CLINICIAN UPDATE

Catheter Ablation of Ventricular Tachycardia

Roderick Tung, MD; Noel G. Boyle, MD, PhD; Kalyanam Shivkumar, MD, PhD

Jentricular tachycardia (VT) most commonly develops in patients with structural heart disease. Myocardial infarction results in collagen replacement interspersed with surviving myocardium, which alters impulse propagation, facilitating re-entry.1 Aside from the postinfarction substrate, scar-mediated VT occurs in patients with nonischemic cardiomyopathy, Chagas disease, sarcoidosis, arrhythmogenic right ventricular cardiomyopathy, and postsurgical congenital heart disease. In structurally normal hearts, VT results from intracellular calcium overload or an abnormal response to adrenergic stimulation, promoting triggered activity or automaticity, respectively.

There are 3 treatment options for VT, although many patients require a combination: an implantable cardioverter-defibrillator (ICD), antiarrhythmic medications, and catheter ablation. An ICD provides abortive "rescue" therapy but cannot prevent the heart from going into VT. Antiarrhythmic therapy has limited efficacy and has the potential for multiple side effects, including proarrhythmia.²

In this Clinician Update, we discuss 3 different VT clinical scenarios that are amenable to catheter ablation to

highlight the range of substratespecific strategies used in the electrophysiology laboratory.

Case 1: Symptomatic Premature Ventricular Contractions With Cardiomyopathy

An 18-year-old man presented with palpitations and fatigue. Over a period of 5 months, he had been unable to play sports owing to dyspnea on exertion. A resting ECG demonstrated sinus rhythm with frequent monomorphic premature ventricular contractions. An echocardiogram revealed an ejection fraction of 35% with global hypokinesis. Previous treatment with β -blockers and flecainide was unsuccessful, and he was referred for evaluation for catheter ablation.

The patient underwent electrophysiological study, and activation mapping was performed in the right and left ventricular outflow tracts to locate the earliest site of origin. A single application of radiofrequency energy at the earliest site below the left coronary cusp resulted in complete abolition of the premature ventricular contractions (Figure 1).

Frequent premature ventricular contractions are an underrecognized, reversible cause of idiopathic cardiomyopathy. A correlation with the burden of premature ventricular contractions with cardiomyopathy has been reported, with higher risk at a burden of >20% on Holter analysis.3 Catheter ablation is recommended for patients with symptomatic monomorphic ventricular ectopy when medications are not effective, tolerated, or desired, particularly in those with diminished systolic function. Ablation can result in elimination of premature ventricular contractions in >80% of cases, with resolution of cardiomyopathy.4,5

Two months later, the patient had a repeat echocardiogram that showed normalization of the systolic function with an ejection fraction of 55%. His fatigue resolved, and he was able to participate in sports again.

Case 2: Recurrent Implantable Cardioverter-Defibrillator Shocks in Ischemic Cardiomyopathy

A 71-year-old man with history of inferior myocardial infarction and an ejection fraction of 25% presented to the emergency department with 4 ap-

From the UCLA Cardiac Arrhythmia Center, David Geffen School of Medicine at UCLA, Los Angeles.

Correspondence to Roderick Tung, MD, UCLA Cardiac Arrhythmia Center, David Geffen School of Medicine at UCLA, 47-123 CHS, 10833 Le Conte Ave, Los Angeles, CA 90095-1679. E-mail rtung@mednet.ucla.edu

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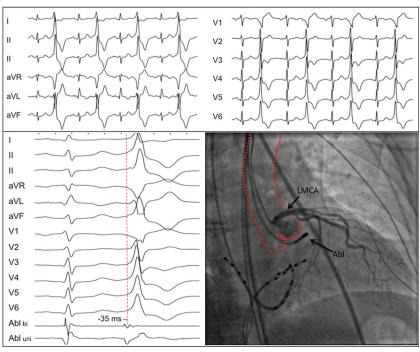


Figure 1. A 12-lead ECG of ventricular bigeminy with left bundle-branch morphology and inferior axis with early precordial transition (**top**). Earliest site of activation (**bottom right**) preceded QRS by 35 milliseconds (Abl bi) with a QS complex with unipolar recording (Abl uni). Successful ablation site (Abl) below the left coronary cusp in the aortic root (red dashed outline) shown during coronary angiography of the left main artery (LMCA).

propriate ICD shocks in a 48-hour period. Amiodarone was initiated, and the patient presented 3 weeks later with lightheadedness; device interrogation showed 35 episodes of VT at a rate of 140 bpm, which were terminated with antitachycardia pacing over the prior 10 days. The patient was referred for catheter ablation.

A basal inferolateral scar was confirmed by contrast-enhanced computed tomography scan (3-dimensional reconstruction), and electroanatomic mapping and late potentials within the scar demonstrated excellent pace-map matches (Figure 2A). Clinical VT was induced, and entrainment mapping demonstrated proof of a critical isthmus with diastolic activity. Ablation at this site resulted in prompt termination of the VT (Figure 2B). Amiodarone was discontinued, and the patient experienced an improved quality of life without any ICD therapies in the following 10 months.

Fewer than 20% of VTs are hemodynamically stable to enable mapping during VT. In these instances, activity during diastole (pre-QRS) is sought because this represents slow conduction within the scar before it exits the circuit and captures the myocardium, represented by the QRS (Figure 2B). Critical isthmuses exhibit specific responses to entrainment mapping6 (Table 1). The majority of ischemic cardiomyopathy patients have multiple inducible VTs, and when VT is not hemodynamically tolerated, a substrate-based ablation strategy dependent on the identification of late potentials (areas of slow conduction) and pace mapping is implemented. Single-center experience and multicenter registries demonstrate an efficacy of 50% to 75% at 6 to 12 months.7

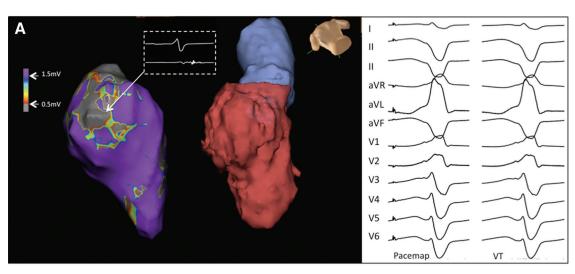
Case 3: Ventricular Tachycardia Storm in Nonischemic Cardiomyopathy With Epicardial Ablation

A 66-year-old woman with idiopathic dilated cardiomyopathy and an ejec-

tion fraction of 25% was admitted for 2 ICD shocks from her biventricular ICD and heart failure. While being treated with diuresis, amiodarone, and inotropes, the patient developed 6 ICD shocks in a 24-hour period. A lidocaine drip was added; the patient was sedated and intubated; and an intraaortic balloon pump was placed.

Because the surface ECG exhibited delayed QRS upstroke or late intrinsicoid deflection suggesting an epicardial focus, a combined epi-endo approach for mapping and ablation was undertaken (Figure 3). Epicardial access was obtained before anticoagulation with heparin following the technique described by Sosa et al,8 and endocardial access was obtained via a transseptal approach on full anticoagulation. Mapping within the pericardial space revealed a significantly greater extent of scar on the epicardium compared with the endocardium in the basal lateral region (see Figure 3). Ventricular tachycardia was induced and was not hemodynamically tolerated, requiring immediate cardioversion. Pace mapping demonstrated a better match from the epicardium than the endocardium. Epicardial ablation was performed at the site of perfect pace map. A second poorly tolerated VT was induced, and pace mapping from the endocardium in the annular scar region revealed the best match. Ablation was performed in this region, and the patient was rendered noninducible. She remained free of VT recurrence for 2 weeks, and her hemodynamic profile improved on inotropes. She was discharged home after a transition to oral medications.

The deleterious effects of ICD shocks, appropriate and inappropriate, in patients with advanced heart failure have been well documented. 9,10 Whether VT is merely a surrogate for pump deterioration or ICD shocks are directly injurious to myocardial function remains unclear. Nevertheless, recurrent VT necessitating ICD therapy is commonly seen with decompensated heart failure and vice versa.



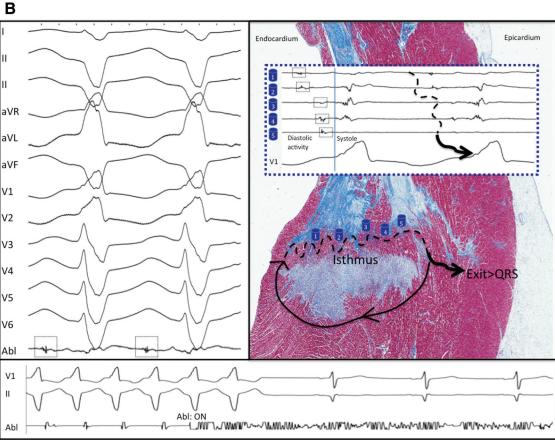


Figure 2. A, Correlation of computed tomography scan and electroanatomic map showing basal inferolateral aneurysmal scar. Left, A late potential within this scar yields a perfect pace map of the targeted ventricular tachycardia (right). B, A 12-lead ECG of ventricular tachycardia with middiastolic activity (boxes) recorded on ablation catheter (Abl; left). Theoretical construct of intramural scar-mediated reentry with diastolic activity recorded in the isthmus (electrodes 1 through 5) before exiting the circuit (bold arrow) between 2 areas of collagen (blue) on trichrome staining of an experimental infarction. Prompt termination of ventricular tachycardia during ablation (Abl:ON) at the site demonstrating concealed entrainment (bottom).

A VT storm is defined as >3 episodes of VT within a 24-hour period. Treatment with intravenous amiodarone, lidocaine, and/or procainamide is first line. Sedation and insertion of an intra-aortic balloon pump are often necessary to decrease adrenergic stimulation and to optimize hemodynam-

ics. In this setting, titration of inotropes must be done with caution. Neuraxial modulation has been shown to be effective in cases refractory to

Table 1. Mapping Techniques for Catheter Ablation of Ventricular Tachycardia

Hemodynamically stable VT

Activation mapping

Idiopathic (triggered or automatic): earliest site of origin

Scar-mediated (reentry): diastolic activity

Presystolic (<30% TCL)=exit

Middiastolic (30%-70% TCL)=isthmus

Early diastolic (>70% TCL)=entrance

Entrainment mapping of isthmus

Concealed fusion

PPI=TCL

S-QRS=EGM-QRS

Hemodynamically unstable VT

Electroanatomic substrate mapping/scar delineation

Pace mapping

Targeting of late potentials

Linear ablation lesions sets

Scar border zones

Scar transection

Connecting scars and anatomic

boundaries, ie, annulus

Mechanical hemodynamic support, ie, IABP, LVAD

VT indicates ventricular tachycardia; TCL, tachycardia cycle length; PPI, postpacing interval; S-QRS, stimulus to QRS; EGM-QRS, electrogram to QRS; IABP, intra-aortic balloon pump; and LVAD, left ventricular assist device.

conventional treatment.¹¹ When control of arrhythmia cannot be achieved, bridging mechanical support, ie, ventricular assist device or extracorporeal membrane oxygenation, may be undertaken to stabilize patients for catheter ablation (Table 2).

Ablation of VT in the setting of a storm has been shown to be effective. ¹² In cases of nonischemic cardiomyopathy, fibrosis tends to be patchier and more basal with variable mural involvement; fewer late potentials are found within scar. ^{13,14} Epicardial scar is frequently more extensive than endocardial scar, and epicardial mapping with ablation is an important adjunct for successful VT ablation. ¹⁵ In cases with prior chest surgery, a limited thoracotomy incision may be neces-

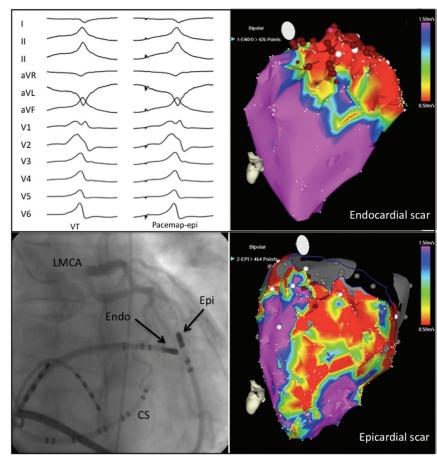


Figure 3. A 12-lead ECG of clinical ventricular tachycardia with a perfect pace map of the ventricular tachycardia from the epicardium. Top left, Combined epicardial (Epi) and endocardial (Endo) mapping in the left anterior oblique projection (bottom left). A coronary sinus (CS) catheter is shown. Electroanatomic mapping demonstrates a greater extent of epicardial (bottom right) scar compared with endocardial scar (top right). Red circles represent areas of radiofrequency application. LMCA indicates left main coronary artery.

sary to access the pericardium and to release adhesions. 16,17

Catheter ablation of VT has evolved significantly over the past 2 decades with conceptual and technological advancements. Patients with advanced

Table 2. Management of Ventricular Tachycardia Storm

B-blockade

Antiarrhythmic drug therapy

Intubation, deep sedation

Mechanical hemodynamic support, ie, IABP, LVAD

Neuraxial modulation: thoracic epidural anesthesia, left stellate ganglionectomy

Catheter ablation

IABP indicates, intra-aortic balloon pump; and LVAD. left ventricular assist device.

cardiomyopathy who develop VT are at high risk for morbidity and mortality; procedural complications, which include stroke (<1%), tamponade (1% to 3%), and death (1%), have been shown to be acceptably low in experienced centers. The results of multicenter registries and Substrate Mapping and Ablation in Sinus Rhythm to Halt Ventricular Tachycardia (SMASH-VT), the first randomized trial in VT ablation,18 have prompted the paradigm shift from use of catheter ablation as a last-resort palliation to a preemptive strategy for the management of recurrent VT.

Disclosures

None.

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