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

Running title: Sapp et al.; Needle ablation of VT

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Journal Subject Codes: Treatment:[22] Ablation/ICD/surgery, Diagnostic testing:[106] Electrophysiology
Abstract

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 utilizing 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 using a needle-tipped catheter. At target sites the needle was advanced 7-9 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 mean LVEF 29% were refractory to multiple antiarrhythmic drugs and had failed 1-4 prior catheter ablation attempts (epicardial in 4). Patients had 1-7 (median 2) VTs present or inducible; 2 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 were free of recurrent VT, and 3 were improved, but had new VTs occur at some point during follow-up. Two died of progression of preexisting heart failure without recurrent VT. Complications included tamponade in 1 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.

Key words: catheter ablation, ventricular tachycardia, intramural reentry, mapping, Intramyocardial Ablation, needle ablation
Background

Catheter ablation is an important therapeutic option for ventricular tachycardia (VT)\(^1,2\) but remains inadequate in some patients despite technical advances including three-dimensional mapping,\(^3\) irrigated radiofrequency ablation,\(^4\) and the availability of epicardial mapping and ablation, and intracoronary ethanol ablation.\(^5\) A common limitation is the existence of reentry substrate that is beyond the reach of ablation using standard techniques,\(^6-8\) in some cases requiring an open surgical approach.\(^9\)

Preclinical work has demonstrated that a catheter with an extendable/retractable electrically active needle can record intramural cardiac electrograms, pace, and create deep myocardial lesions.\(^10\) The addition of intramyocardial saline infusion, likely creating an interstitial ‘virtual electrode’ permits the creation of large, deep myocardial lesions.\(^11\) We report the first human experience with this technique as last resort treatment of therapy-refractory VT.

Methods

Patients

Patients with recurrent VT, despite antiarrhythmic drug therapy and at least one attempt at catheter ablation, were offered catheter ablation using a needle-infusion catheter. Needle ablation was performed under the special access program of health Canada. All patients provided informed consent for use of this “compassionate release” therapy. The institutional research ethics board approved the reporting of the review of medical records and reporting of cases.

Infusion Needle Ablation

Patients were brought to the electrophysiology laboratory in the fasting state. Multipolar electrode catheters and an intracardiac echocardiography (ICE) 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).\(^6\) 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 (\textit{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, Diamond Bar, CA). 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, while when fully deployed it extends 12mm beyond the tip. An adjustable plunge activator on the handle (see \textit{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), as well as between the needle and an inferior vena cava electrode, (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 VT 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 7-9 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% iopamidol (76%)) was injected into the myocardium, after which heparinized saline (2U/mL, 0.9% NaCl) was infused at 2 mL/min for 60 seconds. Myocardial staining with contrast was visualized with fluoroscopy to ensure intramyocardial positioning and rule out perforation, after which temperature-controlled power was delivered for 60-90 sec with temperature limited to 60°C and power limited to <35W.

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

Statistical Analysis
Descriptive statistics were utilized, 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 (EF 29 +/- 11%) associated with nonischemic cardiomyopathy in 6 and ischemic heart disease in 2 patients. All patients had uncontrollable VT despite antiarrhythmic drug combinations and prior catheter ablation and all had failed therapy with amiodarone and one or more other drugs. All patients had undergone prior endocardial
catheter ablation attempts (mean 2, limits 1-4), and 4 had undergone an ineffective epicardial procedure (Table 1).

**Procedures**

Patients had 1-7 inducible or spontaneous monomorphic ventricular arrhythmias (median 2); 2 patients were in incessant VT, 1 with repetitive monomorphic VT, at commencement of the procedure (Table 2). During mapping the needle was inserted at a median of 24 sites per patient (limits 9 to 51). Myocardial staining from the bolus injection of 1 mL 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 due to hemodynamic intolerance and inducibility of multiple VT morphologies, or non-reproducible inducibility. In 6 patients, at least 1 VT was terminated with intramyocardial needle infusion and ablation (figure 3) and rendered non-inducible, 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 1 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 (limits 131-2686) seconds, with median average power of 8-20 Watts (See Table 2); all lesions were delivered using the needle ablation catheter, except 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 prior procedures to conclude that
similar intracardiac sites were targeted with needle ablation which had been previously targeted
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 was not tested because VT had not been reproducibly inducible and only
ventricular flutter was induced, and in 4, further repeat induction testing was not performed after
post-ablation 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 two morphologies and severely depressed ventricular function felt to be in part due to the
arrhythmia. Incessant ventricular ectopy was markedly suppressed with administration of
anaesthesia. Based on limited activation and pace-mapping, ablation was performed at the base
of the posterior papillary muscle, and abolished all ectopy for approximately 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 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 two prior 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 transeptal (using a deflectable sheath) approaches. The transeptal puncture was very difficult due to a very elastic interatrial septum, despite visualization with ICE. 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 mid-septal area. At a site with early activation during VT a His bundle recording was identified (HV 105 msec) 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 ICE, 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. Since surgery was not required we could not verify the site of perforation, but felt that it was most likely related to the difficult transeptal puncture. The patient expired 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 in order to interrupt VT. One patient developed pericardial tamponade described in detail above.

Follow-up

During follow-up of 10-25 months (median 12) 4 patients were free of recurrent VT, 2 patients had occurrence of a new VT, with QRS morphology not seen prior to ablation or during the procedure, that was drug-controlled 7 and 9 months after ablation respectively, and two
continued to have frequent VT recurrence (Table 2). Of the 4 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 prior to ablation. Of the two with late VT recurrence, one had a new VT 7 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 two patients with continued VT early after ablation, the first patient had initial suppression of repetitive monomorphic VT and ectopy, but recurred 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

Radiofrequency 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 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 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 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 radiofrequency 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 follow-up.

Further we confirmed that the needle-tipped catheter permits both recording and pacing from deep within the myocardium. Mid-myocardial 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 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 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 post-procedure, two had early VT, and two had later occurrences. One patient (patient #1) had a recurrence of one of the 2 VT morphologies targeted during the procedure. General anaesthesia 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 utilized. He developed a new VT morphology post-procedure, with a likely exit site remote from the region targeted. Of the two patients with late occurrences of VT, both were on antiarrhythmic amiodarone which was weaned or discontinued following their procedures, after which VTs of new morphologies emerged. We did not pursue an aggressive substrate-based approach on 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 prior animal models and now humans, repeated deployments of the needle for mapping are possible without pericardial bleeding. We did observe one 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 ventricular assist device placement, largely due to continued uncontrollable VT. Although these patients had prior heart failure, and heart failure death is unfortunately common in this patient population, we can not 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 conclusions regarding the safety and efficacy of this technique. Other potential complications, that 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.

**Conflict of Interest Disclosures:** John Sapp is a coholder of patent for needle catheter, rights assigned to Brigham and Women’s Hospital, and reports research funding from Biosense Webster unrelated to this study. Christopher Beeckler is an employee of Biosense Webster. Robert Pike is an employee of Biosense Webster and a coholder of patent for needle catheter. Vikas Kuriachan reports speaker honoraria from Biosense Webster. William Stevenson is a coholder of patent for needle catheter, rights assigned to Brigham and Women’s Hospital. The remaining authors report no conflicts.

**References:**


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Table 1. Baseline Characteristics

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<td>M</td>
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<td>ICM, AVR</td>
<td>NICM</td>
<td>NICM</td>
<td>NICM</td>
<td>NICM, IHD</td>
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<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 weeks</td>
<td>12 VT Rx in 6 weeks</td>
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<td>Epicardial Map/Ablation</td>
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<td>No</td>
<td>No</td>
<td>No</td>
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<td>CRT-D</td>
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<td>Prior AADs</td>
<td>Amiodarone, Sotalol, Lidocaine, Flecaainde, Mxiletine,</td>
<td>Amiodarone, Amiodarone, Mexiletine, Mexiletine,</td>
<td>Amiodarone, Mexiletine, (800mg/d), Mexiletine,</td>
<td>Amiodarone, Amiodarone, Sotalol, Mexiletine, Dofetilide, Flecaainde,</td>
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Abbreviations: NICM, nonischemic cardiomyopathy; ICM, ischemic cardiomyopathy; AVR, aortic valve replacement; IHD, ischemic heart disease; VT, ventricular tachycardia; ICD, implantable cardioverter-defibrillator; CRT-D, cardiac resynchronization defibrillator.
### Table 2. Procedural Findings.

<table>
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<th>Patient</th>
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<th>5</th>
<th>6</th>
<th>7</th>
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<td>No. VTs induced</td>
<td>2</td>
<td>1</td>
<td>4</td>
<td>5</td>
<td>1</td>
<td>2</td>
<td>2</td>
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<td><strong>Target Locations</strong></td>
<td>Base of Posterior Papillary Muscle</td>
<td>Basal Posterior Lateral LV</td>
<td>Basal Superior and Mid LV Septum, Basal RV Septum</td>
<td>Inferior and inferoposterolateral LV</td>
<td>Superior Basal MV annulus, Inferobasal LV Septum, LVOT Septum, RVOT Septum</td>
<td>Inferoposterolateral LV</td>
<td>Basal inferior RV and LV septum</td>
<td>Basal superolateral LV</td>
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<tr>
<td>No. of RF Lesions</td>
<td>12</td>
<td>3</td>
<td>48</td>
<td>43</td>
<td>24</td>
<td>18</td>
<td>21</td>
<td>10</td>
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<tr>
<td>Total RF Time (med, IQR)</td>
<td>471</td>
<td>131</td>
<td>2353</td>
<td>2686</td>
<td>1406</td>
<td>1386</td>
<td>1247</td>
<td>846</td>
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<tr>
<td>RF Power (median, IQR)</td>
<td>14 (12.5-17.5)</td>
<td>11 (11-15.5)</td>
<td>17 (13.75-23)</td>
<td>8 (5-11.25)</td>
<td>14.5 (8.75-17)</td>
<td>19 (15.5-20)</td>
<td>17 (12-21.75)</td>
<td>20 (18.5-21)</td>
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<td>Procedure Duration (min)</td>
<td>536</td>
<td>165</td>
<td>498</td>
<td>572</td>
<td>317</td>
<td>288</td>
<td>223</td>
<td>242</td>
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<tr>
<td>Fluoroscopy Time</td>
<td>53.8</td>
<td>20.3</td>
<td>71.8</td>
<td>58.3</td>
<td>42.6</td>
<td>34.4</td>
<td>25.5</td>
<td>20.5</td>
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<tr>
<td><strong>Endpoint</strong></td>
<td>Abolition of all ectopy, Noninducible</td>
<td>Termination of VT, Noninducible</td>
<td>Rendered 3 VTs noninducible, No further testing after targeting 4th VT</td>
<td>Rendered 4 VTs noninducible, No further testing after targeting 5th VT</td>
<td>Rendered 6 VTs noninducible, No further testing after targeting 7th VT</td>
<td>Not retested, only V Flutter had been inducible</td>
<td>No further testing after targeting 2nd VT</td>
<td>Noninducible</td>
</tr>
<tr>
<td>Complications</td>
<td>None</td>
<td>None</td>
<td>Complete heart block (anticipated)</td>
<td>None</td>
<td>None</td>
<td>Complete heart block</td>
<td>None</td>
<td>None</td>
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<td>Follow-up Duration (months)</td>
<td>25</td>
<td>22</td>
<td>21</td>
<td>12</td>
<td>12</td>
<td>10</td>
<td>10</td>
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<tr>
<td><strong>Follow-Up</strong></td>
<td>Suppressed all ectopy immediately. 3% ectopy on Holter day 3. Recurred at 5 weeks</td>
<td>No recurrence at 25 months off anti-arrhythmics</td>
<td>VT suppressed. Died of progressive heart failure 3 mo post-procedure.</td>
<td>Further VT 9 months later on sotalol; started amiodarone</td>
<td>No recurrence for 7 months until amiodarone tapered; new VT developed</td>
<td>New different frequent VT post-ablation. LVAD implanted. On amiodarone and flecainide</td>
<td>No recurrence at 5 mo. f/u on mexiletine and sotalol, died of progressive heart failure</td>
<td>No recurrence at 10 mo follow-up.</td>
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Abbreviations: LV, left ventricle; RV, right ventricle; MV, mitral valve; LVOT, left ventricular outflow tract; RVOT, right ventricular outflow tract; LVAD, left ventricular assist device. RF Power (median, IQR) indicates median value and interquartile limits of mean powers of each RF lesion.
Figure Legends:

Figure 1. A. The catheter tip is deflectable, and consists of a bipole (dome and ring electrodes), with an extendable/retractable needle embedded with a thermocouple, and capable of infusion. B. The handle is comprised of a standard deflection mechanism with a hypertronics connection, as well as a locking, adjustable-depth plunge mechanism to deploy the needle. A redel connection and infusion port exit the end.

Figure 2. Contrast mixed with heparinized saline was infused shortly after needle deployment and prior to RF delivery at sites of planned ablation to ensure that the tip of the needle remained intramyocardial, and to exclude perforation. Two right ventricular quadripolar electrode catheters are shown, as well as an intracardiac echocardiography probe. Access to the left ventricle is via transeptal puncture through a deflectable sheath.

Figure 3. A: Patient 2 presented with incessant drug-refractory VT and heart failure. The needle was deployed within the myocardium at the basal anterolateral LV. The bipolar electrogram recorded here (NDL; arrow) had a low amplitude early signal preceding the QRS by 30 msec. The endocardial signal (Abl d) at the same site had a corresponding low amplitude, rounded signal with similar timing. RF delivered with saline infusion at this site terminated VT after 5 seconds. Shown are surface ECG leads I, II, III, V1, V5 and bipolar electrograms (NdI = needle to ring electrode, Abl = tip electrode to ring electrode, RVa = right ventricular apical electrogram). B: A high-frequency signal recorded from the needle deployed within the myocardium precedes the signal recorded from the endocardium (arrow). Activation at this site
did not precede the QRS, and RF was not delivered. Shown are electrograms as in panel A, as well as high-pass filtered unipolar electrograms (Ndl uni = unipolar needle electrogram; Abl uni = tip electrode unipolar electrogram).

**Figure 4.** Intramyocardial sites in areas of low endocardial bipolar signal amplitude also demonstrated low amplitude signal, and sometimes fragmentation. Intramyocardial pacing from the needle was possible. In this case (Patient 4), intramyocardial pacing captured with delay and initiated ventricular tachycardia.
Figure 2
Figure 4
Initial Human Feasibility of Infusion Needle Catheter Ablation for Refractory Ventricular Tachycardia

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*Circulation.* published online September 13, 2013;
*Circulation* is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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http://circ.ahajournals.org/content/early/2013/09/13/CIRCULATIONAHA.113.003423

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