Feasibility of a Noncontact Catheter for Endocardial Mapping of Human Ventricular Tachycardia

Richard J. Schilling, MB; Nicholas S. Peters, MD; D. Wyn Davies, MD

Background—Catheter ablation of ventricular tachycardia (VT) is limited by difficulty in identifying suitable sites for ablation. This study assesses use of a system capable of simultaneous endocardial mapping of the human left ventricle to map and guide radiofrequency (RF) catheter ablation of VT.

Methods and Results—A catheter-mounted noncontact multielectrode array was used to reconstruct 3360 electrograms, superimposed onto a computer-simulated endocardial model. Of 24 patients studied, 20 had ischemic heart disease. Exit sites were demonstrated by the noncontact system in 80 (99%) of 81 VTs, with complete VT circuits traced in 17 (21%). In another 37 VTs, 36±30% (mean±SD) of the diastolic interval was identified. Thirty-eight VT morphologies were ablated with 154 RF energy applications. Successful ablation was achieved by 77% of RF applications to relevant diastolic activity identified by the system and was significantly more likely (P<0.0001) than by RF at the VT exit or remote from diastolic activation. Over a mean follow-up of 1.5 years, 14 patients (64%) have had no recurrence of VT, and only 2 target VTs (5.3%) have recurred. Five patients have had recurrence of other VTs.

Conclusions—This noncontact mapping system identified diastolic portions of the circuit in most VTs studied and can safely map and guide ablation of human VT. (Circulation. 1999;99:2543-2552.)

Key Words: ablation n ventricles n tachycardia

Inducible ventricular tachycardias (VTs) are poorly suppressed by medication,1 such failure carrying a poor prognosis.2 Empirical drug therapy risks proarrhythmic effects.3 Palliation with implantable cardioverter-defibrillators (ICDs) is increasing4 but is not ideal for patients being treated for frequent or slow VT.

Catheter ablation may eliminate VT with less risk than surgical ablation.5 For reentrant VT,6,7 ablation targets the diastolic activity that maintains reentry.7,8 Of patients with structural heart disease and VT, 10% have been considered suitable for catheter ablation,9 because hemodynamic intolerance of VT prevents conventional mapping. A percutaneous mapping system producing high-resolution endocardial activation maps of the entire left ventricle (LV)10 has been validated in humans during sinus rhythm.11 We report its first use in catheter ablation of human VT.

Methods

Patients

Twenty-four patients were studied during endocardial LV mapping for ablation of VT (Table 1). Twenty-one were on amiodarone, and 4 were also on another antiarrhythmic agent. Eight patients with ICDs had unacceptable frequent or slow VT. The remaining patients had well-tolerated VT refractory to medical therapy. Two patients had normal LVs, 1 with fascicular and 1 with LV outflow tract tachycardia.

Mapping Procedure

The study was approved by the local ethics committee, whose guidelines were followed. A quadrupolar catheter was placed in the right ventricle, and two 7F 4-mm-tip catheters were passed to the LV via the aorta and transseptally. Contact catheter data and ECGs were recorded simultaneously on another EP system.11

Mapping Protocol

Validation of Reconstructed Electrograms

VT electrograms selected from sites of interest were displayed and compared with contact unipolar electrograms from the same site, as identified by the locator. Off-line analysis used cross-correlation techniques.11,12

Isopotential Mapping

Voltages are displayed as a colored isopotential map on the virtual endocardium. The color scale is adjusted to create a binary display, with negative unipolar potentials in white on a purple background, producing a unipolar activation map. Diastolic depolarization was strictly defined as activity on the isopotential map (Figure 1) that could be continuously tracked back in time from VT exit sites, defined on the map as synchronous with the QRS onset (Figures 1 and 2). Diastolic activity and exit sites were then marked on the virtual endocardium, and a mapping catheter was navigated to them by the locator. If these sites met conventional mapping criteria for ablation,13,14 temperature-controlled radiofrequency energy (RF) was delivered. These sites were then identified by off-line analysis, for which successful ablation was defined as RF terminating VT and partial success as RF changing VT morphology, after both of which the original VT was noninducible.
Statistics
Continuous data are presented as mean and SD and analyzed by Student’s t test. Data of the relation between catheter location and the outcome of RF was analyzed with 2 analysis.

Results
Data were collected from all patients during VT (Table 2). Ablation was completed in 22 patients and was not performed because ventricular fibrillation (VF) was followed by cardiogenic shock in 1 patient and because of facial weakness after transseptal puncture before multielectrode array (MEA) introduction in another.

Validation of Reconstructed Electrograms During VT
Cross-correlation analyzed 7593 (range, 40 to 1731) unipolar VT electrograms. Cross-correlation of contact and reconstructed electrograms was 0.86 ± 0.16 (mean ± SD) (Figure 3). The timing shift that produced the closest electrogram match was −1.67 ± 10.46 ms.

Mapping
Eighty-one of 97 VTs were mapped with the MEA (3.4 per patient). Sixteen VTs were recorded only before MEA introduction. Twenty-four mapped VTs were clinical. Noncontact mapping identified 80 (99%) of 81 VT exits and diastolic activity connecting to this in 54 (67%). In 17 VTs, complete reentrant circuits were mapped (Figure 4A and 4B). Of these, 7 were successfully ablated, all from within mapped diastolic pathways (DPs). The other 10 were not ablated either because of hemodynamic instability (n = 2) or because the VT was induced only once (n = 8). In the remaining 37 VTs, diastolic activity was traced over 36 ± 30% (range, 1% to 95%) of the diastolic interval.

Ablation
One hundred fifty-four RF applications (range, 0 to 34 per patient) ablated a total of 38 (15 clinical) VTs (4 RFs per VT). Four VTs were ablated by 2 RF applications on shared DPs (Figures 5 and 6). Seventy-six RFs were delivered during VT, with 22 successful, 9 partially successful, and 45 unsuccessful. Of RFs applied to a mapped DP, 77% successfully ablated VT. RF delivered to exit sites was significantly less successful (P < 0.005) and failed to eliminate VT in 79%, either changing morphology or causing no change to the VT. RF delivered remote from the VT DP was significantly less successful (P < 0.0001), failing in 91% of applications. RF
applied close to incompletely identified DPs succeeded in 80% of cases (Table 3).

**Complications**

No cardiac complications resulted from MEA use. The first 2 patients developed false femoral artery aneurysms requiring surgical repair, 1 at the site of MEA introduction. Subsequently, anticoagulation was reversed, and sheaths were removed immediately with no further vascular complication. Other procedural complications were cerebrovascular event (n=1) before MEA deployment, cardiogenic shock after VF (n=1), and hemothorax (n=1) after transseptal puncture. A patient with incessant VT and hypotension died 12 hours after the procedure of cardiogenic shock exacerbated by cardiac tamponade from a 100-mL effusion through a right ventricular perforation by a preexisting temporary catheter.

**Follow-Up**

Of 22 patients ablated, 14 (64%) have had no VT over a mean follow-up of 1.5 years (range, 0.6 to 2.5 years). Of 38 target VTs, 2 (5.3%) recurred; both were then successfully ablated. In 2 patients, slower nonsustained target VT recurred at 1 week and 1 year. In 1 patient, documented fast VT recurred after 6 months. Two patients had new VTs >3 months after the procedure.

Four patients have died, 1 of myocardial infarction 3 days after the procedure. A patient with an ICD and dilated cardiomyopathy died 3 months later of incessant VF, with no recurrence of VT before this. Two patients were free of VT but died 6 and 18 months, respectively, after the procedure, 1 of noncardiac and 1 of unknown causes.

For those patients with preexisting ICDs, over a follow-up of 1.19±0.7 years, shock frequency was reduced from 9.2 per year (range, 2 to 23) to 0.16 (P<0.05).

In 7 patients, ICDs were implanted after ablation because of rapid VT during the procedure. No therapy has been delivered by these ICDs. None of the remaining 9 patients have had recurrence of VT. Antiarrhythmics were stopped in the 2 patients with normal hearts and unchanged in the 7 with structural heart disease.

**Discussion**

We report the first use of noncontact mapping of human VT, which identified exit sites in 99% and diastolic portions of VT reentry circuits in 67%. Thirty-eight VTs were ablated,
with a >78% success rate over a follow-up period of 1 year in patients who survived the perioperative period.

The most common cause for clinical VT is coronary heart disease. Catheter ablation of such VTs has been limited, by the time required for sequential endocardial activation mapping, to those with hemodynamically stable VT. The results have been disappointing, with immediate success rates of 71% to 90%, but recurrence is common.

Simultaneous epicardial and endocardial activation mapping has been performed at surgery with multiple electrodes on the epicardium or on a balloon introduced transmurally to the endocardium. These circumstances may modify the substrate, with associated morbidity and mortality. Percutaneous endocardial basket arrays simultaneously map multiple endocardial points. Resolution is limited to those electrodes in endocardial contact and by unequal deployment of the splines.

Noncontact mapping, as described, allows high-resolution simultaneous activation maps of the entire cardiac chamber from just 1 beat of VT. Noncontact endocardial mapping was first described by Taccardi et al in 1987, when olive and cylindrical probes recorded canine ventricular paced electrograms, producing potentials of low frequency and amplitude. To enhance these, principles previously used to reconstruct epicardial maps from skin surface electrograms were applied. Noncontact mapping using cylindrical probes in open-chest dogs with an epicardial echocardiographic geometry matrix demonstrated good correlation between contact and reconstructed electrograms.

The system described here uses a percutaneous catheter-mounted electrode array. It combines inverse-solution mathematics with a catheter location device, creating several advantages over previous systems. First, a representation of cavity geometry is obtained, and second, the progress of a catheter within the cavity can be continuously monitored during mapping.

Despite several theoretical situations in which contact and the corresponding reconstructed electrogram may differ, good correlation between contact and reconstructed electrograms from the same sites have been shown, with accuracy decreasing with increasing endocardium-MEA distance, especially when this distance is >34 mm. Further validation, by cross-correlation comparison of reconstructed and contact electrograms recorded from the same site during VT, is presented in this article, showing a good correlation of 0.86 ± 0.16 and a mean timing error of -1.67 ± 10.06 ms.

The exit site was identified in 99% of VTs. In 67%, the MEA also identified a DP leading to the exit site. This was used as an initial target for confirmation by conventional mapping. Sites found suitable for ablation were validated by comparing the locations of successful and unsuccessful ablation sites with those of the DP on the map. RF delivered on
a mapped DP was significantly more successful than RF delivered on an exit or remote from the DP. The success rates for RF near incompletely identified DPs may reflect the small numbers and the fact that the ablation catheter had been positioned where diastolic activity had been identified by the MEA but did not qualify as relevant DP under our strict definition. Failure of RF applied to DPs may be due to catheter movement, inadequate lesion size, or errors in positioning the catheter or identifying the DP. However, these data suggest that mapped DPs are clinically relevant and are the optimum location for ablation of VT, with a 93% (12/13) success or partial success rate. RF applications to mapped exit sites were partially or completely successful in 50% but more often changed morphology than terminated VT. Of the VT DPs identified entirely and ablated successfully, RF was within or immediately adjacent to the mapped DP in all cases.

There are several possibilities why the MEA failed to identify a complete VT circuit. Perhaps some of the DP electrograms were too small to be detected or were intramural or epicardial and therefore could not be detected by the MEA. In some, the DPs were far from the MEA. Previous validation data indicate that this may affect the accuracy of electrogram reconstruction. However, the locations of parts of some DPs identified completely were >40 mm from the MEA and thus beyond the theoretical distance for optimal reconstruction. It is therefore difficult to be certain of the importance of distance in electrogram reconstruction. Once the current design of the MEA is deployed, it remains stable, so that the initial acquisition of chamber geometry remains valid for the procedure.

A difficulty with identifying diastolic components of some VT reentry circuits is saturation of the maps by LV repolarization. Although high-pass filtering will reduce this, it also attenuates diastolic electrograms and thus may not distinguish between the two. We have attempted to avoid this by our strict definition of relevant diastolic activity, but significant portions of the DP may be hidden by repolarization.

The long-term follow-up in 20 patients who survive indicates that 14 had no VT recurrence and a further 3 had recurrence of new VT morphologies. The recurrence of target VT was low, at 5.4% (2 of 37 VTs), even though 43 VTs induced during the procedures were either not targeted or not targeted by patient.

### TABLE 2. Procedural Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>Procedure/Fluoroscopy Time, min</th>
<th>MEA Deployment Time, min</th>
<th>No. of VTs (Clinical VT)</th>
<th>Cycle Length, ms, Mean (Range)</th>
<th>No. of VTs Ablated (Clinical/Nonclinical)</th>
<th>Recurrence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>520/43</td>
<td>47</td>
<td>4 (1)</td>
<td>426 (250–520)</td>
<td>1/1</td>
<td>1 (self-terminating)</td>
</tr>
<tr>
<td>2</td>
<td>420/46</td>
<td>47</td>
<td>3 (1)</td>
<td>426 (348–538)</td>
<td>1/2</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>435/48.5</td>
<td>47</td>
<td>1 (1)</td>
<td>280</td>
<td>1/0</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>440/89.6</td>
<td>47</td>
<td>2 (1)</td>
<td>292 (264–320)</td>
<td>1/1</td>
<td>New fast VT</td>
</tr>
<tr>
<td>5</td>
<td>570/86.3</td>
<td>47</td>
<td>2 (1)</td>
<td>365 (310–420)</td>
<td>1/1</td>
<td>New VT</td>
</tr>
<tr>
<td>6</td>
<td>495/105</td>
<td>47</td>
<td>6 (0)</td>
<td>467 (374–600)</td>
<td>0/2</td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>585/74.6</td>
<td>47</td>
<td>6 (0)</td>
<td>426 (338–470)</td>
<td>0/4</td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>460/72.1</td>
<td>47</td>
<td>2 (1)</td>
<td>449 (278–620)</td>
<td>1/0</td>
<td>Died (3 d)</td>
</tr>
<tr>
<td>9</td>
<td>315/66.3</td>
<td>47</td>
<td>1 (1)</td>
<td>374</td>
<td>1/0</td>
<td>None</td>
</tr>
<tr>
<td>10</td>
<td>395/89.8</td>
<td>47</td>
<td>3 (1)</td>
<td>512 (474–560)</td>
<td>1/2</td>
<td>New fast VT</td>
</tr>
<tr>
<td>11</td>
<td>435/36.3</td>
<td>47</td>
<td>3 (0)</td>
<td>360 (316–424)</td>
<td>0/2</td>
<td>None</td>
</tr>
<tr>
<td>12</td>
<td>515/61.9</td>
<td>47</td>
<td>1 (4)</td>
<td>382 (322–522)</td>
<td>1/2</td>
<td>None</td>
</tr>
<tr>
<td>13</td>
<td>390/41.2</td>
<td>47</td>
<td>1 (0)</td>
<td>450</td>
<td>0/0</td>
<td>Not ablated</td>
</tr>
<tr>
<td>14</td>
<td>310/43.1</td>
<td>47</td>
<td>1 (1)</td>
<td>370</td>
<td>?/0</td>
<td>Early recurrence</td>
</tr>
<tr>
<td>15</td>
<td>365/73.1</td>
<td>47</td>
<td>6 (2)</td>
<td>483 (370–600)</td>
<td>2/4</td>
<td>Early recurrence of slow, nonsustained VT</td>
</tr>
<tr>
<td>16</td>
<td>425/102.7</td>
<td>47</td>
<td>5 (1)</td>
<td>322 (170–405)</td>
<td>?/0</td>
<td>None</td>
</tr>
<tr>
<td>17</td>
<td>460/108</td>
<td>47</td>
<td>8 (1)</td>
<td>415 (330–446)</td>
<td>1/1</td>
<td>None</td>
</tr>
<tr>
<td>18</td>
<td>395/65.4</td>
<td>47</td>
<td>2 (2)</td>
<td>328 (308–347)</td>
<td>2/0</td>
<td>None</td>
</tr>
<tr>
<td>19</td>
<td>270/44.1</td>
<td>47</td>
<td>1 (0)</td>
<td>410</td>
<td>0/0</td>
<td>Not ablated, died (12 wks)</td>
</tr>
<tr>
<td>20</td>
<td>300/55.1</td>
<td>47</td>
<td>5 (1)</td>
<td>381 (254–352)</td>
<td>0/1</td>
<td>None</td>
</tr>
<tr>
<td>21</td>
<td>260/47.9</td>
<td>47</td>
<td>1 (1)</td>
<td>458</td>
<td>?/0</td>
<td>Died (12 h)</td>
</tr>
<tr>
<td>22</td>
<td>140/19.2</td>
<td>47</td>
<td>1 (1)</td>
<td>332</td>
<td>?/0</td>
<td>None</td>
</tr>
<tr>
<td>23</td>
<td>430/44.8</td>
<td>47</td>
<td>6 (2)</td>
<td>389 (280–528)</td>
<td>?/0</td>
<td>None</td>
</tr>
<tr>
<td>24</td>
<td>290/57.4</td>
<td>47</td>
<td>1 (1)</td>
<td>448</td>
<td>1/0</td>
<td>None</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>400.83 (105.47)/ 77.01 (65.16)</td>
<td>231.45 (114.56)</td>
<td>81 (25)</td>
<td>399.35 (89.76)</td>
<td>38</td>
<td>15/23</td>
</tr>
</tbody>
</table>

? indicates patients who had RF to clinical VT but did not achieve the study criteria for ablation.
ablated successfully. It is possible that they were either clinically irrelevant or dependent on the same substrate as the ablated VTs. Maps have demonstrated different VTs sharing portions of the same DP either in contrarotation or with different exits. Also, where complete DPs could not be identified, it was apparent that the exit sites of several VTs were close, so that they may have shared DPs. Many VTs became difficult to induce or sustain during a study. In this situation, the DP was located by use of VT maps recorded earlier. This area was then conventionally mapped during sinus rhythm when RF was applied, which may have resulted in ablation of VT.

Limitations of the Study
Because this study describes initial experience and development of a novel technology, it is largely descriptive and has a limited number of patients. Our validation by comparing the DPs identified by the system with the location of successful RF sites is not controlled and is subject to observer bias. The limited resolution of conventional percutaneous endocardial mapping and catheter location techniques prevents creation of comparable contact isopotential maps.

In addition, identification of DPs relied on the assumption that all VT exits were endocardial, but some may have been epicardial with endocardial breakthrough identified. Comparison of the locations of successful and unsuccessful RF sites relative to DPs partly addresses this.

Conclusions
Noncontact mapping safely mapped and guided ablation of VT in human LVs. It rapidly identified VT exit sites and thus starting points for conventional mapping. In >50% of cases, it identified some of the DPs and therefore suitable targets for ablation, allowed monitoring of the position of other catheters, and demonstrated a relationship between the site of the DP and the outcome of RF application.

Figure 3. Histogram showing cross-correlation between contact and reconstructed electrograms recorded during VT. Majority of compared electrograms had a perfect correlation (1), and almost all comparisons gave a correlation >0.8, which was considered to be an acceptable value.
Figure 4. A, Maps of VT. Virtual endocardium has been opened along anterior septum. Anatomic labels are as follows: Base and Apex, LV base and apex; Septum, LV septum; Lateral, LV lateral wall. Successful RF site is shown with green dot. Direction of activation is shown with blue arrows. Letters A through E represent positions at which electrograms have been reconstructed and displayed in Figure 4B. Frames 1 and 2 show end of systole. Diastolic activity progresses from base to apex in frames 3 and 4 before wave front splits in frame 5. Anteroseptal wave front moves from base to apex (frame 6) before blocking (bystander) (frame 7). A more posterior wave front goes from base to apex (frames 5 and 6), turns at apical end (frame 6), and progresses basally (frames 7 and 8). Wave front exits DP, resulting in systolic activation (frames 9, 10, 11, 12). B, Waveform window shows reconstructed bipolar electrograms from sites A through E in panel A with different bipole directions, enhancing diastolic potentials, which progress in time from E to A before LV systolic activation. Also shown is a bipolar electrogram from a contact catheter located at RF site in Figure 4A and ECG lead V3. Contact electrogram is timed between electrograms B and A and is saturated because pacing has been used to prove concealed entrainment.
Figure 5. A, VT1 electrograms showing contact electrograms recorded at successful RF site in Figure 6 with late diastolic potentials (dp). Diastolic potentials on distal bipolar leads proximal bipolar. B, VT4 electrograms. Mapping catheter is positioned at same site as in Figures 5A and 6 and shows early diastolic potentials. Timing of diastolic potentials on distal and proximal bipolar is now reversed. C, Electrograms during pacing from VT4 DP from same site as in Figures 5A and 5B and 6, resulting in acceleration of VT4. An increasing stimulus to QRS interval results from rapid pacing, leading to VT4 termination. After 2 cycles, a perfect pace match of VT1 is seen, suggesting reversal of diastolic activation with a shorter pacing to QRS interval. Pacing is closely followed by spontaneous onset of VT1, which confirms accuracy of pace match.
Figure 6. Maps recorded during VTs shown in Figure 5, with 2 VTs using same DP in contrarotation. Virtual endocardium has been opened along anterior septum so that 2 edges are in continuity. Anatomic locations as in previous figures. Activation is shown by white areas. Successful RF site is shown with a green dot. A, VT1. Frame 1 shows DP activation just before exit. Frames 2 through 4 show LV systolic activation. Frames 5 through 8 show activation progressing along DP from apical septum to a basolateral exit. B, VT4. Frames 1 through 4 show activation in DP progressing from basal and lateral (exit site for VT shown in Figure 6A) toward an exit in apical septum. Frames 5 through 7 show LV systolic activation of LV.
TABLE 3. Results of RF Application During VT

<table>
<thead>
<tr>
<th></th>
<th>On DP</th>
<th>On Exit*</th>
<th>Near DP</th>
<th>Remote From DP</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Success</td>
<td>10 (77)</td>
<td>5 (21)</td>
<td>4 (80)</td>
<td>3 (9)</td>
<td>22</td>
</tr>
<tr>
<td>Partial</td>
<td>2 (15)</td>
<td>7 (29)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>9</td>
</tr>
<tr>
<td>Failure</td>
<td>1 (8)</td>
<td>12 (50)</td>
<td>1 (20)</td>
<td>31 (91)</td>
<td>45</td>
</tr>
<tr>
<td>Total</td>
<td>13 (100)</td>
<td>24 (100)</td>
<td>5 (100)</td>
<td>34 (100)</td>
<td>76</td>
</tr>
</tbody>
</table>

On DP indicates that RF was delivered on the mapped DP; on exit, RF delivered at an incompletely identified DP; and remote from DP, RF delivered at a site remote from the mapped DP. Values are n (%).

*P<0.005 vs On DP; †P<0.0001 vs On DP.

References

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