Ventricular Tachycardia Ablation:
Evolution of patients and procedures over 8 years

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Abstract

**Background:** Evolving management of coronary artery disease, heart failure, and the use of Implantable Cardioverter-Defibrillators (ICD) impacts the characteristics of patients with recurrent ventricular tachycardia (VT). We investigated the substrate, procedure and outcome evolution of all patients referred for VT ablation over the past 8 years.

**Methods and Results:** From 1999 to 2006, 493 consecutive patients (358 male, 57±16yo) underwent 623 VT ablations: 131 had no structural heart disease (SHD), 213 ischemic cardiomyopathies (ICMP) and 149 non-ischemic cardiomyopathies (NICMP). Whereas the main substrate is ICMP, the proportion of NICMP has increased from 27% to 35 % (p=0.06) from the 1999-2002 to the 2003-2006 period. The procedure abolished or modified inducible VTs in ≥75% of patients in all groups, but abolition of all monomorphic VTs was achieved in 125(83%) patients without SHD, 180(65%) with ICMP and 99(51%) with NICMP (p<0.0001). During a mean follow-up of 3.3±2.4 years, no deaths occurred in patients without SHD but 75 patients (35%) with ICMP and 26 patients (17%) with NICMP died after a median of 13 months. Multivariate Cox regression analysis found age, ejection fraction and need for pre-procedural mechanical hemodynamic support, predicted mortality.

**Conclusions:** The substrate causing VT in patients requiring ablation is evolving and determines the long term outcome. In the setting of a normal heart, VT ablation is associated with low risk of subsequent mortality with no deaths occurring over a mean follow-up >3 years. In contrast, in patients with SHD and recurrent VT, VT ablation can be helpful to suppress drug refractory VT, but long term mortality remains significant.

**Key-words:** Ventricular Tachycardia, Catheter Ablation, Mortality, Substrate
Introduction

Evolving management of coronary syndromes and heart failure therapies in nonischemic and ischemic diseases impact the substrate causing ventricular tachycardia (VT) with structural heart disease (SHD). The predominant strategy for treating VT remains palliative, utilizing anti-arrhythmic drugs (AAD) and/or Implantable Cardioverter Defibrillators (ICD). Even if ICDs improve survival and reduce sudden death in high risk patients with SHD\textsuperscript{1-3}, 10 to 20\% of patients with ICDs experience “electrical storm” with repeated device therapies \textsuperscript{4,5}. Although AADs reduce episodes, efficacy has been disappointing \textsuperscript{6,7} and side effects are an important problem \textsuperscript{8}. Radiofrequency catheter ablation (RFCA) is an option to control recurrent VT, often as sole therapy in patient without SHD or in combination with an ICD and anti-arrhythmic therapy in scar related VT associated with SHD. Several investigators reported ablation outcomes for series of patients selected for having one predominant morphology of VT. Other studies reporting RFCA for VT considered only patients with one underlying etiology, relatively small numbers of patients, or short follow-up periods. The objectives of this study were to evaluate the evolution of the substrate associated with VT, the procedural evolution and the long-term mortality of all patients with VT ablation over the last 8 years.

Methods

Study Population

From January 1999 to December 2006, 493 consecutive patients (358 (73\%) male, 57 ±16yo) underwent 623 VT ablations at our institution. One hundred thirty-one had no SHD, 213 had ischemic cardiomyopathy (ICMP) with coronary artery disease defined as history of prior myocardial infarction or documented obstructive coronary artery disease and 149 had a non-ischemic cardiomyopathy (NICMP) (Table 1). Approximately 53\% of these patients have
also been reported in smaller cohorts investigating specific mapping and ablation methods. In all patients at least one episode of VT (sustained or non-sustained) was recorded on monitoring: Holter, ICD log or 12 lead ECG. VT was continuous and incessant, or very frequent, meeting a commonly used definition of electrical storm with more than 3 separate VT episodes in the 24 hours prior to ablation, in 136 (22%). Patients failed a mean of 3 ±1 AADs. Amiodarone was used prior to ablation in 14% in the group without SHD, in 84% in the group with ICMP and in 59% in patients with NICMP. A prior ablation had been attempted in 49% (mean of 1.5± 0.7 previous ablations for the group). An ICD had been implanted previously in 87% of patients with ICMP and 69% of patients with NICMP, respectively; and these were biventricular pacing devices in 13% and 12% of cases, respectively.

Written informed consent was obtained from all patients. Procedures and review of medical records were conducted under protocols approved by the institutional human subject protection committee.

VT ablation

The methods used for mapping and ablation were those reported previously but did evolve from 1999 to 2006. Ventricular mapping was performed with 7 or 8 F steerable catheters with either a 8, 4- or 3.5-mm electrode tip (Navi Star or Thermo-Cool, Biosense Webster, Inc. Diamond Bar, CA, USA). Bipolar electrograms were recorded on the electro-anatomic mapping system (filtered at 10 to 400 Hz) (CARTO, Biosense Webster, Inc., Diamond Bar, CA, USA) and a separate digital system (filtered at 30 to 500 Hz; Prucka Engineering Inc). Pace mapping and entrainment mapping used unipolar pacing from the distal electrode with an initial current strength of 10 mA and pulse width of 2 ms.

If VT was not incessant, the ventricle of interest (right, left or both) was mapped during sinus or paced rhythm to identify areas with abnormal electrograms and low-voltage (<1.5 mV)
consistent with scar \cite{10,11,21,22}. If abnormal areas were present the mapping catheter was placed at an abnormal site that had pace-mapping characteristics of an exit or potential isthmus site and VT initiated to assess electrograms during VT, entrainment mapping and potentially RF ablation during VT to assess VT termination as previously described \cite{10,11}. If VT was stable, mapping continued during VT. If the circuit could not be identified ablation was performed through the presumptive exit based on voltage mapping combined with pace-mapping. In patients without structural heart disease a focal source for the arrhythmia was sought from a combination of activation mapping and pace-mapping.

Ablation lesions were created with radiofrequency (RF) current with a maximum power of 50 W (EP Technologies. Inc or Stockert, Biosense Webster Inc) using various types of catheter ablation over the study period (4-mm or 8-mm standard tip, internally or externally irrigated tip catheters).

The acute outcome of the ablation procedure was defined as follows: success—no monomorphic VT was inducible (including VTs that had not been observed to occur spontaneously, often referred to as “non clinical”); indeterminant - monomorphic VT was inducible but was different and usually faster than VTs induced at the beginning of the procedure, suggesting the arrhythmia substrate has been modified, or the acute outcome could not be reliably determined due to inability to reliably induce the arrhythmia, but no arrhythmia was provokable after ablation (particularly in patients without SHD); and failure—VT inducible at the beginning of the procedure remained present and/or inducible.

After ablation, anti-arrhythmic drugs were reduced or discontinued depending on the substrate and the acute success of the procedure.

We report only major complications (resulting in long term disability, requiring intervention, or prolonging hospital stay) and including life-threatening complications (with immediate or short term risk of death).
Data collection and Follow-up

Data were collected from a centralized system that contained complete records of all patients treated and followed at the Brigham and Women’s Hospital. These records provide a detailed history and diagnosis for all patients, including ablation report, emergency room visits and outpatient visits, as well as data recorded during inpatient care. Patients local to the hospital were followed-up in Brigham and Women’s Hospital clinics. Referring cardiologists were contacted for clinical follow-up of their patients. Mortality was assessed from the social security death index queried in October 2007.

Statistical analysis

Baseline characteristics of the patients were compared with the chi-square test for the categorical variables and with a t-test for the quantitative variable depending on the substrate. The event-free survival was graphically displayed according to the method of Kaplan and Meier, with unadjusted comparisons of mortality by the log-rank test. Univariate and multivariate Cox proportional hazards regression analysis was used to evaluate the contribution of cardiomyopathy type (ischemic or non ischemic), age, left ventricular ejection fraction (LVEF), acute outcome, complications related to the procedure, hemodynamic support, AADs and number of VTs induced to mortality. A 2-sided alpha value <0.05 was considered statistically significant.

Statement of responsibility

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

Results

Substrate and clinical presentation

Our idiopathic VT (no SHD) population was composed of 78 patients with typical RVOT VT, 12 with LVOT VT, 10 fascicular VT, 8 RV VT outside the outflow tract, 14 LV other
than outflow tract or fascicular VT and 9 epicardial VTs. Whereas the main substrate for VT due to structural heart disease is ICMP, there has been a trend for an increasing proportion of NICMP from 27% to 35% (p=0.06) from the 1999-2002 to the 2003-2006 period (Figure 1). Patients with ICMP had a mean of 2.2 ±0.9 coronary vessels diseased. Their last myocardial infarction occurred 10 ±8.3 years (median 9 years) before ablation. One hundred and eight (51%) pts with ICMP had prior coronary artery bypass surgery and 70 (33%) had prior percutaneous coronary intervention before ablation. Only 61(29%) patients had not had a revascularization procedure, 31% during the 1999-2002 period vs 26% during the 2003-2006 period (p=NS).

In ICMP, the predominant scar region was the inferior wall but the proportion of patients with septal scars increased over time. (Table 2)

NICMP substrates during this 8 year period, were idiopathic dilated cardiomyopathy (n=106; 54%), valvular heart disease (37; 19%), congenital heart disease (14; 7%), arrhythmogenic right ventricular dysplasia (30; 15%) and sarcoidosis (8; 4%). Their mean left ventricular ejection fraction was 39 ±16%.

In NICMP, the septum, inferior and lateral wall as well as perivalvular areas and epicardium were almost equally involved with sites of low voltage scar giving rise to VT (Table 2). In the 106 patients with idiopathic dilated cardiomyopathy, 46 patients had a predominant basal scar, 10 patients had a predominant apical scar, 21 patients had scar involving the mid left ventricle and 16 pts had no scar. Accurate data on base vs apex location was not available in the 13 remaining patients.

The vast majority of patients with SHD (288 (80%)) had an ICD before referral VT ablation and were referred for recurrent ICD therapies. In the 74 remaining patients with SHD and no ICD at presentation, 19 were referred for frequent recurrent non sustained VT, 43 because of stable sustained VT and 12 due to VT causing syncope. In the 131 patients without SHD, 51
patients were referred for frequent recurrent non-sustained VT, 55 for stable sustained VT and 25 because of VT leading to syncope.

**VT characteristics and procedure**

Of the 623 VT ablation procedures, we had detailed data on VT characteristics available in 587 (94%) [139 (93%) for no SHD, 268 (96%) for ICMP and 180 (92%) for NICMP]. A mean of 2.2 ±1.7 monomorphic VTs per procedure was induced. Of the 587 procedures, unstable VTs (requiring termination for hemodynamic compromise) alone were present in 131 (22%), stable VTs (monomorphic hemodynamically well tolerated) alone were seen in 183 (31%) and both were present in 273 (47%) procedures.

There were several electrophysiologic differences according to underlying heart disease. At the time of the procedure, patients with no SHD often had only PVCs or non-sustained VT inducible [80 patients (58%)], compared to 6 (2%) and 24 (13%) of ICMP and NICMP patients, respectively (p<0.001). Very slow (< 150 beats/min) and very fast (> 200 beats/minute VTs) were more common in patients with structural heart disease.

In the 2003-2006 period, 78% of the VT ablations in patients with SHD were performed using a combination of substrate mapping, pace-mapping, entrainment mapping and activation mapping. Of note epicardial ablations increased from 7% of procedures to 12% (p=0.04) from 1999-2002 to 2003-2006 period. Mechanical hemodynamic support (intra-aortic balloon pump or assist device) was used in 21 procedures.

Procedural outcomes are shown in table 1. VTs were abolished (success) or modified, in 83% of patients without SHD, in 83% of ICM and 79% of NICM patients. Acute success, with abolition of all monomorphic VTs, was achieved most often in patients with no SHD, followed by ICMP and NICMP. None of the patient without SHD was receiving amiodarone at discharge whereas 33% of patients in the ICMP group and 18% of patients in the NICMP group were receiving amiodarone at the time of hospital discharge.
Of the 623 procedures, 48 (7.7%) patients had adverse events during or within 48 hours after the procedure (Table 3). Life-threatening complications were seen in 2 (1.3%) patients without SHD, 13 pts with ICM (4.7%) and 8 pts with NICMP (4.1%).

**Outcome**

Mean follow-up for the entire group was 3.3 ±2.4 years. No deaths occurred in non SHD patients during a mean follow-up of 4.2 ±2.2 years in this group. In contrast, 75 patients (35%) with ICMP and 26 patients (17%) with NICMP died after a median of 13 months [5 pts within 1 week]. Kaplan-Meier curve of survival are displayed in Figure 2. Eight pts (3.8%) with ICMP and 13 pts (8.7%) with NICMP underwent heart transplant 3.3 months (median) after VT ablation.

In univariable Cox proportional hazards analysis age and LV ejection fraction were among predictors of mortality (table 4). ICMP had a 2-fold (95% CI 1.3 - 3.2) increased risk of mortality compared with NICMP. Other univariable risk factors for mortality were complications related to the procedure, the number of failed AADs and the number of VTs induced during the procedure. This increased risk was explained by older age and lower ejection fractions among these patients. Age, left ventricular ejection fraction and mechanical hemodynamic support were independent predictors of mortality in a multivariable model (Table 4). For every year of age, there was a 4.3% mortality increase and for every percent EF increase, a 3.3 % mortality decrease. Those patients requiring mechanical support pre- or peri-procedurally had a four-fold greater independent risk of mortality, with worse prognosis than those suffering a major procedural complication. Arrhythmic storm or incessant VT were not predictors of mortality in this population.

VT recurred after a median of 1 month in 29% of ICMP and 39% of NICMP patients and arrhythmic outcome was not available for 30 of these procedures (6%). Data for recurrent VT
during late follow-up was not reliably obtainable for the patients without SHD due to the referral nature of the population.

Discussion

Main findings

During a mean follow-up of 3.3 ±2.4 years, no deaths were seen after VT ablation in patients without SHD and long term mortality was 35% for ICMP and 17% for NICMP. Age, LVEF as well as necessity for mechanical hemodynamic support during the procedure were independent risk factors for mortality. During VT ablation, patients without SHD more often had only PVCs inducible, whereas patients with ICMP and NICMP had sustained VTs that could be very slow or very fast. A combination of substrate mapping, pace-mapping, activation mapping and entrainment was predominantly used especially in patients with SHD. Whereas the main substrate for VT ablation is ICMP (especially with inferior scar, late after myocardial infarction), the proportion of patients with NICMP is increasing. These patients have more variable scar locations (Table 1).

Long term outcome and predictors of mortality

VT in normal hearts (so called idiopathic VT) is generally felt to be benign, based on small historical series. In our study, there was no death with a follow-up of 4.2 ±2.2 years in this sizable observational group. For patients with SHD, VT ablation is a palliative therapy that can reduce the number of ICD shocks. However, the presence of shocks may indicate more severe disease. This concern is consistent with the mortality rate of 35% at 3 years in the ICMP group. Mortality was somewhat better in the NICMP group, 18% at 3 years, likely related, at least in part, to younger age and generally better LV function in the NICMP patients.
Arrhythmic storm has been shown to predict mortality in NICMP and ICMP. In our study, patients with incessant VT or electrical storm had similar outcomes to those with less frequent VT. It is possible that ablation improved outcome for patients with electrical storm or incessant VT, such that their prognosis was then similar to those with intermittent VT. There are also, however, substantial selection biases that influence our results. The sickest patients may not have been felt appropriate for VT ablation and not been referred. The nature and provocative factors for electrical storm also vary among different studies. In our population, ablation was not performed in patients with a ‘secondary’ cause of arrhythmic storm (such as ischemia and metabolic disorders); whereas in the study of Exner et al., 65.4% of arrhythmic storms were attributed to one or more of these secondary causes.

**Procedure**

In SHD, a combined approach (substrate mapping, pace-mapping, and activation mapping) was usually used (figure 3). When VT is stable, substrate mapping during sinus rhythm can be used to identify regions for further evaluation during VT, minimizing the time spent in VT. Brief entrainment is often possible, even for unstable VTs, to confirm the location of a reentry circuit, potentially allowing ablation with a smaller number of RF lesions than when ablation is guided only by substrate mapping.

Of note, the characteristics of VT, specifically the VT cycle length were different depending on the substrate. Patients without SHD more often have only PVCs at that time of the procedure with more difficulty inducing VT, possibly consistent with the non-reentrant mechanisms thought to be common in this population. Patients with ICMP have more slow VT (CL>400ms) than patients with NICMP probably due to the substantial extent of scar with slow conduction after myocardial infarction.

In patients with SHD, acute success with abolition of all inducible VTs (59%) was similar to previous reports (averaging respectively 38% to 75%). Despite the evolution of therapy,
the acute procedural outcome is failure in 13% of our patients at our referral center and modified substrate (still inducible VT but different from those initially inducible in 28% of procedures.

In the multicenter study of Calkins et al. 28 (using an internally irrigated catheter for ablation of mappable VT ablation in ICMP), ablation of all mappable VTs was achieved acutely in 75% of patients with an 8% risk of major complication. We observed roughly similar acute success rate and complication rates in a more diverse population of patients with structural heart disease, including patients with unstable VTs. Both studies demonstrate that major complications are not negligible in this particularly sick population.

Failure may be due to inaccurate mapping, inadequate lesion creation, or to the presence of deep intramyocardial or epicardial arrhythmia substrate. Concerning the first item, many studies have been published in the last 10 years that have improved our ability to interpret electrograms and identify components of reentry circuits using entrainment and characterize entrainment.10-12, 31, 32 and to better define the substrate based on low voltage areas, unexcitable electrical scar, and electrograms.33-35 Concerning inadequate lesion formation, progresses have been made by using larger ablation electrodes and cooled-tip electrodes that increase lesion size36. However even with irrigated tip catheters ventricular lesions may not be transmural. It is clear that epicardial approaches are required for some patients.13, 37

Deep intramural circuits or foci may still be difficult to eliminate with available approaches.

New technologies, such as needle irrigated RF ablation, that allows deliver energy directly inside the myocardium, that are under investigation will be of interest in this regard 38, 39 and systems for improved catheter navigation seem promising40.

Substrate
In ICMP, there was often a substantial latency between infarction and the VT ablation due to occurrence of multiple, refractory VTs, with a median delay between the last myocardial infarction and VT ablation of 9 years. This observation suggests a role for continued late remodeling occurring after myocardial infarction. The recent observation from the MADIT II trial that appropriate ICD therapy for VT or VF predicts increased mortality also suggests that arrhythmias are a marker for disease severity and possibly remodeling. Thus, the occurrence of frequent VT, resulting in referral for VT ablation may be a marker of a more advanced or malignant disease consistent with mortality rates in our post MI patients of 16%, 24% and 35% at 1, 2 and 3 years after ablation.

There has been an increase in the proportion of patients with NICMP and VT compared to ICMP over the last 8 years. It is possible that more aggressive reperfusion strategies for acute myocardial infarction result in smaller infarcts and reduce the number of infarct survivors that eventually need VT ablation. It is also possible that more NICMP patients with VT are surviving because of a better medical therapy and a greater use of ICDs in that population. Changes in referral patterns may also be responsible for this trend. The recognition that ablation of these VTs can be challenging, may lead to earlier referral to a tertiary center.

**Clinical Implication**

VT ablation may be considered as a reasonable first or second line therapy in patients with idiopathic VT that is symptomatic or sufficiently frequent to raise concern about causing depressed ventricular function. In patients with SHD, ablation can be useful to prevent or reduce recurrent episodes of VT, usually as adjunctive therapy to an ICD. The risks are greater and mortality remains significant after ablation in these patients with recurrent, drug refractory VT. Attention to optimizing treatment of the underlying disease, as well as controlling VT recurrences, seems prudent. A recent study suggested that outcomes may be
better in patients undergoing ablation after initial presentation with VT\textsuperscript{41}. Further studies are needed to help define the role of ablation as the patient population and technologies continue to evolve.

**Limitations**

(1) Our findings are based on a retrospective observational analysis. Although we adjusted for potentially confounding difference, we cannot exclude that other factors have contributed to our findings. (2) Being a referral center for VT ablation, our population is selected and may be skewed toward a sicker VT ablation population, consistent with the relatively high proportion of patients who had prior ablation attempts. It is possible that long term mortality would be better for ablation performed before failure of multiple antiarrhythmic medications. (3) While our findings of no mortality in patients without structural heart disease are reassuring, it should be appreciated that some myopathic processes, such as sarcoidosis and arrhythmogenic RV dysplasia can be subtle in their clinical manifestations and may escape detection. A careful search for underlying disease is warranted in these patients. (4) Patients without structural heart disease often stop seeing their referring cardiologist during late follow-up, such that clinical follow-up for VT recurrences was not available for 71% of our patients without SHD. Use of the social security death index allowed mortality to be obtained for all patients. In SHD patients, follow-up was based on reports of ICD interrogations, which may have been incomplete. (5) The vast majority of our SHD patients had ICDs that undoubtedly extend survival in this patient population. The impact of an ablation strategy on mortality either with or without an ICD can not be assessed from our data. (6) The ablation and mapping technology used in an individual patient was influenced by its availability, uncontrolled patient and physician factors. We did not therefore attempt to compare different ablation technologies overall in this population.
Conclusion

In this large observational series, the proportion of patients with NICMP compared to ICMP with VT requiring ablation is increasing. Procedures targeting these substrates have evolved through the last 8 years with availability of epicardial mapping and common use of a combination of substrate mapping, pace mapping and activation mapping.

In the setting of a normal heart, prognosis is excellent, with no deaths occurring over a mean follow-up >3 years. For patients with recurrent VT due to SHD, ablation is a palliative option which suppresses or decreases drug refractory VT episodes while long term mortality remains significant.

Funding sources

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Conflict of Interest Disclosures:

We report for Dr Frederic Sacher, an educational grant from Biosense Webster (modest),

During the time period over which these patients were studied Dr Tedrow received research funding from Boston Scientific and Medtronic (modest) and received speaking honoraria from Boston Scientific (modest), Medtronic (Modest), and St Jude Medical, Inc (modest),

Dr Bruce Koplan received speaking honoraria from Boston Scientific (modest), Medtronic (Modest), and St Jude Medical, Inc (modest), Dr Laurence Epstein was a consultant to Biosense Webster (modest) and received speaking honoraria from Boston Scientific (modest), Medtronic (Modest), St Jude Medical, Inc (modest) and Biosense Webster (modest), Dr William Stevenson was a consultant to Biosense Webster (modest) and received speaking honoraria from Boston Scientific (modest), Medtronic (Modest), St Jude Medical, Inc (modest) and Biosense Webster (modest).
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Legends

Table 1: Characteristics of patients with Ventricular Tachycardia ablation depending on the substrate

Table 2: Evolution of the patients undergoing Ventricular Tachycardia ablation between 1999-2002 and 2003-2006 according to substrate

Table 3: Complications during, to 48 hours after VT ablation

Table 4: Predictive factors of mortality in patients with CMP undergoing VT ablation. Cut-off value for age and LVEF were the median. (CI: Confidence Interval, LVEF: Left Ventricular Ejection Fraction, ICMP: Ischemic Cardiomyopathy, AAD: Anti-Arrhythmic Drugs, VT: Ventricular Tachycardia)

Figure 1: Frequency of the different VT substrates from 1999 to 2006

Figure 2: Kaplan Meier curves of survival after VT ablation depending on the substrate.

Figure 3: Approach to Mapping and Ablation of VTs performed in our center. (VT: Ventricular Tachycardia; RV: Right Ventricle; RF: Radiofrequency)
### Table 1

<table>
<thead>
<tr>
<th></th>
<th>No SHD</th>
<th>ICMP</th>
<th>NICMP</th>
<th>p-value between ICMP and NICMP</th>
<th>Overall p-value</th>
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<td><strong>Demographic (493 patients)</strong></td>
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<tr>
<td>Age (yo)</td>
<td>46 ±14</td>
<td>67 ±11</td>
<td>52 ±15</td>
<td>p&lt;0.001</td>
<td>p&lt;0.001</td>
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<td>Sex (Male)</td>
<td>55 (42%)</td>
<td>188 (88%)</td>
<td>115 (77%)</td>
<td>p=0.006</td>
<td>p&lt;0.001</td>
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<tr>
<td>LVEF (%)</td>
<td>61 ±5</td>
<td>28 ±13</td>
<td>39 ±16</td>
<td>p&lt;0.001</td>
<td>p&lt;0.001</td>
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<tr>
<td>Number of failed AAD</td>
<td>2 ±1</td>
<td>3 ±1</td>
<td>3 ±1</td>
<td>NS</td>
<td>p&lt;0.001</td>
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<td>Procedure (n=623)</td>
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<td>Number of patients with a prior ablation</td>
<td>58 (39%)</td>
<td>128 (46%)</td>
<td>117 (60%)</td>
<td>P=0.02</td>
<td>p&lt;0.001</td>
</tr>
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<td>Number of VT prior to ablation</td>
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<tr>
<td>- None during the last month</td>
<td>23(15%)</td>
<td>6(2%)</td>
<td>20(10%)</td>
<td>p&lt;0.001</td>
<td>p&lt;0.001</td>
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<tr>
<td>- &gt;1 during the last month but not the week preceding ablation</td>
<td>70(47%)</td>
<td>30(11%)</td>
<td>48(25%)</td>
<td>p&lt;0.001</td>
<td>p&lt;0.001</td>
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<tr>
<td>- &gt;1VT in the week preceding ablation / Arrhythmic Storm</td>
<td>57 (38%)/ 11(7%)</td>
<td>242 (87%)/ 81(29%)</td>
<td>127 (65%)/ 43 (22%)</td>
<td>p&lt;0.001 / NS</td>
<td>p&lt;0.001 / p&lt;0.001</td>
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<tr>
<td><strong>Acute Results</strong></td>
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<tr>
<td>Success</td>
<td>108 (72%)</td>
<td>180 (65%)</td>
<td>99 (51%)</td>
<td>p=0.001</td>
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<tr>
<td>Indeterminate</td>
<td>17 (11%)</td>
<td>79 (28%)</td>
<td>54 (28%)</td>
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<td>Failure</td>
<td>25 (17%)</td>
<td>19 (7%)</td>
<td>42 (21%)</td>
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<td><strong>Radiofrequency time (min)</strong></td>
<td>13 ±10</td>
<td>33 ±22</td>
<td>26 ±21</td>
<td>NS</td>
<td>p&lt;0.001</td>
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<td><strong>Fluoroscopy time (min)</strong></td>
<td>35 ±23</td>
<td>46 ±26</td>
<td>48 ±29</td>
<td>NS</td>
<td>p&lt;0.001</td>
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<tr>
<td><strong>Complications</strong></td>
<td>4(2.7%)</td>
<td>32(11.5%)</td>
<td>12(6.2%)</td>
<td>p=0.03</td>
<td>p=0.002</td>
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### Table 2

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<td></td>
</tr>
<tr>
<td>Age (yo)</td>
<td>68 ±11</td>
<td>65 ±11</td>
<td>0.02</td>
<td>52 ±16</td>
<td>58 ±14</td>
<td>NS</td>
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<tr>
<td>Sex (M)</td>
<td>116</td>
<td>132</td>
<td>0.03</td>
<td>30 (83%)</td>
<td>55 (79%)</td>
<td>NS</td>
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<tr>
<td>LVEF (%)</td>
<td>29 ±12</td>
<td>28 ±13</td>
<td>NS</td>
<td>30 ±11</td>
<td>32 ±12</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Scar Area</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procedure with voltage map</td>
<td>132</td>
<td>134</td>
<td>NS</td>
<td>31</td>
<td>66</td>
<td>NS</td>
</tr>
<tr>
<td>Inferior scar</td>
<td>96(73%)</td>
<td>80(60%)</td>
<td>0.02</td>
<td>10 (32%)</td>
<td>16 (24%)</td>
<td>NS</td>
</tr>
<tr>
<td>Anterior scar</td>
<td>30(23%)</td>
<td>40(30%)</td>
<td>NS</td>
<td>2 (6%)</td>
<td>11 (16%)</td>
<td>NS</td>
</tr>
<tr>
<td>Septal scar</td>
<td>43(33%)</td>
<td>66(49%)</td>
<td>0.004</td>
<td>7 (23%)</td>
<td>18 (27%)</td>
<td>NS</td>
</tr>
<tr>
<td>Lateral scar</td>
<td>31(23%)</td>
<td>38(28%)</td>
<td>NS</td>
<td>2 (6%)</td>
<td>12 (18%)</td>
<td>NS</td>
</tr>
<tr>
<td>Apex</td>
<td>35(27%)</td>
<td>45(34%)</td>
<td>NS</td>
<td>7 (23%)</td>
<td>8 (12%)</td>
<td>NS</td>
</tr>
<tr>
<td>LVOT</td>
<td>4(3%)</td>
<td>1(0.7%)</td>
<td>NS</td>
<td>7 (23%)</td>
<td>14 (21%)</td>
<td>NS</td>
</tr>
<tr>
<td>Epicardial ablation</td>
<td>7(5%)</td>
<td>13(9%)</td>
<td>NS</td>
<td>8 (26%)</td>
<td>18 (27%)</td>
<td>NS</td>
</tr>
</tbody>
</table>
### Table 3

<table>
<thead>
<tr>
<th>Major life-threatening complications</th>
<th>No SHD (n=4 - 2.7%)</th>
<th>ICMP (n=32 - 11.5%)</th>
<th>NICMP (n=12 - 6.2%)</th>
<th>Number (n=48 - 7.7%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tamponade-Cardiac perforation</td>
<td>2</td>
<td>3</td>
<td>3</td>
<td>8 (1.3%)</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>0</td>
<td>5</td>
<td>2</td>
<td>7 (1.1%)</td>
</tr>
<tr>
<td>Acute respiratory distress</td>
<td>0</td>
<td>2</td>
<td>2</td>
<td>4 (0.6%)</td>
</tr>
<tr>
<td>Pulmonary Embolism</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2 (0.3%)</td>
</tr>
<tr>
<td>Stroke</td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>2 (0.3%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>23 (3.7%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Other major complications**

<table>
<thead>
<tr>
<th>Other major complications</th>
<th>No SHD (n=4 - 2.7%)</th>
<th>ICMP (n=32 - 11.5%)</th>
<th>NICMP (n=12 - 6.2%)</th>
<th>Number (n=48 - 7.7%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transient ischemic event</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>3 (0.5%)</td>
</tr>
<tr>
<td>Other embolic event</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>2 (0.3%)</td>
</tr>
<tr>
<td>AV block</td>
<td>1</td>
<td>3</td>
<td>1</td>
<td>5 (0.8%)</td>
</tr>
<tr>
<td>Local vascular complication</td>
<td>0</td>
<td>7</td>
<td>2</td>
<td>9 (1.4%)</td>
</tr>
<tr>
<td>Groin hematoma (requiring blood transfusion)</td>
<td>0</td>
<td>5</td>
<td>0</td>
<td>5 (0.8%)</td>
</tr>
<tr>
<td>Phrenic Nerve Injury</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1 (0.2%)</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>25 (4%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Unadjusted</td>
<td>Adjusted</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------------</td>
<td>-----------------------------</td>
<td>----------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hazard ratio</td>
<td>95% CI</td>
<td>p-value</td>
<td>Hazard ratio</td>
</tr>
<tr>
<td>Age &gt;62yo</td>
<td>2.508</td>
<td>1.622-3.877</td>
<td>&lt;0.0001</td>
<td>2.573</td>
</tr>
<tr>
<td>LVEF &lt;30%</td>
<td>2.212</td>
<td>1.477-3.314</td>
<td>=0.0001</td>
<td>1.477</td>
</tr>
<tr>
<td>ICMP</td>
<td>2.054</td>
<td>1.315-3.209</td>
<td>&lt;0.0001</td>
<td>0.935</td>
</tr>
<tr>
<td>Complications</td>
<td>1.956</td>
<td>1.419-2.698</td>
<td>&lt;0.0001</td>
<td>1.584</td>
</tr>
<tr>
<td>Mechanical</td>
<td>3.352</td>
<td>1.687-6.660</td>
<td>&lt;0.0006</td>
<td>4.333</td>
</tr>
<tr>
<td>Hemodynamic support</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of failed AAD</td>
<td>1.213</td>
<td>1.019-1.444</td>
<td>0.03</td>
<td>1.089</td>
</tr>
<tr>
<td>Number of VT induced</td>
<td>1.184</td>
<td>1.060-1.321</td>
<td>0.0027</td>
<td>1.106</td>
</tr>
<tr>
<td>Prior failed ablation</td>
<td>1.240</td>
<td>0.834-1.843</td>
<td>NS</td>
<td></td>
</tr>
</tbody>
</table>
Unadjusted Survival after VT ablation

<table>
<thead>
<tr>
<th>Follow-up (years)</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>No SHD</td>
<td>131</td>
<td>117</td>
<td>105</td>
<td>89</td>
<td>70</td>
<td>53</td>
<td>35</td>
<td>16</td>
<td>5</td>
</tr>
<tr>
<td>Non Ischemic CMP</td>
<td>149</td>
<td>113</td>
<td>88</td>
<td>68</td>
<td>45</td>
<td>32</td>
<td>17</td>
<td>11</td>
<td>3</td>
</tr>
<tr>
<td>Ischemic CMP</td>
<td>213</td>
<td>158</td>
<td>113</td>
<td>89</td>
<td>67</td>
<td>51</td>
<td>30</td>
<td>16</td>
<td>5</td>
</tr>
</tbody>
</table>
Induce VT / confirm diagnosis

Recurrent VT

Idiopathic VT

Well tolerated VT
- activation mapping
- RF for termination

Untolerated or non sustained VT
- Pace mapping

Induce VT again at good pace mapping site
- possible RF for termination

Scal related VT
- Entrainment from RV apex
- Stop tachycardia with overdrive pacing

Sinus Rhythm Mapping

Identify Scar – Voltage Map
- Late potentials
- Fractionated potentials

Pace-mapping (isthmus, exit)
- QRS morphology
- S-QRS duration- slow conduction
- Unexcitable Electrical Scar

Initiate VT
with ablation catheter at site of suspected isthmus/exit

Stable VT
- entrainment
- electrogram

Ablation of isthmuses / exits during VT

Unstable VT
- entrainment once
- possible RF for termination

Ablation of isthmuses / exits during sinus rhythm

Untolerated or non sustained VT
- Pace mapping

Induce VT atgood pace mapping site
- possible RF for termination
Ventricular Tachycardia Ablation: Evolution of patients and procedures over 8 years
Frédéric Sacher, Usha B. Tedrow, Michael Field, Jean-Marc Raymond, Bruce A. Koplan, Laurence M. Epstein and William G. Stevenson

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