Surgical Ablation of Refractory Ventricular Tachycardia in Patients with Non-Ischemic Cardiomyopathy

Running title: Anter et al.; Surgical Ablation of Refractory VT

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Journal Subject Codes: [22] Ablation/ICD/surgery; [39] CV surgery: other
Abstract:

Background - The surgical approach for the treatment of ventricular tachycardia (VT) has been largely replaced by percutaneous, catheter-based techniques. However, some VT circuits, particularly in patients with non-ischemic cardiomyopathy (NICM), remain inaccessible to percutaneous ablation. Surgical therapy of these VTs is an alternative approach; however its methodology has not been well defined. The purpose of this study was to evaluate the efficacy of preoperative electroanatomic and electrophysiologic characterization of the VT substrate and circuit in order to guide surgical ablation.

Methods and Results - Eight patients with recurrent sustained VT refractory to antiarrhythmic drugs (AAD) underwent endocardial and / or epicardial ablation procedures. Electroanatomical mapping was performed, and the VT substrate, and circuit(s) were defined using voltage, activation, entrainment, and pace-mapping. All 8 patients underwent detailed endocardial mapping; 6 patients also underwent epicardial mapping. Radiofrequency ablation was performed using an open irrigation catheter. Following the unsuccessful percutaneous approach, surgical cryoablation was applied to the sites, previously identified and targeted during the percutaneous procedure. There were no significant peri-operative complications. During a mean follow-up period of 23±6 months (Range: 15-34 months), 6 patients had significant reduction in VT burden as evident by a reduced number of ICD shocks after ablation (6.6 to 0.6 shocks per patient; p=0.026). Two patients died, one from progressive heart failure and one from sepsis.

Conclusions - VT circuits inaccessible to percutaneous ablation techniques are rare but can be encountered in patients with NICM. These VTs can be successfully targeted by surgical cryoablation guided by pre-operative electroanatomic and electrophysiologic mapping.

Key words: Ventricular Tachycardia, Catheter Ablation, Surgical Ablation, Cryoablation
Introduction:

Catheter based radiofrequency (RF) ablation has become an important therapeutic option for treatment of ventricular tachycardias (VTs). Ablation therapy is considered an effective strategy for the treatment of both idiopathic and scar-related VTs.\textsuperscript{1} However, in the latter category, and especially in patients with a non-ischemic substrate, the success of RF ablation is not uniform. The likely reason for this discrepancy is the nature and / or distribution of scar.\textsuperscript{2-4} We have previously shown that in patients with non-ischemic cardiomyopathy, the scar burden is often peri-valvular and frequently epicardial. Although, critical components of the VT circuit in these locations can still be successfully ablated with a conventional approach, in some patients a catheter-based approach is ineffective. In these cases, surgical ablation of the arrhythmia substrate may be the only alternative. However, the existing approach to surgical VT ablation is largely based on the experience reported over 2 decades ago when this procedure was performed exclusively in patients with healed myocardial infarction. Such an approach may not be applicable in the non-ischemic patient population manifesting heterogeneous scar burden.

In this study, we report a our experience of surgical ablation, guided by detailed pre-operative electroanatomic and electrophysiologic characterization of the underlying substrate in patients with non-ischemic cardiomyopathy experiencing drug refractory VT who had failed percutaneous catheter RF ablation.
Methods

Study population

Over a 3-year period (2007-2009), 527 patients underwent 644 VT ablation procedures at our center. Structural heart disease was present in 295 patients (56%). Of these, 144 patients (49%) were categorized as non-ischemic based on the lack prior infarct and absence of coronary disease. In 8 of these patients, all with a non-ischemic substrate, the arrhythmia remained refractory to medications, endocardial and/or epicardial percutaneous RF ablation. These patients underwent a surgical ablation procedure and constitute our study population. All procedures were performed as per the institutional guidelines of the University of Pennsylvania Health System, and all patients provided written informed consent.

Endocardial Mapping

A retrograde trans-aortic approach was used to access the left ventricle (LV) in all cases. A detailed electroanatomic map of the chamber of interest was created during sinus rhythm or right ventricular (RV) pacing. All mapping was performed with a 3.5-mm open irrigation tip catheter (Navistar Thermocool, Biosense Webster, Diamond Bar, CA) maintaining a fill threshold of 15 percent to ensure adequate representation of the entire sampled surface area. The details of the contact electroanatomic mapping system (CARTO™, Biosense Webster, Diamond Bar, CA) have been described previously.² ⁵ Bipolar signals were also acquired on the Prucka (GE) recording system and analyzed separately.

Epicardial Mapping
Epicardial access was obtained with the technique originally described by Sosa et al.\textsuperscript{6} Briefly, under general anesthesia, a Tuohy needle was introduced via a subxiphoid approach to gain access to the pericardium. An 8F sheath was then advanced over a wire into the pericardial space. Mapping was performed using the same methodology as described above.

**Reference Values for Voltage Mapping and Areas of Voltage Abnormalities:**

The reference values for identifying low amplitude endocardial bipolar electrograms were defined as per previously established criteria.\textsuperscript{5} A signal amplitude greater than 1.5mV was categorized as normal. The most abnormal signal amplitude (<0.5mV) comprised "dense scar".

The reference values for defining abnormal electrograms in the epicardium have been established recently.\textsuperscript{7} For this study, normal epicardial electrograms were defined as signal amplitudes greater than $\geq$1.0 mV, recorded at a distance $\geq$1 cm from large epicardial coronary arteries and/or valves. To adequately distinguish abnormal epicardial locations from fat mimicking scar, these areas were also required to demonstrate abnormal electrograms (i.e., fractionated, split and / or late potentials). The extent of abnormal endocardial and epicardial bipolar voltage signals was quantified by measuring contiguous areas of abnormal electrograms using the "area calculation" tool available in the 3-D mapping system.

**Electrophysiological Study and VT Ablation**

During the electrophysiology study, programmed electrical stimulation was performed from $\geq$1 ventricular site at 2 drive cycle lengths using up to 3 extrastimuli. The ECG of all spontaneous and induced VTs were analyzed on the Prucka recording system. When the VT was stable and tolerated, endocardial and / or epicardial activation and entrainment mapping were employed to
characterize the critical components of the circuit and/or the site of origin (if the underlying mechanism was felt to be non-reentrant). If the VT was not well tolerated or not reproducibly initiated, detailed characterization of the underlying substrate was performed by 1) marking areas manifesting late, split or fractionated potentials and, 2) pace-mapping to mimic the VT morphology. The combination of abnormal electrograms during sinus rhythm, delayed stimulus to QRS during pace mapping, and a good (10/12 or better) QRS match of pace-map were used as surrogates of the VT circuit. RF ablation was performed, extending from the border zone to the dense scar while transecting critical components of the VT circuit. When these regions were in close proximity to anatomical boundaries (mitral or aortic valve), the lesion sets were extended to incorporate these inert areas. Typical settings during lesion creation were: power range of 20-50 watts, maximum temperature of 45° for a total duration of 60-180 seconds in order to achieve an impedance drop of 10 to 18 Ohms.

**Cardiac Imaging**

Intracardiac echocardiography (Acuson AcuNav™, 8F ultrasound catheter) was used in all ablation procedures. The transducer was placed in the RV, and abnormal echogenic area, representing scar in the endocardium and/or subepicardium were identified. Cardiac magnetic resonance imaging (MRI) was performed prior to the ablation procedure in 4 of the 8 patients. We have previously described the feasibility and safety of MRI in patients with defibrillators. In brief, informed consent was obtained and cardiac MRI was performed at 1.5T. The defibrillator was interrogated prior to the study and tachycardia detection and therapy disabled. Pacing was programmed to VVI mode at 40bpm in these non-dependent patients. Cardiac MRI sequences were adapted to minimize energy deposition and image artifact. After cardiac MRI, defibrillators
were re-interrogated and returned to previous settings. No clinically relevant changes were seen in any parameters in these 4 patients. Standard criteria in the form of delayed enhancement using a phase-sensitive inversion recovery sequence after gadolinium-DTPA administration were used for identifying ventricular scar.

Surgical VT Ablation technique

Surgery was performed utilizing median sternotomy with cardiopulmonary bypass. Cryothermy was applied under cold cardioplegia. The relevant VT surfaces (endocardial, epicardial, or both) were carefully inspected, and correlation to the pre-acquired electroanatomical map was performed. Endocardial exposure was performed via a trans-aortic valve approach. Moreover, in all patients, the previously placed RF ablation lesions were identified. No additional mapping was performed during the surgical procedure. Cryothermy to the endocardial and/or epicardial locations previously identified was applied using the Surgifrost™ Surgical Cryoablation System (Medtronic CryoCath LP, Quebec, Canada). This consists of a flexible metal probe with an adjustable insulation sheath, which can be molded to conform to the cardiac contours. The system uses Argon gas in order to achieve rapid cooling to a temperature of -150°C. During a 3-minute application time, this creates an ablation lesion as deep as 60 mm (Figure 1).

Follow-up

After completion of the surgical ablation, patients recovered in the hospital as per standard open-heart surgery protocol. They were subsequently followed through regular clinic visits (first month, and every 3 months then after) during which arrhythmia recurrences were assessed by patient interview, 12-lead ECG, and device interrogation. In the event of no arrhythmia
recurrence, modification / discontinuation of the antiarrhythmic regimen was left to the discretion of the treating electrophysiologist.

**Statistical analysis**
Continuous variables are expressed as group mean ±1 standard deviation. Comparisons of continuous variables between groups were analyzed with the Wilcoxon signed rank test. Categorical variables expressed as proportions in different groups were compared by the Chi-square test. A $p$ value of <0.05 was considered statistically significant.

**Results**

**Patient Population**
Baseline characteristics of the 8 patients are listed in Table 1. There were 7 men and 1 woman with a mean age of 58 ± 11 years. The median LV ejection fraction was 35%, and all patients had non-ischemic cardiomyopathy (6 patients with dilated cardiomyopathy and 2 patients with hypertrophic cardiomyopathy). Each patient included in this series had failed medical therapy with at least 1 AAD and had experienced multiple appropriate ICD shocks (median 6, range 3-16) in the preceding 3 months before surgical ablation. Patients underwent a median of 1.5 (range 1-3) endocardial and 1.0 (range 0-2) epicardial RF ablation procedures prior to the surgical ablation.

**Substrate Characterization**
The details of electroanatomic mapping are presented in Table 2. The LV endocardium was mapped in all 8 patients (average of 398±228 sites per subject). Additionally, in 4 patients
manifesting a clinical VT morphology consistent with RV origin (left bundle branch block pattern), electroanatomical mapping of the RV was also performed. Endocardial regions of dense scar occupied small area, ranging from 0 to 12% of the entire LV endocardium (mean 3.7 ± 2.7%). Consistent with our prior observations, these low voltage zones were predominantly in the peri-mitral annulus distribution. Two patients with hypertrophic cardiomyopathy (patients 3 and 8) also demonstrated scar along the base septum. Epicardial mapping was performed in 6 patients. A mean of 491±213 sites (range 219-826 per patient) were sampled in order to create the RV\LV epicardial voltage map. In 2 patients detailed epicardial mapping was not performed: one patient had undergone multiple prior valve replacement procedures and so we anticipated lack of potential pericardial space, and in the second patient, difficult pericardial access resulted in RV perforation requiring emergent surgery.

Electrophysiological Characterization

A total of 24 spontaneous or induced VTs were observed (median of 2 per patient, range 1-8) with a mean cycle length of 334±186ms (Table 3). Four patients had stable and well-tolerated VTs that allowed characterization of the circuit. In the other 4 patients, the VT was not hemodynamically tolerated, and the substrate was characterized by identifying surrogates of the potential VT circuits. The clinical VT (defined as either identical to a 12 lead ECG obtained during spontaneous arrhythmia or matching electrogram morphology and rate, as recorded by the ICD during spontaneous VT) was induced and characterized in all patients. In 5 patients, at least one component of the VT circuit was mapped and targeted from to the endocardium. Additional endocardial lesions were performed with intention to eliminate all induced VTs. A mean of 51±49 minutes of radiofrequency ablation time was applied during the endocardial ablation
procedure. In the 6 patients undergoing epicardial VT mapping, the abnormal substrate was usually opposite to the endocardial ablation lesions, but was separated by thick myocardium that ranged from 15 to 32 mm (as measured by intracardiac ultrasound and/or MRI). Moreover, all 4 patients who underwent cardiac MRI exhibited a mid-myocardial scar (Figure 2). The mid-myocardial scar was identified in the septum in 3 cases and in the inferior wall in 1 case. These MRI findings of mid-myocardial scar correlated with lack of endocardial or epicardial scar at these locations on the bipolar voltage map. However, in one patient, with basal-septal mid-myocardial scar, scar was identified on the epicardial surface.

At the conclusion of the last percutaneous RF ablation procedure, 4 patients had no inducible VT, 3 patients were inducible for the clinical VT, and in 1 patient inducibility could not be assessed due to urgent surgical intervention (subject 7; Table 3). In all 3 patients who were non-inducible at the end of the procedure, ≥1 of the targeted VT recurred within one week.

**Surgical ablation**

The median interval between the last percutaneous RF ablation procedure and surgical cryoablation was 2±4 weeks (range 0-12 weeks). Cryothermy was applied at the previously identified critical VT circuit locations. In 5 patients cryothermy was applied from both the endocardium and epicardium, and in 3 patients from the epicardium alone. The details of the surgical ablation procedure are shown in Table 4. Importantly, in 2 patients (subjects 3 and 8), the clinical VT originated from the thickened (>20mm) basal septum where MRI imaging demonstrated mid-myocardial scar (Figure 2). In this location endocardial and epicardial cryothermy lesions were applied adjacent to the base of the LV septum near the aortic valve,
taking care to spare the left anterior descending coronary artery. In 2 patients (subjects 4 and 5),
the clinical VT was mapped to the epicardial surface and was in close proximity (<1 cm) to the
left anterior descending and circumflex arteries. During surgical exploration, these locations
were targeted with cryoablation after ensuring a minimum separation of ≥0.5 cm from the
coronary vessels. In 1 patient, surgical ablation was performed following complication of the
percutaneous ablation procedure (laceration of the RV). This patient had a normal endocardial
voltage map, and the circuit of the clinical VT did not appear to be endocardial. When the heart
was exposed, epicardial scar was evident on the antero-lateral LV surface, consistent with the VT
morphology. This location was targeted by cryothermy. Overall a median of 5 cryothermal
applications (range 7±4) were made per subject with a mean cryoablation time of 18±6 minutes
per patient. Of note, in 2 patients (subjects 1 and 4), surgical cryoablation was combined with
valve repair (Table 4). For patients who only underwent a surgical cryoablation procedure, the
mean surgical procedure duration was 112 ± 28 minutes (range 82-164 minutes).

**Short and Long Term Outcomes**

The follow-up data is presented in table 5. Two patients died during the index hospitalization (at
6 and 10 weeks after the surgical ablation): one from progressive heart failure and second from
sepsis. In the remaining 6 patients, the median time from surgery to discharge was 7 days (range
5-11 days). Non-invasive programmed stimulation via the right ventricular defibrillator lead was
performed in 4 patients before discharge. VT was induced only in 1 patient. Patients were
discharged on the pre-ablation antiarrhythmic drug regimen: 4 patients were discharged on a
single antiarrhythmic agent, and 1 patient on two antiarrhythmic agents. One patient was
discharged off antiarrhythmic drug therapy. Over a mean follow-up of 23±6 months (range: 15-
34), 4 patients remained free of VT, 1 patient had a single VT episode resulting in 1 ICD shock and 1 patient had three VT episodes, all occurring during the first 3 post-operative months, but none over the remainder 12 months of follow-up (without modification of medical therapy). Overall, there was a significant reduction in burden of VT and ICD therapies. The number of ICD shocks per patient declined from 6.6 shocks in the preceding three months before surgery to 0.6 during the first 3 post surgical months ($p=0.026$).

**Discussion**

We hereby report our surgical ablation experience in patients with recurrent symptomatic VT that is refractory to medical therapy and percutaneous ablation procedures. The surgical ablation procedure was guided by detailed electrophysiological and electroanatomical mapping obtained *a priori* during the percutaneous ablation procedure. The salient findings of this study include: 1) VTs refractory to percutaneous ablation were more common in patients with a non-ischemic substrate, 2) the underlying substrate frequently involved a mid-myocardial or epicardial scar in close proximity to a coronary vessel, and 3) detailed *a priori* electroanatomical and electrophysiological characterization of the arrhythmia / substrate was useful to identify the surgical targets of ablation.

It should be emphasized that this series represents one end of the spectrum: patients with frequent symptomatic VTs that are resistant to antiarrhythmic drug therapy and percutaneous ablation techniques. Surgical ablation for these patients was last resort. In these cases, electroanatomical and electrophysiological guided surgical cryoablation may be useful. Despite, the excellent outcome in 6 patients (75%), two patients died during the follow-up period. These
deaths were not directly related to the ablation procedures, as both patients experienced normal uncomplicated recovery from their surgical procedure. It is, however, possible that these procedures, along with their associated hospital stay may indirectly increased the risk for complications.

The initial experience with surgical VT ablation was described in patients with healed myocardial infarcts and primarily involved endocardial resection of the visible scar. However, this led only to a modest long-term arrhythmia control. Subsequent work by Miller et al showed that the outcome of surgical VT ablation in the setting of healed infarction could be improved by detailed pre and intraoperative mapping. Seminal work by Josephson et al resulted in the development of subendocardial resection, which further enhanced the procedural efficacy of surgical VT ablation in this population, with long-term arrhythmia control rates of ~80%. With improvements in catheter-based ablation technology, success rates for percutaneous VT ablation have improved, and VT arrhythmia surgery has become uncommon. However, in some patients, particularly those with a non-ischemic substrate, catheter ablation may still fail, and surgical approach remains the only alternative. Despite this, studies on the surgical ablation experience in this patient population are lacking.

As our experience shows, patients with non-ischemic cardiomyopathy may have mid-myocardial and / or basal LV epicardial scars that are in close proximity to major coronary arteries. Thus, RF energy may not be effective or safe in these locations. For this sub-group of patients, surgical VT ablation can be beneficial. However, in our opinion, to accomplish successful surgical cryoablation the following are important: 1) detailed characterization of the
underlying arrhythmia mechanism / substrate utilizing percutaneous electrophysiologic /
electroanatomic mapping, 2) cardiac imaging (intracardiac ultrasound / MRI), 3) full sternotomy
for complete visualization of various cardiac surfaces and, 4) cardiopulmonary bypass with cold
cardioplegia. Additionally, precise surgical ablation is also facilitated by identifying and
targeting locations manifesting prior RF lesions. In our experience these sites are easily
recognized in the endocardium but may not be as evident in the epicardium. In the latter location,
they appear as small punctate spots (Figure 3, Panel B) and may be indistinguishable from
epicardial fat or scar. This appearance of epicardial lesions may be a reflection of the inadequacy
of catheter-based RF energy to create effective epicardial lesions via the percutaneous technique.

Limitations

The major study limitations are as follows: 1) the study population is heterogeneous,
consisting of patients with dilated as well as hypertrophic cardiomyopathy. Thus, the arrhythmia
mechanism(s) / substrate may not be uniform; 2) although we performed detailed
electrophysiologic and electroanatomic characterization of the substrate endocardially in all
patients, in 2 patients the epicardium was not well characterized. Nevertheless, in these 2
subjects, surgical ablation was still guided by the information collected during detailed
endocardial mapping; 3) since intra-operative mapping was not performed, we cannot comment
on its utility in our study population, however an hybrid surgical approach with mapping during
surgery is also likely to be useful; 4) programmed stimulation was not performed routinely in all
patients before discharge, and therefore assessment of acute success is based on clinical outcome
5) patients included in this series had incessant VT resistant to antiarrhythmic drug therapy and
percutaneous ablation attempts; and therefore outcome can not be extrapolated to patients in whom less aggressive measures where not fully utilized.

**Conclusion**

Ventricular tachycardia refractory to percutaneous catheter ablation is rare. However, when encountered, it is primarily seen in patients with a non-ischemic substrate. Such VTs can be successfully targeted by surgical cryoablation guided by prior detailed percutaneous electroanatomic and electrophysiologic mapping.

**Conflict of Interest Disclosures:** None.

**References:**


Table 1: Baseline Patient Characteristics

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<tr>
<th>Patient</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>LVEF (%)</th>
<th>NICM</th>
<th>ICD</th>
<th>No. Failed AADs</th>
<th>No. ICD shocks in preceding 3 months</th>
<th>No. of prior endocardial procedures</th>
<th>No. of prior epicardial procedures</th>
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LVEF= left ventricular ejection fraction; NICM= non-ischemic cardiomyopathy, ICD= implantable defibrillator, AAD=antiarrhythmic drugs. *ICD was implanted after ablation procedure.

Table 2: Endocardial and Epicardial Substrate Characteristics

<table>
<thead>
<tr>
<th>Sinus rhythm LV points</th>
<th>LVZ, cm²</th>
<th>% dense scar</th>
<th>LP in dense scar, %</th>
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LVZ= low voltage zone defined as bipolar signal amplitude ≤1.5mV in endocardium and ≤1.0mV in the epicardium; dense scar was defined as a bipolar signal amplitude ≤0.5mV in both the endocardium and epicardium; LP=late potentials.
Table 3: Endocardial and Epicardial Ablation Data

<table>
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<tr>
<th>Patient #</th>
<th>No. of Induced VT</th>
<th>No. of RF Lesions</th>
<th>VT mapping/target identification</th>
<th>Inducible at end of most recent procedure</th>
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<td>No. of Epicardial</td>
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AM=activation mapping; EM=entrainment mapping; LP=late potentials; PM=pace mapping

Table 4: Surgical Cryo Ablation Data

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<td>Yes</td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>Cryotherapy</td>
<td>Yes</td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>0</td>
<td>Cryotherapy</td>
<td>No</td>
<td>Repair of laceration in RV wall</td>
</tr>
<tr>
<td>8</td>
<td>0</td>
<td>Cryotherapy</td>
<td>Yes</td>
<td>None</td>
</tr>
</tbody>
</table>
Table 5: Follow-up Data

<table>
<thead>
<tr>
<th>Patient #</th>
<th>Time from surgery to discharge (days)</th>
<th>NIPS pre-discharge</th>
<th>No. of AADs at discharge</th>
<th>No. of ICD shocks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>11</td>
<td>Not performed</td>
<td>2 (Quinidine, Mexiletine)</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>Non-inducible</td>
<td>1 (Sotalol)</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>8</td>
<td>Not performed</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>Died</td>
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<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>5</td>
<td>7</td>
<td>Non-inducible</td>
<td>1 (Amiodarone)</td>
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</tr>
<tr>
<td>6</td>
<td>Died</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>7</td>
<td>6</td>
<td>Non-inducible</td>
<td>1 (Sotalol)</td>
<td>0</td>
</tr>
<tr>
<td>8</td>
<td>7</td>
<td>MMVT Inducible</td>
<td>1 (Mexiletine)</td>
<td>1</td>
</tr>
</tbody>
</table>

NIPS= non invasive programmed stimulation; ICD= implantable defibrillator, AAD=antiarrhythmic drugs

Figure Legends:

**Figure 1:** Two approaches to epicardial cryoablation. In **Panel A**, the cryoprobe was aligned in its entire length along the anterior interventricular septum ensuring a safe distance from the left anterior descending coronary artery. In **Panel B**, the cryoprobe was coiled to create a circular lesion at the anterolateral LV base between the branches of the circumflex artery. Sep=septum; Lat=lateral wall.

**Figure 2:** Cardiac MRI showing a 4 chamber view of the heart obtained using a phase-sensitive inversion recovery sequence approximately 15 minutes after gadolinium-DTPA administration which demonstrates a mid-myocardial late enhancement in the basal septum (arrow).

**Figure 3:** VT morphology (right bundle, right inferior) and cardiac images in a patient with hypertrophic cardiomyopathy. Endocardial (Endo) and epicardial (Epi) electroanatomic mapping revealed normal voltage and thick basal left ventricle (distance between epi and endo surface >3 cm (**panel A**). At surgical ablation, prior RF lesions were visualized epicardially (**panel B**, black circle) and endocardially, via trans-aortic approach (**panel C**, red arrow). Epi and endo cryothermy applications were made over prior RF lesions resulting in a large confluent basal epicardial lesions (**panel D**, dotted area).
Surgical Ablation of Refractory Ventricular Tachycardia in Patients with Non-Ischemic Cardiomyopathy