Epicardial Radiofrequency Ablation Failure During Ablation Procedures for Ventricular Arrhythmias

Reasons and Implications for Outcomes

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Background—Radiofrequency ablation (RFA) from the epicardial space for ventricular arrhythmias is limited or impossible in some cases. Reasons for epicardial ablation failure and the effect on outcome have not been systematically analyzed.

Methods and Results—We assessed reasons for epicardial RFA failure relative to the anatomic target area and the type of heart disease and assessed the effect of failed epicardial RFA on outcome after ablation procedures for ventricular arrhythmias in a large single-center cohort. Epicardial access was attempted during 309 ablation procedures in 277 patients and was achieved in 291 procedures (94%). Unlimited ablation in an identified target region could be performed in 181 cases (59%), limited ablation was possible in 22 cases (7%), and epicardial ablation was deemed not feasible in 88 cases (28%). Reasons for failed or limited ablation were unsuccessful epicardial access (6%), failure to identify an epicardial target (15%), proximity to a coronary artery (13%), proximity to the phrenic nerve (6%), and complications (<1%). Epicardial RFA was impeded in the majority of cases targeting the left ventricular summit region. Acute complications occurred in 9%. The risk for acute ablation failure was 8.3× higher (4.5–15.0; P<0.001) after no or limited epicardial RFA compared with unlimited RFA, and patients with unlimited epicardial RFA had better recurrence-free survival rates (P<0.001).

Conclusions—Epicardial RFA for ventricular arrhythmias is often limited even when pericardial access is successful. Variability of success is dependent on the target area, and the presence of factors limiting ablation is associated with worse outcomes. (Circ Arrhythm Electrophysiol. 2015;8:1422-1432. DOI: 10.1161/CIRCEP.115.003202.)

Key Words: catheter ablation • complications • epicardium • phrenic nerve • survival rate • tachycardia, ventricular • ventricular premature complexes

Radiofrequency ablation (RFA) is an effective treatment for drug-refractory ventricular arrhythmias (VAs), but endocardial catheter ablation of postinfarction ventricular tachycardia (VT) fails in 20% to 50% of patients. Epicardial substrate is frequently identified in ischemic and especially in nonischemic cardiomyopathies (NICMs). Therefore, epicardial RFA using percutaneous access or a subxiphoid surgical pericardial window has played an increasing role in electrophysiological procedures. However, epicardial access may be challenging when pericardial adhesions are present, and even after successful access, epicardial RFA may be limited or impossible because of the lack of epicardial substrate or proximity to vulnerable anatomic structures. The purpose of the study was to characterize the reasons for epicardial ablation failure after successful epicardial access, to relate these to the targeted epicardial area, and to assess implications of failed or limited epicardial RFA on outcome for VA ablation procedures.

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scanned to refractoriness or a minimum coupling interval of 180 ms, after a basic drive of 600 and 400 ms plus burst pacing was performed preferentially from 2 sites for patients with a history of sustained monomorphic VT (SMVT). SMVT was defined as continuous monomorphic VT for ≥30 s or one that required an intervention for termination (cardioversion, pacing, or ablation). If no SMVT was inducible, programmed stimulation was repeated during intravenous infusion of isoproterenol or epinephrine. If no SMVT was reliably inducible, nonsustained VT or premature ventricular contractions (PVCs) felt likely to be originating from the SMVT region were targeted.

We defined a clinical VT as matching a spontaneous VT for 12-lead ECG morphology and rate (within 20 ms). If 12-lead ECGs of the presenting VT were not available before ablation, induced VTs that were similar in the rate and intracardiac electrogram characteristics to a spontaneous VT detected by an implanted cardioverter-defibrillator were designated presumptive clinical VTs. Undocumented VTs were defined as inducible VTs that did not match spontaneous VTs by the above criteria.1 For PVC ablations, intravenous isoproterenol or epinephrine was given if spontaneous PVCs were absent. At the end of the procedure, the same stimulation protocol was repeated if feasible for all VAs.

Epicardial access was generally attempted if VA was suspected to be of epicardial origin based on findings from previous ablation procedures, QRS morphology and type of heart disease, endocardial mapping, or unsuccessful endocardial ablation and could be performed at the same or a subsequent procedure at the discretion of the treating physician. Epicardial access was attempted using the percutaneous approach as described in the Methods section in the Data Supplement or the surgical window approach as previously reported.9 Coronary angiography was performed before epicardial ablation to assess proximity to coronary arteries except for some cases targeting the right ventricular (RV) free wall. High output pacing (10 mA at 2-ms pulse width) from the ablation catheter was performed to assess proximity to the phrenic nerve.

Table 1. Baseline Characteristics of Patients (n=277)

<table>
<thead>
<tr>
<th>Patient characteristics</th>
<th>No Structural Heart Disease (n=44)</th>
<th>CAD (n=73)</th>
<th>NICM Other Than ARVC (n=125)</th>
<th>ARVC (n=35)</th>
<th>All (n=277)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>46 (38–55)</td>
<td>66 (59–72)</td>
<td>57 (47–67)</td>
<td>47 (35–53)</td>
<td>57 (46–67)</td>
</tr>
<tr>
<td>Male sex</td>
<td>24 (55)</td>
<td>68 (93)</td>
<td>198 (78)</td>
<td>30 (86)</td>
<td>220 (79)</td>
</tr>
<tr>
<td>ICD</td>
<td>11 (25)</td>
<td>69 (95)</td>
<td>112 (90)</td>
<td>31 (89)</td>
<td>223 (81)</td>
</tr>
<tr>
<td>CRT</td>
<td>0</td>
<td>18 (25)</td>
<td>26 (21)</td>
<td>1 (3)</td>
<td>47 (17)</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>60 (50–65)</td>
<td>30 (20–39)</td>
<td>35 (25–45)</td>
<td>55 (47–63)</td>
<td>40 (25–55)</td>
</tr>
<tr>
<td>LVEDD, mm</td>
<td>49 (45–55)</td>
<td>62 (58–68)</td>
<td>59 (53–65)</td>
<td>48 (45–53)</td>
<td>57 (50–64)</td>
</tr>
<tr>
<td>SMVT</td>
<td>27 (61)</td>
<td>70 (96)</td>
<td>119 (95)</td>
<td>34 (97)</td>
<td>250 (90)</td>
</tr>
<tr>
<td>VT storm</td>
<td>0</td>
<td>12 (16)</td>
<td>21 (17)</td>
<td>1 (3)</td>
<td>37 (13)</td>
</tr>
<tr>
<td>Incessant VT</td>
<td>1 (2)</td>
<td>4 (6)</td>
<td>4 (3)</td>
<td>1 (3)</td>
<td>10 (4)</td>
</tr>
<tr>
<td>PVC/NSVT</td>
<td>17 (39)</td>
<td>3 (4)</td>
<td>6 (5)</td>
<td>1 (3)</td>
<td>27 (10)</td>
</tr>
<tr>
<td>PVC-induced VF</td>
<td>1 (2)</td>
<td>0</td>
<td>2 (2)</td>
<td>1 (3)</td>
<td>4 (1)</td>
</tr>
<tr>
<td>Total number of failed AAD (classes I and III)</td>
<td>1 (0–2)</td>
<td>2 (1–3)</td>
<td>2 (1–3)</td>
<td>2 (1–3)</td>
<td>2 (1–3)</td>
</tr>
<tr>
<td>Previous heart surgery</td>
<td>0</td>
<td>18 (25)</td>
<td>4 (3)</td>
<td>0</td>
<td>22 (8)</td>
</tr>
<tr>
<td>Patients with prior VA ablations</td>
<td>37 (84)</td>
<td>65 (89)</td>
<td>78 (62)</td>
<td>26 (74)</td>
<td>206 (74)</td>
</tr>
<tr>
<td>No. of previous VA ablations</td>
<td>1 (1–3)</td>
<td>2 (1–2)</td>
<td>1 (1–2)</td>
<td>2 (1–2)</td>
<td>1 (1–2)</td>
</tr>
</tbody>
</table>

Values are presented as median (25th to 75th interquartile range) or as n (%). AAD indicates antiarrhythmic drug; ARVC, arrhythmogenic right ventricular cardiomyopathy; CAD, coronary artery disease; CRT, cardiac resynchronization therapy; ICD, implantable cardioverter defibrillator; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; NICM, nonischemic cardiomyopathy; NSVT, nonsustained ventricular tachycardia; PVC, premature ventricular contraction; SMVT, sustained monomorphic ventricular tachycardia; and VA, ventricular arrhythmia.
For scar-related VA, quantitative assessment of local voltage was performed with particular focus on the low-amplitude scar region using an electroanatomic mapping system (CARTO3 or XP, Biosense Webster, Diamond Bar, CA) and a 3.5-mm- or 4-mm-tip open-irrigated ablation catheter (NaviStar, NaviStar ThermoCool, ThermoCool SF or ThermoCool Smart-Touch; Biosense Webster), a multipolar, multispline catheter (Pentaray NAV; Biosense Webster), or a decapolar catheter (DECANA V; Biosense Webster). Intracardiac echocardiography was used for anatomic reconstructions when available (64-element, 5.5–10.0 MHz, SoundStar; Biosense Webster).

Areas of low bipolar voltage ($\leq 1.5$ mV), dense scar ($\leq 0.5$ mV), and electrically unexcitable scar (pacing threshold, $>10$ mA at 2-ms pulse width) were identified. After creation of the voltage map, VT was reinitiated with programmed stimulation and entrainment maneuvers were performed at selected sites. Ablation was performed at putative isthmus sites. If the induced VT was hemodynamically unstable, it was terminated with RFA, rapid pacing, or cardioversion, and substrate modification of the scar was then performed targeting regions of slow conduction evident as late or fractionated potentials or stimulus-to-QRS delays $>40$ ms during pace mapping where the paced QRS morphology matched the QRS morphology of an induced VT. Activation and pace mapping were performed for PVC ablation procedures. Irrigated radiofrequency energy was delivered at a power of 25 to 50 W targeting an impedance drop of 10 to 20 $\Omega$. Applications were repeated at target areas

Figure 1. Types of heart disease in all 277 patients. ARVC indicates arrhythmogenic right ventricular cardiomyopathy; and NICM, nonischemic cardiomyopathy.

Figure 2. Epicardial target areas after successful access relative to the type of the disease. ARVC indicates arrhythmogenic right ventricular cardiomyopathy; CAD, coronary artery disease; CS, coronary sinus; LAD, left anterior descending coronary artery; LV, left ventricle; LVOT, left ventricular outflow tract; NICM, nonischemic cardiomyopathy; RCA, right coronary artery; RV, right ventricle; and RVOT, right ventricular outflow tract.
with the aim of rendering the area electrically unexcitable to unipolar pacing (10 mA at 2-ms pulse width).19

**Epicardial Target Area**
For this study, the epicardial cardiac surface was segmented into 9 areas: (1) left ventricular (LV) summit,20 LV outflow tract, and RV outflow tract, other than free wall; (2) basal lateral LV; (3) basal inferior LV; (4) mid anterior LV; (5) mid lateral LV; (6) mid inferior LV; (7) LV apex; (8) RV and RV outflow tract free wall; and (9) inferior RV.

For cases where epicardial radiofrequency has been applied, the epicardial target area was defined as the segment that was predominately targeted with radiofrequency. For cases where no radiofrequency was applied, the target area was defined as the segment that was suspected to contain the VT origin based on mapping data. An epicardial target area was only defined, when access was successful.

**Reasons for Epicardial Ablation Failure**
If no or limited epicardial RFA was performed after successful epicardial access, reasons were classified as follows:

1. Failure to identify an epicardial target (no epicardial substrate present; inability to capture with high output pacing [10 mA and 9-ms pulse width] at the desired target indicating possible thick underlying fat; local electric activity after the surface QRS complex or later than endocardial activity during activation mapping of focal arrhythmias; and target site not part of the reentrant circuit during entrainment mapping) for reentrant arrhythmias when it could be assessed.
2. Proximity to a coronary artery prohibits RFA (within 5 mm as seen during coronary angiography).17
3. Proximity to the left phrenic nerve prohibits RFA (left phrenic nerve capture when pacing from the ablation catheter).

**Acute Outcome**
Acute outcomes for VT ablations are reported as follows:

1. Noninducibility of any SMVT
2. Abolishment of at least 1 initially inducible clinical or presumptive clinical SMVT
3. Clinical or presumptive clinical SMVTs still inducible (ablation failure)
4. No reinduction test performed after RFA.

Acute outcomes for PVC ablation procedures are reported as follows:

1. Abolishment of all spontaneous PVCs
2. Spontaneous PVCs still present but less frequent
3. No effect on spontaneous PVCs (ablation failure)

Acute ablation failure for all VAs was defined as the persistent presence or inducibility of all spontaneous VTs or PVCs after RFA.

**Assessment of Recurrence and Mortality**
For VT ablation procedures, recurrence was defined as any VT resulting in implanted cardioverter-defibrillator shock, rehospitalization, or repeat ablation during the index hospitalization. For PVC ablation procedures, recurrence was defined as recurrence of any PVC requiring treatment for symptom control. Mortality was assessed from the Social Security Death Index. Referring cardiologists and primary care physicians were contacted for clinical follow-up if necessary.

**Statistical Analysis**
Procedural characteristics, acute success, and complications are reported on a per-procedure basis, whereas clinical outcomes were analyzed on a per-patient basis. In cases where patients required repeat ablation, only the first ablation procedure was included in the

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**Table 2. Procedural Characteristics (n=309)**

<table>
<thead>
<tr>
<th>Type of intervention</th>
<th>No RFA</th>
<th>Only endocardial RFA</th>
<th>Only epicardial RFA</th>
<th>Endocardial and epicardial RFA</th>
<th>Transcoronary ethanol ablation</th>
<th>Epicardial cryoablation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>15 (5)</td>
<td>91 (30)</td>
<td>63 (20)</td>
<td>140 (45)</td>
<td>4 (1)</td>
<td>2 (&lt;1)</td>
</tr>
</tbody>
</table>

**Epicardial access**

<table>
<thead>
<tr>
<th>Reason for epicardial access</th>
<th>Epicardial access after endocardial mapping</th>
<th>Epicardial access first</th>
<th>Based on the findings from previous procedures</th>
<th>Based on morphology/type of heart disease</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>208 (67)</td>
<td>101 (33)</td>
<td>69 (23)</td>
<td>28 (9)</td>
<td>4 (1)</td>
</tr>
</tbody>
</table>

**Reason for no/limited epicardial RFA**

<table>
<thead>
<tr>
<th>Reason for no/limited epicardial RFA</th>
<th>Unlimited</th>
<th>Limited</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>181 (59)</td>
<td>22 (7)</td>
<td>106 (34)</td>
</tr>
</tbody>
</table>

**Analysis for clinical outcome. Continuous variables are presented as median (25th to 75th interquartile range) or as n (%). PVC indicates premature ventricular contraction; RFA, radiofrequency ablation; and VT, ventricular tachycardia.**
value of <0.05 was considered statistically significant. Data analysis was performed using IBM SPSS Statistics for Mac, Version 22.0. Armonk, NY.

Results

Baseline and Procedural Characteristics

Epicardial access was attempted during 309 procedures in 277 patients (79% men; median age, 57 [46–67] years). Ablation was performed for VT in 279 (90%) and PVC in 30 cases (10%). Patient characteristics and types of heart disease are summarized in Table 1 and Figure 1.

Figure 2 shows the epicardial target area in different types of heart disease for cases with successful epicardial access (n=291). The outflow tracts were predominant targets in patients with no structural heart disease or arrhythmogenic RV cardiomyopathy (ARVC). The basal, perimitral areas were targeted most frequently in other NICMs, whereas the LV midwall and apex were the major targets in patients with coronary artery disease (CAD; exact numbers are provided in Table I in the Data Supplement).

Epicardial RFA was applied in 203 (65%) procedures. Procedural characteristics are summarized in Table 2. Of the 140 cases where endocardial and epicardial radiofrequency had been applied, radiofrequency durations of individual applications were only available for 64 cases performed after 2010.

During 4 cases (1%), transcoronary ethanol ablation and during another 2 cases, (<1%) epicardial cryoablation were performed.

Factors Limiting Epicardial RFA

Epicardial access was successful in 291 procedures (94%). Unlimited RFA at the target area could be performed in 181 cases (59%). Limited RFA was possible in 22 cases (7%), whereas in 106 cases (34%), RFA was not felt to be possible.

Reasons for limited or no epicardial RFA were unsuccessful access in 18 (6%), failure to identify an epicardial target for ablation in 48 (15%), proximity to a coronary artery in 40 (13%), proximity to the phrenic nerve in 19 (6%), and a complication prohibiting further RFA in 3 (1%) of all procedures. The frequency of limited and no RFA and the limiting factors at different target areas after successful access are shown in Figure 3 (numbers are provided in Table I in the Data Supplement), and limitations dependent on types of heart disease are shown in Figure 4.

Epicardial ablation was deemed not feasible in the vast majority of cases where the VA origin was in the LV summit region, mainly because of proximity to a coronary artery.

Figure 3. The figure shows how often radiofrequency ablation (RFA) was limited or deemed not feasible at different epicardial target areas. The number of procedures for each area is provided in the label for the y axis. The pie charts summarize the limitations for all cases (total numbers in brackets) where no or limited RFA has been applied at target areas. LV indicates left ventricle; RV, right ventricle; and RVOT, right ventricular outflow tract.
unlimited epicardial ablation could be performed. Among the 25 patients who had limited or no epicardial RFA, however, acute ablation failure was seen in 18 (72%) patients. The risk of ablation failure was 10.3× higher (1.0–109.0; \( P=0.05 \)) after no or limited epicardial ablation compared with unlimited ablation.

Patients were followed up for a median time of 15 months (interquartile range, 2–45). The combined end point of VT recurrence or death occurred in 69 of 158 patients (44%) with unlimited ablation, compared with 73 of 119 (61%) with no or limited ablation. Kaplan–Meier analysis demonstrated an early divergence between the 2 curves as shown in Figure 8 (\( P=0.001 \)). There was no difference in overall mortality between the 2 groups (\( P=0.285 \)). Subgroup analysis is summarized in Table 4 and Figure I in the Data Supplement. Only 2 patients with ARVC had limited or no ablation, precluding statistical assessment for this group. No patient without structural heart disease died during follow-up. For patients with LV-scar related VT, the combined end point of recurrence or death occurred in 27 of 47 (57%) patients from the cohort with a target area protected by an anatomic structure compared with 17 of 34 (50%) when no epicardial target could be identified and 55 of 108 (51%) of those with targets that could receive unlimited ablation.

**Discussion**

In this single-center study, we report procedural characteristics and outcome of 309 procedures for VAs that included an epicardial access attempt. It is well known that epicardial access is sometimes challenging, especially after previous cardiac surgery and alternatives to the subxyphoid approach have been described.9,23,24 Even after successful epicardial

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*(example is in given Figure 5) or failure to identify an epicardial target. Generally, proximity to a coronary artery was the predominant anatomic reason for limited or no RFA at the anterior areas of the LV. At the lateral areas, proximity to the left phrenic nerve was frequently limiting RFA as shown in Figure 6. Overall, the phrenic nerve was the limitation in 19 cases. In 6 patients, RFA was successful despite initial phrenic nerve capture after displacing the phrenic nerve using a vascular or esophageal balloon that was inserted into the epicardial space as described previously.21,22 In another 8 cases, it was decided not to attempt phrenic nerve protection because some ablation was possible at adjacent sites. In 5 cases, the operator decided not to attempt phrenic nerve protection and did not apply ablation in the region (2 of these procedures were done before methods for phrenic nerve protection were described).

**Complications**

Acute complications were observed in 29 of 309 procedures (9%) and are shown in Table 3. Epicardial bleeding (>80 mL) often after RV puncture was seen in 18 cases (6%). Bleeding stopped spontaneously or after reversal of heparin anticoagulation. Two patients required blood transfusion, and 1 patient had pulseless electric activity after development of tamponade, requiring cardiopulmonary resuscitation and emergency surgery to repair an RV laceration. One patient required thrombus aspiration for acute thrombotic occlusion of the left anterior descending coronary artery during angiography. Ten complications were related to endocardial ablation (Table 3). Two patients developed incessant VT and ventricular fibrillation and required cardiopulmonary resuscitation and emergent LV assist device placement.

**Acute Outcome and Clinical Follow-Up**

Ablation abolished all SMVTS or targeted VAs in 138 (45%), abolished some of the VAs in 61 (20%), failed in 75 (24%), and was not assessed acutely in 35 (11%). Figure 7 summarizes acute outcomes for all VA ablation procedures. Overall, the risk of acute ablation failure was 8.3× higher (4.5–15.0; \( P<0.001 \)) after no or limited epicardial ablation compared with unlimited ablation, accounting for multiple procedures per patient. When only considering VT cases, the risk of ablation failure was 6.6× higher (3.5–12.5; \( P<0.001 \)). No reinjection test was performed after RFA in 10% and 12%, respectively, of the 2 groups mainly because the hemodynamic consequences of the reinjection test were of concern. In another 3% and 1%, respectively, the clinical VT could not be induced during the procedure, and RFA was guided by substrate mapping only.

Epicardial ablation for LV scar-related VT because of CAD or NICM was attempted during 210 procedures (21 redo procedures). In this population, at least 1 clinical or presumptive clinical VT could be abolished in 93 of 122 cases (76%) after unlimited epicardial ablation compared with 20 of 35 cases (57%) when no epicardial target could be identified and 22 of 53 cases (42%) when the target site was protected by an anatomic structure.

For PVC/nonsustained VT ablations, acute ablation failure (no effect on PVCs) was seen in 1 of 5 cases (20%) where
access, however, RFA is not always possible. In this large series, we systematically analyzed reasons for epicardial RFA failure relative to the epicardial target area. The majority of included patients had nonischemic cardiomyopathies, followed by patients with CAD, ARVC and no structural heart disease. Considering the overall incidence of these heart diseases, however, an epicardial approach was most likely to be chosen in patients with NICM and ARVC as reported previously. Patients without any structural heart disease and especially those with VAs originating from the LV outflow tract and LV summit region may be over-represented in this series because of the referral nature of the population to our tertiary-care center.

After successful access, unlimited RFA at the epicardial target area could only be performed in 62% of cases. Limited RFA was possible in 8%, and in 30%, RFA was not felt to be possible at all. Reasons for limited or failed epicardial ablation included failure to identify an epicardial target for RFA, a complication or—dependent on the target area—proximity to a coronary artery or the phrenic nerve. The epicardial target areas depended on the presence and the type of heart disease as shown in Figure 2. The outflow tracts were predominant targets in patients with no structural heart disease or ARVC. The basal, perimitral areas were targeted most frequently in other NICMs, whereas the LV midwall and apex were the major targets in patients with CAD. Figures 3 and 4 show success rates and reasons for impeded epicardial RFA according to the target area and according to the type of heart disease.

Coronary artery narrowing or occlusion caused by RFA has been described previously. Proximity to a coronary artery frequently impeded RFA along the anterior wall of the LV and especially in the LV summit region, which is known to be a challenging area for RFA. RFA targeting this region was successful only in the minority of cases (11%). The other main reason for failed RFA in this area was failure to identify an epicardial target for RFA because of overlying fat or a deep intramural focus out of reach for radiofrequency energy. Figure 5 shows an example of a patient undergoing ablation for symptomatic PVCs originating in the LV summit region, where RFA could not be applied from either the great cardiac or the epicardium because of proximity to coronary arteries. Endocardial RFA had no effect on the PVCs, and consequently, the procedure failed. Surgical cryoablation can offer a treatment option in such cases. Surgical dissection of the epicardial fat tissue and mobilization of the coronary arteries can potentially localize the arrhythmogenic foci and allow for ablation as described previously.

The left phrenic nerve that passes behind the lateral wall of the LV can also prohibit epicardial RFA. Injury to the phrenic nerve and consequent diaphragmatic paralysis has been reported after epicardial RFA for VT. Proximity to the left phrenic nerve was the main limitation for RFA at the

![Figure 5. A. Example of a patient undergoing ablation for symptomatic premature ventricular contractions (PVCs). B and C. Activation mapping showed the earliest electric activity in the epicardial left ventricular (LV) summit region. D and E, Radiofrequency ablation (RFA) was neither possible from the great cardiac vein nor from the epicardial space because of the proximity to the coronary arteries. RFA from the corresponding endocardial site in the left ventricular outflow tract (red tags) had no effect on the PVCs. CS indicates coronary sinus; LAD, left anterior descending coronary artery; LAO, left anterior oblique view; LCC, left coronary cusp; LCX, left circumflex coronary artery; NCC, noncoronary cusp, RAO, right anterior oblique view; and RCC, right coronary cusp.](http://circep.ahajournals.org/doi/abs/10.1161/CIRCEP.115.005264?journalCode=cep)
lateral LV wall and can potentially be seen anywhere from the base to the apex and even anteriorly and inferiorly. In this series, the phrenic nerve was protected using a vascular or esophageal balloon that was inserted into the epicardial space in 6 cases targeting the lateral LV. Without that maneuver, RFA application in that area would have been prevented even more frequently. Figure 6 shows findings from a patient with NICM undergoing an ablation procedure for drug-refractory, hemodynamically untolerated VT. Phrenic nerve capture (black tags) impeded RFA targeting late potentials within a large lateral epicardial scar. RFA could be performed in this case after displacing the phrenic nerve using an esophageal balloon. Others have used instillation of saline, air, or a combination of air and saline to attempt to move the phrenic nerve away from the epicardium, but this can result in transient hemodynamic impairment, and whether radiofrequency lesions are as effective when the pericardium is filled with saline or air is not certain.

Failure to identify an epicardial target for RFA after successful access was the limitation in 15% of cases. To this point, indicators of epicardial substrate are limited. Hence, the absence of arrhythmia origin on the endocardium does

### Table 3. All Complications Related to the Ablation Procedure (n=309)

<table>
<thead>
<tr>
<th>Complication</th>
<th>Further Interventions Required</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute complications related to epicardial access/mapping/ablation</strong></td>
<td></td>
</tr>
<tr>
<td>Intrapercardial bleeding (&gt;80 mL)</td>
<td>Transfusion/volume administration 4</td>
</tr>
<tr>
<td>PEA after tamponade</td>
<td>Drain, CPR, and surgical repair</td>
</tr>
<tr>
<td>LAD occlusion during angiography</td>
<td>PCI</td>
</tr>
<tr>
<td><strong>Other acute complications</strong></td>
<td></td>
</tr>
<tr>
<td>RV rupture after endocardial steam pop</td>
<td>Surgical repair 2</td>
</tr>
<tr>
<td>Stroke</td>
<td>Thrombus extraction 1</td>
</tr>
<tr>
<td>Fatal pulmonary embolism</td>
<td></td>
</tr>
<tr>
<td>Development of incessant VT/VF/cardiogenic shock</td>
<td>CPR, emergent LVAD placement 2</td>
</tr>
<tr>
<td>Right iliac artery dissection</td>
<td>Stent placement</td>
</tr>
<tr>
<td>Groin hematoma</td>
<td>Transfusion 2</td>
</tr>
<tr>
<td><strong>Delayed complications</strong></td>
<td></td>
</tr>
<tr>
<td>Major pericardial reaction—pericarditis</td>
<td>Hospitalization</td>
</tr>
<tr>
<td>RCA occlusion 2 wk after procedure</td>
<td>PCI</td>
</tr>
<tr>
<td>Mixed metabolic and respiratory acidosis</td>
<td>Prolonged hospitalization 2</td>
</tr>
<tr>
<td>Pericardial effusion &gt;4 wk after procedure</td>
<td>Pericardiocentesis 2</td>
</tr>
<tr>
<td>Cardiac decompensation (volume overload)</td>
<td>Prolonged hospitalization 2</td>
</tr>
</tbody>
</table>

Values are presented as n (%). CPR indicates cardiopulmonary resuscitation; LAD, left anterior descending coronary artery; LVAD, left ventricular assist device; PCI, percutaneous coronary intervention; PEA, pulseless electric activity; RCA, right coronary artery; RV, right ventricle; VF, ventricular fibrillation; and VT, ventricular tachycardia.
Acute complications related to the epicardial approach remain an important concern and occurred in 9% of cases, most commonly bleeding into the pericardial space. These results should be interpreted in the context of the procedures being performed at an experienced center. A recent multicenter study involving 290 procedures from 6 centers reported an acute success rate of 71% for epicardial VT ablation procedures with a similar complication rate and reasonable outcome. In our single-center study involving 309 procedures for VT and PVC, we found that acute outcome is highly dependent on the target area. Our data point out that the anticipated epicardial target area for RFA must be taken into account when making a decision on whether epicardial access is justified. Especially for VA originating in the LV summit region, epicardial access is often not helpful and success rates are rather low. On the other hand, VT ablation was rarely limited for patients with ARVC, where the epicardial RV was the main target area.

Recurrence rates after limited or no epicardial RFA were significantly higher for patients without structural heart disease and patients with NICM but not for patients with CAD. In this patient group, epicardial RFA may have limited effect in the presence of intramural reentrant circuits. Because epicardial RFA was unlimited in the vast majority of patients with ARVC, no reliable statement can be made on the effect of limited epicardial RFA in patients with ARVC. For patients with CAD or NICM undergoing ablation procedures for LV scar–related VT, recurrence rates were higher if the target was protected by an anatomic structure compared with cases where no target could be identified, suggesting that phrenic nerve protection strategies have value. The somewhat better success rate for those without at epicardial target may reflect the presence of intramural arrhythmia substrate, such that endocardial ablation was effective in some despite evidence of the arrhythmia presence after endocardial mapping and ablation.

**Limitations**

This is a retrospective study of patients referred to a tertiary-care center for ablation. Results, including complication rates, may be different for other centers or operators. The study...
summarizes ablation procedures performed for 16 years, and advances in VT ablation techniques over time may have influenced the results and need to be acknowledged when interpreting our data. Our population is selected and may be skewed toward a cohort of patients with more advanced disease, consistent with the relatively high proportion of patients who had ≥1 previous ablation attempts. In some patients with large scars, the targeted epicardial area may have overlapped multiple segments as defined for this study. In such cases, the predominantly involved area was selected. The decision to not ablate at a site because of proximity of a coronary artery or the phrenic nerve is subjective and was left to the operator. Although guidelines have recommended a distance of 5 mm to be maintained between the ablation catheter and coronary arteries, the data supporting this guidance are limited, and others may be willing to ablate closer to the vessel. Similarly, we did not ablate at sites where pacing captured the phrenic nerve, on the assumption that it would be at risk. It is possible that phrenic nerve protection maneuvers could be performed in more of these patients. Although total radiofrequency time was available for all procedures, separation into endocardial and epicardial radiofrequency time was only possible for procedures performed after mid 2010. The referral nature of our center did not allow us to adequately assess asymptomatic VT recurrences in this retrospective study. The definition of recurrence used allowed us to provide accurate follow-up data and describe recurrence rates consistently. Holter ECG data were not systematically available for patients after PVC ablation procedures. Therefore, we have no information on asymptomatic PVC recurrence.

Conclusions

Epicardial RFA for VAs is frequently limited or impossible even after successful epicardial access, and impeded epicardial RFA is associated with greater chance of acute procedural failure and higher recurrence rates. The main limitations of epicardial RFA include failure to identify a target for RFA and proximity to a coronary artery or the left phrenic nerve. Variability of success of epicardial ablation procedures is dependent on the target area, and complication rates are significant. These factors should be taken into account when considering whether to attempt epicardial access for catheter ablation procedures.

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Disclosures

Dr Epstein received consulting fees from Boston Scientific, Medtronic, and Spectranetics. Dr John received consulting fees from St. Jude Medical. Drs Michaud and Tedrow received consulting fees from St. Jude Medical and research funding from Boston Scientific and Biosense Webster. Dr. Stevenson is a coholder of a patent for needle ablation that is consigned to Brigham and Women’s Hospital. The other authors report no conflicts.

References


Table 4. Recurrence or Death and Overall Death Rates in Subgroups

<table>
<thead>
<tr>
<th></th>
<th>No Structural Heart Disease</th>
<th>CAD</th>
<th>NICM (Other Than ARVC)</th>
<th>ARVC</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Recurrence or death (%)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unlimited ablation</td>
<td>1/17 (6)</td>
<td>22/41 (54)</td>
<td>33/67 (49)</td>
<td>13/33 (39)</td>
<td>69/1158 (44)</td>
</tr>
<tr>
<td>No/limited ablation</td>
<td>20/27 (74)</td>
<td>17/32 (53)</td>
<td>34/58 (59)</td>
<td>2/2 (100)</td>
<td>73/1119 (61)</td>
</tr>
<tr>
<td><strong>Overall death</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unlimited ablation</td>
<td>0/17</td>
<td>12/41 (29)</td>
<td>11/67 (16)</td>
<td>0/33</td>
<td>23/158 (15)</td>
</tr>
<tr>
<td>No/limited ablation</td>
<td>0/27</td>
<td>8/32 (25)</td>
<td>12/58 (21)</td>
<td>1/2 (50)</td>
<td>21/1119 (18)</td>
</tr>
</tbody>
</table>

Presented are rates of recurrence or death and overall death after unlimited and no/limited radiofrequency ablation. Kaplan–Meier curves are provided in the Results section in the Data Supplement. ARVC indicates arrhythmogenic right ventricular cardiomyopathy; CAD, coronary artery disease; and NICM, nonischemic cardiomyopathy.


Epicardial Radiofrequency Ablation Failure During Ablation Procedures for Ventricular Arrhythmias: Reasons and Implications for Outcomes
Samuel H. Baldinger, Saurabh Kumar, Chirag R. Barbhaiya, Saagar Mahida, Laurence M. Epstein, Gregory F. Michaud, Roy John, Usha B. Tedrow and William G. Stevenson

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Supplemental Methods: Subxyphoid Epicardial Access

The choice of an anterior or posterior puncture for epicardial access was based on the operator’s preference. Before 2014 the technique described in detail by Sousa et al. was used for epicardial access.\(^1\) In brief a 6-inch, 17G or 18G, 152-mm Tuohy needle (Hakko Cooperative Ltd, Chikuma-shi, Naganoken, Japan) was advanced below the xiphoid process under fluoroscopic guidance to the border of the cardiac silhouette. Injection of a small amount of contrast was used to identify the pericardium and confirm entry into the pericardial space. A guidewire was placed into the epicardial space, and a long sheath was advanced over the wire. The ablation catheter was then positioned in the epicardial space through the long sheath.

In 2014 we switched to the needle in needle technique as described by Kumar et al.\(^2\) This technique uses an 18G Cook needle (Percutaneous Entry Thinwall Needle, 18G, 7 cm, Cook Medical, Bloomington, IN) and a 21G micropuncture (Cook Medical) or long spinal needle (Chiba Biopsy needle 21G, 20 cm, Cook Medical). Subxiphoid insertion of an 18G Cook needle is made to a point beneath the sternum outside the cardiac silhouette. A 21G micropuncture needle is inserted through the 18G Cook needle and is used to enter the pericardial space. A 0.018G guidewire with a floppy tip (Hi Torque Steelcore 18 guidewire with microglide coating, 0.018 inch, 190 cm, Abbott Vascular, Santa Clara, CA) is advanced through the 21G needle into the pericardial space. Both needles are removed and a micropuncture dilator is advanced into the pericardial space over the 0.18G guidewire and then exchanged for a 6 French (Fr) or 8Fr dilator.

The guidewire is exchanged for a floppy-tip 0.35-inch wire (Cook Medical) and an 8Fr sheath with dilator is inserted over the 0.35-inch wire. When maintaining the option for a second pericardial access, a second 0.35- or 0.32-inch wire is inserted into the 8Fr sheath, the 8Fr sheath is removed and the sheath desired for introducing the catheter, usually a long steerable sheath (Agilis, St. Jude Medical, Minneapolis, MN) is inserted.

<table>
<thead>
<tr>
<th>Targeted Area</th>
<th>Total</th>
<th>Type of Heart Disease</th>
<th>Epicardial RFA</th>
<th>Reason for no / limited RF-ablation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>no heart disease</td>
<td>CAD</td>
<td>NICM (other than ARVC)</td>
</tr>
<tr>
<td>LV summit</td>
<td>56</td>
<td>21 (38)</td>
<td>8 (14)</td>
<td>26 (46)</td>
</tr>
<tr>
<td>LV basal lateral</td>
<td>30</td>
<td>3 (10)</td>
<td>5 (17)</td>
<td>22 (73)</td>
</tr>
<tr>
<td>LV infero-basal</td>
<td>47</td>
<td>2 (4)</td>
<td>17 (36)</td>
<td>28 (60)</td>
</tr>
<tr>
<td>LV mid anterior</td>
<td>19</td>
<td>5 (26)</td>
<td>7 (37)</td>
<td>6 (32)</td>
</tr>
<tr>
<td>LV mid lateral</td>
<td>27</td>
<td>0</td>
<td>7 (26)</td>
<td>20 (74)</td>
</tr>
<tr>
<td>LV mid inferior</td>
<td>34</td>
<td>2 (6)</td>
<td>19 (56)</td>
<td>13 (38)</td>
</tr>
<tr>
<td>LV apex</td>
<td>16</td>
<td>0</td>
<td>9 (56)</td>
<td>7 (44)</td>
</tr>
<tr>
<td>RV / RVOT free wall</td>
<td>49</td>
<td>12 (25)</td>
<td>1 (2)</td>
<td>4 (8)</td>
</tr>
<tr>
<td>RV inferior</td>
<td>13</td>
<td>2 (15)</td>
<td>1 (8)</td>
<td>3 (23)</td>
</tr>
<tr>
<td>all</td>
<td>291</td>
<td>47 (16)</td>
<td>74 (26)</td>
<td>129 (44)</td>
</tr>
</tbody>
</table>

Values are presented as n (%), percentages are calculated per row (targeted area)
LV: left ventricle, RV: right ventricle, RVOT: right ventricular outflow tract, CAD: coronary artery disease, NICM: non-ischemic cardiomyopathy, ARVC: arrhythmogenic right ventricular cardiomyopathy, RF: radiofrequency, RFA: radiofrequency ablation
Supplemental Figure: Recurrence free survival and overall survival in subgroups

No structural Heart Disease

Coronary Artery Disease

Non-ischemic Cardiomyopathy (other than ARVC)

Recurrence free Survival

Overall Survival

No deaths in patients without structural heart disease

Log-Rank $p < 0.001$

Log-Rank $p = 0.580$

Log-Rank $p = 0.007$

Log-Rank $p = 0.590$

Log-Rank $p = 0.240$