Non-Contact Mapping to Guide Radiofrequency Ablation of Atypical Right Atrial Flutter

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OBJECTIVES
This study was aimed at evaluating the efficacy of non-contact mapping and ablation of non-incisional atypical right atrial (RA) flutters.

BACKGROUND
The majority of atypical RA flutters were reported in patients after surgical incision of the RA.

METHODS
The study group consisted of 15 patients (61 ± 13 years, 8 males) with atypical atrial flutter (AFL). The RA activation during AFL was delineated using a non-contact mapping system (EnSite 3000 with Precision Software, Endocardial Solutions, St. Paul, Minnesota). The narrowest part of each reentrant circuit was targeted using radiofrequency energy.

RESULTS
In all 15 patients, non-contact mapping showed AFLs confined to the RA with RA activation time accounting for 100% of the cycle length (210 ± 19 ms). During single-loop re-entry in seven patients, the activation wave front circulated around the central obstacle (CO) in the anterolateral wall with conduction through the channel between the CO and the crista terminalis (CT). During figure-of-eight re-entry in eight patients, simultaneous upper and lower loop re-entry through the conduction gap in the CT was found in four patients, and simultaneous upper loop and free-wall single-loop re-entry was observed in four patients. Radiofrequency ablation of the free-wall channel and/or CT gap was effective in eliminating these AFLs in 13 patients. During a follow-up of 16.8 ± 3.8 months, two patients had recurrence of left AFL, and one had recurrence of atrial fibrillation.

CONCLUSIONS
Atypical RA flutters could arise from single-loop or double-loop figure-of-eight re-entry. Radiofrequency ablation of the free-wall channel and/or the CT gap was effective in eliminating these arrhythmias. (J Am Coll Cardiol 2004;44:1080–6) © 2004 by the American College of Cardiology Foundation

Electrophysiologic study. Informed written consent was obtained from all patients. As described previously, all antiarrhythmic drugs except amiodarone were discontinued for at least five half-lives before the study (12). In all patients, a 7-F, 20-pole, deflectable Halo catheter with 10-mm paired spacing (Cordis-Webster, Inc., Baldwin Park, California) was positioned around the tricuspid annulus to record the RA activation in the lateral wall and CTI simultaneously. A 7-F, deflectable, decapolar catheter with 2-mm interelectrode distance and a 5-mm space between each electrode pair was also inserted into the coronary sinus via the internal jugular vein. The position of the proximal electrode pair at the ostium of the CS was confirmed with contrast injection. A 9-F sheath placed in the left femoral vein was used to introduce the non-contact mapping catheter.

If spontaneous atrial arrhythmia was found at the onset of study, the non-contact mapping was performed to investigate the reentrant circuit. If sinus rhythm was present at baseline, low RA and CS pacing (twice diastolic threshold, 8 beats followed by bursts) until 2-to-1 atrial capture was performed to induce atrial arrhythmias.

Non-contact mapping system. The non-contact mapping system (EnSite 3000, Endocardial Solutions, St. Paul, Minnesota) was used to delineate the RA activation in the lateral wall and CTI simultaneously (2004;44:1080–6).
Minnesota) was described in detail previously (12–15). In brief, the system consists of a non-contact catheter (9-F) with a multi-electrode array (MEA) surrounding a 7.5-ml balloon mounted at the distal end. Raw data detected by the MEA are transferred to a silicon graphics workstation via a digitalized amplifier system.

Before deployment of the MEA, patients were administered 3,000 IU heparin boluses to maintain activated clotting time between 250 and 300 s throughout the study. The MEA catheter was deployed over a 0.035-inch guide wire, which had been advanced to the superior vena cava (SVC). The system locates any catheter in relation to the MEA using a “locator” signal, which serves two purposes. It is used to construct a three-dimensional computer model of the virtual endocardium providing a geometry matrix for the inverse solution. Geometric points are sampled at the beginning of the study during sinus rhythm or AFL. The locator signal is also used to display and track the position of the catheter on the virtual endocardium and allows marking of anatomic locations identified using fluoroscopy and electrogram characteristics.

Using mathematical techniques to process these potentials, the system is able to reconstruct more than 3,000 unipolar electrograms simultaneously and superimpose them onto the virtual endocardium, producing isopotential maps with a color range representing voltage amplitude. Reconstructed electrograms can also be selected from sites on the virtual endocardium and displayed individually. Morphologic components in the virtual electrogram correlate with the contact electrogram as previously validated (13–15).

During review of the recorded data, we always started the high-pass filter setting at 2 Hz to preserve the slow conduction on the isopotential map. We also drew the virtuals on the map color spot to analyze the unipolar electrograms and made 1-to-1 color settings to the electrogram deflections of interest. If the atrial electrograms were overlapped with the T-wave, we increased the high-pass

<table>
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<th>Abbreviations and Acronyms</th>
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<tr>
<td>AFL = atrial flutter</td>
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<td>CO = coronary sinus</td>
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<td>CS = crista terminalis</td>
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<td>CT = cavotricuspid isthmus</td>
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<td>IVC = inferior vena cava</td>
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<td>MEA = multi-electrode array</td>
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<td>RA = right atrial/atrium</td>
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<td>RF = radiofrequency</td>
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<td>SVC = superior vena cava</td>
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Figure 1. The 12-lead surface electrocardiograms of atypical right atrial flutters. (A) Single-loop re-entry. The flutter waves are positive in leads II, III, aVF and positive in lead V1. (B) Type I figure-of-eight re-entry. The flutter waves are positive in leads II, III, aVF and biphasic in lead V1. (C) Type II figure-of-eight re-entry. The flutter waves are negative in leads II, III, aVF and positive in lead V1.
Table 1. Previous Re-Entry Characteristics RF Ablation and Ablation of Atrial Flutter

<table>
<thead>
<tr>
<th>Patient</th>
<th>Gender</th>
<th>CVD</th>
<th>Age (yrs)</th>
<th>Use of Amiodarone</th>
<th>AF History</th>
<th>Electrocardiogram</th>
<th>24-h Holter monitoring</th>
<th>Clinical symptoms</th>
<th>Electrophysiologic characteristics</th>
<th>RESULTS</th>
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<td>Four patients had spontaneous incessant atypical AFLs.</td>
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<td>In 11 patients, sustained atypical AFLs were induced with burst pacing at cycle lengths of 200 to 250 ms from the CS or low lateral RA.</td>
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<td>The mean cycle length of atypical AFLs was 210 ± 19 ms. During sustained atypical AFL, RA activation was earlier than CS activation.</td>
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<td>Non-contact mapping showed AFLs confined to the RA, with RA activation time accounting for 100% of the cycle length in all 15 patients.</td>
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<td>Seven patients had single-loop re-entry, and eight patients had double-loop figure-of-eight re-entry.</td>
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<td>Single-loop re-entry (n = 7). The activation wave front propagated through the channel between the CT and central obstacle (CO), which was composed of a functional block area in the anterolateral wall combined with the RA appendage, activated the low anterior wall, ascended to the high anterior wall, turned around the SVC, activated the septal and posterior wall, and crossed over the top of the CT to complete the reentrant circuit (Fig. 2). The virtual unipolar electrograms in the CO showed double potentials.</td>
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<td>Type I figure-of-eight re-entry (n = 4). In all patients, non-contact mapping showed simultaneous upper loop</td>
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<td>RF ablation. Radiofrequency ablation was performed using a 4-mm electrode-tipped ablation catheter connected to an EPT-1000 generator. For single-loop re-entry, the channel between barriers of the reentrant circuit was ablated. For double-loop figure-of-eight re-entry, the narrowest part of each reentrant circuit was targeted separately. Sequential point-by-point energy applications (50 W, 60°C, 40 s) were performed to produce a linear lesion. Successful ablation was defined as termination and non-inducibility of the targeted tachycardia. Bidirectional conduction block along the ablation lines was verified by pacing during sinus rhythm and shown by non-contact mapping.</td>
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<td>Follow-up. After hospital discharge, all patients were followed up closely in the outpatient clinic. Long-term efficacy was assessed clinically on the basis of the resting surface electrocardiogram, 24-h Holter monitoring, and clinical symptoms.</td>
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<td>Definitions. “Double potentials” were defined as virtual unipolar atrial electrograms with two discrete deflections per beat separated by an isoelectric baseline.</td>
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<td>“Lower loop re-entry” was defined as a CTI-dependent atypical AFL involving the lower portion of the RA (11).</td>
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<td>“Upper loop re-entry” was defined as an atypical AFL involving the upper portion of the RA with transverse conduction through the conduction gap in the crista terminalis (CT) (12).</td>
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<td>Statistical analysis. Continuous data are expressed as mean ± SD.</td>
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re-entry and lower loop re-entry. In two patients, the activation wave front crossed the CT gap (common pathway) in the posterior-to-anterior direction and bifurcated into two wave fronts (Fig. 3). One propagated upward in the anterolateral wall, turned around the SVC, and spread downward in the septal wall (upper loop re-entry), whereas the other propagated downward in the anterolateral wall, traveled through the CTI, and spread upward in the septal...
wall (lower loop re-entry). Two wave fronts joined in the region of the CS ostium and proceeded in the posterior wall to complete the reentrant circuit. The other two patients had a reverse activation sequence with the common pathway still located in the CT gap.

Type II figure-of-eight re-entry (n = 4). In all patients, the activation wave front propagated through the channel (common pathway) between the CT and CO (a functional block area in the anterolateral wall combined with the RA appendage) and bifurcated into two wave fronts. One crossed the CT gap, spread upward in the posterior wall, and turned around the SVC (upper loop re-entry), whereas the other turned around the CO, spread upward in the anterior wall to the roof (free-wall re-entry), and joined the wave front from the opposite reentrant circuit (Fig. 4).

RF ablation and follow-up. Two patients did not tolerate the RF ablation procedure, because of chest pain. In seven patients with single-loop re-entry, the channel between the CT and CO was successfully ablated with a mean of 10 ± 4 pulses. In three patients with type I figure-of-eight re-entry, RF applications were first delivered in the CTI. After a mean of 3 ± 1 pulses, the tachycardia changed into upper loop re-entry only. After subsequent ablation of the CT gap with a mean of 5 ± 2 pulses, the tachycardia was eliminated. In three patients with type II figure-of-eight re-entry, RF applications were first delivered in the CT gap. After a mean of 7 ± 2 pulses, the tachycardia changed into free-wall re-entry. In subsequent ablation of the channel between the CT and CO with a mean pulse of 9 ± 1, the tachycardia was abolished. There was no complication during the ablation procedure.

During a follow-up of 16.8 ± 3.8 (range 10 to 23) months, two patients had recurrence of left AFL. In seven patients with a history of atrial fibrillation, only one had recurrence of atrial fibrillation during the follow-up period.

DISCUSSION

Major finding. The present study demonstrated that atypical RA flutters could arise from single-loop or double-loop figure-of-eight re-entry. During single-loop re-entry in seven patients, the activation wave front circulated around the CO composed of a functional block area and the RA appendage. During figure-of-eight re-entry, simultaneous upper and lower loop re-entry through the CT gap was found in four patients, and simultaneous upper loop and free-wall re-entry through the common channel between the CT and the CO was observed in four patients. Radio-
frequency ablation of the free-wall channel and/or the CT gap was effective in eliminating these AFLs.

**Single-loop re-entry.** Single-loop re-entry with the CO in the RA free wall has been demonstrated in sterile pericarditis and atrial enlargement models (16–18). In the left atrial enlargement model, Yamauchi et al. (18) found single-loop RA re-entrant circuits in 7 of 10 dogs. The most common form is a re-entrant circuit rotating around the SVC and RA appendage. After placement of an incision from the SVC to the inferior vena cava (IVC) just lateral to the CT, AFL recurred with the circuit rotating around the RA appendage and a line of functional block (18).

The characteristics of single-loop re-entry in this study seem to reflect a similar re-entrant circuit rotating around the SVC and RA appendage with the CT as a lateral boundary. Therefore, RF ablation of the channel between the CO near the RA appendage and CT could eliminate this tachycardia. Use of non-contact mapping simplified the characterization of the re-entrant circuit and permitted the precise localization of ablation lines.

**Figure-of-eight re-entry.** The figure-of-eight re-entry was first described by El Sherif et al. (19), who mapped the canine ventricular arrhythmia in the subacute phase of myocardial infarction. The counterclockwise wave fronts rotated around the two separate arcs of functional conduction block and coalesced into a common wave front that conducted slowly between the arcs of block (19). The arcs of block developed as a result of ischemia-induced spatial nonhomogeneous lengthening of refractoriness (19).

The atrial figure-of-eight re-entry has been described in the canine sterile pericarditis model (20,21). Schoels et al. (20) demonstrated that 2 of 11 dogs (4 episodes) showed double-loop re-entry during sustained AFL. One had the reentrant circuits located in the lower RA wall, and the other had the re-entrant circuits involving the left atrial free wall (20). Uno et al. studied 10 episodes of induced, sustained AFL in nine dogs (21). In six episodes, there were two re-entrant circuits, with one in the RA free wall and the other involving the atrial septum; both circuits shared a pathway in the RA free wall (21).

Clinical dual-loop RA re-entrant tachycardias were first described by Shah et al. (4) in five patients with surgical closure of an ostium secundum atrial septal defect. Five figure-of-eight tachycardias had a counterclockwise loop around the tricuspid valve sharing a common anterior channel with a clockwise loop around the lateral atriotomy.

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**Figure 4.** Isochronal map showing type II figure-of-eight re-entry in the right posterior oblique view. The activation wave front propagated through the channel between two central obstacles. One is located in the crista terminalis (virtual 10 to 13), and the other is located in the anterolateral wall combined with the right atrial appendage (virtual 14 to 17). The counterclockwise wave front rotated around the anterior free-wall obstacle, whereas the clockwise wave front rotated around the crista terminalis. IVC = inferior vena cava; RAA = right atrial appendage; SVC = superior vena cava; TV = tricuspid valve.
scar (4). Using electroanatomic mapping, Ouyang et al. (22) have identified dual-loop reentrant circuits in 74% of left atrial macro-re-entrant tachycardias in 23 patients. The common isthmus of both loops was protected by two electrically silent areas, or one electrically silent area and an atriotomy scar (22).

In the present study, we first used non-contact three-dimensional mapping to delineate RA figure-of-eight re-entry in patients without an atriotomy scar. Type I showed simultaneous upper loop re-entry and lower loop re-entry. The activation wave fronts rotated around two functional/anatomic obstacles; one was the upper CT combined with the SVC, and the other was the lower CT combined with the IVC. Radiofrequency ablation of the CTI and the CT gap was effective in eliminating lower loop re-entry and upper loop re-entry, respectively. Type II showed simultaneous upper loop re-entry and anterior free wall re-entry. The activation wave fronts revolved around two separate COs; one was the CT, and the other was the functional block area combined with the RA appendage and SVC. The channel between the CT and the anterior wall CO was a common pathway. Radiofrequency ablation of the CT gap and the channel was effective in abolishing upper loop re-entry and anterior free-wall re-entry, respectively.

Conclusions. Atypical AFL could arise from single-loop or double-loop figure-of-eight re-entry in the RA. The conduction channel between the CT and CO and/or the CT gap were critical pathways for maintenance of AFL. Radiofrequency ablation of these critical pathways was effective in eliminating these atrial arrhythmias.

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REFERENCES