Noncontact Mapping to Guide Ablation of Right Ventricular Outflow Tract Tachycardia

Paul A. Friedman, MD, FACC,* Samuel J. Asirvatham, MD,* Suellen Grice, RN,* Michael Glikson, MD,† Thomas M. Munger, MD, FACC,* Robert F. Rea, MD, FACC,* Win K. Shen, MD, FACC,* Arshad Jahanghir, MD,* Douglas L. Packer, MD, FACC,* Stephen C. Hammill, MD, FACC*

Rochester, Minnesota and Tel Hashomer, Israel

OBJECTIVES
The aim of this study was to determine whether noncontact mapping is feasible in the right ventricle and assess its utility in guiding ablation of difficult-to-treat right ventricular outflow tract (RVOT) ventricular tachycardia (VT).

BACKGROUND
In patients without inducible arrhythmia, RVOT VT may be difficult to ablate. Noncontact mapping permits ablation guided by a single tachycardia complex, which may facilitate ablation of difficult cases. However, the mapping system may be geometry-dependent, and it has not been validated in the unique geometry of the RVOT.

METHODS
Ten patients with left bundle inferior axis VT, no history of myocardial infarction and normal left ventricular function underwent noncontact guided ablation; seven had failed previous ablation and three had received a defibrillator. All noncontact maps were analyzed by a blinded reviewer to determine whether the arrhythmia focus was epicardial and to predict on the basis of the map whether arrhythmia would recur.

RESULTS
The procedure was acutely successful in 9 of 10 patients. During a mean follow-up of 11 months, 7 of 9 patients remained arrhythmia-free. Both patients in whom the blinded reviewer predicted failure had arrhythmia recurrence: one due to epicardial origin with multiple endocardial exit sites and one due to discordance between site of lesion placement and earliest activation on noncontact map.

CONCLUSIONS
Mechanisms of ablation failure in RVOT VT include absence of sustained arrhythmia, difficulty with substrate localization and epicardial origin of arrhythmia. In this study, noncontact mapping was safely and effectively used to guide ablation of patients with difficult-to-treat RVOT VT. (J Am Coll Cardiol 2002;39:1808–12) © 2002 by the American College of Cardiology Foundation

In patients with left bundle inferior axis ventricular tachycardia (VT) and a structurally normal heart, arrhythmia usually arises from abnormal triggered activity in the right ventricular outflow tract (RVOT). Radiofrequency (RF) catheter ablation can eliminate tachycardia in 83% to 100% of patients (1). In some patients with clinically problematic arrhythmia and failed ablation, absence of VT or clinical ectopy despite adrenergic and electrical stimulation in the electrophysiology laboratory limits localization of the arrhythmogenic substrate. Recently, a noncontact mapping system capable of recording endocardial potentials from >3,000 sites simultaneously to create a three-dimensional (3-D) map of electrical activation has been introduced into clinical practice (2–5). This system permits generation of a potential map from a single premature ventricular complex and may help guide difficult ablation procedures. Noncontact mapping has been validated in the right atrium and left ventricle and approved for use in the U.S. in the right atrium alone. Whether it can be used in the unique geometry of the right ventricle (RV) has not been established. We sought to determine whether this new mapping system could be employed in the RV to guide ablation in patients with difficult-to-ablate RVOT VT. Additionally, due to the high-density data acquisition afforded by noncontact mapping, we sought to characterize the mechanism of procedural failure in those patients in whom ablation was unsuccessful.

METHODS
Patient characteristics. Between July 1999 and October 2000, 10 patients with left bundle inferior axis VT, no history of myocardial infarction and a normal left ventricular ejection fraction were referred to our laboratory for catheter ablation and underwent noncontact mapping. Patients had a history of VT for 5.4 ± 5.5 years and had failed 3 ± 1 antiarrhythmic medications. In all patients, echocardiography demonstrated normal left ventricular function. Seven patients had previously undergone unsuccessful ablation elsewhere. Pace mapping was used to guide previous ablation in four patients and not used in one; information regarding pace mapping was not available in two patients. Three patients with fast or syncopal VT and unsuccessful previous ablation had received an implantable defibrillator before referral; one of these patients had magnetic resonance imaging abnormalities suggestive of arrhythmogenic RV dysplasia (Table 1). In one patient without an implantable cardioverter defibrillator (ICD) at the time of noncontact ablation, nondiagnostic RV abnormalities were noted on computed tomography scan.

From the *Division of Cardiovascular Disease, Mayo Clinic, Rochester, Minnesota; and the †Sheba Medical Center, Tel Hashomer, Israel.

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Electrophysiology and mapping study. Programmed stimulation was performed with up to three extrastimuli from two RV sites. The following pharmacologic stimulation was used in an effort to induce VT: isoproterenol (all patients), epinephrine (four patients), aminophylline (two patients), adenosine (one patient), atropine (one patient) and phenylephrine (one patient).

The details of the Ensite 3000 mapping system (Endocardial Solutions, Saint Paul, Minnesota) have been previously described (2). The system utilizes a noncontact mapping catheter that contains 64 filaments with 0.025-in insulation breaks at specified sites to form a multielectrode array. This array was advanced through the vasculature in low profile to the RVOT over a guide wire. A standard deflectable 4- or 5-mm tip ablation catheter was then placed in the pulmonary artery and slowly withdrawn until sharp bipolar electrograms were recorded on the distal electrodes, signifying the pulmonary valve-RVOT junction. Using this position as a fluoroscopic landmark, the noncontact array was then deployed at least 1 cm below the valve (Fig. 1).

With the multiple electrode array (MEA) in position, a geometry of the RV was created. The location of the mapping catheter relative to the MEA was determined by the system using a low-current “locator” signal. Location positions were automatically acquired 10 times per second. With each passage of the mapping catheter to a new, more distant location from the MEA, the position was taken as the endocardial boundary. Sweeping a mapping catheter along the RV endocardium resulted in 3-D chamber geometry. Care was taken to create a high-density RVOT geometry and to “tag” at least two points at the junction of the RVOT and pulmonary valve, as well as points at the RV apex and His bundle positions.

Upon geometry completion, global RV potential voltage maps from a single premature ventricular complex could be created. These were displayed on the computer workstation as a 3-D endocardial potential map that changed in time as the computer-generated “movie” of cardiac activation advanced. In all patients, 12-lead electrocardiograms (ECG) of any RVOT ectopy, nonsustained VT or sustained VT were reviewed to ensure a morphology identical to the clinical arrhythmia. When the morphology of the recorded complexes matched the clinical arrhythmia, isopotential maps were generated to identify the earliest site of activation. Virtual electrograms were reconstructed at sites of

### Abbreviations and Acronyms

- **3-D**: three-dimensional
- **ECG**: electrocardiogram
- **ICD**: implantable cardioverter defibrillator
- **MEA**: multiple electrode array
- **RF**: radiofrequency
- **RV**: right ventricle/ventricular
- **RVOT**: right ventricular outflow tract
- **VT**: ventricular tachycardia

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<th>Follow-Up Months</th>
<th>Follow-Up Success</th>
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**Abbreviations**

- **BB**: beta blocker
- **CaB**: calcium channel blocker
- **ICD**: implantable cardioverter defibrillator
- **NK**: not known
- **VT**: ventricular tachycardia

**Drugs**

- **BB**: beta blocker
- **CaB**: calcium channel blocker
- **ICD**: implantable cardioverter defibrillator
- **NK**: not known
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earliest activation on the isopotential maps to look for a unipolar QS pattern (Fig. 2). Once earliest activation was identified, the site was labeled on the 3-D map and the locator signal used to navigate the ablation catheter to it in real time during normal rhythm when sustained tachycardia was not inducible.

A blinded reviewer (S. J. A.) who did not participate in any of the electrophysiologic procedures analyzed all non-contact maps to determine whether the arrhythmia focus was epicardial and to predict whether arrhythmia would recur. A focus was defined as epicardial if virtual electrograms at the earliest activation site had an R-wave (as opposed to a QS pattern) and the color map at this site had a broad (as opposed to discrete, focal) appearance. Comparison of the arrhythmia potential map with the labeled ablation sites (tagged on the geometry) was used to predict success.

All noncontact studies were performed with a specially trained nurse operating the noncontact computer system under the direction of the attending electrophysiologist and in the absence of representatives from the manufacturer of the mapping system.

Follow-up. All patients were seen in the clinic or contacted by telephone to determine clinical status. Patients with symptoms suggestive of arrhythmia recurrence were further assessed by ECG, event recorder or Holter monitoring. Patients with implantable defibrillators underwent device interrogation.

RESULTS

The noncontact mapping system was successfully deployed in the RVOT in all 10 consecutive patients. Deployment of the MEA catheter in the RVOT required 14 ± 9 min
(range 6 to 30 min), and geometry creation took 23 ± 11 min (range 11 to 41 min). In 5 of 10 patients, only isolated ectopy or transient arrhythmia was present at the time of noncontact study. No patient had electrocardiographic characteristics suggestive of left ventricular or sinus of Valsalva arrhythmia origin (6–8). Isopotential movies demonstrated point-source ectopy in all patients; re-entry was not seen as the cause of arrhythmia in any of the patients. Noncontact guided ablation was acutely successful in 9 of 10 patients. The one patient with acute procedural failure had no further ectopy after deployment of the MEA so that no RF energy was delivered. Earliest activation sites were anteroseptal (n = 5), posteroseptal (n = 4) and midseptal (n = 1) RVOT. One patient had two different early sites (anteroseptal and posteroseptal), and both were ablated. In one patient with frequent ectopy, the noncontact system failed to identify the ultimately successful ablation site. In that patient, activation time mapping of the ectopy, assisted by the noncontact locator system, resulted in successful ablation. The average number of RF energy deliveries until success was achieved (no further ectopy or VT) was 3.7 ± 2.6. There were no complications.

During a mean follow-up of 11 ± 6 months, 7 of the 9 ablated patients (77.5%, 95% confidence intervals: 46.5% to 100%) remained free of arrhythmia recurrence, 6 in the absence of antiarrhythmic drugs. One patient with an implantable defibrillator was treated with propafenone; device interrogation and electrogram analysis after an ICD shock revealed a supraventricular arrhythmia. Of the four patients with no inducible arrhythmia at noncontact study, 3 had no further ectopy during long-term follow-up (Table 1). The blinded reviewer predicted procedural failure in two patients; both had recurrent arrhythmia. In one patient (Patient 4 in Table 1), ablation lesions were placed at a site removed from the earliest activation on the noncontact map as determined by review. In the second patient (Patient 8), the earliest activation was from an epicardial focus, which then propagated along two discrete pathways with subsequent breakout at two endocardial sites. Ablation had been directed at both endocardial sites.

**DISCUSSION**

**Main findings.** We found noncontact mapping to be a useful adjunct to guiding ablation in a consecutive series of patients with RVOT VT. This patient population reflected a particularly challenging ablation group: 70% of patients had failed previous ablation, 30% of patients had received ICD therapy before referral and 50% had no sustained VT to guide mapping. Despite this, noncontact-guided ablation was acutely successful in 90% of this population, with no recurrence in 78% of acutely successful patients during a mean follow-up of 11 months.

**Mechanism of failure in RVOT VT ablation.** Although catheter ablation has proven highly effective for most patients with RVOT VT, there are several mechanisms that may account for procedure failure. In the absence of inducible arrhythmia or frequent ectopy, pace mapping has been used to guide ablation. Wen et al. (9) found that use of pace mapping alone for ablation site selection was a predictor of tachycardia recurrence in patients with idiopathic RVOT VT. Pace mapping may be limited by anodal capture, by capture of a large region of myocardium or by similar appearing pace maps obtainable with stimulation from a relatively large region of myocardium. The resolution of pace mapping is approximately 5 mm to 10 mm, even with the use of strict criteria (10). This may be a limiting factor because typical RF lesions are 5 mm in diameter (11). Additionally, pacing rate differences as small as 80 ms from the coupling interval of spontaneous ectopy or from the VT cycle length result in rate-dependent changes in QRS morphology that can be confounded with site-dependent morphology changes (10). The difficulty in ablating RVOT VT in the absence of sustained arrhythmia is supported by the present study. In five of the seven patients with previous failed ablation, failure was attributed to a transient or noninducible arrhythmia. Our long-term success in seven of nine patients based on mapping spontaneous arrhythmia activity (as opposed to pace mapping) supports the usefulness of defining the site of spontaneous activity.

**Use of noncontact mapping in the RVOT.** Use of non-contact mapping in the RVOT has not been previously reported. Theoretical limitations include mechanical obstruction of the pulmonic valve and mapping limitations due to the odd-shaped RV chamber geometry. We were able to deploy the MEA and create geometries in all patients without complication. However, one patient had no further ectopy after MEA deployment, possibly due to inadvertent “bumping” of the focus during deployment. Additionally, in another patient, the noncontact map failed to identify the
appropriate target. This may have stemmed from inadvertent MEA contact with the RVOT and contact ectopy or from difficulty interpreting the noncontact map using an early version of the system software. Despite these limitations, the system was successfully employed in 80% of patients.

**Study limitations.** Pace mapping has been used to guide ablation in the absence of inducible arrhythmia. We did not randomize patients to pace mapping versus noncontact mapping to determine which technique is superior. However, pace mapping was used to guide the initial ablation in at least four of the seven patients with previous failed ablation. Pace mapping was performed in 7 of 10 patients after noncontact mapping, at the site predicted by the noncontact map. It demonstrated a 12 of 12 match in all but 2 patients (both with 11 of 12 match), both of whom had late arrhythmia recurrence. Because the noncontact map locator signal may facilitate pace mapping (by cataloging points at which maps are performed and redirecting the catheter to promising sites), these techniques may be complementary. The small size of our study limits the strength of our conclusions.

**Conclusions.** In conclusion, mechanisms of ablation failure in RVOT VT include absence of sustained arrhythmia, difficulty with substrate localization and epicardial origin of arrhythmia. In this study, noncontact mapping was safely and effectively used to guide ablation of patients with difficult-to-treat RVOT VT.

**Reprint requests and correspondence:** Dr. Paul A. Friedman, Division of Cardiovascular Diseases, Mayo Clinic, 200 First Street Southwest, Rochester, Minnesota 55905. E-mail: pfriedman@mayo.edu.

**REFERENCES**