Catheter Ablation of Multiple Ventricular Tachycardias After Myocardial Infarction Guided by Combined Contact and Noncontact Mapping

Hanno U. Klemm, MD, MSc*; Rodolfo Ventura, MD*; Daniel Steven, MD; Christin Johnsen, MD; Thomas Rostock, MD; Boris Lutomsky, MD; Tim Risius, MD; Thomas Meinertz, MD; Stephan Willems, MD

Background—Insights gained from noncontact mapping of ventricular tachycardia (VT) have not been systematically applied to contact maps. This study sought to unify both techniques for an individualized approach to the patient with multiple ischemic VTs irrespective of cycle length.

Methods and Results—For 12 consecutive patients with chronic myocardial infarction and recurrent VT, bipolar contact maps were acquired during sinus or paced rhythm. Additional noncontact maps were obtained during 48 induced VTs (cycle length 192 to 579 ms). Endocardial exit sites were superimposed on contact maps and verified by pace-mapping. Radiofrequency lesions were extended for critical borders defined by multiple neighboring exits and followed the isovoltage contour line of contact maps. Nine critical borders were identified in 8 patients and constituted the substrate for 31 VTs. The voltage at exit sites was 0.8 mV (range 0.1 to 2.3). Noncontact maps revealed 23±18% of isthmus conduction. Thirty-seven (77%) of all and 83% of clinically documented VTs were rendered noninducible irrespective of cycle length by application of 27 radiofrequency lesions (range 18 to 56). Spontaneous transitions between distinct VTs along critical borders were demonstrated in 4 patients. Pace-mapping reproduced the QRS morphology of 81% of VTs and was associated with successful ablation (P<0.01). Noninducibility of any sustained VT was reached for 8 (67%) patients. During 15 months (range 5 to 28) of follow-up, 8 patients remained without recurrence, and VT episodes were reduced in the other 4 patients (P<0.01). VT cycle length was not predictive for acute or long-term success.

Conclusion—The combined approach of contact and noncontact mapping effectively defines critical borders as the substrate of multiple VTs without limitation for unstable VTs. (Circulation. 2007;115:2697-2704.)

Key Words: catheter ablation ▪ mapping ▪ tachycardia

A number of criteria for mapping and ablation of ventricular tachycardia (VT) after remote myocardial infarction have been established.1-3 Both activation mapping4 and approaches during sinus rhythm5,6 have been shown to be effective for VT with a distinct diastolic pathway. A mature scar was found to constitute the underlying substrate of VT in most of these cases with slow conduction at an isthmus site.7

Clinical Perspective p 2704

Catheter ablation of VT, however, can still be challenging, especially in cases of intolerable arrhythmia when mapping conditions are limited. Furthermore, the relevance of “non-clinical” and fast VTs for long-term success is uncertain. Thus, a need exists to develop alternative approaches to VT ablation and thus facilitated delineation of the arrhythmogenic substrate.8 The purpose of this study was to demonstrate the combination of electroanatomic mapping with a noncontact mapping system and to provide an individualized basis for ablation that is not limited by cycle length (CL) and targets all inducible VTs.

Methods

Patients

Twelve patients referred to the University Heart Center Hamburg for first-time VT ablation were studied. All patients suffered from recurrent ventricular tachycardia refractory to antiarrhythmic treatment. The subjects consisted of 11 men and 1 woman with a median age of 65 years (range 58 to 78) (Table 1). All patients had a history of myocardial infarction with thrombolysis in 1 case and primary percutaneous coronary intervention in 8 cases. Coronary artery bypass graft surgery was performed in 4 subjects. The mean left ventricular ejection fraction was 33% (range 20 to 50). An implantable cardioverter-defibrillator (ICD) had been implanted before ablation in 5 patients. The remaining 7 patients received a device before they were discharged from the hospital. Frequent episodes of VT before or that continued shock deliveries after implantation of an ICD were present in all patients. Intracardiac electrograms could be stored by all ICDs and documentation of VT was available as 12-lead
ECG for 8 and as intracardiac electrogram for 4 patients. Documentation of spontaneous VT was available for all patients. One patient presented 2 documented spontaneous VTs. Coronary angiography was performed in all cases to exclude acute ischemia. Before ablation, 7 patients were on metoprolol, 4 were taking amiodarone, and 1 patient was not on any drug regimen. In all cases, antiarrhythmic drugs were unsuccessful in the prevention of VT. For ablation, metoprolol was discontinued at 5 half-lives before the beginning of procedure, whereas amiodarone was continued. After catheter ablation, antiarrhythmic therapy was continued unmodified as before. For all patients on amiodarone, treatment had reached a steady state at 200 mg intake per day at the time of the ablation procedure and during follow-up.

Ventricular Tachycardia–Induction Protocol

Electrophysiological studies were performed in the postabsorption state. Systemic arterial blood pressure was continuously recorded in addition to bipolar electrograms with the conventional electrophysiology system (Labsystem Pro, Bard Electrophysiology, Lowell, Mass). A 5F quadripolar diagnostic catheter (Viking, Bard) was inserted into a femoral vein and placed at the right ventricular apex and outflow tract. The inducibility of VT was tested by programmed ventricular stimulation with up to 3 extrastimuli and a shortest coupling interval of 180 ms. Two basic drive CLs of 510 ms and 440 ms were applied.

In the case of induction of a sustained VT, overdrive stimulation at 80% of the spontaneous CL was performed for up to 5 s. Repeated overdrive stimulations included ramps with sequential shortening of the stimulation CL at 10-ms steps up to a minimum of 200 ms. If VT continued or ventricular flutter or fibrillation was induced, a biphasic cardioverter/defibrillator shock of 200 J restored sinus rhythm (M-Series, Zoll, Chelmsford, Mass).

For all induced monomorphic VT, either by extrastimuli or overdrive pacing, a 12-lead ECG was examined. VTs were preliminarily assumed as different and eligible for further analysis if the QRS morphology varied in at least 1 of the 12 leads. The VT isthmus and exit sites were subsequently identified by analysis of virtual electrograms (see Analysis of Virtual Electrograms below). In case of a separate exit site, the VT was considered a unique entity for mapping and ablation (Figure 1). In contrast, transitional beats between stable morphologies were not individually treated and a minimum of 3 identical beats was required to define a target VT. Induction of a previously spontaneous VT was assumed if the CL and morphology of the 12-lead ECG or intracardiac electrogram obtained by repeated interrogation of the ICD matched the documented VT.

### Mapping and Ablation Setup

The noncontact mapping system that was used (EnSite 3000, St. Jude Medical, St. Paul, Minn) has been described previously. Briefly, the system is based on a 64-polar multielectrode array mounted on an inflatable 7.5-mL balloon. The multielectrode array was advanced over a guide wire into the left ventricle with the use of a retrograde

---

**TABLE 1. Patient Characteristics**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, y</th>
<th>LVEF, %</th>
<th>CAD †</th>
<th>Time Post-MI, y</th>
<th>History of T-lysis, PCI, or CABG</th>
<th>AAD</th>
<th>No. of Inducible VTs</th>
<th>Range of VT-CL, ms</th>
<th>No. of Fast VTs</th>
<th>Episodes 3 Months Preablation, n/mo</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>66</td>
<td>30</td>
<td>2</td>
<td>33</td>
<td>P</td>
<td>Met</td>
<td>3</td>
<td>219 to 308</td>
<td>2</td>
<td>40</td>
</tr>
<tr>
<td>2</td>
<td>67</td>
<td>20</td>
<td>3</td>
<td>32, 14</td>
<td>P</td>
<td>Ami</td>
<td>2</td>
<td>335 to 381</td>
<td>0</td>
<td>Incessant</td>
</tr>
<tr>
<td>3</td>
<td>58</td>
<td>50</td>
<td>2</td>
<td>7</td>
<td>P</td>
<td>Met</td>
<td>4</td>
<td>289 to 378</td>
<td>0</td>
<td>18</td>
</tr>
<tr>
<td>4</td>
<td>61</td>
<td>25</td>
<td>2</td>
<td>10</td>
<td>T, P</td>
<td>Ami</td>
<td>7</td>
<td>250 to 579</td>
<td>1</td>
<td>22</td>
</tr>
<tr>
<td>5</td>
<td>61</td>
<td>20</td>
<td>3</td>
<td>27, 25, 16</td>
<td>C</td>
<td>Ami</td>
<td>11</td>
<td>305 to 560</td>
<td>0</td>
<td>138</td>
</tr>
<tr>
<td>6</td>
<td>60</td>
<td>40</td>
<td>3</td>
<td>24</td>
<td>C</td>
<td>Met</td>
<td>2</td>
<td>219 to 274</td>
<td>1</td>
<td>27</td>
</tr>
<tr>
<td>7*</td>
<td>69</td>
<td>50</td>
<td>2</td>
<td>2</td>
<td>C</td>
<td>Met</td>
<td>4</td>
<td>247 to 271</td>
<td>1</td>
<td>16</td>
</tr>
<tr>
<td>8</td>
<td>65</td>
<td>30</td>
<td>3</td>
<td>10, 8</td>
<td>C</td>
<td>Ami</td>
<td>2</td>
<td>342 to 408</td>
<td>0</td>
<td>15</td>
</tr>
<tr>
<td>9</td>
<td>64</td>
<td>40</td>
<td>1</td>
<td>4</td>
<td>P</td>
<td>Met</td>
<td>3</td>
<td>250 to 463</td>
<td>1</td>
<td>18</td>
</tr>
<tr>
<td>10</td>
<td>78</td>
<td>30</td>
<td>2</td>
<td>3</td>
<td>C</td>
<td>Met</td>
<td>3</td>
<td>231 to 365</td>
<td>1</td>
<td>23</td>
</tr>
<tr>
<td>11</td>
<td>68</td>
<td>35</td>
<td>3</td>
<td>14</td>
<td>P</td>
<td>None</td>
<td>3</td>
<td>323 to 335</td>
<td>0</td>
<td>30</td>
</tr>
<tr>
<td>12</td>
<td>62</td>
<td>50</td>
<td>2</td>
<td>1</td>
<td>P</td>
<td>Met</td>
<td>4</td>
<td>192 to 304</td>
<td>3</td>
<td>55</td>
</tr>
</tbody>
</table>

AAD indicates antiarrhythmic drugs; Ami, amiodarone; CABG, coronary artery bypass grafting (C); CAD, coronary artery disease; LVEF, left ventricular ejection fraction; Met, metoprolol (discontinued prior to electrophysiological study); MI, myocardial infarction; PCI, percutaneous coronary intervention (P); and T-lysis, thrombolysis (T).

*Female patient.
†Number of vessels functionally stenotic or revascularized.
Figure 2. Contact voltage map of patient 5 that shows superimposed VT exits (E) defined by noncontact mapping and applied ablation lesions. Left, exit sites were defined by the onset of the reconstructed unipolar QS deflection that preceded the VT QRS complex. The color range represents bipolar voltage amplitudes (peak-to-peak) with control sliders set from 0 (red) to 1.5 mV (purple). The modified posterior view shows exits of distinct VT entities as defined by QRS morphology; exits are numbered in the order of induction. A critical border was identified with exits 1 to 9 (excluding E4 and E6) located on the same aspect of an extended septal to inferior myocardial scar area. Median voltage of the critical border was 0.8 mV. The ablation lesions followed the contour of the contact map to approach VT1 to VT9 (excluding VT4 and VT6). The exit of VT10 was located on the anteroseptal aspect and is therefore not visible. Right, 1 cycle of each VT is displayed centered on the QRS complex. The numbers below correspond to the VT exit sites in the left panel. Please note the left bundle branch block pattern for the septal and apicoseptal VT exits (E4, E6, and E11).

The actual voltages at a specific point in time are displayed as isopotential maps with each voltage represented by a different color. The color range can be adjusted as desired for analysis. Within a recorded sequence the investigator is free to view the isopotential map for any point in time or step forward or backward in time as needed. A digital high-pass filter can retrospectively be applied at any time during the analysis and greatly influences the detection of the earliest electrical activity. T-waves are also well recorded by unipolar electrograms and can be the source of a shift of the isoelectric baseline when repolarization reaches electrical end-diastole. High-pass filters are therefore adjusted at the lowest value that minimizes the shift, usually in the range of 0.5 to 4 Hz. A 150-Hz inverse Chebyshev low-pass filter was used to minimize artifacts of the locator signal. In addition to the propagation of isoelectric wave fronts, virtual electrograms were manually reviewed to locate areas with QS(r') morphology. A unipolar QS(r') morphology that preceded QRS onset is regarded as a source of activation from which wave fronts spread out to activate surrounding myocardium. In the case of VT activation mapping, this corresponds to the VT exit where isthmus conduction enters healthy myocardium. A late r' deflection may be present and reflects a far field from remote activated areas. After the exit site is defined, isthmus conduction was traced back in time with careful adjustments of the voltage color scale sliders to obtain the earliest endocardial activation that exceeded baseline noise. The site of earliest electrical activity and the exit were marked on the virtual geometry for each tachycardia.

Pace-mapping was performed during sinus or paced rhythm by bipolar pacing (up to 10 mA, 2 ms) at the exit sites with the use of the distal pair of electrodes of the quadripolar catheter at a rate similar to the VT CL in order to confirm the validity of the analysis. All exit sites revealed by contact mapping were superimposed on the previously created contact maps. Local bipolar voltages at the exit sites were obtained from the Ensite landmarks map, which uses a distance-weighted interpolation of voltages from surrounding points. Because points were sampled at high density at scar borders, at least 3 points were located within 10 mm of each exit site.

**Definitions**

The CL was defined as RR interval and expressed in milliseconds. The duration of the electrical diastole was defined as CL-QRS duration. The amount of isthmus conduction mapped was defined as the time interval from earliest detected electrical activity that resulted...
TABLE 2. Mapping and Ablation Results

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>No. of Contact Points</th>
<th>Surface Area,* cm²</th>
<th>Scar Area,† cm²</th>
<th>No. of Critical Borders</th>
<th>Median Bipolar Voltage of Critical Border, mV</th>
<th>Earliest Activity (Median Time to QRS), ms</th>
<th>Percentage of Isthmus Defined (range)</th>
<th>No. of RF Applications</th>
<th>Linear Lesions, cm</th>
<th>Procedure End Point</th>
<th>Total Postablation Episodes During Follow-Up, n</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>288</td>
<td>214</td>
<td>123</td>
<td>0</td>
<td>18 (8 to 21)</td>
<td>17 (16 to 30)</td>
<td>53 (30 + 20 to 29)</td>
<td>36</td>
<td>0</td>
<td>0</td>
<td>2 VTs</td>
</tr>
<tr>
<td>2</td>
<td>493</td>
<td>235</td>
<td>175</td>
<td>1</td>
<td>0.7 (0.6 to 0.7)</td>
<td>7 (3 to 10)</td>
<td>8 (4 to 13)</td>
<td>10</td>
<td>6</td>
<td>0</td>
<td>3 VTs</td>
</tr>
<tr>
<td>3</td>
<td>462</td>
<td>209</td>
<td>47</td>
<td>2</td>
<td>1.2 (0.3 to 2.1)</td>
<td>39 (26 to 79)</td>
<td>26 (17 to 68)</td>
<td>37 + 33</td>
<td>1</td>
<td>1 vs VT</td>
<td>3 VTs</td>
</tr>
<tr>
<td>4</td>
<td>474</td>
<td>211</td>
<td>80</td>
<td>1</td>
<td>1.0 (0.5 to 1.7)</td>
<td>20 (7 to 96)</td>
<td>30 (0 to 55)</td>
<td>26</td>
<td>34 + 18</td>
<td>2 VTs</td>
<td>0 VTs</td>
</tr>
<tr>
<td>5</td>
<td>452</td>
<td>352</td>
<td>197</td>
<td>1</td>
<td>0.8 (0.7 to 2.3)</td>
<td>24 (8 to 75)</td>
<td>21 (4 to 46)</td>
<td>33</td>
<td>70 + 23 to 16 + 21</td>
<td>3 VTs</td>
<td>2 VTs</td>
</tr>
<tr>
<td>6</td>
<td>307</td>
<td>153</td>
<td>51</td>
<td>0</td>
<td>18 (15 to 21)</td>
<td>20 (18 to 21)</td>
<td>18</td>
<td>21 + 20</td>
<td>0 VTs</td>
<td>0 VTs</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>581</td>
<td>223</td>
<td>63</td>
<td>1</td>
<td>0.8 (0.5 to 1.2)</td>
<td>15 (9 to 26)</td>
<td>15 (10 to 28)</td>
<td>27</td>
<td>53 + 13</td>
<td>Epi nsVT</td>
<td>0 VTs</td>
</tr>
<tr>
<td>8</td>
<td>504</td>
<td>278</td>
<td>157</td>
<td>0</td>
<td>51 (14 to 87)</td>
<td>38 (18 to 57)</td>
<td>34</td>
<td>31 + 30</td>
<td>0 VTs</td>
<td>0 VTs</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>302</td>
<td>170</td>
<td>59</td>
<td>0</td>
<td>NA</td>
<td>43 (29 to 80)</td>
<td>36 (14 to 73)</td>
<td>26</td>
<td>24 + 28</td>
<td>1 fast VT</td>
<td>0 VTs</td>
</tr>
<tr>
<td>10</td>
<td>377</td>
<td>175</td>
<td>51</td>
<td>1</td>
<td>1.0 (0.9 to 1.2)</td>
<td>28 (2 to 44)</td>
<td>22 (2 to 63)</td>
<td>38</td>
<td>46 + 29</td>
<td>0 VTs</td>
<td>0 VTs</td>
</tr>
<tr>
<td>11</td>
<td>495</td>
<td>170</td>
<td>78</td>
<td>1</td>
<td>1.2 (1.2 to 2.1)</td>
<td>39 (14 to 39)</td>
<td>29 (12 to 33)</td>
<td>21</td>
<td>43 + 39</td>
<td>0 VTs</td>
<td>0 VTs</td>
</tr>
<tr>
<td>12</td>
<td>512</td>
<td>144</td>
<td>66</td>
<td>1</td>
<td>0.4 (0.4 to 1.8)</td>
<td>4 (1 to 9)</td>
<td>10 (2 to 16)</td>
<td>19</td>
<td>40</td>
<td>VFlut</td>
<td>13 VTs</td>
</tr>
</tbody>
</table>

Data in parentheses are ranges. NI indicates not inducible; Epi, epicardial; ns, nonsustained; and VFlut, ventricular flutter.

*Surface of the reconstructed left ventricular cavity.
†Based on a 1.5-mV scar definition.

in a continuous conduction to the VT exit site to QRS onset as measured from the surface ECG. A fast VT was considered any VT with a CL <250 ms. A myocardial region that contained multiple VT exits in close vicinity to the edge of a myocardial low-voltage area was defined as critical border (Figure 2). A good pace map was defined as the reproduction of the QRS morphology during VT in all 12 ECG leads with a maximum of 1 minor configuration difference. An epicardial origin of a VT was presumed if initial R waves were present on all virtual electrograms and no QS morphology was detectable. Epicardial VTs were not targeted by ablation.

**Ablation Strategy**
For VTs caused by a solitary isthmus that was not part of a critical border, short linear ablation lines were aimed at the VT exit sites perpendicular and centered in relation to the preceding isthmus activation. The initial length of the ablation line was limited to 2 cm but could be extended during subsequent ablation if the target VT was still inducible. In the case of demonstration of a critical border, multiple VTs were simultaneously targeted by application of an extended ablation line. The ablation lesion followed the isovoltage contour line of the superimposed contact map and contained all identified exit sites.

**Follow-Up**
Patients were discharged after a minimum of 72 hours of observation after ablation. Periodic evaluations took place on an outpatient basis every 3 months or on any occurrence of ICD therapy delivery. Antiarrhythmic therapy was unchanged during follow-up and maintained as before ablation. During follow-up, ICDs were interrogated, and data about clinical status, VT recurrences, VT CL, drugs, and mortality were collected for all patients. In cases of admission in other institutions, colleagues were asked to send clinical documentation that included ECG.

**Statistics**
Continuous data are expressed as medians and ranges in parenthesis. Under the assumption that induced VTs are independent entities, subgroups of VTs were compared with the 2-tailed Mann-Whitney test. Categorical data on pace mapping and patient-based data on procedural results and follow-up were represented as absolute frequencies of events. Subsequent comparisons were performed with 2×2 cross-tabulations and Fisher exact test. The Wilcoxon signed ranks test was used to compare the numbers of episodes before and after ablation. A value of P<0.05 was considered statistically significant. Statistical analyses were performed with commercially available software (SPSS 12, SPSS Inc., Chicago, Ill).

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

**Results**
VTs were inducible in all patients, and unipolar electrogram data were collected during a total of 48 VTs. The median number of inducible VTs per patient was 3 (range 2 to 11). The median CL for all VTs was 335 ms (range 192 to 579). The median electrical diastole was 111 ms (range 24 to 347). Nine fast VTs were inducible in 6 patients (Table 1). Endocardial exit sites were identified for 47 VTs. An epicardial origin was assumed for 1 of the 4 induced VTs of patient 7 and not targeted for ablation. Criteria for a good pace-map were met during stimulation at the VT exit site in 39 (81%) cases. The total median procedure time was 4.5 h (range 3.8 to 6.2). For catheter placement, geometry reconstruction, and confirmation of catheter position during ablation, 36 min (range 21 to 86) of fluoroscopy were required.

**Bipolar Voltage at Exit Sites**
Detailed electroanatomic voltage maps were constructed and contained 468 (range 288 to 581) sampled points. Areas with <1.5 mV bipolar voltage measured 96 cm² (range 47 to 197). In 7 patients a single critical border was identified (Figure 2).
One patient showed 2 critical borders at opposite sites of an extended myocardial scar area (Table 2). For the remaining patients solitary exit sites at distant locations of the low-voltage border were the substrate for all induced VTs.

Exit sites were identified at the border of low-voltage areas with a median voltage of 0.8 mV (range 0.1 to 2.3). Critical borders consisted of the exit sites of 31 VTs along bipolar isovoltage lines (Figure 2). The voltage of the lines was variable with a median of 0.9 mV (range 0.3 to 2.3). Median bipolar voltages at 16 solitary exit sites, 0.8 mV (0.1 to 2.0), were not significantly different from critical borders ($P=0.47$). Furthermore, no correlation was found between CL and voltage at the exit site (Pearson correlation $R=-0.001$, $P=0.92$).

**Isthmus Characterization**

Electrical activity consistent with isthmus conduction within myocardial scar and border zones was recorded for a variable interval that preceded the onset of the QRS complex. The median interval from earliest electrical activity detected by virtual electrograms as the beginning of the local QS deflection to QRS onset was 21 ms (range 0 to 96). This corresponds to 18% (range 0% to 73%) of the diastolic pathway. The earliest detection of isthmus conduction was 27 ms (range 3 to 296) ms for VTs that fulfilled the criteria of a good pace map. This was significantly longer than a median of 3 ms (range 0 to 24) ms measured if QRS morphology could not be reproduced by pacing at the exit site ($P<0.01$). The angle between detectable isthmus conduction and the scar border varied markedly without an identifiable predominant pattern. For 11 (23%) VTs isthmus conduction was mappable for <10 ms before the endocardial exit with limited information on the orientation of the pathway. A large negative potential caused by recorded T-waves partially obscured early isthmus conduction despite variations of the high-pass filter in 12 VTs. This was resolved for all VTs when activation was traced to the exit site where the potential of the depolarizing myocardium became more negative than the T-wave far-field. Therefore, the presence of T-waves imposed no limitation on the detection of exit sites.

**Ablation Results**

A median of 27 (range 18 to 56) RF lesions were applied. A total of 37 (77%) VTs were rendered noninducible. The ablation end point of noninducibility of any sustained VT was reached in 8 patients (67%). Complete noninducibility of any sustained or nonsustained VT/VF was obtained in 6 patients (50%). Documented spontaneous VTs were successfully ablated in 10 patients (83%). Only fast VTs or ventricular flutter remained inducible in 2 patients (17%). Ablation of the 39 VTs showed a good match of the QRS morphology during pace mapping was successful in 35 cases. In contrast, only 2 of the 8 VTs without a satisfactory pace map were not inducible after ablation ($P<0.01$). For individual VT exits, the median size of the linear lesion was 24 mm (range 13 to 39). Critical borders were ablated along the isovoltage line that extended a median of 45 mm (range 33 to 70). These lesions were substantially longer than lines that targeted solitary exits ($P<0.01$). Fast VTs were equally distributed between critical borders (45%) and solitary exits (55%). Ablation success was not dependent on the CL with the same efficacy for fast CLs (78%) and longer (>250 ms) CL (79%) VTs ($P=1.0$).

Spontaneous transitions between distinct VT morphologies with intermediate exits along the critical border were identified and characterized in 4 patients (Figure 3). With 1 exception, the CL of VTs that showed transitions, 256 ms (range 219 to 405), was faster than the median CL, and only a small change in CL, 11 ms (range 3 to 31), was noted between the initial and the subsequent VT. In patients with documented transitions, nonsustained VTs remained inducible after ablation. This was found only in 2 of the 8 patients without transitions ($P=0.06$). After incomplete creation of the ablation lesion, a detour of the electrical activation that took a different exit within the critical border was found in 3 patients during reinduction (Figure 4). The modified activation was blocked in 2 cases by extension of the linear ablation line.

**Complications**

Because of extensive peripheral artery disease, a transseptal approach had to be used in patient 5. For this patient, ablation was discontinued after creation of 4 linear lesions caused by cardiogenic shock after ventricular fibrillation. A transient mechanical total AV block occurred in patient 8 after placement of the multielectrode array–catheter that required constant right ventricular pacing. The block resolved spontaneously 48 hours after the procedure.

---

**Figure 3.** Same patient as in Figure 2. Spontaneous transition of 1 VT morphology into another incorporated 11 intermediate exit sites identified by beat-wise noncontact mapping. Left, cutout of a myocardial scar area that contained a critical border. The bipolar low-voltage area is darkly shaded. The color range represents the unipolar voltages at QRS onset of VT7. Activation is centered at exit E7. Consecutive intermediate beats are labeled alphabetically (a to k) at the respective exit sites. VT8 is sustained with reproducible identification of E8 for the following beats. Right, the corresponding ECG tracing is shown with marks set at the onsets of intermediate beats.
Follow-Up
After a follow-up of 15 months (range 5 to 28), 8 patients (67%) had no recurrence of any VT/VF. One patient (patient 1) suffered a cluster of ICD discharges caused by polymorphic VT 9 days after ablation without any further episodes. Two patients (patients 3 and 12) presented with recurrences of monomorphic VT 4 and 6 months after discharge, respectively. Patient 5 died of congestive heart failure 2 days after ablation; VTs that showed small alterations of the QRS-complex as compared with the morphologies during ablation were recorded. One of the 4 patients with recurrences was among the 8 patients who reached the ablation end point. The remaining 3 patients with recurrences, however, failed to reach ablation end point ($P=0.07$). The ablation end point is presumably predictive of a favorable follow-up, with a theoretical positive predictive value of 88%. The induction of fast VTs was not associated with recurrence during follow-up as compared with VTs that showed a longer CL ($P=1.0$). A significant reduction of the total number of episodes from 25 (range 15 to incessant) preablation down to a median of 0 (range 0 to 32) during follow-up ($P<0.01$) could be demonstrated (Figure 5). Antiarrhythmic drug treatment remained unchanged during follow-up in all patients.

Discussion
Main Findings
The present study demonstrated the benefits of a combination of contact and noncontact mapping for catheter ablation of multiple and fast VTs that cover a broad range of CL (192 ms to 579 ms). Exit sites identified by noncontact mapping are located within the border zone of the myocardial scar defined by conventional voltage scar criteria. These are helpful to narrow down the area in which ablation lesions should be created. Noncontact mapping allows the definition of numerous exit sites and transition states between individual VT morphologies. The study also showed the relevance of extended ablation along critical borders shared by multiple exit sites to include a majority of inducible VTs. Favorable acute and long-term results were obtained and associated with the selected ablation end point.

Figure 4. Frames of sequential unipolar isopotential maps are shown after creation of a linear ablation lesion at a critical border of patient 10. The activation sequence was observed during reinduction of VT. Exit sites of 2 VTs (E1 and E2) were included in the line; exit E3 is a remote site discontinuous to the critical border. The activation wavefront is depicted as the leading edge of the unipolar potentials. White and purple represent activated and resting myocardium, respectively. The aortic valve is simplified by a sphere (AV) in this posteroinferior view. The position of His-bundle activation (HIS) recorded during sinus rhythm with the mapping catheter was also labeled. Frame 1, diastolic isthmus activation approaches the ablation line. Frame 2, the previous pathway that exited at E1 is blocked. Frame 3, the activation takes a detour with a shifted exit closer to E2 and activates the left ventricle. Frame 4, myocardium distal to the ablation line is now activated late.

Figure 5. Reduction of VT episodes per month by radiofrequency ablation in the patient cohort. The number of episodes per month is calculated from all episodes within each 3-month interval before and up to 12 months after ablation. A significant reduction after ablation is evident ($P<0.01$). No episodes were detected after 6 months after ablation.
Individual Definition of Myocardial Scar Areas

Bipolar voltage criteria of diseased myocardium are well established. However, patients with coronary artery disease and previous myocardial infarction show extended left ventricular areas of low voltage. A universal definition of a critical voltage for diseased myocardium and dense scar of 1.5 mV and 0.5 mV, respectively, as proposed in the study by Marchinski et al., has been challenged by results from recent studies that suggest a broader voltage range of critical sites. Also, the low-voltage zone can be further characterized by conducting channels. The addition of noncontact mapping to traditional contact maps facilitates an individual approach that defines the critical voltage for each patient. However, the voltage of exit sites found in the present study, 0.8 mV (range 0.1 to 2.3), underscores the relevance of the thresholds of 1.5 mV and 0.5 mV as boundaries previously introduced as an orientation to guide catheter mapping for VT ablation.

Identification of the Protected Isthmus

In concordance with previously published data, earliest isthmus activity was recorded at a broad range from 0 to 96 ms before the QRS complex. This had minor consequences for the definition of critical borders but may limit ablation of solitary exit sites. In an early study by Schilling et al., mapping of the complete pathway was demonstrated in 21%, and an extensive definition of the isthmus was obtained for the remaining VTs. The pathway was, however, not connected to the exit in 23%. These VTs may correspond to the tachycardia with negative deflections in the virtual electrograms were required to detectable channels or unexcitable tissue within scar areas and can complicate the definition of the diastolic pathway. A clear-cut definition of epicardial pathways cannot be made with either technique and remains subject to epicardial mapping. Termination of tachycardia was unavailable as proof of success because all ablation was performed during sinus or paced rhythm. No attempts were made to induce VT via left ventricular stimulation. Because of the superposition of T-wave repolarization, we regard the observation of the point of maximum negativity that travels through scar tissue as not reliable enough to accurately localize the isthmus. Local negative deflections in the virtual electrograms were required to define isthmus conduction.

In the present study, only 1 VT did not present an endocardial exit that could be approached for ablation. At the earliest endocardial activation site, the initial deflection of the unipolar electrograms was positive, which suggested an approaching wave compatible with epicardial reentry. Pace mapping at this site did not match the target VT. On the contrary, in the other 8 cases of pace mapping mismatch at the presumed exit side, unipolar electrograms of the earliest myocardial activation presented with negative deflections, which supports an endocardial exit of the VT. The time from detectable activation to QRS onset, however, was short, and catheter ablation was unsuccessful at this site. Potentially, this is an expression of an epicardial or intramural reentry with a localized endocardial breakthrough.

Ablation Strategy

The ablation strategy was based on the hypothesis that any inducible sustained VT circuit is of relevance within a slowly changing substrate. Other approaches based on fragmented low-amplitude potential or late activation during sinus rhythm have been used for the ablation of unstable VTs. These characteristics have been shown to be present at successful ablation sites. They can, however, also be demonstrated at sites not involved in the reentry circuit and are therefore not specific for a distinct VT. The value of pace mapping to identify exit sites is also limited by the observation that pace mapping in agreement with other studies was successful in only 81% of cases. On the other hand, pacing at distant sites can result in a good match. Several strategies have guided the placement of linear lesions. We preferred a perpendicular line with respect to the final diastolic pathway to ablation along the isthmus. The observation of several adjacent exits and transition states was the rationale for this approach. No attempts were made to interconnect scar areas or to connect a line to an anatomic boundary. During 15 months (range 5 to 28) of follow-up, only 1 (13%) of the 8 patients who reached the end point experienced a recurrence of VT. This outcome emphasizes the importance of considering all inducible VTs for ablation. A direct comparison with recent studies is limited by differences in the definition of target VT, ablation end point, and criteria for recurrence. Ablation of unstable VT can be effective in the long term in 75% of patients. Studies that use detectable channels within scar areas or for tolerated and nontolerated VT reported recurrences in 23% and 29% of patients, respectively. In the present investigation, all induced VTs were approached and any episode of VT/VF was considered a recurrence that represented the most relevant aspect.

Study Limitations

Both strategies, contact and noncontact mapping, are subject to specific limitations. Although contact mapping is mainly restricted in spatial resolution, the noncontact technique has intrinsic limitations with regard to the signal-to-noise ratio. The latter is important for small signals (eg, slow conduction within scar areas) and can complicate the definition of the diastolic pathway. A clear-cut definition of epicardial pathways cannot be made with either technique and remains subject to epicardial mapping. Termination of tachycardia was unavailable as proof of success because all ablation was performed during sinus or paced rhythm. No attempts were made to induce VT via left ventricular stimulation. Because of the low number of cases, interpretation of statistical test is limited. The long-term follow-up and a larger patient cohort will further elucidate the relevance of extended ablation at critical borders.

Conclusion

The combination of contact and noncontact mapping unifies advantages of both techniques. Critical borders can be defined from recordings of VTs and successfully ablated regardless of hemodynamic stability. They are identified as sources of multiple and, after incomplete ablation, probably newly arising VTs. Spontaneous transitions to different VT morphologies indicate scar areas with a continuity of exit sites and difficult ablation setting.

Disclosures

Drs Klemm, Rostock, Lutomsky, and Willems report having received lecture honoraria from St. Jude Medical, St. Paul, Minn. The other authors report no conflicts.
Ablation of ventricular tachycardia after remote myocardial infarction remains challenging because of the complexity of the underlying substrate. Multiple tachycardias of varying hemodynamic stability can usually be induced in a single patient. This study demonstrates the combined use of well-established bipolar contact maps acquired during sinus rhythm and noncontact activation maps. Although bipolar voltage thresholds for injured myocardium and dense scar have been suggested, coverage of an extended scar area by ablation is not practical. To guide the placement of ablation lesions, detection of the late isthmus conduction that precedes QRS onset was performed by noncontact mapping. It was demonstrated that tachycardia exit sites, defined as the origin of the rapid activation spread within healthy myocardium, are located at the border of the infarct area. Multiple tachycardias and spontaneous transitions between distinct morphologies were found to originate from closely spaced sites, which gave rise to the definition of a critical border. These were approached by a single linear ablation lesion that followed the outline of the bipolar low-voltage zone and covered all relevant exit sites. The ability to reproduce the tachycardia QRS by pace-mapping at the exit sites was shown to be associated with successful ablation. The definition of critical borders by combination of both mapping techniques may facilitate ablation of multiple and unstable arrhythmias. Effective ablation strategies are of growing importance in view of the increasing number of patients who suffer from recurrent shock deliveries by implanted cardioverter-defibrillators.
Catheter Ablation of Multiple Ventricular Tachycardias After Myocardial Infarction Guided by Combined Contact and Noncontact Mapping
Hanno U. Klemm, Rodolfo Ventura, Daniel Steven, Christin Johnsen, Thomas Rostock, Boris Lutomsky, Tim Risius, Thomas Meinertz and Stephan Willems

_Circulation_. 2007;115:2697-2704; originally published online May 14, 2007; doi: 10.1161/CIRCULATIONAHA.106.668673
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2007 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/115/21/2697

**Permissions:** Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Circulation* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

**Reprints:** Information about reprints can be found online at:
http://www.lww.com/reprints

**Subscriptions:** Information about subscribing to *Circulation* is online at:
http://circ.ahajournals.org/subscriptions/