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REVIEW ARTICLE

Catheter Ablation of Ventricular Tachycardia as an Adjunct Therapy to Reduce ICD Shocks

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ABSTRACT. *Patients with cardiomyopathy have improved long-term survival when implantable cardioverter-defibrillator (ICD) therapy is added to standard medical therapy. However, ICDs do not prevent arrhythmias and they provide benefit by delivering painful shocks. Furthermore, ICD shocks have been associated with poor quality of life and increased mortality. Medical therapy alone is not sufficient in eliminating shocks, and the use of antiarrhythmics provides modest benefit. Catheter ablation of ventricular tachycardia is possible in patients with both ischemic and non-ischemic cardiomyopathy. This procedure provides an adjunctive therapy to antiarrhythmics and has been shown to reduce ICD shocks. This article reviews the background of and role for catheter ablation of ventricular tachycardia as an adjunct therapy to reduce the frequency of ICD shocks. It highlights current data and an ongoing trial to address this important clinical dilemma.*

KEYWORDS. *catheter ablation, implantable cardioverter-defibrillator, ventricular tachycardia.*

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Introduction

Implantable cardioverter-defibrillators (ICDs) reduce mortality in patients with stable ischemic and non-ischemic cardiomyopathy.[1–5] Their life-saving therapy is primarily in the form of high-energy shocks. However, ICDs do not reduce the frequency of ventricular arrhythmias and may deliver therapy for non-life-threatening arrhythmias. ICD shocks can negatively impact patients by creating anxiety about future shocks and reducing quality of life.[6] Furthermore, ICD shocks have been associated with an increase in mortality, a finding that has been demonstrated for both appropriate and inappropriate shocks.[7] Medical therapy with β-blockers and Class III (Vaughan-Williams) antiarrhythmics may reduce the frequency of ICD shocks, but can be pro-arrhythmic and cause toxic side effects.

Catheter ablation of ventricular tachycardia (VT) has historically been used primarily for drug refractory ventricular arrhythmias in patients with ICDs. However, advances in electro-anatomical mapping systems, techniques to identify ablation sites during sinus rhythm, and the use of hemodynamic support devices has broadened the applicability of catheter ablation for ventricular arrhythmias. When performed in centers with high procedural volumes, the rates of complications remain relatively low.[8]

Frequency of ICD shocks

The frequency and time-course of ICD shocks depends on several factors including the arrhythmogenic substrate, the use of antiarrhythmic medications, and device detection and therapy programming. Quantifying the frequency of ICD shocks for ventricular arrhythmias is confounded by therapies delivered for supraventricular arrhythmias or non-life threatening ventricular arrhythmias. Earlier trials had relatively high rates of ICD therapies. For instance, in the AVID trial[1] patients with a history of prior VT had an incidence of ICD therapies of 68% at 1 year and 85% at 3 years.

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Changes in device programming can have a substantial impact on decreasing the frequency of appropriate ICD shocks. The PREPARE trial[9] evaluated the effectiveness of a ''strategic'' programming arm with the combined use of prolonged detection intervals (30 out of 40 beats), high heart rate cutoffs (182 beats/minute), supraventricular tachycardia discriminators, antitachycardia pacing, and high output first shock on the frequency of ICD therapies. The control arm of this trial was historical control data from the EMPIRIC and MIRACLE-ICD trials.[10,11] The primary endpoint of the trial was the combined endpoint of ICD shocks (appropriate and inappropriate), syncope related to arrhythmia, and untreated sustained VT/ventricular fibrillation (VF). This trial found that the incidence of first shock for VT/VF at 12 months was significantly lower in the ''strategic'' programming arm than in the control arm (5.4% versus 9.4%, $p<0.01$). Mortality was also decreased in the PREPARE arm at 12 months (4.9% versus 8.7%, $p<0.01$).

The ADVANCE III trial[12] also evaluated the effect of increasing the number of intervals that were needed to detect a tachyarrhythmia on the frequency of ICD shocks. When only appropriate ICD shocks were considered, the use of prolonged detection intervals was associated with 19 shocks per 100 patient-years versus 30 shocks per 100 patient-years in the standard programming group. The MADIT-RIT trial[13,14] used prolonged detection times or high rate cutoffs to decrease the frequency of ICD shocks. In the conventional arm, 22% of patients had a first occurrence of appropriate therapy during follow-up compared with 6% in the prolonged detection time arm ($p<0.001$) and 9% in the high rate cutoff arm ($p<0.001$). The majority of this benefit was driven by a reduction in antitachycardia pacing. Inappropriate therapies were also higher in the conventional arm.[13] In fact, seven inappropriate therapies induced ventricular arrhythmias that required appropriate therapy. In this trial, 12% of patients had appropriate ICD therapies during a mean follow-up of 1.4 years. Patients who had appropriate ICD shocks had a mean number of 1.29 shocks per arrhythmia episode. Appropriate shocks were associated with an increase in overall mortality (HR 6.25; 95% CI 3.13–12.75 $p<0.001$). Other factors associated with an increase in mortality were inappropriate ICD therapy and randomization to the conventional programming arm.[14]

To reduce the frequency of appropriate and inappropriate ICD therapies, our practice is to broadly approach ICD programming with conservative parameters. While device programming should always incorporate patient-specific considerations, we generally advocate using long detection intervals and high rate cutoffs.

Impact of ICD shocks

While ICD shocks are often life-saving, they are by no means a benign therapy. ICD shocks are painful and have been associated with an increase in mortality,[7] an increase in clinician visits, and decrease in quality of life for patients.[6]

Effect on mortality

One of the paradoxes of ICD therapy is that while these devices improve mortality in patients with stable cardiomyopathy, ICD shocks themselves are associated with an increased risk of death. This finding has been shown for both appropriate and inappropriate ICD shocks.[7,13–17] As previously mentioned, the MADIT-RIT trial demonstrated a significant increase in mortality associated with any inappropriate ICD therapy or appropriate ICD shocks.[14] The increase in mortality for appropriate ICD shocks is higher than inappropriate therapy, ranging from two- to sixfold compared with patients who do not receive shocks.[17] In general, this finding has been independent of other common predictors of outcome, including etiology of cardiomyopathy, ejection fraction, and New York Heart Association class. The effect on mortality may be ''dose-dependent'', as one study found that an increase in mortality was seen only in patients who received more than five shocks, with no difference in mortality in patients receiving fewer than five shocks. ATP (adenosine triphosphate) alone has not consistently been shown to worsen outcomes. This finding poses the critical question of whether ICD shocks themselves are detrimental to myocardial function or rather a marker for more severe underlying disease. There is evidence that high-energy shocks can lead to electroporation of cellular membranes and acutely worsen myocardial function[18] and ICD shocks may potentially be proarrhythmic.[19]

Effect on quality-of-life

Patients with ICDs who receive shocks have consistently demonstrated increased stress and anxiety and decreased quality of life.[6,20,21] In the AVID cohort,[6] patients who received ICD shocks had an increase in concerns about receiving more shocks and reductions in their sense of well-being and physical state. Patients with more than two shocks had worse quality of life assessments than those with fewer shocks. In a sub-analysis of the CIDS data, patients with ICDs had a higher perceived quality of life than those randomized to receive amiodarone. However, this effect was not seen in patients who received five or more shocks.[20] Other data suggest that younger patients may be more at risk for adverse psychological effects from living with ICDs than older patients.[22]

Medical therapy to reduce ICD shocks

While most patients with cardiomyopathy have an indication for β-blockers at baseline, the use of additional antiarrhythmic medications to reduce ventricular tachycardia may be limited by the presence of significant structural heart disease. In many patients with ICDs, Vaughan-Williams Class IC agents such as flecainide and propafenone are often of limited use. Typically, medications such as β-blockers, Class III agents (sotalol, dofetilide, and amiodarone), or Class IB agents (mexiletine) are used as adjunctive therapies to reduce the

likelihood of tachycardia and subsequent ICD shocks. Class IA agents like quinidine may have a role in Brugada syndrome but are often used as a last resort. The safety and efficacy of sotalol in preventing ICD shocks was first studied in 1999 in a cohort of 302 patients randomized to receive either sotalol or placebo.[23] The use of sotalol reduced the risk of death from any cause or the delivery of a first appropriate ICD shock by 44% ($p = 0.007$) at 1 year. The frequency of shocks in the sotalol group was 1.43 (± 3.53) per year versus 3.89 (± 10.65) in the placebo group ($p = 0.008$). One of the limitations of this study is that only about one-fourth of patients in both groups were taking β-blockers at baseline.

The comparative effectiveness of amiodarone, sotalol, and β-blockers was evaluated in the OPTIC study (Optimal Pharmacological Therapy in Cardioverter Defibrillator Patients).[24] In this study, patients were randomized to treatment with amiodarone plus β-blockers, β-blockers alone, or sotalol. The primary endpoint was ICD shocks. Patients receiving amiodarone plus β-blockers had a reduction in the risk of shock at 1 year compared with β-blockers alone (HR, 0.27; 95% CI, 0.14–0.52; $p < 0.001$) and sotalol (HR, 0.43; 95% CI, 0.22–0.85; $p = 0.02$). The use of sotalol had a non-significant trend towards fewer shocks than β-blockers alone (HR, 0.61; 95% CI, 0.37–1.01; $p = 0.055$). The annualized rate of shocks in the β-blocker alone group was 4.32 (± 15.76) versus 0.93 (± 3.50) in the sotalol group and 0.51 (± 2.67) in the amiodarone plus β-blocker group. Patients in the group receiving amiodarone did have higher rates of pulmonary- and thyroid-related adverse events, as well as more symptomatic bradycardia.

VT ablation

Catheter ablation in patients with cardiomyopathy has become an accepted therapeutic option for patients with recurrent ventricular tachycardia. This approach can be used in patients with both ischemic[25–28] and non-ischemic[29] cardiomyopathy. Comparative data regarding the effectiveness of ablation in ischemic versus non-ischemic patients is lacking. While short-term outcomes may be similar between these two groups, one prospective study found that long-term arrhythmia recurrence was significantly higher in patients with non-ischemic cardiomyopathy (HR 1.62; 95% CI 1.12–2.34; $p = 0.01$).[29] When an epicardial approach is incorporated, the relative long-term success may be more comparable.[30]

Indications for VT ablation are listed in Table 1.

Ablation of stable VT

The ideal situation for VT ablation is one where sustained tachycardia can be mapped using standard electrophysiologic principles. This requires hemodynamic stability during potentially prolonged periods of tachycardia. Catheter mapping of hemodynamically tolerated VT requires identification of critical areas within myocardial scar that serve as requisite portions of tachycardia circuits. Careful analysis of candidate sites during periods of concealed entrainment can identify areas that have post-pacing intervals that approximate tachycardia cycle length, stimulus to QRS intervals that equal EGM (Intracardiac Electrogram) to QRS intervals, and stimulus to QRS intervals of $<70\%$ of the tachycardia

Table 1: Indications for catheter ablation of ventricular tachycardia

Indications for catheter ablation of ventricular tachycardia
Structural heart disease
Ablation is recommended for
1. Symptomatic ventricular tachycardia (VT) treated by an implantable cardioverter-defibrillator despite antiarrhythmic therapy or when antiarrhythmic therapy cannot be tolerated.
2. Incessant VT/VT storm.
3. Frequent VT (or PVCs (Premature ventricular contractions) or NSVT (Nonsustained ventricular tachycardia)) causing ventricular dysfunction.
4. Bundle branch reentry or fasicular VT.
5. Recurrent polymorphic VT or VF with a trigger that can be targeted for ablation.
Ablation should be considered in
1. Patients with sustained VT despite antiarrhythmic therapy.
2. Patients with recurrent sustained VT, prior myocardial infarction (MI), and ejection fraction (EF) $<30\%$ as an alternate to amiodarone therapy.
3. Patients hemodynamically tolerated sustained monomorphic VT, prior MI, and EF $>35\%$, even if not on antiarrhythmic therapy.
Patients without structural heart disease
Ablation is recommended for
1. Monomorphic VT causing severe symptoms.
2. Monomorphic VT when antiarrhythmic medications are not effective or poorly tolerated.
3. Recurrent polymorphic VT or VF with a trigger that can be targeted for ablation.
Catheter ablation of VT is contraindicated
1. When mobile thrombus is present when endocardial mapping of that chamber is planned.
2. Asymptomatic PVCs or NSVT not resulting in ventricular dysfunction.
3. VT due to transient, reversible causes.

Adapted from Aliot et al.[31]

cycle length. All of these measurements predict favorable locations for radiofrequency ablation[27] (**Figures 1–3**). While entrainment mapping during sustained tachycardia is preferable, this is not always possible. In both ischemic and non-ischemic patients, the majority of inducible tachycardias are not hemodynamically tolerated.[32]

Ablation of unstable VT

When mapping during ventricular tachycardia is not possible, additional approaches are required. Mapping techniques during sinus rhythm are important and may be successfully used to ablate clinical tachycardias.[33] Using non-invasive imaging modalities such as echocardiography, computed tomography (CT), and magnetic resonance imaging (MRI) to localize areas of infarct or scar can help in the planning of the ablation procedure to identify areas of interest prior to the procedure.[34,35] Contrast-enhanced CT can be used to define areas of myocardial hypoperfusion and wall motion abnormalities that correlate well with regions of low-voltage scar on electro-anatomical mapping systems.[34]

Identification of low-voltage bipolar electrograms is an important step when using a substrate-based ablation approach. Low-voltage regions are associated with arrhythmogenic substrate required for reentrant tachycardias. A commonly accepted guideline is that recorded electrograms with voltages of <0.5 mV indicate scar, while voltages of 0.5–1.5 mV indicate transition zones (peri-scar regions), and voltages of >1.5 mV indicate healthy myocardium[32] (**Figure 4**). Once a detailed scar map is made, additional techniques are used to further define candidate locations for ablation. Near-threshold pace mapping can be used to localize regions within or connected to a critical isthmus. Areas with prolonged stimulation-QRS times are associated with regions remote from a terminal exit point, whereas areas with short stimulation-QRS times and excellent 12-lead pace maps identify exit points. Exit points are typically located within voltage map transition zones.

One difficulty with substrate-based ablation is defining an appropriate procedural endpoint. Non-inducibility of the clinical rhythm is ideal, but often the clinical tachycardia is difficult to induce at baseline. An additional technique that offers a target for ablation and can be used as a predictive endpoint is the elimination of local abnormal ventricular electrograms (LAVAs).[36] LAVAs are low-amplitude late-systolic potentials that are recorded within areas of scar during sinus rhythm. Ablation and elimination of LAVAs can be successfully achieved in the majority of patients and is associated with a reduction in recurrent tachycardias.[37] Similar approaches to elimination of arrhythmogenic substrate that can be identified during sinus rhythm have been

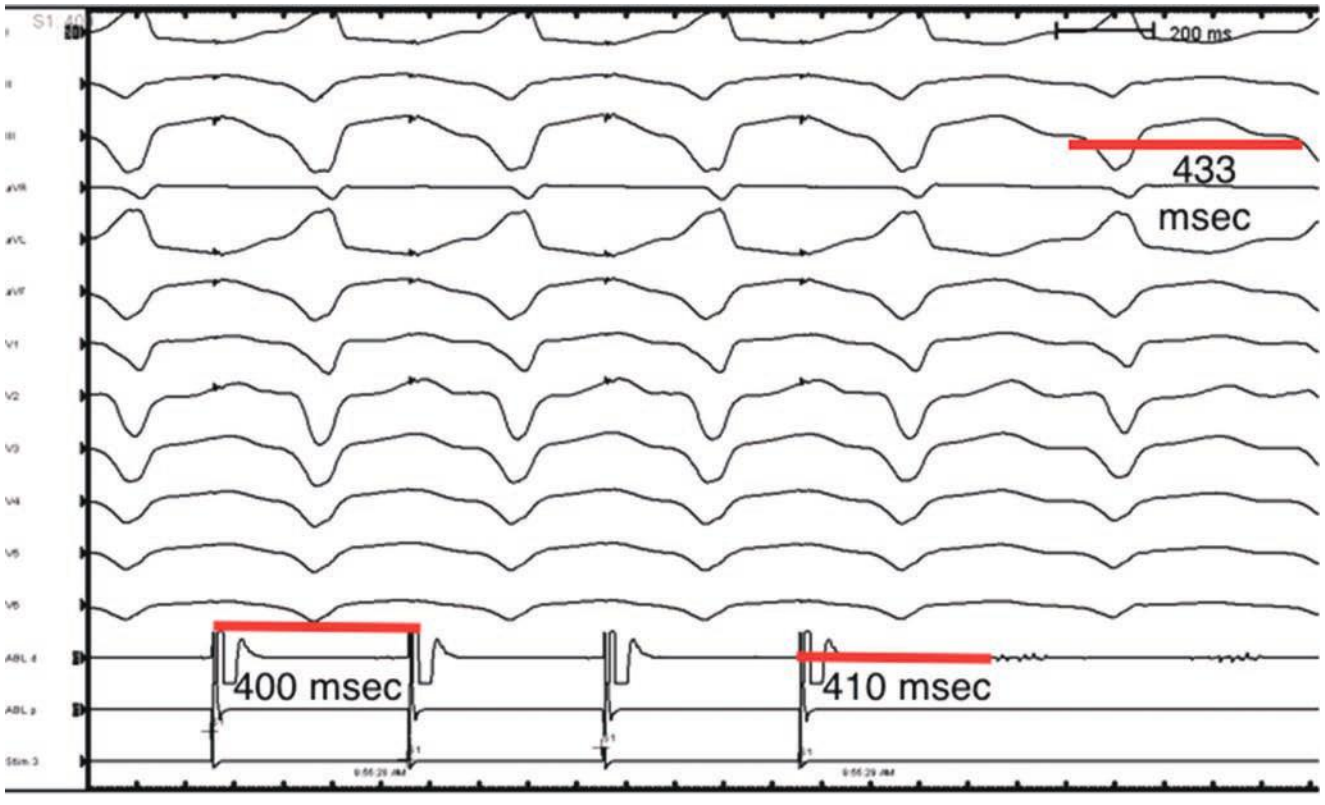

Figure 1: Example of concealed entrainment of ventricular tachycardia from a common pathway isthmus. TCL: 433 ms; pacing rate: 400 ms; PPI: 410 ms (TCL–PPI: 23 ms). The mid-diastolic location of the recorded electrograms and prolonged stimulus-electrogram time indicates that the recorded location is roughly halfway between the entrance and exit points of the protected isthmus (PPI: post-pacing interval; TCL: tachycardia cycle length).

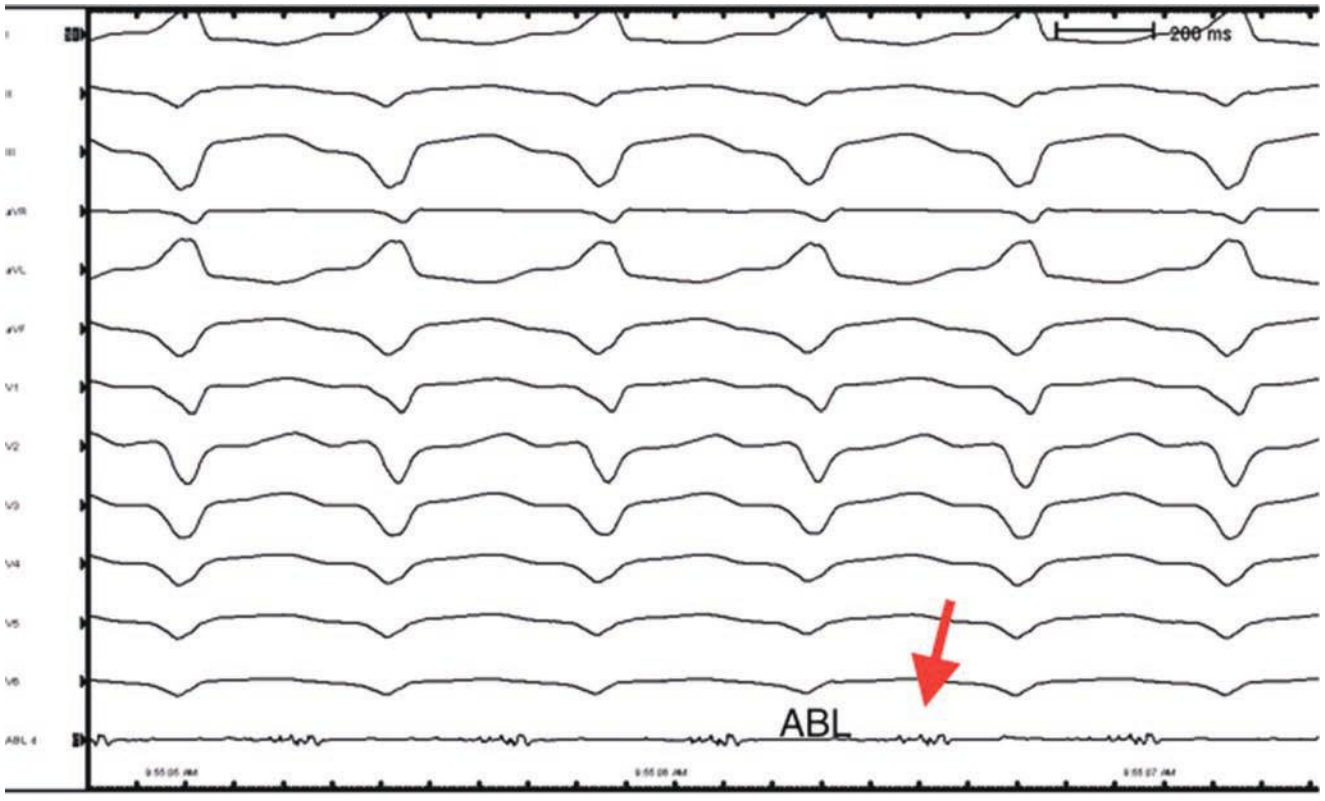

Figure 2: Mid-diastolic electrograms during sustained ventricular tachycardia. Note the electrogram is low-amplitude and high-frequency with a long duration of recorded signal that encompasses much of diastole.

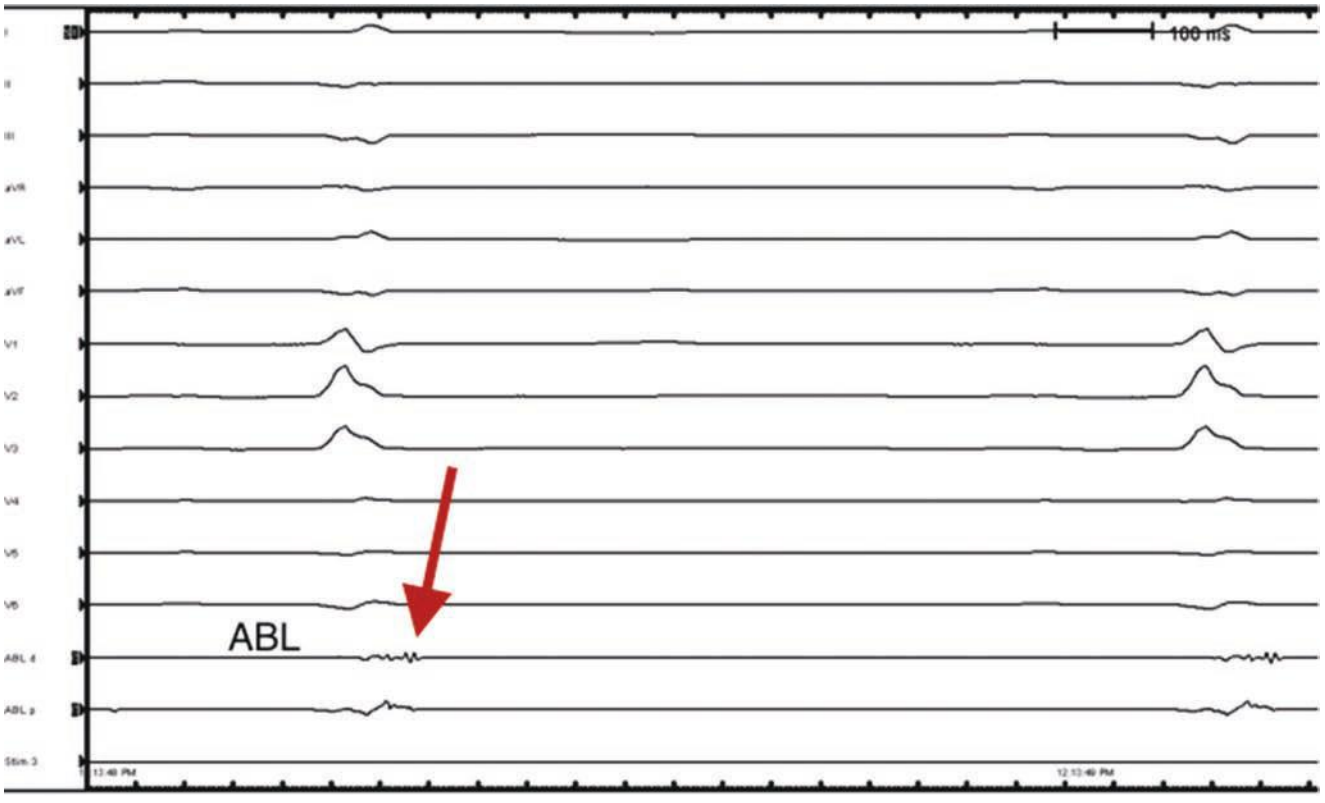

Figure 3: Late-systolic signals recorded during sinus rhythm. These low-amplitude signals were recorded in a region of scar on an electro-anatomical map. The electrograms extend >100 ms after the onset of the surface QRS complex.

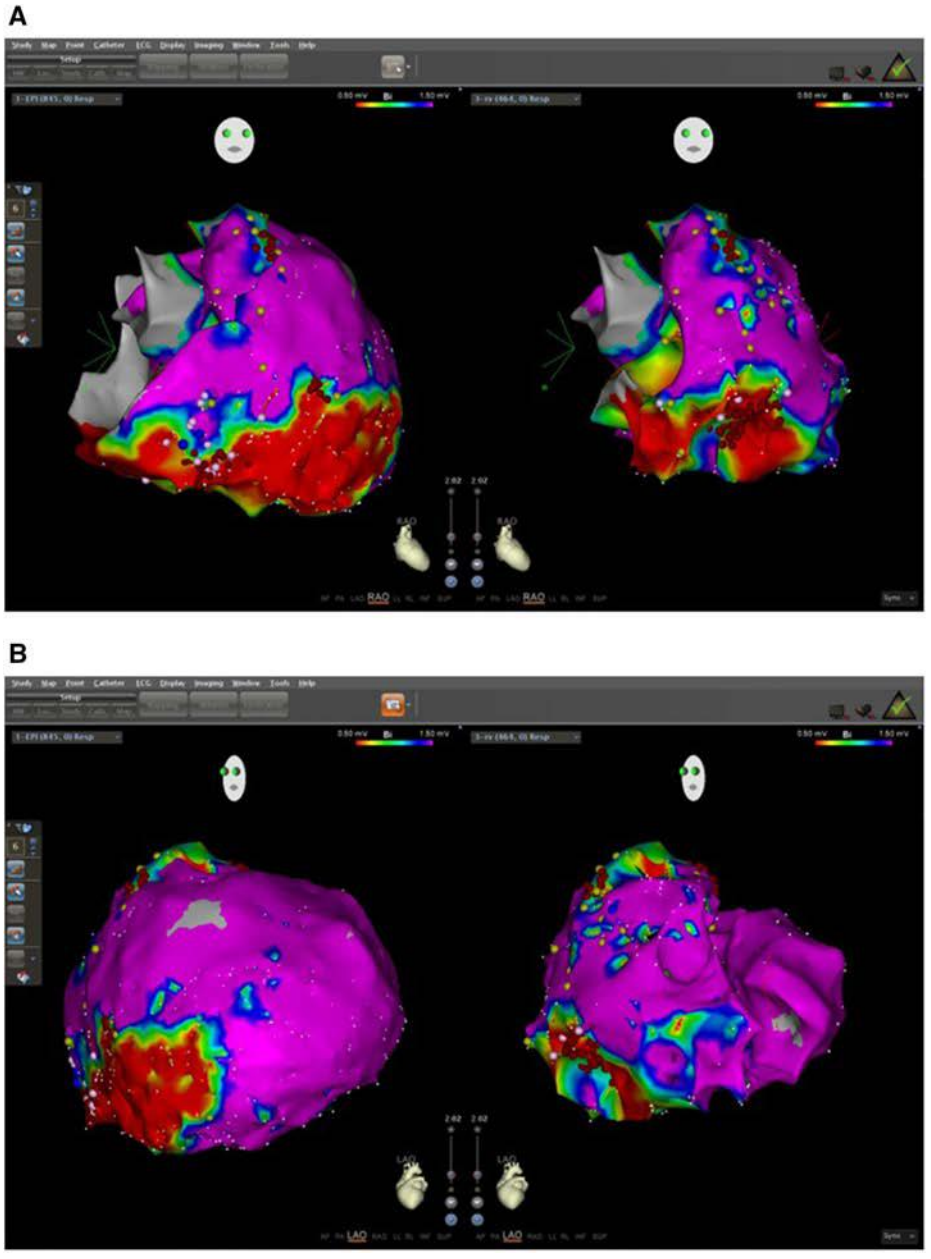

Figure 4: Voltage maps of the right ventricle in a patient with ARVC in the RAO (A) and LAO (B) views. In both panels, the left image is an epicardial map and the right image is an endocardial map. Purple indicates areas of normal voltage and red indicates areas of low voltage (scar). Transition zones are indicated by multicolored areas. ARVC: arrhythmogenic right ventricular cardiomyopathy; LAO: left anterior oblique; RAO: right anterior oblique.

described and improve long-term outcomes when compared to the endpoint of non-inducibility of clinical arrhythmias.[38,39]

The use of hemodynamic support

The use of hemodynamic support during ablation with percutaneous left ventricular assist devices allows for ablation of tachycardias that would otherwise be poorly hemodynamically tolerated. This is particularly important when activation and entrainment mapping of the tachycardia is desired. However, given the increase in cost and complication rates[40] when using hemodynamic support devices, their use should be carefully considered. Devices commonly used for this purpose include the TandemHeart (Cardiac Assist, Inc, Pittsburg, PA) and Impella (Abiomed Inc, Danvers, MA) devices. Intra-aortic ballon pumps (IABPs) are less helpful because of the difficulty in timing of inflation of the balloon with the tachycardia. Reddy et al.[40] evaluated the relative effectiveness of IABP, TandemHeart, and Impella devices at assisting with ablation of VT. They found that when operators used either a TandemHeart or Impella versus IABP that more unstable VTs could be mapped and

ablated (1.05 ± 0.78 versus 0.32 ± 0.48; $p < 0.001$), more VTs could be terminated with ablation (1.59 ± 1.0 versus 0.91 ± 0.81; $p = 0.007$), and fewer VTs required shocks to terminate the rhythm (1.9 ± 2.2 versus 3.0 ± 1.5; $p = 0.049$). The complication rate in the group using the TandemHeart or Impella devices was greater (32% versus 14%, $p = 0.0143$). Similar benefits have been seen when performing ablations with implanted left ventricular assist devices or cardiopulmonary bypass.[41]

Epicardial VT ablation

Because myocardial scar is a three-dimensional substrate, identification of a critical isthmus or the entirety of late activation potentials is not always possible by mapping the endocardial surface alone. This occurs when critical portions of the tachycardia circuit traverse the mid-myocardial or epicardial ventricular tissue. Pre-procedural clues to an epicardial origin of a tachycardia include epicardial scar on MRI and certain QRS characteristics during VT. Increases in total QRS duration, pseudodelta wave, intrinsicoid deflection, and shortest precordial lead RS complex are associated with epicardial exit points.[42] In addition, transmurality of scar on pre-procedure CT, MRI, or scintigraphy suggests patients may benefit from an empiric epicardial approach.[43] During endocardial mapping, identification of low voltage unipolar endocardial signals have been associated with epicardial VT substrate.[44]

Epicardial access is typically obtained using a subxiphoid approach. Anticipation of the area of interest for ablation will dictate whether an anterior- or posterior-biased approach is taken when obtaining access. While the use of a Touhy needle is often used for access, the recently described use of a micropuncture technique is associated with fewer access-related complications.[45] Although right ventricular puncture is understandably the most common complication of epicardial access, other unusual complications can include liver injury, intraperitoneal bleeding, and coronary lacerations.[46] Once guidewire access of the pericardial space is obtained, a deflectable sheath can be inserted to allow for fairly easy access to most components of the epicardial surface. Voltage maps are created using guidelines similar to those used when mapping the endocardial surface.

Additional considerations unique to mapping the epicardial surface are the development of pericardial or pleural effusions and injuries to the phrenic nerves and coronary arteries. Identifying and recording regions of phrenic nerve capture when pacing from the ablation catheter can help prevent phrenic nerve injury during ablation, which can be permanent when it occurs. Additional techniques have been described to physically deflect the phrenic nerve when it lies on an area of interest for ablation.[47] Coronary angiography is recommended to outline the epicardial course of large coronary branches, particularly when ablating near the ventricular base.

The need for epicardial ablation varies depending on the arrhythmogenic substrate. While a portion of patients with ischemic cardiomyopathy may require epicardial ablation,[43] patients with arrhythmogenic right ventricular cardiomyopathy[48] and non-ischemic cardiomyopathy[49] have a higher likelihood of requiring this approach. Using a combination of endocardial and epicardial ablation during the same procedure is often required. Di Biase et al.[50] showed that concomitant epicardial and endocardial scar homogenization during a single procedure reduced the recurrence of arrhythmia from 47% to 19% ($p = 0.006$) (mean follow-up 25 months).

Complications of ablation

The rate of complications from ablation can vary widely depending on patient characteristics, approach to ablation (endocardial versus epicardial), the use of ancillary equipment (hemodynamic support devices), and center experience. Complications may include vascular injury, myocardial infarction, stroke, tamponade, or death. One large retrospective review of hospital discharge records found that adverse events occurred in 8.5% of patients, major adverse events in 3.0%, and death in 1.1% of patients. Centers with >25 procedures/year have fewer complications than centers with lower volumes (6.4% versus 8.8%, $p = 0.008$).[51]

Outcomes after VT ablation

The precise impact of VT ablation on mortality is not clear. In the Euro-VT trial,[26] which evaluated the effectiveness of ablation on 63 patients with ischemic cardiomyopathy, no deaths occurred acutely or within 30 days of follow-up. In a larger trial of 231 patients with ischemic cardiomyopathy, the procedural mortality was 3% with a 1-year mortality of 18%.[28] Tung et al.[52] evaluated a large international cohort of ischemic and non-ischemic cardiomyopathy patients undergoing ablation and found a 10% mortality rate at 1 year. Another 3% of patients underwent cardiac transplantation.

Dinov et al.[29] compared outcomes of VT ablation in patients with non-ischemic versus ischemic cardiomyopathy. They prospectively evaluated 227 patients (28% non-ischemic) undergoing catheter ablation for VT over a 1-year follow-up period. VT-free survival was 40.5% in patients with non-ischemic cardiomyopathy versus 57% in patients with ischemic disease. Other data suggest similar outcomes after ablation in non-ischemic versus ischemic patients.[30]

Several predictors of favorable outcomes after ablation have been identified. Non-inducibility has been associated with improved arrhythmia-free mortality and all cause mortality in both ischemic[29,53] and non-ischemic patients.[29,54] Tung et al.[52] found that freedom from VT recurrence after ablation is associated with improved transplant-free survival at 1 year.

Finally, timing of ablation may be important on patient outcomes after ablation. Dinov et al.[55] evaluated the impact of early referral for ablation on acute and long-term outcomes. They compared patients undergoing ablation within 1 month, between 1 month and 1 year, and >1 year after the first documented episode of VT.

Acute success (non-inducibility) was achieved in >70% in all three groups. However, the group undergoing early ablation had a lower rate of VT recurrence in 2 years of follow-up (37.3% versus 61.9% and 64.5% respectively). While recurrence-free survival was highest in the group undergoing early ablation, overall survival was not significantly different between groups. Only VT recurrence predicted increased mortality (HR 1.91; $p = 0.037$).

Ablation versus medical therapy to reduce ICD shocks

There are limited data directly comparing the effectiveness of catheter ablation to medical therapy in the treatment and prevention of ICD shocks. This first such study was published in 1998 in abstract form only. Epstein et al.[56] evaluated 73 patients randomized to undergo catheter ablation versus 32 patients treated with medical therapy. The overall cohort was predominantly ischemic, with drug-refractory VT (mean number of drugs failed >2). In a 6-month follow-up period, the ablation group experienced a 49% VT recurrence rate versus 75% in the medical therapy arm.

Two prospective clinical trials evaluating the effect of catheter ablation of ventricular tachycardia on the frequency of ICD shocks are the VTACH and SMASH-VT trials. The VTACH trial[57] randomized patients with prior myocardial infarction, ejection fraction <50%, and a history of stable monomorphic ventricular tachycardia to catheter ablation followed by ICD implant versus ICD alone. The ablation strategy for this trial was targeted ablation of the clinical arrhythmia with an endpoint of non-inducibility of the rhythm. For unstable rhythms, substrate-based ablation was performed. The primary endpoint was time to first episode of VT/VF. The time to recurrent VT/VF was significantly longer in the ablation group compared to the control group (18.6 months versus 5.9 months). Furthermore, after 2 years of follow up, the percentage of patients free of recurrent VT/VF was significantly higher in the ablation group (47% versus 29%). The number of appropriate ICD shocks per patient/year was significantly higher in the control group (3.4 versus 0.6). Complications from the ablation procedure were uncommon.

Secondary endpoints of VT storm, syncope, or death were not different between groups in the VTACH trial. Interestingly, the primary endpoint was driven strongly by significant improvement in outcomes in the ablation group in patients with ejection fractions >35%, while those patients with lower ejection fractions did not experience a significant difference in the primary endpoint.

The SMASH-VT trial[58] was a three-center study that evaluated the efficacy of catheter ablation in patients with prior myocardial infarction who were undergoing ICD implantation for ventricular fibrillation, unstable ventricular tachycardia, or syncope with inducible VT. Patients were randomized to substrate-based ablation coupled with ICD implantation versus ICD alone. The primary endpoint was survival free from appropriate ICD therapies for VT/VF with secondary endpoints of appropriate ICD shock, death, or VT storm. After 2 years of follow-up, 12% of patients in the ablation group received appropriate ICD therapy compared with 33% in the control group. There was a trend towards a decrease in mortality in the ablation arm that did not reach statistical significance. Complications from the ablation procedure were uncommon.

Importantly, the rates of VT/VF in the control arms of VTACH and SMASH-VT were significantly different (71% versus 33%) over similar follow up periods. The reason for this difference is not clear. VTACH and SMASH-VT have several other factors that limit their applicability to general clinical practice. First, they both included patients with prior myocardial infarction and did not include patients with non-ischemic cardiomyopathy. Second, they used different ablation strategies, with VTACH focusing on ablation during the clinical tachycardia and SMASH-VT using a substrate-based ablation during sinus rhythm.

Bunch et al.[59] performed a retrospective comparison of patients with appropriate ICD shocks treated with medical therapy versus ablation. This study included patients with both ischemic and non-ischemic cardiomyopathy. They found that patients with ICD shocks treated with medical therapy alone had increased mortality and higher rates of hospitalization for congestive heart failure than patients treated with ablation. The mortality rates in the ablation group were similar to an additional cohort that had ICDs but no history of shocks. This study is limited by retrospective design, the possibility of selection bias for patients receiving ablation, and the inability to accurately quantify complication rates from ablation procedures.

A meta-analysis of available data evaluating the efficacy of ablation as an adjunct to medical therapy for VT found an overall 38% reduction in the number of shocks in patients treated with ablation compared with medical therapy alone (relative risk 0.62; 95% CI 0.51–0.76; $p < 0.001$). There was a non-significant trend towards improvement in mortality with ablation. Procedural complication rates were 6.3%.[60]

Emerging data

The STAR-VT (Substrate Targeted Ablation using the FlexAbility™ Ablation Catheter System for the Reduction of Ventricular Tachycardia) trial is currently enrolling patients. This trial is a prospective, randomized, multicenter trial evaluating the safety and efficacy of empiric ablation of VT in patients identified with inducible or spontaneous monomorphic VT. The primary endpoint of the trial is freedom from any ICD shock at 1 year. Secondary endpoints are number of cardiovascular hospitalizations or emergency room visits. Anticipated enrollment is 1,450 patients in 50 US centers (with additional international centers). Patients will be randomized 1:1 to receive either ICD plus medical therapy (control) or the addition of VT ablation (therapy). The STAR-VT trial is unique in that it will include patients with both ischemic and non-ischemic cardiomyopathy

Table 2: Key trials comparing catheter ablation of ventricular tachycardia to medical therapy in reducing ICD shocks

Study	Epstein et al.[56] (abstract)	Reddy et al.[58]	Kuck et al.[57]	STAR-VT
Design	Randomized, prospective; 83% had prior MI. Catheter ablation versus medical therapy.	Randomized, prospective. Prior MI, spontaneous VT/VF. Catheter ablation plus ICD versus ICD alone.	Randomized, prospective. Prior MI, EF $\leq$50%. Catheter ablation plus ICD versus ICD alone.	Randomized, prospective
Location	United States	United States	Europe	International
Major endpoints	VT recurrence	Survival free from ICD therapy	Time to first VT/VF recurrence	Freedom from ICD shocks
Follow-up	6 months	22.5 $\pm$ 5.5 months	22.5 $\pm$ 9.0 months	Unknown
Key findings	VT recurred in 49% of ablation group versus 75% of medical therapy group at 6 months.	31% of control group versus 9% of ablation group received shocks. No significant difference in mortality.	Survival free from VT/VF in 47% of ablation group versus 29% of control group at 2 years.	Currently enrolling

EF: ejection fraction; ICD: implantable cardioverter-defibrillator; VT: ventricular tachycardia.

and will evaluate both endocardial and epicardial ablation. The catheter used in this trial is a novel irrigated catheter with a flexible mesh tip that allows for preferential irrigation on the aspect of the catheter tip that is best in contact with the myocardium. Enrollment in STAR-VT is anticipated through 2016.

Key trials comparing ablation to medical therapy in the reduction of ICD shocks are highlighted in Table 2.

Conclusion

While ICDs provide proven survival benefit in patients with cardiomyopathy, this benefit comes at the expense of painful shocks. These shocks have negative psychological and medical consequences. While medical therapy may reduce the likelihood of recurrent shocks, ventricular tachycardia ablation provides an additional therapeutic strategy. The role of this procedure in reducing ICD shocks is evolving, with additional data forthcoming.

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