Noncontact Mapping and Radiofrequency Catheter Ablation of Fast and Hemodynamically Unstable Ventricular Tachycardia After Surgical Repair of Tetralogy of Fallot

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Objectives
The goal of this work was to assess efficacy of radiofrequency (RF) ablation of fast ventricular tachycardia (VT) in patients after surgical repair of tetralogy of Fallot (TOF) guided by noncontact mapping.

Background
Ventricular tachycardias after repair of TOF are associated with significant morbidity and mortality.

Methods
Ten patients after surgical repair of TOF underwent electrophysiological study for hemodynamically unstable VT using the noncontact mapping system. Dynamic substrate mapping was performed and activation was recorded during basic rhythm and induced VT (mean cycle length 269 ms) using color-coded isopotential maps and reconstructed unipolar electrograms.

Results
A total of 13 VTs were induced in the 10 patients. In 11 of 13 VTs, a macro–re-entrant mechanism was identified; 2 had a focal origin. For macro–re-entrant VT, RF current lesion lines were created between areas of residual conduction; in 2 patients, no RF current was delivered due to high risk of atrioventricular block. Focal applications were performed for the focal VTs. Ventricular tachycardia was not inducible after RF application in the 8 patients in whom ablation was attempted (100%, 80% of all patients). An internal cardioverter-defibrillator had already been implanted in 2 patients and was recommended to the rest of the group. During follow-up (mean 35.4 months), 6 of 8 patients with a successful procedure were still free of VT, and 2 patients had recurrence of VT with a different cycle length.

Conclusions
In patients with fast and unstable VT after surgical repair of TOF, noncontact mapping helped to identify the tachycardia substrate and allowed for effective and safe treatment by RF ablation.

Complete intracardiac repair of tetralogy of Fallot (TOF) has now been available for more than 40 years with excellent short- and long-term results (1,2). During long-term follow-up, tachyarrhythmias like atrial flutter and ventricular tachycardia (VT) have been noted in these patients resulting in significant morbidity and mortality (2,3).

Electrophysiological mechanism responsible for VT after surgical correction of TOF is typically a re-entrant circuit within the right ventricle (RV) around suspected scar tissue or prosthetic materials used during surgical repair. Due to high heart rates and abnormal RV function, VT is often accompanied by compromised hemodynamic tolerance.

Thus, conventional mapping techniques that require sequential electrogram acquisition and entrainment mapping can only be applied in selected patients, generating either case reports or very small patient series (4–9).

Electroanatomical mapping has been used to generate sinus rhythm voltage maps and identify areas of low voltage and conduction barriers critical to initiation and perpetuation of VT (10,11). Even using this sophisticated technique, however, propagation path can often not be assessed in some patients with fast VT circuits. This limitation may, at least in part, be overcome with the use of the noncontact mapping system that allows simultaneous acquisition of electrical activation during a single heartbeat. To that end, the present study describes endocardial mapping and radiofrequency (RF) ablation using the noncontact mapping system in a series of patients with fast and hemodynamically unstable VT after surgical repair of TOF.
Methods

Patients. Ten consecutive patients underwent endocardial mapping of recurrent episodes of VT after surgical repair of TOF with the noncontact mapping system (EnSite 3000, St. Jude Medical Inc., St. Paul, Minnesota) at both institutions. Demographic data of the patients are listed in Table 1. Age of the patients ranged from 14 to 42 (mean 29.1) years, and the mean body weight was 66.1 (range 40.8 to 104) kg. In 2 patients, an implantable cardioverter-defibrillator (ICD) had already been implanted before the procedure (Patients #6 and #10). Indications for electrophysiological study included nonsustained VT on Holter monitor in 4 patients, spontaneous sustained VT in 4 patients requiring external cardioversion, and frequent ICD discharges for monomorphic VT in 2 patients. In both facilities, noncontact and electroanatomical systems were available. A conscious decision was made for every patient to use the noncontact system due to the pre-procedural findings of fast VT.

The study protocol was approved by the human investigation committees of both institutions.

Electrophysiological study. An arterial line was placed in every patient for continuous blood pressure control. A quadripolar electrode catheter was placed at the His bundle for an anatomic landmark. A biplane angiogram of the RV was performed to delineate endocardial anatomy in right posterior oblique 30° and left anterior oblique 60° projections.

Noncontact mapping. The 9-F multielectrode balloon array of the noncontact mapping system (EnSite Array, St. Jude Medical Inc.) was introduced in the RV outflow tract over a stiff 0.035-inch guidewire with the tip of the guidewire placed in the peripheral pulmonary artery (Fig. 1). During the whole procedure, the activated clotting time was maintained >300 s. Right ventricular anatomy was reconstructed as previously reported (12,13), using a steerable 7-F mapping and ablation catheter (Marinr, Medtronic, Minneapolis, Minnesota). Areas with diminished or absent electrical activation or fractionated potentials at the roving catheter were noted on the created anatomy.

Subsequently, a dynamic substrate map was established during sinus rhythm using a high-pass filter of 2 Hz (14). Substrate was defined as an area of consistently low peak negative voltage exhibiting <35% of the largest unipolar deflection recorded on the RV endocardium during the duration of the surface QRS complex (Fig. 2). These areas of low voltage were considered to be areas of potential conduction block and substrates for the development and perpetuation of RV macro-re-entrant VT. The isopotential color-coded maps were studied during several cardiac cycles of the sinus rhythm to identify the propagation of ventricular activation along the labelled anatomical landmarks and suspected low-voltage areas (Fig. 3). Findings were validated by placing the reconstructed virtual electrograms into these areas looking for fractionated or low amplitude signals.

Ventricular tachycardia induction (Fig. 4) was performed at least twice in each of the individual patients in order to determine one of the end points after ablation (see the following text). After recording by the noncontact mapping system, VT was terminated by overdrive pacing or DC cardioversion due to unstable hemodynamics. Patients' hemodynamics were assessed by continuous blood pressure control via the arterial line and were defined as unstable when systolic blood pressure was <40 mm Hg.

Spread of activation within the RV was studied by analyzing color-coded isopotential maps (Fig. 5) using filter settings for high pass at 0.5 to 2 Hz and for low pass at inverse chebychev 150 Hz. In macro-re-entrant tachycardias, activation could be traced within the RV throughout the whole tachycardia cycle. For focal VT, eccentric propagation from one area in the RV was observed.

Table 1  Electrophysiological Characteristics of the 10 Patients

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yrs)</th>
<th>Body Weight (kg)</th>
<th>VT Cycle Length (ms)</th>
<th>Area of RF</th>
<th>Successful Ablation</th>
<th>ICD Implantation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>41</td>
<td>104</td>
<td>280</td>
<td>VSD patch ↔ AFW</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>18</td>
<td>77</td>
<td>210 ↔ 220</td>
<td>AFW</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>3</td>
<td>17</td>
<td>41</td>
<td>245</td>
<td>AFW</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>4</td>
<td>36</td>
<td>63</td>
<td>320</td>
<td>TVA ↔ RVOT</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>32</td>
<td>56</td>
<td>300</td>
<td>TVA ↔ RVOT</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>6</td>
<td>38</td>
<td>62</td>
<td>290</td>
<td>Anterior RVOT</td>
<td>+++</td>
<td>+ (before the procedure)</td>
</tr>
<tr>
<td>7</td>
<td>14</td>
<td>55</td>
<td>240</td>
<td>VSD patch ↔ TVA</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>8</td>
<td>42</td>
<td>67</td>
<td>220</td>
<td>Anterior RVOT</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>9</td>
<td>16</td>
<td>78</td>
<td>300</td>
<td>VSD patch ↔ TVA (close to His bundle)</td>
<td>No</td>
<td>–</td>
</tr>
<tr>
<td>10</td>
<td>37</td>
<td>58</td>
<td>250</td>
<td>VSD patch ↔ TVA (close to His bundle)</td>
<td>No</td>
<td>+ (before the procedure)</td>
</tr>
</tbody>
</table>

AFW = anterior free wall; ICD = implantable cardioverter-defibrillator; RF = radiofrequency; RVOT = right ventricular outflow tract; TVA = tricuspid valve annulus; VSD = ventricular septal defect; VT = ventricular tachycardia.
In macro-re-entrant VT, propagation was traced on the color-coded isopotential map to identify a protected zone of the re-entrant circuit between surgical and/or anatomical barriers. In focal or micro-re-entrant VT, localization of the earliest endocardial activation was assessed on the color-coded isopotential map and by the presence of a QS configuration of the local virtual electrograms at that location.

**RF current application.** In macro-re-entrant VT, RF was applied during basic rhythm between natural or surgical barriers at a target temperature of 65°C. Energy was delivered in a point-by-point start-stop fashion using a 4-mm tip ablation catheter (Marinr, Medtronic) for at least 30 s in each location. In focal VT, the area of earliest endocardial activation was targeted for ablation. No irrigated tip catheters were used for any procedure.

Ablation success for any particular VT was defined by lack of re-inducibility. In macro-re-entrant VT, completeness of linear RF lesion lines was also validated during spontaneous rhythm and during pacing from adjacent sites along the induced line by the presence of double potentials and analysis of the color-coded isopotential maps (Fig. 6) (15).

### Results

Thirteen different monomorphic VTs (mean cycle length 269 ms, range 210 to 320 ms) (Table 1) could reproducibly be induced. One patient had 2 different forms of macro-re-entrant VT (Patient #2). Patient #6 had initially successful ablation of a focal/micro-re-entrant VT. During follow-up, a VT occurred with a different cycle length and morphology. A second study was performed, during which 2 different macro-re-entrant VTs were induced and finally treated (Table 1).

Eleven of 13 VTs were considered to be macro-re-entrant, and 2 were considered focal or micro-re-entrant. All tachycardias had a left bundle branch block pattern. An inferior axis was evident in 11 VTs, and 2 VTs had a leftward axis. In the 4 patients with documented spontaneous VT requiring external cardioversion, the induced tachycardia was identical to the clinically documented arrhythmia.

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**Figure 1** Position of the Multielectrode Balloon Array

Noncontact mapping of ventricular tachycardia in an 18-year-old boy (Patient #2) after surgical repair of tetralogy of Fallot (left: 30° right anterior oblique; right: 60° left anterior oblique). The multielectrode balloon array of the noncontact mapping system (MEA) has been inflated in the right ventricular outflow tract; the steerable mapping and ablation catheter (Map) is positioned at the anterior right ventricular free wall supported by a long precurved sheath. Additional catheters have been placed at the His bundle position (His) and in the coronary sinus (CS).

**Figure 2** Dynamic Substrate Map

Dynamic substrate map (DSM) of the right ventricle (patient as in Fig. 1) during sinus rhythm; geometry of the right ventricle has been reconstructed (30° right anterior oblique view). The scale on the left side shows the ratio of the peak negative voltage through the cardiac cycle. Negative voltage is displayed on the endocardial surface with white illustrating low-peak negative voltage (<25% of the largest unipolar deflection) and purple the most negative peak voltage (>57.5%). The zone between 25% and 57.5% is displayed in intermediate colors. Two areas of low-peak negative voltage (<35%) can be identified between the right anterior wall and the right ventricular outflow tract and between the tricuspid valve annulus and the right ventricular apex. A critical channel of electrical conduction can be suspected between the 2 low-voltage areas (arrows). Aneurysm = right ventricular outflow tract aneurysm; anterior = anterior right ventricular free wall; lateral = right ventricular lateral wall; RVA/Apex = right ventricular apex; TV6 = tricuspid valve annulus at 6 o’clock position; 6 to 10 = localization of virtual catheter electrograms.
on 12-lead electrocardiogram. In the remaining 6 patients who had nonsustained VT, the induced tachycardias had the same cycle length with a variation of ±20 ms.

Endocardial mapping and RF ablation. Reconstruction of the endocardial surface of the RV could be accomplished in all patients. Anatomy correlated nicely with the RV angiography. There was no obvious relationship between the angiography/the anatomy reconstructed by the non-contact mapping system reconstruction and the induced VT cycle length or pattern of VT circuit, respectively. All VTs had an RV origin. In all patients, induced VT resulted in unstable hemodynamics with a systolic blood pressure <40 mm Hg.

MACRO–RE-ENTRANT TACHYCARDIAS (n = 11). The area of the shortest isthmus in the circuit to target is displayed in Table 1. In 2 patients (Patients #9 and #10), the critical isthmus involved tissue in close proximity, approximately 5 to 10 mm, to the location of the His bundle. In order to avoid induction of atrioventricular block, no RF current was delivered. In one patient (Patient #5), the critical isthmus was identified between the tricuspid valve annulus and the RV outflow tract. After induction of an RF ablation line between the 2 structures, no ventricular ectopy could be induced. However, repeat pacing on each side of the line revealed residual conduction with very low amplitude signals at the line probably reflecting conduction within the epicardial layers of the RV wall.
In all macro–re-entrant tachycardias, the ablation lines that needed to be created extended from 10 to 40 mm.

**MICRO–RE-ENTRANT TACHYCARDIAS (n = 2).** The area of earliest endocardial ventricular activation is displayed in Table 1. The inferred sizes of these areas were approximately 5 and 8 mm.

**Summary of results.** The procedure was considered acutely successful in 8 of 10 patients (80%) and in 11 of 13 VTs (85%). Repeat programmed ventricular stimulation revealed non-inducibility in these 8 patients. In 2 patients, no RF ablation was performed due to risk of atrioventricular block. Proof of complete conduction block along the ablation line could be demonstrated for all but 1 of the targeted macro–re-entrant tachycardias.

**Procedure characteristics.** The mean duration of the procedure (skin to skin) was 390 (range 223 to 518) min, mean fluoroscopy time 53.9 (range 14 to 105) min. No complications were noted.

**Further considerations.** As this approach to fast and potentially life-threatening VT is new and no follow-up data were available, ICD implantation was recommended to all patients irrespective of the results of the procedure.

**Follow-up.** After the ablation procedure, 3 of 4 patients who had experienced spontaneous sustained VT requiring external cardioversion received an endocardial ICD. In the fourth patient who had no ablation performed due to risk of atrioventricular block (Patient #9), the parents refused ICD implantation. All 4 patients who had nonsustained VT on Holter before the procedure denied ICD implantation.

During follow-up (mean 35.4, range 3 to 52 months), 6 of 8 patients with a successful procedure remained free of VT. In 1 of 3 patients with a newly implanted ICD (Patient #7), occurrence of a VT with a different cycle length was documented and successfully terminated by the ICD. The remaining patient (Patient #6) had 2 new macro–re-entrant VT. After the second procedure, this patient is still free of VT.

**Discussion**

The noncontact mapping system has been used to guide mapping and successful ablation of both idiopathic VT and VT after myocardial infarction (16,17). However, this series
is the first to report feasibility and efficacy of using the noncontact mapping system in patients after surgical repair of TOF with hemodynamically unstable VT. Radiofrequency catheter ablation was successful for 85% of the tachycardia substrates. In patients with stable hemodynamic during sustained VT after surgical correction of TOF, catheter ablation using traditional mapping techniques has been shown to be technically feasible (4) with a comparable success rate and limited follow-up. Except for this, success rates were significantly lower. Success rates of 60% using electroanatomical mapping and/or conventional mapping techniques (5) and of 50% using the noncontact mapping and/or the electroanatomical mapping (6) in patients with VT after surgical correction of congenital heart disease were reported. However, due to the variety of VT substrates and mapping techniques in these studies, a direct comparison of prior results with the present study remains difficult.

In the present study, mean VT cycle length of 269 ms was significantly lower compared with that seen in previous studies reporting on hemodynamically stable VT with a mean cycle length of 377 ms (4). In 2 case reports using the electroanatomical mapping system, VT cycle length was 340 ms (10) and 480 ms (11), respectively.

Pacing and entrainment techniques, which are often critical for a success when using contact mapping, were not feasible in the patients reported here due to high ventricular rates. Efficacy could be demonstrated by both non-inducibility of the VT and pacing from adjacent sites along the induced lines. In only 1 patient complete conduction block along the line could not be accomplished, possibly due to a subepicardial or epicardial location of the critical structure. However, no VT was inducible in this patient after the ablation procedure.

In 2 patients, the critical isthmus was identified in close proximity to the His bundle. Radiofrequency ablation was not performed to avoid atrioventricular block. If these patients had been studied more recently, cryotherapy would have been a safer therapeutic alternative, which has been reported in RV outflow tract tachycardia (18). However, cryoenergy was not available at the time of study.

Follow-up. Long-term follow-up data after RF ablation of VT in patients after surgical repair of TOF are lacking. In the present study, we noted VT recurrence with a different cycle length and morphology in 2 of 8 patients after an initially successful procedure. Patient #6 remained free of VT after the second procedure.

As this approach to fast and potentially life-threatening VT is new and no follow-up data were available, ICD implantation was recommended to all patients irrespective of the result. There was recurrent macro-re-entrant VT in 1 patient. This was either a proarrhythmic effect of ablation, or more likely, a circuit that was not detected at the earlier study. In either event, it reinforces uncertainty and skepticism about ablation being absolutely curative.
Study limitations. Due to unstable hemodynamics, ablation could only be performed during sinus rhythm. Thus, termination of the ongoing VT could not be assessed. However, in macro–re-entrant VT, ablation success was documented by both non-inducibility of VT and demonstration of the completeness of the linear ablation lines. A more precise identification of low voltage areas can probably be achieved by electroanatomical mapping (Carto, Biosense Webster, Diamond Bar, California). However, in hemodynamically unstable VT, electroanatomical mapping of the activation sequence seemed to be difficult or even technically not feasible. Therefore, noncontact mapping may represent the preferable technique for catheter ablation of hemodynamically unstable VT after surgical repair of TOF.

Conclusions

In patients with fast and unstable VT after surgical repair of TOF, noncontact mapping allowed for an understanding of the tachycardia substrate, and for effective and safe RF ablation therapy.

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REFERENCES