How We Ablate Ventricular Tachycardia in Non-ischemic, left Ventricular Cardiomyopathy

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ABSTRACT. The ablation of ventricular tachycardia (VT) in non-ischemic cardiomyopathy (NICM) is one of the most challenging procedures in electrophysiology. As survival with NICM continues to improve, electrophysiologists will undoubtedly be faced with increasing numbers of patients with refractory VT. In this article, we outline our current approach to VT ablation in this population at the hospital of the University of Pennsylvania. We approach each procedure first with a thorough pre-procedural evaluation with the goals of optimizing the patient’s clinical status and non-invasive characterization of the underlying substrate. During the procedure itself, it is critical to distinguish the substrate in as much detail as possible from the endocardium, as scar in NICM may be patchy and frequently involves the mid-myocardium and/or epicardium. In all cases, we aim to achieve as much as possible from the endocardium, even though an epicardial approach will also be required in a substantial proportion. Entrainment mapping of VTs in NICM can only be accomplished in a minority of cases due to multiple induced morphologies and hemodynamic instability. In these instances, a careful lesion set must be developed with the goal of interrupting critical components of the VT circuit(s).

KEYWORDS. dilated catheter ablation, tachycardia, ventricular cardiomyopathy.

Introduction

The technology and techniques available for ventricular tachycardia (VT) ablation have grown considerably in recent years and, as a consequence, the success rates for catheter ablation of VT have also improved. Much of the focus has centered on ablation of VT in patients with ischemic heart disease, in whom there is a relatively fixed, well-defined, endocardial substrate. Increasingly, however, these lessons are being adapted to ventricular ablation in other cardiomyopathies, particularly in non-ischemic cardiomyopathy (NICM).

Sustained, monomorphic VT is less common in NICM than among patients with ischemic heart disease. Nonetheless, the rate of appropriate implantable cardioverter-defibrillator (ICD) discharges among patients in the DEFINITE trial, who had pre-existing non-sustained VT or frequent premature ventricular complexes, approached 9% per year. Nor is VT always a manifestation of end-stage heart failure; many patients with NICM have well-compensated heart failure, with VT being the dominant problem affecting quality of life. Improvements in pharmacologic and non-pharmacologic therapy for NICM continue to extend patient survival for this condition. As a consequence, the NICM population at risk for the development of VT will continue to increase and VT in this setting will increasingly be encountered by electrophysiologists.

Ablation of VT in NICM is challenging. Among published series, the success rates have ranged between 50% and 70%, which is less than that reported for ischemic VT. There are still enormous gaps in our knowledge of the pathophysiology of VT in NICM, and the technology available for ablation of VT in these patients is far from perfected. Notwithstanding these obstacles, ablation is frequently the only available therapy for patients who have failed antiarrhythmic therapy. The purpose of this review is to outline our
current approach to ablation of VT in patients with NICM. We will focus our discussion on left ventricular NICM, including idiopathic and secondary cardiomyopathies, but many of the principles may be equally applied to ablation of right ventricular disease processes and ischemic heart disease.

Pre-procedure evaluation

Our pre-procedural assessment focuses on two essential goals: 1) optimizing clinical status prior to ablation, and 2) non-invasive evaluation of the arrhythmia substrate.

Optimization prior to ablation

Many patients with NICM and VT will have concurrent acute exacerbations of heart failure. The exacerbation of heart failure may be the triggering event for VT in some, whereas in others therapy for VT (by virtue of negative inotropy) or VT itself may have led to decompensation. Whatever the causal link, it is imperative that cardiac function, and particularly volume status, be optimized prior to ablation. We routinely involve the heart failure service in the pre- and post-procedural care of patients with severe NICM presenting for VT ablation. Some patients will require admission to the coronary care unit for tailored hemodynamic therapy prior to ablation. The decision about the optimal timing of ablation is complex and must weigh the risks of ongoing VT and its associated therapies with the need for adequate cardiac optimization.

Patients with NICM frequently have indications for chronic anticoagulation. If epicardial or right ventricular (RV) mapping, with their inherently higher risk of bleeding complications, is anticipated then we reverse their anticoagulation prior to the ablation, with the need for bridging therapy assessed on an individual basis. Where possible, we try to hold any antiarrhythmic drugs (AADs) for five half-lives prior to the procedure to maximize inducibility of VT. This is not a rigid approach and AADs are often continued in the presence of VT storm or if the AAD has rendered the VT more tolerable from a hemodynamic point of view.

Substrate

Our assessment of the arrhythmia substrate begins with the 12-lead electrocardiogram (ECG). The underlying rhythm can provide useful clues. The presence of advanced atrioventricular block should raise the suspicion for septal involvement of the disease process. The presence of a left bundle branch block (LBBB) also implies septal or diffuse left ventricular involvement, with increased QRS durations associated with a greater burden of fibrosis. Transmural fibrosis is not typically seen in NICM; nevertheless, Q waves on the underlying ECG indicate severe fibrosis, regardless of the underlying etiology. The 12-lead ECG of VT, when available, is of critical importance. First, it localizes the exit site, which helps to delineate the likely underlying substrate abnormality. Measures of delayed intrinsocoid deflection can provide evidence of an epicardial exit, but the accuracy of these findings is impaired in the presence of AADs, especially in the acute setting. We rely more on morphologic characteristics of the tachycardia and specifically on the presence of Q waves or QS complexes to suggest an epicardial exit (Figure 1). In the absence of myocardial infarction, Q waves in leads I or AVL suggest a lateral epicardial exit, whereas Q waves in leads II, III or AVF suggest an inferior epicardial exit. The common form of bundle branch re-entry should be suspected for any LBBB morphology VT with a leftward or left superior axis, especially if similar in morphology to any underlying LBBB.

We obtained a baseline transthoracic echocardiography in all patients to assess LV size and function as well as to identify potential secondary causes of LV cardiomyopathy such as amyloidosis or non-compaction. An accurate assessment of LV diastolic dimensions is helpful in selecting the appropriate ablation catheter curve. If the presence of LV thrombus cannot be ruled out with standard imaging then echo contrast is used. The presence of concomitant valvular disease that may preclude a retrograde approach to the LV or the insertion of an intra-aortic balloon pump (IABP) is also assessed.

Where possible, we also obtain pre-procedural cardiac magnetic resonance imaging (MRI) to evaluate areas of delayed gadolinium enhancement indicative of extensive fibrosis. Cardiac MRI can not only localize the presence of fibrosis but also further characterize the distribution as involving the endocardium, mid-myocardium and/or epicardium. When extensive mid-myocardial or epicardial delayed enhancement is seen, the likelihood of epicardial ablation to achieve arrhythmia control increases dramatically. Although not routinely available in all centers, we perform cardiac MRI even in patients with pre-existing devices. The relative safety of 1.5-tesla imaging in the presence of ICDs has been previously documented.

If prior coronary artery evaluation has not been performed previously, we obtain coronary angiography prior to the ablation to rule out significant concomitant stenoses that may influence tolerability of VT induction. In general, functional non-invasive stress imaging is inadequate to rule out coronary stenoses in patients with significant LV dysfunction. If epicardial mapping and ablation are anticipated, then coronary angiography may be deferred until the ablation procedure.

Intraprocedure considerations

Anesthesia

At our center, there is ongoing debate among the electrophysiologists about optimal anesthesia for ablation of VT. We routinely employ conscious sedation with a short-acting opioid as the dominant agent. A nurse anesthetist is present for the duration of the procedure. The advantages of conscious sedation relate primarily
to improved hemodynamic stability with relatively slow VT, allowing for entrainment mapping. Although never rigorously proven, enhanced inducibility is also more likely with sedation than with general anesthesia. Nevertheless, conscious sedation has risks including: 1) a greater potential for patient movement either spontaneously or with cardioversion/defibrillation with subsequent potential loss of mapping information, 2) an increased risk of aspiration, and 3) a decreased likelihood that the patient will tolerate extremely long procedures from a comfort perspective. The need for epicardial access does not preclude the use of conscious sedation. Although epicardial access is easier during apnea (with standard positive pressure ventilation) or jet
ventilation, it can be performed with reasonable safety during spontaneous respiration.

In general, we attempt conscious sedation as our first option, when feasible. Certainly if the patient has a difficult airway, is prone to obstruction, or is susceptible to aspiration, then conscious sedation is not an option. The nature of the clinical VT(s) may also dictate which anesthetic approach is used. If there are known to be multiple clinical VT morphologies that are poorly tolerated, then general anesthesia is favored. Of course any anesthetic staff for the procedure should ideally have familiarity in providing anesthesia to patients with severe cardiac dysfunction.

**Access and equipment**

When possible, we use the retrograde approach to the left ventricle. A 9F arterial sheath is placed to accommodate an 8F irrigated ablation catheter while also allowing for reliable arterial pressure tracings. We favor braided sheaths, either short (24 cm) or long (65 cm—for very tortuous or diseased aortoiliac vessels) to facilitate transmission of torque to the catheter tip. Venous sheaths are also inserted to accommodate RV apical and His quadripolar catheters and the intracardiac echocardiography (ICE) catheter. We place an additional 8F venous sheath to allow for RV endocardial mapping or epicardial mapping via the cardiac venous system. Anticoagulation with heparin is initiated to maintain an activated clotting time (ACT) between 250 and 350 s for LV endocardial mapping and ablation.

We use ICE for all procedures to assess anatomy, assist with ablation, and for continuous monitoring of any intracardiac complications during the procedure. The real-time anatomical information provided by ICE can assist greatly in catheter positioning and ensuring good endocardial contact at the time of ablation (Figure 2; showing good contact with or without lesion in NICM). ICE is also essential for accurately mapping the papillary muscles and aortic cusps.

**Hemodynamic assessment and support**

We routinely perform a right heart catheterization at the beginning of any VT ablation procedure if the patient has any component of current or recent decompensated heart failure. Such baseline measurements provide a benchmark to assess how the procedure is being tolerated from a hemodynamic perspective at later time points. In high-risk patients, we leave the PA catheter in place for the duration of the procedure. VT ablation entails the exposure of the patient to many insults that may impair cardiac contractility, including anesthetic agents, volume loading with irrigated ablation, and recurrent cardioversion/defibrillation. We pay close attention to negative trends in pulmonary pressures, wedge pressure and cardiac output. If a negative trend cannot be quickly corrected, then the procedure is aborted.

The use of IABPs plays an important role in VT ablation in ischemic heart disease but may also provide enhanced afterload reduction in NICM. The presence of an IABP does not facilitate mapping of poorly tolerated VTs but may allow for more rapid recovery of cardiac function after termination of VT. We place IABPs up front in patients with NICM who are at high risk of decompensation with repetitive inductions of VT. It should be emphasized that a retrograde approach to the LV can still be used in the presence of an IABP.

The advent of percutaneous, mechanical assist devices can allow for mapping of hemodynamically unstable VT. Our center has performed procedures with active mechanical support with success, although a transseptal approach to the LV must be used. However, given their resource-intense nature, we reserve these devices for patients who have failed conventional ablation and who have multiple, poorly tolerated VT morphologies.

**Characterization of the substrate from the LV endocardium**

Even when epicardial mapping and ablation are anticipated, it is our practice to gain as much information and accomplish as much mapping and ablation from the endocardium as possible. Endocardial mapping and ablation are safer for the patient and pose fewer logistical concerns than epicardial mapping and ablation.

We typically begin by performing LV voltage mapping. A detailed electroanatomic map is of critical importance in NICM, where voltage abnormalities may be patchy rather than confluent. Particular care must be
taken to identify the valvular structures, by electrogram (EGM) or by ICE, as fibrosis in NICM typically extends from the atrioventricular groove or aortic annulus. For bipolar endocardial mapping of either the left ventricle or right ventricle, we use the standard voltage cutoff of <1.5 mV to indicate abnormality. It should be realized, however, that this cutoff has not been validated in patients with NICM and may be insensitive at identifying patchy or mid-myocardial scar. Therefore, we pay close attention to the presence of fractionation or late potentials, even when the bipolar endocardial voltage is normal. Sites of abnormality are tagged for later reference. Unipolar voltage maps provide critical additional information in NICM, owing to the ability to detect an epicardial scar (and likely mid-myocardial scar by extension) from the endocardium (Figure 3). As previously validated by our institution, a unipolar cutoff of <8.3 mV for the LV endocardium is used to identify abnormal signals. When bipolar and unipolar maps are compared in NICM, the unipolar abnormality is typically much larger.

Previous work by our institution has identified two predominant patterns of scar in NICM, which may overlap. The first pattern is involvement of the basal lateral wall, and the second is involvement of the interventricular septum. Fibrosis typically extends from the atrioventricular (AV) groove/perivalvular area. Exceptions do exist and may represent more focal disease processes, such as focal myocarditis or sarcoidosis. Even if bipolar voltages are normal, a unipolar abnormality on the basal lateral wall alerts us to the very likely need for epicardial mapping and ablation. Similarly the presence of septal scar means we will likely need to map the RV septum, and possibly the epicardium. Before proceeding to epicardial puncture, we attempt to first map the coronary venous system. If difficulty is encountered, we use coronary sinus venography to facilitate advancing the ablation catheter to areas of interest.

**Induction, mapping, and ablation of VT**

Most VT in NICM is re-entrant with the majority being scar related. Nonetheless, it is important to remember that a significant proportion of patients may have either bundle branch re-entry (BBR) VT or a focal tachycardia.

**Figure 3: Endocardial bipolar and unipolar voltage maps in NICM with septal involvement.** The bipolar and unipolar endocardial voltage maps for the LV septum and RV septum in a patient with NICM are displayed. Note the bipolar scar is patchy but the unipolar map suggests more extensive fibrosis of the mid-myocardium. The bipolar voltage cutoff for scar in the RV and LV was 1.50 mV. The unipolar voltage cutoff for scar was 5.5 mV in the RV and 8.3 mV in the LV. Abbreviations: LV, left ventricle; RV, right ventricle; AV, aortic valve; MV, mitral valve; PV pulmonic valve.
Any atypical features of VT, such as acceleration, non-reproducible induction with programmed stimuli or frequent initiation and termination prompt us to formally evaluate the arrhythmia mechanism.

The presence of a His catheter provides valuable information on retrograde His activation and the sequence of right bundle branch activation during suspected BBR. We routinely entrain from the RV apical catheter to ensure that a diagnosis of BBR is not missed.

While entrainment mapping remains our preferred choice of mapping strategy in NICM, it can be difficult to accomplish for a variety of reasons. First, patients with NICM often have multiple inducible VT morphologies and, in our experience, frequently switch morphologies with attempts at entrainment. Furthermore, the substrate is less likely to consist of a large dense scar, meaning slow and tolerated VTs are the exception, with most inducible VTs being poorly tolerated. When entrainment is possible, it is important to establish whether any part of the endocardium is involved in the circuit. Unlike, ischemic cardiomyopathy, outer loop sites provide valuable information. For VTs originating from the basal lateral wall, we perform entrainment at sites around the mitral annulus, even in areas of normal voltage. If sites around the mitral annulus are outer loop for one or more VTs, even when no protected sites are identified on the endocardium, then we focus ablation in areas of abnormal substrate on the lateral wall and link this to the mitral annulus. Similarly, demonstration that the superior or inferior septum is an outer loop for one or more VTs suggest the protected components may lie within the septum, even if they cannot be demonstrated from the endocardium. We then focus our ablation efforts on the septum from the left ventricle and the right ventricle. It is important to remember that, even when one component of the circuit is demonstrated on the endocardium, the area of slow conduction may be transmural with other protected sites in the mid-myocardium or epicardium.

When entrainment is not possible, we rely on pace mapping to guide substrate ablation. At any site of abnormal signal during mapping, endocardial or epicardial, we pace (preferably at the same cycle length of the clinical tachycardia) to aid localization of the exit site for any known VT. As in ischemic cardiomyopathy, we also try to identify areas with long stimulus-to-QRS intervals that may represent critical parts of a macro-reentrant circuit. As the scar in NICM most commonly originates from the base, we attempt to produce lesion sets incorporating exit sites but that are also confluent with the mitral or aortic annuli.

We exclusively use bidirectional, irrigated ablation catheters with power of 30–50 Watts and temperature limits of 45°C. On the LV endocardium, we target impedance drops of 12–18 Ohms, and, in areas of mid-myocardial scar, continue ablation for 120–240 s to achieve maximum lesion depth.

**Epicardial mapping and ablation**

Our general philosophy is to obtain as much information and perform as much ablation as possible from the endocardium. Nonetheless, it is not infrequent that patients with NICM will have minimal endocardial substrate abnormalities and no critical sites for VT identified on the endocardium.

However, the decision to proceed with epicardial mapping and ablation has many logistical concerns. If extensive LV endocardial ablation has been performed, is it safe to reverse and interrupt anticoagulation or will the patient be exposed to an increased risk of embolic complications? If the procedure has already been lengthy, how much longer will the patient tolerate mapping and ablation? Although ideally we like to proceed with epicardial mapping and ablation during the same procedure, occasionally it is more prudent to delay accessing the epicardium as a separate procedure later that admission.

We obtain epicardial access using the Sosa method, which has been previously well described. We place a quadriipolar catheter at the true RV apex, as visualized by ICE, as a radiographic marker for the endocardium. To avoid injury to the right ventricle, we aim for puncture lateral to the RV catheter in the left anterior oblique (LAO) projection. Typically, we use a posterior approach to avoid coming near the acute angle of the RV. An extra-stiff J guidewire is advanced into the epicardium and we look to see that the wire spans the cardiac silhouette in the right anterior oblique and LAO views to ensure that it has not been inadvertently placed in the endovascular space. After confirmation, a 23 cm 8F sheath is advanced into the epicardial space. We employ a 40 cm steerable, short-curved sheath if catheter manipulation is difficult.

When mapping the epicardium, voltages of <1.0 mV are considered abnormal. However, to differentiate fat from truly abnormal substrate, areas with fractionated/split EGMs or true late potentials, are tagged specifically within the mapping system. We focus specifically on defining the AV groove and the superior extension of ventricular myocardium extending towards the outflow tract. As with the endocardium, ideally entrainment is performed. Again, it is important to establish whether the mitral annulus is part of one or more VTs exiting on the basal lateral wall. However, only the minority of patients with dominantly septal involvement will have demonstrable targets for ablation on the epicardium. While pace mapping, we also try to define the course of the left phrenic nerve across the anterolateral, lateral and inferolateral walls of the LV.

The general strategy for substrate ablation is the same for the epicardium as for the endocardium; however, the course of the coronary arteries and the phrenic nerve must be taken into consideration (Figure 4). We develop an ideal ablation strategy, then perform coronary angiography to localize the major epicardial vessels. During angiography, the ablation catheter is left at a site of particular interest for ablation. To accurately define the course of the coronaries with respect to the substrate for ablation, typically we perform multiple injections with the ablation catheter placed at various critical positions. As yet, we have no reliable way to incorporate angiographic imaging within an electroanatomic mapping.
system. We always aim to perform ablation at least 1 cm away from any major epicardial vessel. Prior to ablation, high output pacing is performed to ensure no phrenic capture is observed. In patients with lateral epicardial fibrosis, the phrenic nerve may run through the area to be targeted for ablation. Two strategies can be employed to minimize the chance for phrenic injury: either air and/or saline can be injected directly into the epicardial space or a large valvuloplasty balloon can be placed via a second pericardial sheath. Both approaches aim to increase the distance between the visceral and parietal pericardium.

**Figure 4:** Epicardial substrate ablation targeting the basal lateral wall. This patient had multiple inducible ventricular tachycardias (VTs) and extensive basal lateral scar on the epicardium. Limited entrainment of two VTs suggested they both coursed around the mitral annulus. An isthmus site was identified for one VT (white marker) and terminated with ablation at that site. Further substrate ablation was performed around the extent of the scar, anchoring to the atrioventricular groove. The lesion set had to take into consideration the course of the left circumflex coronary artery (arrows).

**Procedure endpoints**

Our dominant endpoint for ablation is non-inducibility of VT. Complete non-inducibility of monomorphic VT, with triple extrastimuli with RV and LV endocardial stimulation, is often difficult to achieve in NICM. At a minimum, we try to ensure the clinical VT is no longer inducible.

However, non-inducibility is an imperfect measure of the success of ablation and more reproducible endpoints are sorely needed. During the procedure, we try to also demonstrate additional endpoints, where possible, to show we have adequately affected the substrate with ablation. First, we ensure non-capture of the local myocardium with high output pacing (20–50 amps at 1.0 ms) immediately after ablation. When performing substrate ablation, we often try to create areas of inexcitability within the scar using surrounding lesion sets, as shown by the inability to exit the scar with high output pacing after ablation. Elimination of late potentials within areas of dense scar is another endpoint for substrate ablation, although this can be more difficult to achieve than in ischemic scar, as late potentials may originate from the mid-myocardium.
Post-procedure care

Endocardial procedures

All sheaths are removed when the ACT approaches 180–200 s. Anticoagulation is resumed 6 h after sheath removal, as long as no bleeding complications have occurred. Anticoagulation is continued for 24–48 h. For patients without indications for long-term anticoagulation, aspirin is administered for 6 weeks post ablation.

Epicardial procedures

For epicardial procedures, a pig-tail catheter is typically left in the epicardial space; however, if the aspirates are clear it may be removed to avoid the discomfort of an indwelling catheter overnight. Corticosteroid is administered after all irrigation fluid has been aspirated, to minimize pericardial adhesions. After 15 min, the pig-tail catheter is attached to a negative pressure drain. All patients are monitored overnight in the coronary care unit and anticoagulation is held. Transthoracic echocardiography is obtained the next morning and the pig-tail, if left in place, is removed if no significant effusion is seen. Echocardiography is repeated 3–4 h after removal, and if no further fluid has accumulated anticoagulation is initiated if extensive epicardial ablation had been performed or if there are other indications present.

Post-procedure stimulation

If patients have not had a recurrence of VT at 48–72 h and they are clinically stable, then we perform non-invasive programmed stimulation under conscious sedation using their ICD. If the clinical VT remains inducible or if other VTs are readily inducible with single or double extrastimuli, then repeat ablation is considered. Given the lower success rates for VT ablation in NICM, this post-procedure testing is critical in identifying patients who would benefit from repeat procedures. The decision to continue antiarrhythmics other than beta-blockade is individualized based on the number of inducible VT morphologies during the procedure, nature of the substrate, and residual inducibility at non-invasive testing.

Conclusion

It is important to reiterate that ablation of VT in NICM is very challenging, and outcomes are not as promising as those in ischemic VT. Nonetheless, with perseverance, and frequently with multiple procedures, we have achieved reasonable success with ablation in this population. While the elimination of all inducible VT may be unrealistic in many patients, frequently elimination of the dominant clinical VTs can be achieved in order to prevent further ICD therapy.

References