Endocardial Activation Mapping of Ventricular Tachycardia in Patients
First Application of a 32-Site Bipolar Mapping Electrode Catheter

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Background—Localization of early activated endocardial areas during ventricular tachycardia (VT) is mandatory for performance of surgical or radiofrequency catheter interventions. The use of a multielectrode catheter may shorten the procedure time and increase the accuracy of the procedure compared with single-electrode mapping techniques. This study was performed to evaluate the safety and efficacy of a 32-bipolar-electrode mapping catheter in patients.

Methods and Results—The basket-shaped mapping catheter (BMC), integrated with a computerized mapping system, allowed on-line reconstruction of endocardial activation maps. Twenty patients with VT were studied before surgery (n=4) or radiofrequency catheter ablation (n=16). End-diastolic left ventricular (LV) volume was 280±120 mL, with an LV ejection fraction of 33±14%. The volume encompassed by the BMC was 164±27 mL (130 to 200 mL); the deployment time was 46±11 minutes. Endocardial activation time during sinus rhythm was 105±34 ms; 14±5 electrodes could be used to stimulate the heart. Cycle length of VT was 325±83 ms. Earliest endocardial activation was recorded 58±42 ms before the onset of the surface ECG. Complications were pericardial effusion (n=2) and transient cerebral disorientation (n=1).

Conclusions—Percutaneous multielectrode endocardial mapping in patients with VT is feasible and relatively safe. The use of this technique shortens the time patients have to endure VT. (Circulation. 1998;98:2168-2179.)

Key Words: reentry ■ tachycardia ■ mapping ■ ischemia ■ heart diseases

Reentry is the underlying mechanism of most ventricular arrhythmias in patients with ischemic heart disease. Surviving cell layers within the infarcted myocardium play a key role in the induction and perpetuation of reentry.1-5 These cell layers are often localized within the subendocardium of the left ventricle (LV) or intramurally within the interventricular septum.1,2,6,7 Drug therapy, still the first-choice treatment modality in most patients, is associated with high recurrence rates.8,9 If arrhythmias recur, surgical resection or radiofrequency catheter ablation may serve as alternative treatment modalities.10-13 Before treatment, endocardial activation mapping to localize the arrhythmogenic site13-17 is mandatory. Current mapping techniques are limited. Single-electrode mapping or the use of multiple catheters, during which the deployment time was 46±11 minutes. Endocardial activation time during sinus rhythm was 105±34 ms; 14±5 electrodes could be used to stimulate the heart. Cycle length of VT was 325±83 ms. Earliest endocardial activation was recorded 58±42 ms before the onset of the surface ECG. Complications were pericardial effusion (n=2) and transient cerebral disorientation (n=1).

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A multielectrode catheter inserted percutaneously may allow detailed and reproducible endocardial mapping of VT within a short period of time. This study was performed to evaluate the safety and efficacy of the use of a novel multielectrode mapping catheter in patients with VT.

Methods
Twenty patients were studied between December 1995 and November 1997. This study was approved by the institutional ethics committee. Informed consent was obtained from all patients. Before the mapping procedure, patients underwent neurological and cardiac examinations, including 24-hour Holter monitoring, echocardiography (either transthoracic or transesophageal) to rule out the presence of LV pedunculated thrombi, and coronary plus biplane LV angiography. Patients underwent an electrophysiological study before the procedure. During the mapping procedure, heparin (activated coagulation time of 2.5 to 3 times the baseline value) was given intravenously. Systemic blood pressure and heart rate were monitored continuously.

“Basket” Mapping Catheter
The 8.5F mapping catheter (Cardiac Pathways) consists of an open-lumen catheter shaft with a collapsible, basket-shaped, 32-bipolar-electrode array on the distal end (Figure 1A). The catheter is constructed of 8 equidistant metallic arms, each containing 4 electrode pairs with an interelectrode spacing of 1 to 1.5 cm. Three arms contain radiopaque markers for localization (Figure 1B). The catheter (50, 130, 170, or 200 mL) was inserted into the LV through an 11F guiding catheter, which was introduced percutaneously into the right femoral artery and positioned in the LV. The size of the
basket catheter was chosen on the basis of the LV end-diastolic volume and the length of the LV (calculated from the LV angiogram). The central lumen of the catheter was flushed (4 U heparin/mL saline) with an infusion pump (during insertion, 100 mL/h; after deployment, 50 mL/h). A transthoracic echocardiogram was made to study the deployment of the basket and to rule out aortic regurgitation caused by the catheter.

**Mapping System**

The mapping system (Cardiac Pathways) consists of an acquisition module connected to a SPARC 20 computer (Sun Computers). The system is capable of simultaneously processing (1) 32 bipolar electrograms from the basket catheter, (2) 16 bipolar/unipolar electrogram signals, (3) a 12-lead ECG, and (4) a pressure signal. Color-coded activation maps are reconstructed on-line. Signals are sampled at 3 kHz/channel with a resolution of 14 bits. Signals are stored on optical disk for off-line analysis. Activation marks are generated automatically with either a peak or slope algorithm. The peak algorithm places activation marks on the maximum amplitude; the slope algorithm places the activation marks on the maximum absolute slope (dV/dt). The activation times can be adjusted manually.

**Programmed Electrical Stimulation**

The hearts were stimulated with a pulse width of 2 ms by use of a constant-current stimulator (Medtronic). The output of the stimulator could be directed to any of the electrode pairs of the basket catheter or to any of the 16 bipolar electrode leads.

To test the contact between the electrodes and the endocardium, the heart was stimulated through each of the basket electrode pairs at an output of 2.0 mA, at a rate of 20% above the intrinsic sinus rate, and checked for ventricular capture. Next, the output was set at 5.0 mA, and the pacing protocol was repeated. Programmed electrical stimulation applying up to 3 extrastimuli at 2 times diastolic threshold was used to induce VT.

**Radiofrequency Ablation Procedure**

In 16 patients, a radiofrequency ablation procedure was performed immediately after removal of the basket catheter. A standard ablation catheter (Steerecath, EP Technology) was used. Detailed localization included the reconstruction of pace maps during stimulation from the tip of the ablation catheter, stimulation during tachycardia to reveal concealed entrainment, and the characterization of locally recorded electrograms. Radiofrequency current was delivered for 60 seconds and repeated until the target tachycardia was no longer inducible.

**After the Procedure**

The arterial sheath was removed 4 to 6 hours after the procedure. The patients then received heparin for at least 24 hours. Neurological examination, an echocardiogram, and a chest radiograph were obtained within 24 hours after the procedure.

**Results**

**Patient Characteristics**

Twenty patients (all male, 64±7 years old, Table 1) with drug-refractory VT were studied before surgical resection (n=4) or radiofrequency catheter ablation (n=16).

**Introduction and Deployment of the Mapping Catheter**

The mapping catheter was inserted successfully in all patients. No significant changes were observed in heart rate or blood pressure during the procedures. No sustained arrhythmias were elicited by the insertion of the catheters. Insertion and deployment of the catheters took 9 to 16 minutes (11±2.6 minutes). Although it was difficult to manipulate the basket within the LV, it was possible to rotate the catheters slightly and adjust the deployment in most patients. A satisfactory fit within the LV was obtained in each patient. However, in all patients, the arms of the mapping catheter were unevenly distributed over the endocardial surface. Transthoracic ultrasound examinations during the procedures did not reveal aortic regurgitation in any of the patients.

**Endocardial Electrical Activity**

The noise level was <50 to 75 μV. An example of the electrical activation recorded during sinus rhythm (SR) (A) and during VT (B) is given in Figure 2. Except for 2 leads showing no electrical activity (E2, E6), the signals were of acceptable quality with a noise level of <75 μV. Although movement of the electrodes may occur during contraction of the heart, movement artifacts did not hamper analysis of the recordings. The signal-to-noise ratio was acceptable even in case of low-amplitude fragmented activation.
Recording During SR
During SR, electrical activation could be recorded from 31 ± 1 of the 32 available electrode pairs (Figure 3, Table 2).

Stimulation
Stimulation via the basket electrodes was performed in all patients but one. Patient 16 developed VT during pacing from the right ventricular apex. After cardioversion, the catheter had moved back into the aorta and had to be removed. Fourteen (65) electrode pairs (43%) could be used to stimulate the heart, with an output of 5 mA (Table 2, Figure 4). Stimulation was not always possible when the electrode pairs were localized within infarcted regions or when the electrodes were located near the base of the heart.

Induction of VT
The initiation of VT is shown in Figure 5. In the lower panel, 3 ECG tracings and 4 intracardiac electrograms are shown. VT (cycle length [CL], 410 ms) was initiated after the second premature stimulus (S3). During pacing (S1; CL, 600 ms) from the right ventricular apex, a zone of late activation can be detected around electrode rows D and E (posterobasal part of the LV). The corresponding intracardiac tracings from this region (lower panel) show progressive delay of activation of this part of the ventricle during the consecutive premature beats (S2, S3) and become increasingly fragmented. Ultimately, tachycardia was initiated by an impulse propagating slowly through this area (electrode D1-2). The same pattern of induction of tachycardia could be reconstructed in 12 of 20 patients. Because of the limited resolution of the electrode, an exact reconstruction of the reentrant pathway was not possible. In the remaining patients, it was not possible to reconstruct the initiation of tachycardia either because of the limited resolution of the catheter or because the tachycardia originated from the right side of the intraventricular septum.

Mapping of VT
VT (CL, 325 ± 83 ms) was induced in all patients (Table 2, Figure 6). Earliest endocardial activation was recorded 58 ± 42 ms (0 to 176 ms) before the onset of the surface ECG. In 17 of 20 patients, VT was terminated by burst pacing within 60 seconds after induction. Cardioversion was necessary in 3 patients because of hemodynamic deterioration. In patients studied before surgery, a reasonable correlation was found between the earliest endocardial activated area during percutaneous mapping, and the area was removed during surgery. In 2 patients, the findings were confirmed by endocardial mapping during surgery (using a multielectrode

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MI indicates myocardial infarction; VF, ventricular fibrillation; RF, radiofrequency ablation; and ICD, implantable cardioverter and defibrillator.
Figure 2. Endocardial electrograms recorded during SR (A) and VT (B). During SR, small Purkinje deflections preceding endocardial activation were detected in tracings A3, B2, and B3.

Figure 3. ECG and endocardial activation map during SR. Polar view is reconstructed for 1 heartbeat. Outer circle is located near base of LV. Inner circle is positioned within LV apex. Map is reconstructed from data obtained within yellow time window. Missing recordings (X) are interpolated from neighboring electrodes. Electrode pairs are marked by corresponding numbers. Radiopaque markers (dashes) on rows A, B, and C are marked. The 0-ms reference line is displayed in surface ECG. Activation times are indicated by color code displayed at top. Earliest endocardial activation started at electrode A2 (near septum), with spread of endocardial activation radially over LV endocardium. Area around electrode D2–3 is activated late during cardiac cycle.
During VT1 (CL, 215 ms; panel B), activation started 42 ms before the onset of the surface ECG, with early activation recorded from electrodes C1-2, B1-2, and A1-2. During VT2 (CL, 252 ms; panel C), activation started 67 ms before the onset of the surface ECG, with early activation recorded from electrodes A7-8 and B7-8. In the lower panels (D, E, and F), the electrograms recorded from electrodes A1-2 to B7-8 are displayed. During SR, fragmented activation was recorded in all electrodes. Purkinje deflections were recorded in B5-6 and B7-8. During VT1, the areas around electrodes A1-2 and B1-2 were activated relatively early in the cycle, with a gradual spread of activation to electrodes A7-8 and B7-8. During VT2, the areas around electrodes A7-8 and B7-8 were now activated relatively early, whereas the areas around A1-2 and B1-2 were activated relatively late.

The activation map recorded during tachycardia before ablation is shown in Figure 9A. Early activation is recorded around electrode H5-6. Mididiastolic potentials preceding myocardial activation can be detected 170 ms before onset of the surface ECG (panel B). During the ablation procedure (panel C), mididiastolic potentials preceding myocardial activation can be detected. During ablation at this site, tachycardia stopped within 2 seconds and was no longer inducible.

### Adverse Events

The catheters were deployed for 46±11 minutes (20 to 65 minutes). No thrombus formation could be detected on any of the catheters. No embolic complications were observed. Ultrasound studies did not reveal mitral valve or aortic valve damage in any patient. In 2 patients, pericardial effusion developed. In patient 1, 4 hours after the procedure, 500 mL of fluid was removed by pericardiocentesis. At surgery for endocardial resection (10 days later), no perforation site could be identified. In pericardiocentesis. At surgery for endocardial resection (10 days later), no perforation site could be identified. In the other 2 patients, VT was not inducible during surgery. In these 2 patients, the location of the endocardial resection was based on the findings obtained during the percutaneous mapping procedure (although a larger part of the endocardium was removed). Radiofrequency catheter ablation was successful in 14 of 16 patients (88%). In 3 of 16 patients (19%), the site of successful RF application was located on the right side of the intraventricular septum. In 11 of 13 patients (85%), the localization of the arrhythmia initiation by catheter mapping was consistent with the results obtained by the pericardial effusion. In 2 patients, pericardial effusion developed. In patient 1, 4 hours after the procedure, 500 mL of fluid was removed by pericardiocentesis. At surgery for endocardial resection (10 days later), no perforation site could be identified. In

### Table 2. Procedure Data

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**LVEDV** indicates left ventricular end-diastolic volume; **RBBB**, right bundle-branch block; and **LBBB**, left bundle-branch block.
Figure 4. Four activation maps (patient 2). A, Activation map obtained during pacing via electrode A1 is shown; 2 missing electrodes are marked by X. Activation times are indicated according to color code (displayed at top). Endocardial activation started at electrode A1 (near septum), with spread of endocardial activation radially over LV. Area around electrodes C1–3 was activated late (±70 ms). This part was located within infarcted area. B, C and D, Stimulation electrodes E4, H4, and C3 were stimulated. Although activation patterns were similar during pacing from different sites, total activation times differed markedly (70 to 120 ms). Besides localization with regard to infarcted area, differences in activation times were probably caused by geometric factors or by different contribution of conducting system to spread of activation.
Figure 5. Recordings obtained during initiation of VT.
Figure 6. Example of VT (patient 1). A, Twelve-lead ECGs recorded during SR and VT (CL, 275 ms), respectively. Activation was recorded from 29 of 32 electrodes. Detection algorithm needed adjustment in ~25% of recordings, especially at sites with low-amplitude fragmented activation. C, Activation map is shown as polar view. Earliest endocardial activation is localized near electrodes B1 and B2, with activation spreading radially over LV (total activation, ~70 ms). B, Electrograms recorded from B1 and B2 clearly show 2 deflections during each tachycardia cycle.
patient 14, 1 week after stent implantation, when the patient was still receiving aspirin and ticlopidine, pericardial effusion developed during the procedure. At emergency surgery, a small perforation was detected in the lateral wall of the LV; the patient’s recovery was uneventful. In patient 7, cerebral disorientation was observed the first 12 hours after the procedure; the patient’s recovery was uneventful. No vascular complications were observed.

Discussion
This study reports the safety and efficacy of percutaneous endocardial mapping with a multielectrode basket-shaped catheter in patients with VT.

Mapping of VT
Reentry is an important mechanism of VT in patients with ischemic heart disease.1,2,6 Because of limitations of single-catheter mapping techniques, it has been difficult to elicit the mechanisms of tachyarrhythmias in patients during percutaneous mapping.14 The mapping catheter used in this study may be useful to unravel some of the mechanisms of VT in patients. Local electrophysiological characteristics contributing to induction or perpetuation of reentry may now be studied in detail.

We demonstrated areas of fragmented early endocardial activation in almost all patients. These areas play an important role in the induction and perpetuation of reentry, as demonstrated either by concealed entrainment during pacing4,13 or by the fact that the tachycardias were terminated during radiofrequency application at those sites. Although because of the limited resolution of the mapping catheter used in this study, a high-resolution reconstruction of reentrant circuits was not possible, in the majority of patients reentry seems to be the underlying mechanism of the VT. The fast

Figure 7. Fast VT. A, Activation map during SR. B, Activation map recorded during VT1 (CL, 215 ms). C, Activation map recorded during VT2 (CL, 252 ms). Bottom, electrograms recorded from region of interest (A1-2 to B7-8).
Figure 8. Thirty-two bipolar electrograms recorded during VT2 (Figure 7). Left, Activation map and 12-lead ECG. Bottom, Corresponding intracardiac electrograms. Gray zone corresponds to blue zone displayed in surface ECG. Line marking onset of surface ECG is drawn (black arrows). Fragmentation of electrograms often hampered marking of local activation. ● indicates activation mark; dashed lines, bad signal marking.
reconstruction of endocardial activation maps resulted in a reduction of the time patients must endure tachycardia compared with single-point mapping techniques or the insertion of multiple electrodes into the LV. In this study, the duration of tachycardia totalled maximally 60 seconds.

The spatial resolution of the basket catheter is still limited (≈1 cm along the arms of the catheter and ≥1 cm between the arms). This resolution will be sufficient to locate the earliest activated endocardial site before surgical resection, because a larger part of the endocardium will be resected during this procedure. In case of a catheter ablation procedure, in which only a small area is treated, the spatial resolution is not always sufficient. The outcome of this study is in accordance with this perception. The results of the mapping procedure guided the operator toward the earliest activated endocardial regions, but detailed localization, using pace mapping and entrainment studies, was necessary to localize precisely the regions of interest. Asymmetric mapping devices with a locally enhanced spatial density of electrodes may be required to study these regions of interest precisely.

**Mapping System**

The mapping system allows simultaneous recording of electrical activation from multiple sites and fast reconstruction of endo-
cardial activation maps. The algorithms used to construct activation maps did result in an accurate detection of 75% of the activation times. Manual adjustment was necessary when low-amplitude fragmented signals or noisy signals were recorded.

At this time, only limited 2-dimensional graphic features are available. To enhance the graphic representation, 3-dimensional (3D) reconstruction of activation would be advantageous.

Mapping Techniques

The main objective of endocardial mapping is to reveal sites activated early (with respect to the surface ECG) during tachycardia, to reveal middiastolic potentials, and to identify sites demonstrating concealed entrainment. These sites are targets for either surgical or catheter interventions. Current methods for mapping include single-point mapping, the insertion of multiple catheters, and the use of endocardial multielectrode balloons during surgery. Single-point mapping, although a reliable technique, is inherently less accurate, time-consuming, and not suitable for the mapping of fast tachycardia. The insertion of multiple catheters yields an increased spatial resolution but entails a lengthy procedure, and activation maps are difficult to reconstruct. The use of multielectrode balloons is restricted to surgery. A recently developed technique combines a magnetic 3D localization system with a single roving electrode that allows the reconstruction of high-density (<1 cm) endocardial activation maps. The combination of 3D geometry with electrical activation may guide the operator toward a target site for RF ablation. However, this is also a long procedure.

Limitations of the Technique

Although almost all patients suffered from large myocardial infarctions and most ventricles were seriously deformed, all catheters were deployed successfully. However, the limited torque capabilities of the mapping catheter hampered the correct placement of the catheter. These problems may be improved by adding steerability to the catheter shaft, but the manipulation of the catheter will continue to require extensive training. Furthermore, because of deformation of the ventricles, not all electrodes were in close contact with the endocardial surface (only 44% of the electrodes could be used to stimulate the heart). Electrodes located near the postero-basal part of the LV were often not suitable for pacing; however, local electrical activity could be recorded from most of these sites. These limitations may be resolved by designing specially shaped catheters for different LV shapes. Another limitation of LV endocardial mapping may be that reentrant circuits located either on the right side of the intraventricular septum or intramurally cannot be reconstructed in detail.

Limitations of This Study

By virtue of its capability to reduce the procedure time and duration of tachycardia, the basket catheter allows mapping of hemodynamically unstable tachycardia. However, this study was (with a few exceptions) limited to patients who tolerated tachycardia for a prolonged period. It is another limitation that the protocol of this safety and feasibility study did not allow simultaneous insertion of a radiofrequency ablation catheter, which hampered the correlation between the activation maps and the site of successful ablation. Furthermore, entrainment of tachycardia using the mapping catheter electrodes to stimulate the heart was not studied systematically.

Conclusions

Percutaneous endocardial mapping with the basket catheter is feasible and relatively safe. The technique facilitates endocardial mapping during tachycardia and considerably shortens the time patients have to endure VT. Potentially, the simultaneous deployment of a basket catheter and an ablation catheter will allow the treatment of fast and hemodynamically unstable VT.

References

Endocardial Activation Mapping of Ventricular Tachycardia in Patients: First Application of a 32-Site Bipolar Mapping Electrode Catheter
Martin J. Schalij, F. Paul van Rugge, Machiel Siezenga and Enno T. van der Velde

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