Preshock phase singularity and defibrillation outcome: Another piece to solve the jigsaw puzzle?

Nipon Chattipakorn, MD, PhD

From the Cardiac Electrophysiology Unit, Department of Physiology, Cardiac Electrophysiology Research and Training Center, Faculty of Medicine, and Biomedical Engineering Center, Chiang Mai University, Chiang Mai, Thailand.

Sudden cardiac death is a major cause of death in most industrialized nations around the world, and ventricular fibrillation (VF) is known to be mainly responsible for this fatality.1 Despite the fact that VF has been known for over a century as a lethal culprit often perpetrating sudden cardiac death, electrical defibrillation is still the only known effective clinical therapy for this fatal arrhythmia.2 In the past few decades, the use of an implantable cardioverter-defibrillator has been shown to decrease the mortality rate significantly in patients with prior myocardial infarction and depressed ejection fraction.1 Together with the mortality reduction, our understanding on how the shock terminates VF has been much improved.3 This is partly due to the development of investigating tools such as multichannel electrical and optical cardiac mapping as well as cardiac computer simulations that allow investigators to study electrical activity in the heart in two or three dimensions, which mainly contributes to the advancement in the field of defibrillation.4–8 Despite the fact that many of the “defibrillation puzzle” pieces are in place, a few still remain elusive.3 Why some shocks fail and others of the same strength succeed in effecting defibrillation is not well understood.

Reentry is known to maintain VF and give rise to reinitiation of VF after a failed defibrillation attempt.5,9 When a shock fails to terminate VF, reentry has been seen to follow and be responsible for failed defibrillation.9 Since a phase singularity (PS) represents the existence of reentry, the number of PSs has been thought to be associated with defibrillation outcome.10,11

Influence of shock strength on postshock PS and defibrillation outcome

Influence of shock strength on defibrillation has been extensively investigated, and its important role in postshock activation patterns was suggested.3,10,11 Previous electrical mapping studies in dogs demonstrated that the earliest postshock activation appeared early (i.e., short isoelectric window) after weak shocks (well below the defibrillation threshold) and were associated with failed defibrillation, whereas strong defibrillation shocks had a long isoelectric window after the shock and were generally associated with successful defibrillation.4,5 When shocks at a strength near the defibrillation threshold were used and failed to defibrillate, repetitive responses were observed before degenerating into VF.7,12–15 In contrast, most optical mapping studies in isolated, perfused rabbit hearts reported that there was no isoelectric window after defibrillation shocks and that reentry was responsible for failed defibrillation.6,16,17 The discrepancy among these findings has been proposed as largely due to different shock strengths used among studies, since the shock strength used in those optical mapping studies was frequently much weaker than the defibrillation threshold.3,15

The influence of shock strengths on activation patterns after defibrillation shocks has been strongly emphasized and supported by recent optical mapping reports in isolated pig hearts using shocks at various strengths from 100 to 900 V.10,11 In that report,10 it was demonstrated that (1) postshock interval or an isoelectric window did not exist at low strength shocks but existed and monotonically increased as shock strength increased; (2) the number of postshock PSs progressively decreased (and reached zero) as defibrillation shock strength increased, whereas the number of preshock PSs during VF did not differ among those shocks; and (3) low-strength shocks were not able to defibrillate as the number of postshock PSs was still high and reentry was observed as a postshock activation pattern for failed defibrillation. In that study, however, defibrillation only reached a 100% success rate at 900 V, even though postshock PSs were no longer observed from 600 to 900 V and only a focal pattern of epicardial activation was observed in failed defibrillation.10 These findings indicate that shock strengths play a major role in determining the number of postshock PSs and postshock activation patterns; however, the number of postshock PSs did not critically determine defibrillation outcome.
Postshock activation patterns and defibrillation outcome for near-\text{DFT}_{50} shocks

Although a number of studies have been carried out to investigate defibrillation mechanisms over the past decades, there are only a few that used a shock strength of around 50\% defibrillation success (\text{DFT}_{50}) to investigate the relationship between myocardial responses to the shock and defibrillation outcome.\textsuperscript{7,12–14} Since the nature of defibrillation is probabilistic and shock-strength dependent, investigators have been trying to find the determining factors by which \text{DFT}_{50} shocks of the same strength sometimes succeed while at other times they fail to defibrillate. Electrical and optical cardiac mapping studies in pigs have demonstrated that when the strength of the shock was kept constant at \text{DFT}_{50}, three types of defibrillation outcome, that is, successful defibrillation type A or type 1 (immediate resumption of sinus rhythm); successful type B or type 2 (a few repetitive responses before sinus rhythm); and failed defibrillation, could be observed.\textsuperscript{7,12} At this \text{DFT}_{50}, repetitive responses similar to type B successful shocks were often observed after failed defibrillation.

In electrical mapping studies, investigated parameters immediately after the shock indicated no differences between type B success and failed defibrillation and that the first postshock activation between the two was almost indistinguishable, whereas successive cycles demonstrated divergent patterns.\textsuperscript{7,13} In failed defibrillation episodes, the intercycle interval (i.e., an interval between onsets of successive cycles) was found to be shorter, whereas the wavefront conduction time (i.e., time that each cycle takes to traverse the heart) was longer for the first five successive cycles after the shock, when compared with those in type B success. All of these findings suggest that electrophysiological variables immediately after the shock might not be a crucial factor in determining the outcome of defibrillation. Instead, the number and rapidity of postshock cycles could be determining factors of defibrillation outcome for near-\text{DFT}_{50} shocks. However, electrical state at shock onset was not investigated in those studies.

In an optical mapping study using near-\text{DFT}_{50} shocks, similar findings to previous electrical mapping results were demonstrated.\textsuperscript{12} However, the electrical state of the heart at the time of the shock was different between type B success and failed defibrillation. These findings indicated that the phase of VF action potentials at shock onset is crucial in determining defibrillation outcome. However, immediate myocardial responses to the shock (i.e., depolarization pattern of the first postshock cycle) were no different between type B success and failed defibrillation. These findings, again, could be interpreted to mean that the number and rapidity of postshock activations rather than cardiac state at the time of the shock may be important factors in determining defibrillation outcome at \text{DFT}_{50} shocks. In those studies, however, type A success episodes were often excluded from data analysis and the preshock PS was never investigated for its role in determining defibrillation outcome.

Preshock PS and defibrillation outcome for near-\text{DFT}_{50} shocks

In this issue of Heart Rhythm, Hayashi et al\textsuperscript{18} reported for the first time an important finding concerning the relationship between preshock PS and defibrillation outcome at near-\text{DFT}_{50} shocks. They studied the process of ventricular defibrillation in isolated, perfused rabbit hearts using an optical mapping system that provides an elegant method to dynamically monitor electrophysiological activation and recovery of a significant portion of the ventricular heart muscle. They found that when shocks near the DFT were applied either early (10 seconds) or late (1 minutes) after VF was induced, the number of PSs present just before shocks was related to postshock activation dynamics. Their major findings that a low number of preshock PSs were associated with type A success reemphasize the importance of electrical states of myocardium at the time of the shock when determining defibrillation outcome. However, because of the complex nature of defibrillation, Hayashi et al found that the number of preshock PSs was no different between failed and type B successful defibrillation. This suggested that there are possibly parameters other than cardiac state at shock onset that determined defibrillation outcome for type B success and failed defibrillation.

In summary, the article by Hayashi et al\textsuperscript{18} has helped to explain the discrepancy in previous findings in terms of shock strength influence, activation patterns and electrical state of myocardium at the time of the shock, and defibrillation outcome. Do the findings of the Hayashi et al study add a piece to the defibrillation puzzle? In a word, yes. Their results solidify further the notion that electrophysiological activity just before the shock importantly influences the postshock activation dynamics and defibrillation outcome. Their findings suggest the possibility that these dynamics could be actively and reliably controlled in specific regions of the heart,\textsuperscript{19–21} significant and clinically important reductions in DFT might be achieved.

References


