Initial Human Feasibility of Infusion Needle Catheter Ablation for Refractory Ventricular Tachycardia

John L. Sapp, MD, FRCPC, FHRs; Christopher Beeckler, BSc; Robert Pike, PhD; Ratika Parkash, MD, FRCPC, FHRs; Christopher J. Gray, MD, FRCPC; Katja Zeppenfeld, MD; Vikas Kuriachan, MD, FRCPC, FHRs; William G. Stevenson, MD, FHRs

Background—Ablation of ventricular tachycardia (VT) is sometimes unsuccessful when ablation lesions are of insufficient depth to reach arrhythmogenic substrate. We report the initial experience with the use of a catheter with an extendable/retractable irrigated needle at the tip capable of intramyocardial mapping and ablation.

Methods and Results—Sequential consenting patients with recurrent VT underwent ablation with the use of a needle-tipped catheter. At target sites, the needle was advanced 7 to 9 mm into the myocardium, permitting pacing and recording. Infusion of saline/iodinated contrast mixture excluded perforation and ensured intramyocardial deployment. Further infusion was delivered before and during temperature-controlled radiofrequency energy delivery through the needle. All 8 patients included (6 male; mean age, 54) with a mean left ventricular ejection fraction of 29% were refractory to multiple antiarrhythmic drugs, and 1 to 4 previous catheter ablation attempts (epicardial in 4) had failed. Patients had 1 to 7 (median, 2) VTs present or inducible; 2 were incessant. Some intramyocardial VT mapping was possible in 7 patients. A mean of 22 (limits, 3–48) needle ablation lesions were applied in 8 patients. All patients had at least 1 VT terminated or rendered noninducible. During a median of 12 months follow-up, 4 patients were free of recurrent VT, and 3 patients were improved, but had new VTs occur at some point during follow-up. Two died of the progression of preexisting heart failure without recurrent VT. Complications included tamponade in 1 patient and heart block in 2 patients.

Conclusions—Intramyocardial infusion-needle catheter ablation is feasible and permits control of some VTs that have been refractory to conventional catheter ablation therapy, warranting further study. (Circulation. 2013;128:2289–2295.)

Key Words: catheter ablation  ■  tachycardia, ventricular
echocardiography catheter were placed from the femoral veins. Left ventricular access was gained via transeptal puncture and a deflectable sheath (Agilis large curl, St. Jude Medical, St. Paul MN), or via a retrograde aortic approach. Left ventricular substrate mapping was initially performed with a standard 3.5-mm irrigated tip mapping catheter and an electroanatomic mapping system (Carto; Biosense Webster, Diamond Bar, CA). VT was induced with programmed ventricular stimulation. After a voltage map was created, the standard mapping catheter was replaced with a needle-tipped ablation catheter (Figure 1). The deflectable catheter has a distal bipole with an extendable/retractable 27-gauge nitinol needle. The needle has an embedded thermocouple and has a central lumen through which saline can be infused. A position sensor within the tip is compatible with an electroanatomic mapping system (Carto; Biosense Webster). The needle depth can be adjusted and locked in the extended or retracted position. In its fully retracted position, it is entirely within the catheter tip, whereas, when fully deployed, it extends 12 mm beyond the tip. An adjustable plunge activator on the handle (see Figure 1) permits the depth of extension to be preset and locks the needle in position when deployed. Recording and pacing are possible from both the external electrodes and from the needle. The recording system was configured to permit bipolar recordings between the needle and ring electrode (filtered 30–500 Hz), and between the needle and an inferior vena cava electrode, as well (filtered 30–500 Hz, and separately, filtered 0.5–500 Hz).

During catheter manipulation, the needle was kept retracted and irrigated with 0.9% saline (with 2 U/mL heparin) at 1 mL/min. Target sites were sought within areas of reduced bipolar or unipolar signal amplitude and were thought to be in or near components of ventricular circuits based upon endocardial substrate and pace mapping, or entrainment mapping, or relatively early activation during the arrhythmias. The catheter tip was placed at sites of interest with attempted orientation roughly perpendicular to the endocardial surface. Infusion was discontinued, and the needle was extended 7 to 9 mm into the myocardium. Intramyocardial electrograms were recorded from the needle and pacing was performed (10 mA, 2-ms pulse width). At potential target sites, 1 mL of infusate (50% NaCl solution [with 2U/mL heparin], 50% iodamidol [76%]) was injected into the myocardium, after which heparinized saline (2 U/mL, 0.9% NaCl) was infused at 2 mL/min for 60 s. Myocardial staining with contrast was visualized with fluoroscopy to ensure intramyocardial positioning and to rule out perforation, after which temperature-controlled power was delivered for 60 to 90 s with temperature limited to 60°C and power limited to <35 W.

After ablation, programmed stimulation with up to 3 extrastimuli following basic drives of 600 and 400 ms were performed with a shortest extrastimulus coupling interval of 180 ms. Repeat programmed stimulation was not performed if there was concern that it would aggravate the patient’s hemodynamic status.

Statistical Analysis
Descriptive statistics were used, including mean, median, and standard deviation when appropriate.

Results

Patients

Eight patients with recurrent VT were included (6 male), age 54 (limits, 13–70). Ventricular function was reduced in all patients (ejection fraction, 29±11%) associated with nonischemic cardiomyopathy in 6 patients and ischemic heart disease in 2 patients. All patients had uncontrollable VT despite antiarrhythmic drug combinations and previous catheter ablation, and therapy with amiodarone and one or more other drugs had failed in all patients. All patients had undergone previous endocardial catheter ablation attempts (mean, 2; limits, 1–4), and 4 patients had undergone an ineffective epicardial procedure (Table 1).

Procedures

Patients had 1 to 7 inducible or spontaneous monomorphic ventricular arrhythmias (median, 2); 2 patients were in incessant VT, 1 with repetitive monomorphic VT, at the commencement of the procedure (Table 2). During mapping, the needle was inserted at a median of 24 sites per patient (limits, 9–51). Myocardial staining from the bolus injection of 1 mL of saline/contrast was easily appreciated in every patient (Figure 2). When not observed, the needle was assumed not to be intramyocardial and was retracted and repositioned. No pericardial staining, suggesting perforation, was encountered.

In 7 patients, some intramyocardial mapping with the needle electrode was possible for at least 1 VT (VT was not reproducibly inducible in 1 patient). In each of these, a VT was identified with earlier intramyocardial signal than the adjacent endocardial activation time, but mapping during VT was limited because of hemodynamic intolerance and the inducibility of multiple VT morphologies, or nonreproducible inducibility. In 6 patients, at least 1 VT was terminated with intramyocardial needle infusion and ablation (Figure 3) and rendered noninducible, including both patients with incessant VT. This and further substrate-based ablation rendered 20 VTs noninducible in 8 patients. In 5 patients, repeat testing induced another VT, which was targeted with ablation, but further testing was not performed because of concern about hemodynamic status.

A median of 19 (limits, 3–48) lesions were delivered per patient over a median of 1316 s (limits, 131–2686), with a
Table 1. Baseline Characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>M</td>
<td>F</td>
<td>M</td>
<td>M</td>
<td>F</td>
<td>M</td>
<td>M</td>
<td>M</td>
</tr>
<tr>
<td>Age, y</td>
<td>13</td>
<td>40</td>
<td>68</td>
<td>61</td>
<td>48</td>
<td>63</td>
<td>66</td>
<td>70</td>
</tr>
<tr>
<td>Disease</td>
<td>NICM</td>
<td>NICM</td>
<td>ICM</td>
<td>ICM, AVR</td>
<td>NICM</td>
<td>NICM</td>
<td>NICM</td>
<td>NICM, IHD</td>
</tr>
<tr>
<td>EF</td>
<td>25</td>
<td>16</td>
<td>30</td>
<td>45</td>
<td>30</td>
<td>20</td>
<td>44</td>
<td>23</td>
</tr>
<tr>
<td>VT presentation</td>
<td>Incessant</td>
<td>Incessant</td>
<td>VT storm</td>
<td>VT storm</td>
<td>VT storm</td>
<td>VT storm</td>
<td>70 VT Rx in 3 wk</td>
<td>12 VT Rx in 6 wk</td>
</tr>
<tr>
<td>No. previous ablations</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Epicardial map/ablation</td>
<td>1</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>1</td>
<td>1</td>
<td>No</td>
<td>1</td>
</tr>
<tr>
<td>Previous AADs</td>
<td>Amiodarone, sotalol, flecainide</td>
<td>Amiodarone, lidocaine</td>
<td>Amiodarone, mexiletine</td>
<td>Amiodarone, mexiletine</td>
<td>Amiodarone, sotalol (800 mg/d), mexiletine</td>
<td>Amiodarone, mexiletine</td>
<td>Amiodarone, sotalol, mexiletine, dofetilide, flecainide</td>
<td>Amiodarone, sotalol, mexiletine</td>
</tr>
</tbody>
</table>

AAD indicates antiarrhythmic drug; AVR, aortic valve replacement; CRT-D, cardiac resynchronization defibrillator; EF, ejection fraction; ICD, implantable cardioverter-defibrillator; ICM, ischemic cardiomyopathy; IHD, ischemic heart disease; NICM, nonischemic cardiomyopathy; Rx, therapies; and VT, ventricular tachycardia.

median average power of 8 to 20 W (see Table 2); all lesions were delivered by using the needle ablation catheter, with the exception of 3 lesions in patient 4, for which a standard irrigated catheter was used because of a mechanical obstruction of the lumen of the needle catheter. In 4 patients, we had adequate data from previous procedures to conclude that similar intracardiac sites were targeted with needle ablation that had been previously targeted by using conventional ablation. Mean total procedure and fluoroscopy times were 355±157 (limits, 165–572) and 41±19 minutes, respectively. At the end of the procedure, 3 patients had no inducible VT, 1 patient was not tested because VT had not been reproducibly inducible and only ventricular flutter was induced, and in 4 patients, further repeat induction testing was not performed after postablation testing had induced another VT, which was targeted with further ablation (see Table 2).

Some specific examples are illustrative. The first patient had frequent ventricular ectopy of 2 morphologies and severely depressed ventricular function that was felt to be due in part to the arrhythmia. Incessant ventricular ectopy was markedly suppressed with administration of anesthesia. Based on limited activation and pace mapping, ablation was performed at the base of the posterior papillary muscle and abolished all ectopy for >48 hours. Subsequently, the arrhythmia recurred (below).

The second patient had nonischemic cardiomyopathy, incessant VT with pulmonary edema. Endocardial mapping identified a site of earliest focal endocardial activation that was after the QRS onset. Insertion of the needle at that site (Figure 3A), revealed presystolic electrical activity and the first application of radiofrequency (RF) terminated VT for the first time since her initial presentation. She was rendered free of inducible VT and had no further VT during follow-up over 17 months.

The third patient had ischemic heart disease, heart failure, and recurrent VT storm despite amiodarone and mexiletine and 2 previous endocardial ablation procedures. Four VTs were inducible, all of which appeared to exit from the interventricular septum. The left ventricle was mapped via the retrograde aortic and transseptal (using a deflectable sheath) approaches. The transseptal puncture was very difficult owing to a very elastic interatrial septum, despite visualization with intracardiac echocardiography. Sites within the basal septum where intramyocardial activation preceded endocardial signals were targeted with needle ablation, rendering 2 VTs noninducible. The other 2 VTs were mapped to the midseptal area. At a site with early activation during VT, a His bundle recording was identified (HV 105 ms) and, as anticipated, RF here resulted in heart block. Further RF was required from the adjacent right side of the septum to render the fourth VT noninducible. There was no pericardial effusion seen at the end of the procedure with intracardiac echocardiography, but the patient developed hypotension 20 minutes after withdrawal of all catheters and sheaths, and pericardial tamponade was diagnosed, which resolved with pericardiocentesis. During the procedure, all needle deployments were within the septum, and contrast injection did not demonstrate extracardiac staining at any time. Because surgery was not required, we could not verify the site of perforation, but we felt that it was most likely related to the difficult transeptal puncture. The patient died 3 months later of progressive heart failure without recurrent VT.

Complications

Two patients developed heart block during RF delivery at the basal septum. In both cases, this was anticipated and accepted to interrupt VT. One patient developed pericardial tamponade described in detail above.

Follow-Up

During follow-up of 10 to 25 months (median, 12), 4 patients were free of recurrent VT, 2 patients had occurrence of a new
VT, with QRS morphology not seen before ablation or during the procedure, that was drug-controlled 7 and 9 months after ablation, respectively, and 2 continued to have frequent VT recurrence (Table 2). Of the 4 patients with no recurrence, 2 died of heart failure without recurrent VT after 3 and 5 months, respectively; both had a history of heart failure and depressed ventricular function before ablation. Of the 2 patients with late VT recurrence, one had a new VT 9 months after the procedure after amiodarone had been reduced. The other patient developed a new VT 9 months after the procedure after a diarrheal illness while being treated with sotalol alone; amiodarone was resumed. Of the 2 patients with continued VT early after ablation, the first patient had an initial suppression of repetitive monomorphic VT and ectopy, but had a recurrence by 5 weeks. The second developed a new VT morphology entirely different from any targeted during the procedure within 1 week of the procedure and subsequently underwent implantation of a ventricular assist device for progressive heart failure.

### Discussion
RF catheter ablation has been remarkably successful for the treatment of many arrhythmias. The small lesions created by standard RF ablation contribute to its safety, but larger lesions are required for ablation of many ventricular arrhythmias. Irrigated RF ablation creates larger lesions, but it still has incomplete efficacy for ablation of VT when the arrhythmogenic substrate is deep to the endocardial surface. Epicardial mapping and ablation can be effective in some, but it is also limited when the target substrate is intramural, or when epicardial access is not possible or is limited, as in the presence of pericardial adhesions. Transcoronary ethanol ablation is another option, but it can be limited by coronary anatomy and also has significant risks. Surgical ablation can be an option as well, but requires thoracotomy. Intramyocardial needle ablation with saline infusion has been demonstrated to create deep RF ablation lesions in animal models. The intramyocardial saline may function as an expanding virtual electrode in the tissue and may also cool the needle. We report the first use of this technique for the creation of therapeutic ablation lesions in 8 patients with refractory VT. Remarkably, very frequent treatment-refractory ventricular arrhythmias were brought under acute control in 6 patients, 4 of whom remained free of recurrent VT during a median of 12 months of follow-up.

### Table 2. Procedural Findings

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Target locations</th>
<th>No. RF lesions</th>
<th>Total RF time</th>
<th>RF power, median (IQR)</th>
<th>Fluoroscopy time, min</th>
<th>Complications</th>
<th>Follow-up duration, mo</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Base of posterior papillary muscle</td>
<td>12</td>
<td>471</td>
<td>14 (12.5–17.5)</td>
<td>53.8</td>
<td>None</td>
<td>25</td>
<td>Suppressed all ectopy immediately. 3% on Holter day 3. Recurred at 5 wk</td>
</tr>
<tr>
<td>2</td>
<td>Basal posterior lateral LV</td>
<td>3</td>
<td>131</td>
<td>11 (11–15.5)</td>
<td>20.3</td>
<td>None</td>
<td>22</td>
<td>No recurrence at 25 mo off antiarrhythmics</td>
</tr>
<tr>
<td>3</td>
<td>Basal superior and mid LV septum, basal RV septum</td>
<td>48</td>
<td>2353</td>
<td>17 (13.75–23)</td>
<td>71.8</td>
<td>None</td>
<td>21</td>
<td>VT suppressed. Died of progressive heart failure 3 mo postprocedure.</td>
</tr>
<tr>
<td>4</td>
<td>Inferior and interpapillary septum</td>
<td>43</td>
<td>2686</td>
<td>8 (5–11.25)</td>
<td>58.3</td>
<td>None</td>
<td>12</td>
<td>Further VT 9 mo later on sotalol; started amiodarone</td>
</tr>
<tr>
<td>5</td>
<td>Superior basal MV annulus, inferobasal LV septum, LVOT septum, RVOT septum</td>
<td>24</td>
<td>1406</td>
<td>14.5 (8.75–17)</td>
<td>42.6</td>
<td>None</td>
<td>12</td>
<td>No recurrence for 7 mo until amiodarone tapered; new VT developed</td>
</tr>
<tr>
<td>6</td>
<td>Inferoposterolateral LV</td>
<td>18</td>
<td>1386</td>
<td>19 (15.5–20)</td>
<td>34.4</td>
<td>None</td>
<td>10</td>
<td>New different VT postablation. LVAD implanted. On amiodarone and flecainide</td>
</tr>
<tr>
<td>7</td>
<td>Basal inferior RV and LV septum</td>
<td>21</td>
<td>1247</td>
<td>17 (12–21.75)</td>
<td>25.5</td>
<td>Complete heart block</td>
<td>10</td>
<td>No recurrence at 5 mo. f/u on mexitiline and sotalol, died of progressive heart failure</td>
</tr>
<tr>
<td>8</td>
<td>Basal superolateral LV</td>
<td>10</td>
<td>846</td>
<td>20 (18.5–21)</td>
<td>20.5</td>
<td>None</td>
<td>10</td>
<td>No recurrence at 10 mo follow-up.</td>
</tr>
</tbody>
</table>

*RF power, median (IQR) indicates the median value and interquartile limits of mean powers of each RF lesion.*
Further, we confirmed that the needle-tipped catheter permits both recording and pacing from deep within the myocardium. Midmyocardial near-field signal was demonstrably earlier than that of the endocardium in cases where activation mapping was possible (Figure 3). Occasionally, far-field endocardial signal correlated with near-field intramyocardial signal (Figure 3A). Intramyocardial pacing was also possible (Figure 4). The bipolar needle electrogram, recording the potential difference between the needle and the ring electrode, includes contribution from myocardium in contact with the length of the needle and may consequently include greater far-field contribution and capture of a potentially larger volume of tissue during pacing.

We also demonstrate that the injection of a contrast–saline mixture in the myocardium is well tolerated and produces a visual indication of intramyocardial needle deployment. It is likely that contrast infusion will expose perforation of the needle into the pericardial space, although we did not recognize any instances of appearance of the contrast in the pericardial space in these patients. It is possible that the distribution of the contrast may bear some relation to the area subject to the RF current and, therefore, to lesion size. Among the 4 patients who experienced VT postprocedure, 2 had early VT, and 2 had later occurrences. One patient (patient 1) had a recurrence of 1 of the 2 VT morphologies targeted during the procedure. General anesthesia resulted in marked suppression of ectopy, severely limiting mapping to a pace-mapping approach. Ectopy recurred within days to weeks, having previously been incessant, suggesting that it was affected by ablation, but that the critical substrate was not permanently abolished. The second patient with early occurrence did not have reproducibly inducible VT during his procedure, but had repeatedly inducible polymorphic VT requiring defibrillation. A combination of limited activation mapping during brief catheter-induced VT and substrate mapping was thus used. The patient developed a new VT morphology postprocedure, with a likely exit site remote from the region targeted. Of the 2 patients with late occurrences of VT, both were on antiarrhythmic amiodarone that was weaned or discontinued following their procedures, after which VTs of new morphologies emerged. We did not pursue an aggressive approach.
substrate-based approach during this early human experience with this catheter.

With ablation technologies that increase lesion size, the potential for complications is a major concern. Needle ablation is unlikely to be different in this regard. Cardiac perforation and damage to functioning myocardium are important considerations. We speculate that the small diameter of the needle (27 gauge) reduces the risk of significant bleeding if perforation occurs. As we observed in previous animal models and now in humans, repeated deployments of the needle for mapping are possible without pericardial bleeding. We did observe 1 case of tamponade after the procedure; we cannot definitively exclude the needle catheter as the cause of the perforation, although the timing of the event, after sheaths and catheters were removed, and the fact that the ablation targeted the septum in this patient, suggests that it may not have been related to the needle ablation. Two patients died of heart failure, and another subsequently required the placement of a ventricular assist device, largely because of continued uncontrollable VT. Although these patients had previous heart failure, and death from heart failure is unfortunately common in this patient population, we cannot exclude a contribution of larger ablation lesions to subsequent continued heart failure. Confining ablation lesions to regions of scar and attempting to target the lesions to the arrhythmogenic substrate remains relevant for reducing the risk of damage to functioning myocardium.

The small size of this series limits the conclusions regarding the safety and efficacy of this technique. Other potential complications, which we did not detect but remain concerns, are entry of the needle into a coronary vessel or valve leaflet. We were also unable to confirm lesion size in these patients, or draw meaningful conclusions on the potential effects on ventricular function.

Conclusions

Intramyocardial infusion-needle catheter ablation is feasible and permits control of some VTs that have been refractory to conventional catheter ablation therapy. Further studies are warranted.

Acknowledgments

Catheters were provided free of charge by Biosense Webster.

Disclosures

Dr Sapp is a coholder of a patent for the needle catheter, rights assigned to Brigham and Women’s Hospital, and reports research funding from Biosense Webster unrelated to this study. C. Beecker is an employee of Biosense Webster. Dr Pike is an employee of Biosense Webster and a coholder of a patent for the needle catheter. Dr Kuriachan reports speaker honoraria from Biosense Webster. Dr Stevenson is a coholder of a patent for the needle catheter, rights assigned to Brigham and Women’s Hospital. The remaining authors report no conflicts.

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CLINICAL PERSPECTIVE

Recurrent scar-related ventricular tachycardia can be a difficult management problem, associated with significant patient morbidity and the risk of adverse outcomes. Catheter ablation can be very helpful, often in conjunction with antiarrhythmic drug therapy. One of the most significant limitations of catheter ablation, however, is the inability to create sufficient myocardial ablation lesions to interrupt arrhythmogenic substrate that is deep to the endocardium. This series demonstrates the first human experience with an extendable/retractable needle at the tip capable of creating deeper ablations and mapping intramyocardial substrate. In patients with frequent ventricular tachycardia refractory to both catheter ablation and antiarrhythmic drugs, use of the needle catheter permitted intramyocardial mapping and delivery of intramyocardial ablation in all patients, and clinical improvement in most patients. Complications included heart block in 2 patients, which was anticipated based on the location of the ventricular tachycardia substrate, and cardiac perforation in 1 patient, which was thought to be related to the transseptal puncture. Use of this catheter appears to be feasible with an acceptable complication risk and the possibility of benefit in patients with treatment-refractory ventricular tachycardia.
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