Idiopathic fascicular left ventricular tachycardia: Linear ablation lesion strategy for noninducible or nonsustained tachycardia

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BACKGROUND Idiopathic “fascicular” left ventricular tachycardia (IFLVT) is frequently not inducible or nonsustained at the time of planned catheter ablation. The mechanism of the arrhythmia has been suggested to be reentry involving a sizable area of the LV inferior septum extending from base toward the apex.

OBJECTIVE We tested the ability of a series of radiofrequency lesions delivered in a linear fashion to the inferior-mid septum to control ventricular tachycardia not amenable to standard mapping ablation strategies.

METHODS Programmed stimulation both at baseline state and with isoproterenol after heart rate was increased by at least 25% was performed in all patients. The patients included in the study were either non-inducible or only had brief nonsustained VT not amenable to “traditional” mapping. A detailed electroanatomic map of the LV was performed in sinus rhythm. The location of the linear lesion along the inferior septum was guided by the presence of Purkinje potentials, with pacemapping as an additional guide. A linear lesion was placed perpendicular to the long axis of the ventricle approximately midway from the base to the apex in the region of the mid to inferior septum. Radiofrequency lesions were delivered using a 4mm tip catheter at 50 Watts and 52 degrees for 60 –90 seconds.

RESULTS Of 122 consecutive patients who underwent ablation of idiopathic VT from 1999 to 2003, 15 had IFLVT based on standard diagnostic criteria. Six of the 15 patients (40%) had nonsustained or no inducible VT in the EP lab. The number of RF lesions ranged from 7 to 15 (mean 9). The length of the effective linear lesion ranged from 1.2 to 2.2 cm (mean 1.7 cm). Development of left posterior fascicular block was noted in two of the six patients. However, despite the absence of development of left posterior fascicular block in the other four patients, no VT or premature ventricular beats could be induced after ablation using the same provocation maneuvers as performed in the baseline state. No spontaneous arrhythmias occurred during follow-up to 16 ± 8 months (range 6 to 30 months).

CONCLUSION In patients with difficult to induce or nonsustained VT with the typical right bundle branch block pattern and a superiorly directed axis on 12-lead ECG, RF energy ablation delivered in a linear fashion approximately midway to two thirds toward the apex along the mid to inferior septum and perpendicular to the plane of the septum is safe and effective for VT control.

KEYWORDS Ventricular tachycardia; Ablation; Fascicular

Introduction

Idiopathic fascicular left ventricular tachycardia was first described as an electrocardiographic entity by Zipes et al1 and subsequently as a clinical entity by Lin et al.2 The tachycardia originates in the region of the left posterior fascicle of the left bundle and is characterized by a right
bundle branch block morphology with left-axis deviation that is superiorly directed. Pharmacologic studies have found that these tachycardias are often sensitive to verapamil. The mechanism of the sustained arrhythmia has been suggested to be reentry involving a sizable area of the left ventricular inferior septum extending from base toward the apex, with the superficial endocardial Purkinje network participating in the circuit. Application of radiofrequency energy during ventricular tachycardia (VT) to the mid or inferior apical left ventricular septum targeting presystolic Purkinje potential is highly effective in treating this arrhythmia. However, a major limitation of this technique is that the VT must be inducible and sustainable to allow mapping. Unfortunately, idiopathic fascicular left ventricular tachycardia frequently is not inducible or is nonsustained at the time of planned catheter ablation. Given the anticipated anatomically determined nature and location of the VT circuit in this setting, we hypothesized that creation of radiofrequency lesions in a linear fashion targeting the endocardial mid-inferior septum of the left ventricle (LV) would be an effective strategy for targeting nonsustained or noninducible idiopathic left ventricular tachycardia.

Methods

A total of 122 patients underwent ablation of idiopathic VT at our center between January 1999 and December 2003. Of this group, 88 (72%) of the 122 patients had a site of origin localized to the outflow tract region. Of the remaining patients, 15 had fascicular VT based on standard diagnostic criteria.

Of the 15 patients with fascicular VT, nine had inducible and sustainable VTs that were successfully ablated using “traditional” methods. The remaining six patients (one female; age range 17 to 46 years) had structurally normal hearts and clinically documented VT but had only brief nonsustained or noninducible VT in the electrophysiology laboratory. Twelve-lead ECG from each of these patients documented a wide complex tachycardia with a typical right bundle branch block morphology and left-axis deviation that was superiorly directed, consistent with a left posterior fascicular VT (Figure 1). These six patients are the subject of this report.

The study patients underwent electrophysiologic evaluation after providing informed consent. All procedures were performed following the institutional guidelines of the University of Pennsylvania Health System. After discontinuation of the antiarrhythmic agents for at least five half-lives, vascular access was obtained via the right femoral vein and artery. Three standard 6Fr Josephson quadripolar electrode nondeflectable catheters were placed percutaneously via the femoral veins to the high right atrium, His-bundle region, and right ventricular apex. A deflectable 4-mm CARTO ablation catheter (Biosense-Webster, Diamond Bar, CA, USA) was placed via the right femoral artery and advanced retrograde into the LV. A bolus of heparin (80 U/kg) was
administered intravenously with access of the systemic circulation and titrated to maintain an activated clotting time between 250 and 300 seconds.

Baseline electrophysiologic measurements were obtained. Programmed stimulation at twice diastolic threshold amplitude with 2-ms pulse width from the high right atrium and the right and left ventricles was performed with up to three extrastimuli, followed by ventricular burst pacing for 15 intervals at cycle lengths of 350 to 250 ms. If VT was not induced, an infusion of isoproterenol (1–10 μg/min) was initiated and titrated until a heart rate increase >25% from baseline or maximum tolerated dose (up to 10 μg/min) was achieved. The mean isoproterenol dose was 5 μg/min (range 2–10 μg/min). Repeat programmed stimulation was performed once an adequate heart rate response was noted. If VT was induced and sustained and was hemodynamically tolerated, three-dimensional electroanatomic activation mapping was performed using the CARTO system. Right and left ventricular voltage mapping was performed during sinus rhythm to confirm the absence of abnormal myocardium characterized by confluent areas of low voltage (<1.5 mV) and to establish a template for localizing radiofrequency lesion placement. If the VT was either noninducible or nonsustained despite the described methodologies, mapping of the LV to localize regions with Purkinje potentials was performed during sinus rhythm (Figures 2 and 3). Pace mapping at sites where Purkinje potentials were recorded was performed and compared to the clinical VT (Figures 4 and 5). Attempts to reinduce VT via ventricular programmed electrical stimulation were repeated using the protocol described from the mapping catheter at the sites where Purkinje potentials were localized during sinus rhythm to confirm their presence during VT.

Mapping and ablation

A detailed (>150 points) three-dimensional electroanatomic map of the LV was performed in each patient. Radiofrequency energy lesions were placed perpendicularly to the long axis of the ventricle, approximately midway from the base to the apex in the region of the mid to mid-inferior apical septum of the LV (Josephson site 2/3), and further guided by the presence of Purkinje potentials (Figure 6). Pace mapping before each radiofrequency energy delivery was performed and compared with the clinically documented VT. However, a “perfect” pace map was not a necessary requirement for energy delivery. The maximum line length extended from the mid septum to the junction of the septum with the inferior free wall of the LV. Each radiofrequency lesion was delivered via a 4-mm-tip electrode for 60 to 90 seconds at 50 W achieving a maximum temperature of 52°C.

Programmed stimulation and isoproterenol infusion were repeated after a single point lesion in three patients and after the line was completed in all six patients. Successful ablation was defined as noninducibility of any VT or premature ventricular complexes (PVCs) both with and without isoproterenol using the same protocol as preablation.

Figure 4  Pace map obtained at the site of Purkinje potential recordings (left) closely resembling, but not a perfect match to, the clinical premature ventricular complexes (right).

Figure 5  Different patient demonstrating a perfect pace map (12 of 12 lead match) obtained at the site of Purkinje potential recordings (left) compared with the clinical ventricular tachycardia (right).
Baseline intracardiac measurements demonstrated normal AH and HV intervals. Two of the six patients had undergone previous ablation attempts at other institutions. Three of the patients had easily inducible VT with burst pacing at baseline. However, after initial activation mapping, the VT became nonsustained despite all attempts at aggressive pharmacologic stimulation and programmed stimulation. Two of the patients had only brief nonsustained VT that was induced. The last patient had VT that terminated and became noninducible while mapping along the apical inferior portion of the left ventricular septum. The number of RF lesions ranged from 7 to 15 (mean 9). The length of the effective linear lesion ranged from 1.2 to 2.2 cm (mean 1.7). Development of left posterior fascicular block was noted in two of the six patients. However, despite the absence of development of left posterior fascicular block in the other four patients, no VT or PVCs were inducible postablation using the same provocation maneuvers as performed in the baseline state and after a single point lesion in the three patients tested. Mean fluoroscopy time was 39.6 minutes (range 22–68). Mean duration time as defined by catheter insertion to time of catheter removal was 75 minutes (range 55–145). There were no procedure-related complications. Of the six patients who underwent the ablation procedure using the technique described, none experienced recurrence after follow-up of 16 ± 8 months (range 6–30). Follow-up in all but the last patient studied far exceeds the anticipated spontaneous VT frequency noted prior to the study.

**Discussion**

We describe a new technique for ablation of idiopathic left posterior fascicular VT. Although many patients have inducible sustained VT in the electrophysiology laboratory, the VTs in some patients are noninducible or are only brief and characterized by nonsustained paroxysms that preclude detailed activation mapping. Pace mapping alone can be successful in some of these patients. However, the limitations of pace mapping and the difficulty in achieving a 12 of 12 ECG match forced us to consider an alternative strategy. Based on the evidence supporting (1) a reentrant mechanism for the VT, (2) a circuit that likely is of considerable size involving the apical to basal extent of the inferior left interventricular septum, and (3) a circuit that either incorporated or was in close proximity to the Purkinje network, we hypothesized that such a circuit of considerable dimension should be effectively interrupted by a linear ablation lesion that transected this region.

In a consecutive series of six patients, we found that we could provide VT control by targeting a region of the left ventricular mid-inferior septal endocardium marked by the presence of Purkinje potentials in sinus rhythm and the best pace map match of the VT and then creating a linear lesion of significant dimension perpendicular to the long axis of the septum in this region from the mid septum to the inferior wall.

**Background and rationale**

VT occurring in the absence of structural heart disease or any identifiable cause accounts for approximately 10% to 15% of clinical VTs. An uncommon type of idiopathic VT can originate from the left posterior fascicle of the left bundle branch. Various techniques involving activation mapping and identification of the earliest pre-QRS Purkinje potentials during VT have been described, and focal ablation at these sites has been shown to be highly effective. However, many of the previously described mapping techniques are dependent on inducibility of sustained VT. Because many of the fascicular VTs involve a rather superficial circuit in the inferior apical region of the septum, it is not unusual for the VT to be rendered nonsustained or even noninducible by virtue of mechanical “bump” during the mapping process. This observation has also been described by others. Furthermore, VT sometimes is nonsustained or is not inducible despite pharmacologic provocation. These obstacles often lead to aborted radiofrequency ablation procedures or VT recurrences after initial apparent success.

The underlying mechanism of idiopathic fascicular left ventricular tachycardia is still not well understood. Although most findings have supported a reentrant mecha-
nism.\textsuperscript{14–18} Other studies have suggested triggered activity as the underlying mechanism.\textsuperscript{1,19,20} Several characteristics of idiopathic fascicular left ventricular tachycardia support re-entry as the mechanism: (1) VT can be induced and terminated by ventricular programmed stimulation, (2) an inverse relationship exists between the coupling interval of the ventricular extrastimulus and the first tachycardia return cycle, (3) entrainment with progressive fusion with shorter pacing cycle lengths can be demonstrated, and (4) VT can be entrained with concealed fusion over a region of the septum with entrance sites at the more basal septum and exit sites described toward the more mid to apical inferior septum.

Characteristics supporting a smaller reentrant or possibly a triggered mechanism include (1) the His bundle can be activated in an anterograde fashion without influencing the tachycardia and (2) the ventricle can be captured by extra-stimuli without resetting the tachycardia, suggesting that much of the ventricle does not participate in the arrhythmia circuit.\textsuperscript{1,15} A false tendon extending from the posteroinferior LV to the septum found in patients with idiopathic fascicular left ventricular tachycardia also has been postulated to be responsible for this tachycardia.\textsuperscript{21} The exact mechanism by which the false tendons may potentiate the VT is unclear, but the hypotheses include (1) conduction through the false tendon as part of the circuit and (2) stretch of the Purkinje fiber network on the interventricular septum caused by the tendon.

As indicated, characteristically for the sustained VT, detailed mapping is performed to identify the earliest pre-QRS Purkinje potential. Successful ablation has occurred with single point lesions targeting these sites. Pace mapping has met with some success for targeting more limited forms of VT. Some of the possible explanations for the difficulties encountered and the “imperfect” pace map are the variable current strength that is used for pacing, the variability in the conduction system, and, more importantly, the amount of surrounding myocardium “captured.” Furthermore, it is possible to obtain a pace map with a similar QRS morphology at a site remote from the origin of the tachycardia due to selective capture of the Purkinje network. Because of these limitations of point lesion application based on pace mapping alone, a new strategy for providing more uniform success for unmappable forms of this VT was desirable.

Linear ablation lesions for control of unmappable VT in patients with ischemic and nonischemic cardiomyopathy have been described.\textsuperscript{22–25} It is believed that these lesions guided by pace maps, which mimic and approximate the exit site of VT, are successful because they interrupt a circuit of considerable dimension. This technique can also be applied to patients with idiopathic fascicular left ventricular tachycardias that are difficult to induce or that are nonsustained or noninducible for which “traditional” mapping techniques may not work. We found that a linear lesion transecting through the inferior mid to mid-apical left ventricular septum guided by electroanatomic mapping, the best pace map, and the presence of Purkinje potentials in sinus rhythm and self-limited VT was effective in eliminating tachycardia in these patients. Achievement of left posterior fascicular block on surface ECG was not a necessary prerequisite for a successful endpoint, suggesting that conduction through the posterior fascicle is not a necessary prerequisite for the development of clinical VT. The success of the procedure supports a reentrant mechanism for these VTs, with the circuit having significant apical to basal dimension along the inferior septum and thus vulnerable to the effects of the linear lesion.

**Conclusion**

In patients with difficulty to induce or nonsustained VT with the typical right bundle branch block pattern and a superiorly directed axis on 12-lead ECG, radiofrequency energy ablation delivered in a linear fashion approximately midway to two thirds toward the apex along the mid to inferior septum and perpendicular to the plane of the septum is safe and effective for VT control.

**References**