Left Ventricular Apex Ablation Decreases the Upper Limit of Vulnerability

Nipon Chattipakorn, MD, PhD; Parwis C. Fotuhi, MD; Xiangsheng Zheng, MD; Raymond E. Ideker, MD, PhD

Background—After shocks with an \( \approx \) 50% probability of success for the upper limit of vulnerability (ULV\(_{50}\)) of strength, the first few activations appear focally on the epicardium at almost the same site at the left ventricular (LV) apex in both successful and failed induction of ventricular fibrillation (VF). We tested the hypothesis that subendocardial ablation at this early site would decrease the shock strength required for the ULV\(_{50}\).

Methods and Results—Ten S1 stimuli were delivered from the right ventricular apex at a 300-ms coupling interval in 5 pigs. Biphasic shocks were delivered from right ventricular–superior vena cava electrodes after the last S1 stimulus. The ULV\(_{50}\) was determined using an up/down protocol with T-wave scanning. Radiofrequency ablation was performed endocardially at the apical LV. The ULV\(_{50}\) was determined again 30 minutes after ablation. To determine the importance of the ablation region, this protocol was repeated in another 5 pigs with ablation at the LV base. Delivered voltage (401 ± 60 versus 323 ± 50 V) and energy (11 ± 3 versus 7 ± 2 J) for the ULV\(_{50}\) were significantly decreased after LV apex ablation by 19% and 34%, respectively. However, no difference existed in ULV\(_{50}\) before and after LV base ablation. Lesions at both the LV apex and base were subendocardial and ranged from 0.8 to 1.1 cm in diameter.

Conclusions—Subendocardial ablation at the apical LV markedly decreases ULV\(_{50}\), which suggests that the activation originating from this postshock early site is responsible for VF initiation and that interventions to electrically silence this site can influence the outcome of VF induction by ULV shocks. (Circulation. 2000;101:2458-2460.)

Key Words: ablation ■ fibrillation ■ shock

Studies on how ventricular fibrillation (VF) occurs and how a shock halts VF have been performed for decades.\(^2\) Knowledge of VF induction and defibrillation has greatly increased with the advent of electrical and optical systems that can map activation simultaneously from hundreds of sites.\(^3,4\) Several studies suggest that VF induction by shocks during the vulnerable period occurs by the same mechanism that causes VF to recur after shocks that fail to defibrillate.\(^3,5\)

Because VF induction is probabilistic and dependent on shock strength and timing, we recently compared the activation sequences after shocks of the same strength delivered at the same time during the vulnerable period that induced VF 50% of the time, ie, those that had a 50% probability of success for the upper limit of vulnerability (ULV\(_{50}\)). The activation pattern for the first few postshock cycles after shock-induced VF episodes did not differ from that of episodes in which VF was not induced.\(^6,7\) These activations first and repeatedly appeared on the epicardium from the same site at the left ventricular (LV) apex, after which they either died out in episodes in which VF was not induced or arose progressively faster and degenerated into fibrillation in VF episodes.\(^5,7\) These results suggest that this small arrhythmogenic region at the LV apex, where the shock potential gradient is weak for the right ventricular (RV) apex and superior vena cava shocking electrode configuration we used,\(^8\) is important in determining shock outcome. To test this hypothesis, we performed radiofrequency ablation at this region in an attempt to silence this arrhythmogenic source. We tested the hypothesis that the ULV\(_{50}\) shock strength is decreased after ablation at the LV apex. To determine the importance of the ablation region, we also measured ULV\(_{50}\) before and after ablation at the LV base in another group of animals as a control.

Methods

Animal Preparation

Ten pigs (25 to 30 kg) of either sex were studied. The animals were cared for according to the Guide for Care and Use of Laboratory Animals. Animals were anesthetized and monitored and their chest was opened as described previously.\(^6\)

S2 shocks were biphasic, truncated, exponential waveforms (Ventritex Corp) and were delivered from electrodes on 2 catheters (Guidant Corp). A 34-mm electrode catheter was inserted into the RV apex (cathodal first phase), and a 68-mm electrode catheter was positioned at the superior vena cava–right atrial junction (anodal first phase). Delivered voltage and current were displayed, and total delivered energy was calculated by a waveform analyzer (DATA 6100, Analogic Inc). S1 stimuli were constant current, 5-ms, monophasic pulses delivered from the catheter tip in the RV apex.
ULV Determination

Initially, the intrinsic R-R interval and the pacing threshold for S1 were measured. The beginning, peak, and end of the T-wave were identified with an oscilloscope from limb lead II, as described previously. Ten S1 stimuli were delivered 3 times, and the average coupling interval (CI) between the last S1 and the beginning, peak, and end of the T-wave were determined. These intervals were recalculated after every 5 VF episodes.

Ten S1 stimuli at 5 to 10 times the pacing threshold were delivered at an interval of 300 ms. Shock leading edge voltage was initially 500 V. The first shock was delivered at the peak of the T-wave. Subsequent S1-S2 CIs were set to scan the T-wave in 10-ms steps, as described previously. Shock strength was adjusted using a modified up/down protocol. Successive shocks were separated by 15 s. The lowest shock strength that did not induce VF at any CI was defined as the ULV.

Ten shocks 10 V below the ULV were delivered using the same S1-S2 CI that last induced VF during ULV determination. When the shock induced VF, a rescue shock of 20 to 30 J was delivered, and the next S2 shock was not given for 4 minutes. If the number of VF episodes induced by the 10 shocks was not in the range of 4 to 6, the protocol was repeated with the shock strength slightly altered. Thus, the S2 shock strength of the last group was ~ULV50. ULV10 was determined before and 30 minutes after ablation was performed.

Ablation Protocol

A metal pin was inserted from the epicardium to mark the site near the LV apex where the earliest postshock activation occurred, as observed in our previous study. A 7-French ablation catheter with a 4-mm tip electrode (EP Technologies, Inc) was advanced to this pin under fluoroscopic guidance. Radiofrequency energy was delivered for 30 to 40 s while temperature was maintained at 70°C to 80°C (monitored with a thermocouple embedded in the electrode tip). After each ablation, the catheter was moved slightly, and ablation was repeated to make a lesion neighboring the previous lesion. This process was repeated 7 times in an attempt to create a single focal lesion ~1 cm in diameter. To determine the importance of the ablation region, the ablation protocol was performed at the LV base in another 5 pigs to create a lesion similar to that created in the apex.

Animals were euthanized by KCl injection into the heart at the end of the study. The heart was removed, rinsed, and stored in formalin for >10 hours. The maximal diameter of the lesion on the endocardium and its intramural width and depth were measured.

Analyses

Differences in the ULV50 before and after ablation and in the lesion size at the LV apex and base were analyzed with a paired t test. Significance was identified at P≤0.05. All values are mean±SD.

Results

Heart weight was 184±35 g. ULV50 delivered voltage, current, and energy were significantly decreased (P<0.001) after ablation at the LV apex. Delivered voltage decreased by 19% (401±60 versus 323±50 V) and total energy by 34% (11±3 versus 7±2 J) (Figure 1A). The current decreased from 8±1 to 7±1 A, whereas impedance (52±4 versus 53±4 Ω) did not change. ULV50 before ablation always became ULV10 (ie, a shock strength that never induced VF when delivered during the T-wave) after ablation. Table 1 shows the delivered voltage for ULV50 in each animal with ablation at the LV apex; these data confirm that the large mean differences in the ULV50 before and after ablation were not due to a disproportionate effect of a minority of animals.

ULV50 delivered voltage (421±50 versus 428±56 V), total energy (11±2 versus 11±1 J), and current (7±1 versus 7±1 A) before and after the LV base ablation were not significantly different (Figure 1B). Impedance was also constant (63±8 versus 64±7 Ω).

A single consolidated lesion <1 cm³ was always present subendocardially at the apex (Figure 2A) and base (Figure 2B). Maximal diameters of the lesion at the LV apex and base were not significantly different (Table 2).

Discussion

The major finding of this study was that subendocardial ablation at the LV apex of <1% of the heart mass significantly decreased the ULV50. After shocks with the same lead configuration near the ULV50 or near the defibrillation threshold, the first few postshock activations almost always first appeared on the epicardium overlying this site. In the present study, subendocardial ablation was performed at this site in an attempt to remove the source of postshock activation. Our results indicate that this intervention affects this arrhythmogenic site, resulting in a decrease in the ULV. The lack of change in the ULV with ablation of the LV base indicates that the lowering of the ULV is not a generalized effect of ablation in the minority of animals.

| TABLE 1. Delivered Voltage Before and After Ablation at LV Apex |
|-----------------|----------------|----------------|
| Animal Number  | Before Ablation, V | After Ablation, V |
| 1               | 418±2          | 346±3          |
| 2               | 401±2          | 346±3          |
| 3               | 476±3          | 344±2          |
| 4               | 406±2          | 368±4          |
| 5               | 310±3          | 237±2          |
response to ablation but is specific for ablating and, hence, electrically silencing the site of earliest postshock activation. These results are consistent with the critical point hypothesis,11 which states that to not induce VF, a shock must be strong enough so that the entire myocardium must be exposed to a potential gradient field at or above the critical value. Thus, ablating a portion of the tissue where the shock field is weak could have decreased the ULV by allowing a shock field below the critical value to exist in the necrosed tissues without inducing an arrhythmia there.

Although the source of the postshock activations is not known, it has been postulated that Purkinje fibers are one possible source.12 Subendocardial ablation could interrupt these fibers, preventing VF induction and lowering the ULV. It is also possible that ablation interrupts an intramural reentrant circuit preventing VF. Further investigation is needed to explore the source of postshock activation. Regardless of the cause of this postshock activation, this study strongly indicates the importance of the small region giving rise to this activation for VF induction.

**TABLE 2. Lesion Size**

<table>
<thead>
<tr>
<th>Ablation Site</th>
<th>Surface, mm</th>
<th>Width, mm</th>
<th>Depth, mm</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV apex</td>
<td>8±3</td>
<td>11±3</td>
<td>10±3</td>
</tr>
<tr>
<td>LV base</td>
<td>9±3</td>
<td>10±3</td>
<td>10±2</td>
</tr>
</tbody>
</table>

**Limitations**

1. The study was performed in normal pig hearts; thus, the results could differ in diseased hearts and/or in other species.
2. The results are likely specific to the defibrillation lead system used. Therefore, the ablation site may be in other locations for other lead configurations.
3. Although some data suggest the postshock early site at the ULV is not greatly altered by changing the S1 pacing site as long as the S2 electrodes remain the same,13 the results may differ with different S1 pacing sites.
4. ULV correlates well with the defibrillation threshold in the absence of ablation lesions14; however, the effects of ablation on defibrillation could differ from those on ULV.
5. Although it is unlikely that ablation of the functioning myocardium would be performed clinically to reduce the defibrillation threshold, the results of this study suggest that the location of a myocardial scar might be used to help determine the optimal defibrillation lead position.

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**References**

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