes, CCs were delivered without refibrillation (group 2). From the ECG and intracardiac electrodes, the RR interval preceding CCs, the shortest cycle length during the first 2 CCs (short), and the preceding cycle length (long) were measured. A similar study was conducted in 3 more animals without intracardiac catheters (protocol B). In protocol A, the mean RR before CC was 665±292 ms in group 1 and 769±316 ms in group 2. CCs stimulated ventricular beats in all 35 episodes. The short and long intervals were shorter in group 1 (215±31 and 552±210 ms) than in group 2 (402±153 and 699±147 ms) (P=0.009 and P=0.04, respectively). The prematurity index (short/RR) was lower in group 1 than in group 2 (0.35±0.09 vs 0.58±0.21; P<0.01). A short interval <231 ms predicted refibrillation with 88% sensitivity and 91% specificity. In protocol B, CCs were required in 11 episodes, causing ventricular stimulation in all of them and ventricular fibrillation within the first 2 CCs in 3.

**Conclusions**—Under some conditions, CC during resuscitation can stimulate the ventricles and initiate ventricular fibrillation by a long-short sequence. (*Circ Arrhythmia Electrophysiol. 2008;1:282-289.*)

**Key Words:** resuscitation ■ chest compression ■ commotio cordis ■ refibrillation ■ ventricular capture

Despite significant advances in resuscitation and efforts to increase awareness regarding sudden cardiac arrest (SCA), there remains an epidemic of arrhythmic death,1 claiming the lives of 350 000 to 450 000 Americans yearly. Out-of-hospital SCA is also a leading source of health care costs in the United States.2 Survival rates for out-of-hospital SCAs are poor, ranging from 3% to 18%, although with the increasing availability of automatic external defibrillators and a focus on early defibrillation, improvements are now seen.3–11

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During cardiopulmonary resuscitation (CPR), refibrillation is common and often recurrent. The incidence has been reported to be as high as 79%,12 but its impact on patient outcome is not clear. Van Alem et al12 showed a linear association between mortality and the number of episodes of refibrillation.

The primary objective of this study was to investigate episodes of refibrillation in a swine model of long-duration ventricular fibrillation (LDVF) to determine whether refibrillation is associated with chest compressions (CCs).

**Methods**

In a study designed to investigate the defibrillation threshold (DFT) in successive episodes of LDVF, CCs were performed after successful shocks in most to achieve return of spontaneous circulation (ROSC). In the first 2 swine studied, refibrillation was frequently observed and often closely followed initiation of CCs. These observations led us to record continuously from 2 intracardiac monophasic action potential (MAP) electrodes placed, starting with the third animal, to determine whether CCs were electrically stimulating the heart and inducing VF. The results of the DFT study will be presented in another publication. Here, we report the results in the last 8 animals, beginning with the third animal of the original study, dealing with the relationship between CCs and VF recurrence (protocol A). We then performed a second study in 3 more animals in which no catheters were present in the ventricles during CCs to evaluate the possibility that the results we observed in the first study were caused by the mechanical stimulation of intracardiac catheters on the ventricular walls (protocol B).
Animal Preparation
All of the animals were managed in accordance with the guidelines established by the American Heart Association on research animal use and the protocol was approved by the University of Alabama at Birmingham Institutional Animal Care and Use Committee. The animal preparation was similar in both protocols unless otherwise specified. Twelve (24±3 kg) domestic farm swine (8 in protocol A and 3 in protocol B) were sedated initially with an intramuscular injection of telazol (4.4 mg/kg) and xylazine (2.2 mg/kg). Each animal was intubated, restrained in dorsal recumbency, and mechanically ventilated with a mixture of room air, oxygen, and isoflurane (1.5% to 2.5%). Isoflurane mixture levels were adjusted throughout the experiment to maintain a deep surgical plane of anesthesia. Animals were given intravenous normal saline throughout the experiment. Arterial blood gas values, electrolyte levels, and core body temperature were determined every 30 minutes and before each episode of VF was induced and were maintained within normal limits. Heart rate, lead II ECG, and arterial blood pressure were monitored continuously. A 6-French sheath was placed in the femoral artery for blood pressure monitoring and in the femoral vein for intravenous infusion of fluids and medications, if necessary. All pigs had adhesive electrode pads (Quik-Combo, Medtronic Physio-Control, Redmond, Wash) applied to the shaved cutaneous surfaces of the left and right lateral thorax. The right and left electrodes were placed in a parasternal position with the superior edge of the electrode at the second thoracic interspace.

In protocol A, 6-French MAP catheters (EP Technologies) were advanced into the right and left ventricular apices to allow electric induction of VF and continuous intracardiac recordings. The MAP catheters were connected to a recording system via isolated DC couplers, allowing continuous recording during delivery of shocks with no loss of data. In protocol B, MAP catheters were not used, and an 8.5-French 79.4-cm sheath (55°; Convoy Advanced Delivery Sheath Kits, Boston Scientific, Boston Mass) was placed in the right femoral vein and advanced under fluoroscopic guidance into the inferior vena cava–right atrial junction. A His catheter was bent to form a 45° to 60° angle and advanced through the long sheath. This allowed the catheter to be blindly placed in the right ventricle to allow electric induction of VF and to be pulled back out of the heart before the initiation of CCs. Marks were made in the catheter assuring that a few millimeters of the tip would be left outside of the catheter for bipolar recording. A second catheter was introduced into the esophagus and left at the level of the inferior vena cava–right atrial junction to obtain a bipolar recording.

The remainder of the study, including the number and length of VF inductions and resuscitation efforts, were identical for protocols A and B.

Study Design
The DFT was initially obtained using a standard up-down protocol.13 Alternating episodes of short- (10 seconds) and long-duration (2.5 minutes) VF were induced in each animal, up to 12 episodes (6 episodes of short- and 6 of long-duration VF).

Fibrillation/Defibrillation
VF was induced by delivering a burst of 60-Hz alternating current through the right ventricular MAP electrode (protocol A) or tip of a His catheter positioned in the right ventricular free wall (protocol B). During VF, the ventilator was turned off so that shocks would consistently be delivered at end expiration, providing stable trans-thoracic impedance. Exponentially descending biphasic truncated shocks were delivered using an external defibrillator (LifePak 20, Physio-Control). After 10 seconds (short-duration) or 2.5 minutes (long-duration) of VF, shocks of ascending energy were delivered until successful defibrillation was achieved, with no use of “rescue shocks.” The initial shock was 50% of the DFT initially assessed. If refibrillation was observed, shocks of 200, 300, and 360 joules were delivered as necessary to defibrillate.

Resuscitation
If ROSC was not observed within 10 to 20 seconds after defibrillation, CCs were begun. CCs were interrupted briefly after 1 minute for reassessment of spontaneous circulation. In the episodes where defibrillation occurred during delivery of CCs, defibrillation was performed after 1 minute of compressions and resumed after a successful shock if there was no ROSC.

Once ROSC was achieved, 5 minutes (short-duration VF) or 30 minutes (LDVF) were allowed for the animal to recover hemodynamic and metabolic stability after each VF episode.

Chest Compressions
CCs were administered with a commercially available pneumatic device, the Lucas CPR (Jolife, Lund, Sweden), which delivers CCs at a rate of approximately 100 cycles per minute, with chest incursion of 5 cm. The first CC delivered has a cycle length of 960 ms, whereas all succeeding cycle lengths are 575 ms.

Data Analysis
Only those VF episodes that required CCs to achieve ROSC were included in the data analysis. Although in some episodes CCs were required for >1 minute, for statistical analysis, only the first minute of CCs was included. Data analysis was performed separately for protocols A and B (Figure 1).

The episodes requiring CCs in protocol A were divided in 2 groups: 1 in which refibrillation occurred during the first 2 CCs (group 1, n=9) and 1 in which refibrillation was not seen during CCs (group 2, n=26). The following intervals were measured using the ECG and MAP electrode recordings: the RR interval preceding initiation of CCs in those episodes in which a spontaneous rhythm was present before CCs were given, the shortest cycle length during the first 2 CCs (short), and the preceding cycle length (long) (Figure 2).

The prematurity index was calculated by dividing the duration of the short segment by the RR segment (both in ms) in each of the cases in which a spontaneous rhythm was present before CCs were begun. Short/long was similarly obtained by dividing the short segment by the long segment (in ms) for each case.

For protocol B, we were unable to correctly identify all of the intervals because of significant artifacts from the CCs seen in the ECG and the bipolar lead (IVC esophagus) recording.

Definitions
Ventricular capture (by CCs) was defined as a ventricular depolarization that was synchronous with the initial upstroke of the blood pressure caused by compression of the chest (Figure 2).
CC triggered VF was defined as VF initiated after ventricular capture seen with the first or second cycle of CCs.

Statistical Analysis
SPSS (SPSS Inc, Chicago, Ill) was used for statistical analysis. Continuous variables were expressed as mean±SD, and discrete variables were presented as frequencies and percentages. The Wilk-Shapiro test and histogram analysis found all studied continuous variables to be normally distributed. Proportions were compared with the χ² test or Fisher exact test as appropriate. To analyze whether the durations of the intervals measured were different among studied groups, a linear mixed effects model using the swine number as a random subject effect and the occurrence of VF as a fixed effect was fitted to the following response variables: short, long, prematurity index, short/long, short/RR, mean blood pressure, QRS width, and metabolic parameters. Statistical significance was defined as a 2-tailed probability value <0.05. The discriminative ability of the short interval was assessed by calculating the area under the receiver operating characteristic (ROC) curve.

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agreed to the manuscript as written.

Results
In protocol A, 47 episodes of LDVF were induced in the 8 animals (Figure 1). In 12 episodes, ROSC was achieved without the need of CC; ROSC was achieved after the first minute of CPR in 22 episodes, after the second minute in 5 episodes, and after the third minute in 8 episodes. Of the 35 episodes of VF that required CCs after successful defibrillation, refibrillation was seen in 10. In 9 of these episodes (distributed among 4 animals), VF occurred within the first 2 CCs of the first minute of CPR (group 1; n=9). In the remaining 26 episodes, refibrillation was not seen during CCs but happened once before initiation of CCs (group 2). The 9 episodes included in group 1 were seen as early as the second induced episode of LDVF in 2 animals. However, because these 2 animals spontaneously achieved ROSC after the first LDVF episode, this second episode was the first episode that required CCs. In the other 2 animals, CC-triggered VF occurred after 4 and 5 episodes of LDVF.

The mean metabolic parameters before each episode of LDVF for groups 1 and 2 are depicted in Table 1. None of the parameters were statistically significantly different in a linear mixed effects model. In protocol A, CCs were started 21±14 seconds after successful shocks due to a lack of ROSC.

Ventricular capture was observed in all episodes of LDVF in all 3 animals in protocol B (Figures 2 and 3). Although ventricular capture by CCs was not always seen in a 1:1 ratio, it was observed for the first 2 CCs for all episodes in group 1 and for over 60% of the CCs during the first 10 CCs for all episodes in group 2. Ventricular capture was seen in all of the 11 episodes for which CCs were required for the
3 animals in protocol B. Examples of intermittent ventricular capture by CCs are shown for animals from protocols A and B in Figure 3.

As shown in Table 2, the short and long intervals were significantly shorter in group 1 (215±31 and 552±210 ms) than in group 2 (402±153 and 699±147 ms) (P=0.009 and P=0.04, respectively), and the prematurity index (short/RR) was significantly smaller in group 1 (0.35±0.09) than in group 2 (0.58±0.21) (P=0.006).

The discriminative ability of the short interval in predicting refibrillation with initiation of CCs was assessed, and the area under the ROC curve was 0.94±0.03. The ROC curve, sensitivities, specificities, and predictive values of different cutoffs for the short segment with regard to discriminating between initiation or no initiation of refibrillation with CCs are given in Figure 4. A short interval ≤231 ms predicted refibrillation with 88% sensitivity and 91% specificity. Eight of 9 episodes in group 1 and 1 of 23 episodes in group 2 had a short interval ≤231 ms (P=0.0001).

In protocol B, of 18 episodes of VF, CCs were required in 11. There were 3 cases of refibrillation initiating within the first 2 CCs in protocol B (Figure 5).

In group 1, a second and third minute of CCs were required after 3 episodes. In one episode, CCs were resumed without induction of VF, whereas with the other 2, it was seen after initiation of the second and third minute of compressions. In one of these episodes, refibrillation occurred during the first 2 CCs for both the second and third minute of CPR; in another episode, it occurred during the first 2 CCs in the second minute of CPR, whereas in the third minute, CCs were reinitated without immediate induction of VF, which was observed 42 seconds later (Figure 6).

**Discussion**

The major findings of this study are as follows. CCs can electrically stimulate the ventricles. The electric stimulation of the ventricles by CCs can induce VF. In the large majority of VF inductions, a ventricular capture caused by a CC was followed by a spontaneous ventricular activation that set the stage for a short cycle to occur when the next CC captured the ventricles. Thus, the mechanism of VF initiation was the creation of a long-short activation sequence. In our swine model, a short cycle less or >231 ms was the best predictor by ROC curve of whether VF occurred.

**Table 1. Baseline Metabolic Parameters Before Each LDVF Episode**

<table>
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Figure 3. Intermittent ventricular capture by CCs. Panel A shows ventricular depolarizations recorded by the MAP electrode and also seen on the surface ECG closely following CCs (arrowheads) for the first 6 cycles, whereas no stimulation is seen with the following 5 CCs. In panel B, intermittent capture is seen in an animal with no intracardiac electrodes during CCs (protocol B); the 2nd, 9th, 10th, and 11th CCs do not stimulate the ventricles, although capture is seen with the other compressions on the ECG and bipolar lead recording. The tracing labeled “Lucas Device” corresponds to each CC delivered, with the ascending limb of the tracing marking compressions. Both panels show 8 seconds of data.
Ventricular Capture With CCs
Cardiac stimulation by external mechanical forces was initially described by Zoll et al in 1976. It is now well described in different settings with a wide range of externally applied mechanical forces, from a precordial thump to chest-wall impact with baseballs (commotio cordis). However, we are unaware of previous reports of ventricular capture with the force generated by CCs. The amount of mechanical energy necessary to generate a detectable cardiac effect has not been well defined, although Zoll et al have proposed 0.04 to 0.7 joules to be necessary.

Precordial thumps have been used clinically to attempt cardioversion as well as to cause ventricular capture during asystole. Such use for resuscitation is now discouraged in the American Heart Association guidelines for CPR and emergency cardiovascular care. At least 2 cases of VF caused by thumps delivered during the vulnerable period of the cardiac cycle have been documented. Given the paucity of reports of VF provoked by precordial thumps, the exact incidence of this unique proarrhythmia is unknown.

Commotio cordis is the second leading cause of death in youth athletes in the United States and has been extensively studied. The importance of the velocity of the baseball and of the timing of the impact (upper and lower limits of vulnerability) in the generation of VF has been described in a swine model. The mechanism of ventricular capture by commotio cordis has been reported to be activation of mechano-sensitive ion channels.

In our study, ventricular capture by CCs was present in all animals and in all episodes examined. It was frequently seen in a 1:1 ratio, although periods of loss of capture were seen in all of the animals, both those with and those without catheters in the ventricles during CCs (Figure 3).

Refibrillation Initiated by CCs
Although precordial thumps and commotio cordis can cause arrhythmia by stimulating the ventricle during the vulnerable period, the clinical association between CCs and refibrillation has been investigated in only a few publications. In an abstract, Capucci et al reported “14 cases where VF was directly associated with CCs” of 135 consecutive cases of SCA with VF as the initial rhythm. The abstract did not document what type of association was seen or how it was defined. Hess and White retrospectively studied a similar population of patients with witnessed SCA and resuscitated by automatic external defibrillators and did not find a significant interaction between CCs and refibrillation. In the latter study, however, VF recurred during CCs in 16 of 32 patients and one of the figures illustrates VF onset closely after reinitiation of CCs.

In this study, we observed refibrillation after 10 episodes of LDVF. After one of the episodes, VF happened spontaneously before initiation of CCs. After 9 episodes of LDVF, refibrillation closely followed initiation of CCs. Analysis of the episodes of refibrillation associated with CCs (group 1) revealed long-short activation sequences with coupling intervals significantly shorter than those seen in the episodes in which CCs caused ventricular capture without initiating VF (group 2).

A long-short activation sequence is a well-recognized mechanism by which VF may be initiated. It is classically described in torsades de pointes, but the pattern also happens in other scenarios, such as pacing-induced arrhythmias. A recent retrospective analysis of electrograms of patients enrolled in the Pacing Reduces Shocks for Fast Ventricular Tachycardia II (PainFREE Rx II) and Clinical Investigation of the Medtronic EnTrust Implantable Cardioverter Defibrillator Trial (EnTrust) clinical trials emphasizes
the importance of long-short sequences for VT/VF initiation. In 8% to 15% of patients in that study, VT/VF might have been “initiated by long-short sequences that are actively facilitated by bradycardia pacing operation.”

The mechanism by which a long-short sequence initiates VF is thought to be that the abrupt changes in the ventricular cycle length lead to increased dispersion of repolarization and the short cycle causes stimulation of the myocardium during the vulnerable period, creating unidirectional conduction block necessary for the initiation of re-entry that degenerates into VF.

Our results suggest that during CPR, CCs can cause ventricular capture that can interact with a spontaneous activation to create a long-short sequence leading to VF by this mechanism.

**Mechanical CC Device**

It remains to be determined whether ventricular capture with CCs occurs with other types of CCs than those given by the mechanical CC device used in this study. The Lucas device is a commercially available pneumatic device that delivers CCs at a rate of ~100 bpm (575 ms between compressions), except between the first and second compressions, when the interval is ~960 ms. The proarrhythmic phenomenon described in this study may have been facilitated by this longer interval between the first and second compressions, which allowed time for the spontaneous depolarization that created the long-short pattern. However, VF was also seen in 1 episode during CCs at the faster cycle length of 575 ms 42 seconds after the initiation of CCs, with a similar long-short pattern (Figure 6). This example suggests that any type of CCs that causes ventricular capture could induce VF.

**Clinical Implications**

During resuscitation in humans, the incidence of refibrillation has been reported to be as high as 79%, and in one study, the number of VF episodes correlated with mortality. The same study documented a median time of 75 seconds from refibrillation to delivery of a shock for automatic external defibrillators and 43 seconds for manual defibrillators operated by paramedics. It is not known if a delay in shocking VF recurrences while CPR is being delivered is associated with worse outcomes. Another study, however, observed no association between the occurrence or the time to refibrillation and achievement of ROSC. The current American Heart Association guidelines for cardiopulmonary resuscitation emphasize CCs and recommend a 1-shock protocol with immediate resumption of CCs and CPR for 5 cycles of 30 CCs and 2 ventilations (~2 minutes) before brief interruption to recheck the rhythm or pulse. Although concerns that CCs during a postshock organized rhythm might cause refibrillation have been raised, this possibility has not been substantiated.

Even if, in humans, CCs cause ventricular capture and lead to a similar proarrhythmic mechanism as seen in our study, it is important to stress that our data do not indicate that CCs are detrimental. This study was not designed to analyze if CC-related refibrillation worsens outcome. If our findings

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**Figure 5.** Episode of refibrillation seen in an animal that did not have intracardiac electrodes during CCs (protocol B). Ventricular capture is seen with the first CCs, and VF arises after the second CC is delivered. The tracing labeled “Compression Device” corresponds to each CC delivered, with the ascending limb of the tracing marking compressions. Eight seconds of data are shown.

**Figure 6.** Refibrillation arising 42 seconds after initiation of chest compression. Eight seconds of data are shown.
translate into humans, it is possible that the prevention of ventricular capture during CCs or the timing of ventricular capture during CCs to prevent long-short sequences or a compression-on-T-wave phenomenon might be beneficial. It is even possible that the timing of ventricular capture by CCs could be controlled to increase cardiac output if the heart can muster any contractile force. However, both of these possibilities are speculative and require experimental testing.

It is also important to note that other mechanisms for defibrillation have been demonstrated in animal models. Reperfusion arrhythmias, the most well studied, can be reproduced even when ROSC is achieved by methods other than CCs.32 Our study, however, indicates yet another mechanism for refibrillation.

**Limitations**
This study was performed on pig hearts that were healthy before the induction of VF, limiting the clinical applicability of our findings to humans, because most individuals with SCA have preexisting heart disease. In this model, VF was electrically induced, limiting the generalization of our results. In animal models, resuscitation from ischemic VF is more difficult than in electrically induced VF, and refibrillation is more common.33 It is not known if CCs would yield similar results in an ischemic VF model. Furthermore, 2.5 minutes had elapsed after VF induction when defibrillation attempts started, a rather short duration when compared with clinical resuscitation studies.34,35 In humans, when defibrillation is performed for VF of such duration, a high survival rate is seen.4,36

CCs were delivered using a pneumatic device. CCs delivered manually or by other devices were not tested, so it is not known if our findings can be generalized to other types of compression.

The resuscitation protocol used in this study differs significantly from the guidelines put in force by the American Heart Association.18 In our study, CCs were initiated only if ROSC was not observed and interrupted after 1 minute for reassessment, a significant difference from the new guidelines’ emphasis on effective delivery of CCs while minimizing interruptions.

The primary limitation of protocol A, the possibility that ventricular stimulation could have been caused by intracavitory stimulation by the MAP catheters, was overcome by conducting protocol B with no catheters in the ventricles during CCs. Ventricular capture was also seen in this group of animals and, refibrillation within the first 2 CCs occurred in 3 episodes (Figure 5).

Although the spontaneous depolarization after the activation stimulated by the first CC might have represented electrophysiological instability of the myocardium and the initiation of VF, there is evidence that the activation stimulated by the second CC initiated the VF. In group 2, a spontaneous depolarization was also seen after the first compression, but the coupling interval was such that the following activation (stimulated by the second CC) produced a much longer “short cycle.” These spontaneous depolarizations were also never seen in salvos. Furthermore, careful analysis of all episodes in group 1 revealed that the beat delimiting the short cycle was always synchronous with the second CC delivered. Hence, although the mechanism of the spontaneously occurring beat remains to be elucidated, our study supports a mechanistic association between the long-short sequence and the onset of VF.

**Conclusion**
The mechanical force generated by CCs from a pneumatic device can stimulate the heart. Under certain conditions during resuscitation, ventricular stimulation produced by CCs can initiate VF by a long-short sequence.

**Acknowledgments**
We thank Frank L. Vance and Reubin L. Collins for assistance with the experimental preparation.

**Sources of Funding**
This work was supported by National Institute of Health grants HL 28429, HL 66256, and HL 85370.

**Disclosures**
None.

**References**


**CLINICAL PERSPECTIVE**

Sudden cardiac arrest is a leading cause of death in the United States. Survival rates are poor, although an improvement since the advent of automatic external defibrillators and emphasis on early defibrillation has been seen. During cardiopulmonary resuscitation, recurrence of ventricular fibrillation is common, but the true impact on mortality is unknown. The current American Heart Association guidelines for cardiopulmonary resuscitation emphasize chest compressions (CCs), and although concerns that CCs during a postshock organized rhythm might cause refibrillation have been raised, this possibility has not been substantiated. It is known that application of mechanical force to the chest can induce cardiac depolarization, and it is thought to be responsible for commotio cordis. The present study investigated episodes of refibrillation after long-duration ventricular fibrillation in an animal model. We show that after defibrillation, CCs can electrically stimulate the ventricles, and under certain conditions, induce ventricular fibrillation. Ventricular fibrillation was seen when cardiac stimulation from CCs resulted in long-short cardiac cycles, and a CC stimulated the heart during the “vulnerable period” of repolarization. If CC-induced refibrillation occurs in humans, strategies to optimize CCs after defibrillation that avoid a compression-on-T-wave phenomenon may reduce refibrillation during cardiopulmonary resuscitation.
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Circ Arrhythm Electrophysiol. 2008;1:282-289; originally published online September 13, 2008; doi: 10.1161/CIRCEP.108.767855
Circulation: Arrhythmia and Electrophysiology is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 1941-3149. Online ISSN: 1941-3084

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