Focal Atrial Tachycardia
New Insight From Noncontact Mapping and Catheter Ablation

Satoshi Higa, MD; Ching-Tai Tai, MD; Yenn-Jiang Lin, MD; Tu-Ying Liu, MD; Pi-Chang Lee, MD; Jin-Long Huang, MD; Ming-Hsiung Hsieh, MD; Yoga Yuniadi, MD; Bien-Hsien Huang, MD; Shih-Huang Lee, MD; Kwo-Chang Ueng, MD; Yu-An Ding, MD; Shih-Ann Chen, MD

Background—This study investigated the electrophysiologic characteristics, atrial activation pattern, and effects of radiofrequency (RF) catheter ablation guided by noncontact mapping system in patients with focal atrial tachycardia (AT).

Methods and Results—In 13 patients with 14 focal ATs, noncontact mapping system was used to map and guide ablation of AT. AT origins were in the crista terminalis (n=8), right atrial (RA) free wall (n=3), Koch triangle (n=1), anterior portion of RA–inferior vena cava junction (n=1), and superior portion of tricuspid annulus (n=1); breakout sites were in the crista terminalis (n=5), RA free wall (n=5), middle cavotricuspid isthmus (n=2), and RA–superior vena cava junction (n=2). ATs arose from the focal origins (11 ATs inside or at the border of low-voltage zone), with preferential conduction, breakout, and spread to the whole atrium. After applications of RF energy on the earliest activation site or the proximal portion of preferential conduction from AT origin, 13 ATs were eliminated without complication. During the follow-up period (8±5 months), 11 (91.7%) of the 12 patients with successful ablation were free of focal ATs.

Conclusions—Focal AT originates from a small area and spreads out to the whole atrium through a preferential conduction. Application of RF energy guided by noncontact mapping system was effective and safe in eliminating focal AT.

(Circulation. 2004;109:84-91.)

Key Words: mapping ■ tachycardia ■ ablation

Most focal atrial tachycardias (ATs) can be successfully eliminated by catheter ablation, with low complication and recurrence rate.1–5 Although the details of electrophysiologic mechanisms and electropharmacological characteristics of focal AT have been demonstrated, the knowledge about activation pattern of focal AT is very limited.4,5 Recently, noncontact mapping system has been demonstrated to facilitate the identification of ectopic focus, because it is able to reconstruct the precise geometry in the atrium and ventricle and localize the ectopic beats.6,7 The purposes of this study were to demonstrate the electrophysiologic characteristics, activation patterns, and results of catheter ablation in patients with focal AT using noncontact mapping system.

Methods

Patient Characteristics
This study included 13 consecutive patients (7 men and 6 women; age 45±23 years) with clinically documented AT who were referred for electrophysiologic study and catheter ablation guided by the noncontact mapping system. Two patients had cardiomyopathy, and 1 patient had coronary artery disease (Table 1).

Catheter Position and Electrophysiologic Study
Informed written consent was obtained from all patients. As described previously, the patients were studied in the postabsorptive, nonsedated state.4,5 All antiarrhythmic drugs were discontinued for at least 5 half-lives before the study. A 7F, deflectable, decapolar catheter with 2-mm interelectrode distance and 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 coronary sinus was confirmed with contrast injection. A 9F sheath placed in the left femoral vein was used to introduce the noncontact mapping catheter.

Rapid right atrial stimulation (paced cycle length from 600 ms until 2:1 capture was noted) and right atrial extrastimuli (single or double) were used for induction and termination of AT, and they were repeated 2 to 4 times to ensure reproducibility of the responses. If programmed electrical stimulation failed to induce AT, isoproterenol (at graded dosages from 1 to 4 µg/min) was infused intravenously until AT developed or the sinus rate increased to 20% above the resting value. The electrophysiologic criteria used for diagnosis of focal AT have been reported.1–5

Received May 7, 2003; de novo received July 21, 2003; revision received September 29, 2003; accepted September 30, 2003.
From the Division of Cardiology, Department of Medicine, National Yang-Ming, University School of Medicine, Taipei Veterans General Hospital, Taiwan. Dr Higa is a research fellow from Okinawa University, Okinawa, Japan.
Dr Chen has received research support (including free catheters and software analysis) from ESI Co.
Correspondence to Shih-Ann Chen, MD, Division of Cardiology, Department of Medicine, Taipei Veterans General Hospital, 201, Sec. 2, Shih-Pai Road, Taipei, Taiwan. E-mail epsachen@ms41.hinet.net
© 2004 American Heart Association, Inc.

Circulation is available at http://www.circulationaha.org

DOI: 10.1161/01.CIR.0000109481.73788.2E
Response to Adenosine
In 6 patients (cases 2, 3, 8, 11, 12, and 13), an intravenous bolus of adenosine (3 to 12 mg) was given to observe the effects of adenosine on terminating AT.

Noncontact Mapping System
The noncontact mapping system (EnSite 3000 with Precision Software, Endocardial Solutions) has been described in detail previously.\(^6\)\(^-\)\(^10\) In brief, the system consists of a noncontact catheter (9F) with a multielectrode 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.

The MEA catheter was deployed over a 0.035-inch guide wire, which had been advanced to the superior vena cava (SVC) (Figure 1). It is used to construct a 3D computer model of the virtual endocardium, providing a geometry matrix for the inverse solution. The system is able to reconstruct more than 3000 unipolar electrograms simultaneously and superimpose them onto the virtual endocardium, producing isopotential maps with a color range representing voltage amplitude.

During review of the recorded data, we always began analysis with a default high-pass filter setting of 2 Hz to preserve components of slow conduction on the isopotential map. Color settings were adjusted so that the color range matched 1 to 1 with the millivolt range of the electrogram deflection of interest. We also interactively placed virtual electrodes on the map color contours to analyze the corresponding noncontact unipolar electrograms. Occasionally, conduction of activation wavefront was sufficiently slow that we moved the high-pass filter down to 1.0 to 0.5 Hz.

Definitions
Origin of AT was defined as the earliest site showing a single spot of isopotential map and a QS pattern of noncontact unipolar electrogram. Breakout site of AT was the earliest site that showed an rS pattern with sudden increase of peak negative potential of noncontact unipolar electrogram after AT depolarized. The preferential conduction was the initial direction of depolarization away from an origin. Double potentials were noncontact unipolar atrial electrograms with 2 discrete deflections per beat either separated by an isoelectric baseline or a low amplitude interval.\(^10\) Low voltage zone (LVZ) was an area with \(\leq 30\%\) amplitude of noncontact unipolar electrogram peak negative potential.

Validation of Noncontact Electrogram
Contact electrograms were recorded from 99 randomly chosen locations around the chamber during sinus rhythm and AT. The EnGuide navigation signals were simultaneously recorded from each.

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, y</th>
<th>Gender</th>
<th>Associated SHD</th>
<th>ATCL, msec</th>
<th>AT Onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>75</td>
<td>M</td>
<td>None</td>
<td>366</td>
<td>Spontaneous</td>
</tr>
<tr>
<td>2</td>
<td>50</td>
<td>F</td>
<td>Cardiomyopathy</td>
<td>378</td>
<td>Incessant</td>
</tr>
<tr>
<td>3</td>
<td>22</td>
<td>M</td>
<td>Cardiomyopathy</td>
<td>399</td>
<td>Incessant</td>
</tr>
<tr>
<td>4</td>
<td>50</td>
<td>F</td>
<td>None</td>
<td>333</td>
<td>Atrial stimuli</td>
</tr>
<tr>
<td>5</td>
<td>57</td>
<td>F</td>
<td>None</td>
<td>227</td>
<td>Atrial stimuli + Isop</td>
</tr>
<tr>
<td>6</td>
<td>48</td>
<td>F</td>
<td>None</td>
<td>466</td>
<td>Atrial stimuli + Isop</td>
</tr>
<tr>
<td>7</td>
<td>49</td>
<td>F</td>
<td>None</td>
<td>342</td>
<td>Atrial stimuli</td>
</tr>
<tr>
<td>8</td>
<td>16</td>
<td>M</td>
<td>None</td>
<td>448</td>
<td>Spontaneous</td>
</tr>
<tr>
<td>9</td>
<td>77</td>
<td>M</td>
<td>None</td>
<td>590</td>
<td>Incessant</td>
</tr>
<tr>
<td>10</td>
<td>35</td>
<td>M</td>
<td>None</td>
<td>362</td>
<td>Spontaneous + Isop</td>
</tr>
<tr>
<td>11</td>
<td>14</td>
<td>M</td>
<td>None</td>
<td>433</td>
<td>Incessant</td>
</tr>
<tr>
<td>12</td>
<td>75</td>
<td>M</td>
<td>CAD</td>
<td>385</td>
<td>Spontaneous + Isop</td>
</tr>
<tr>
<td>13</td>
<td>17</td>
<td>F</td>
<td>None</td>
<td>423</td>
<td>Spontaneous</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mean±SD</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>45±23</td>
<td>394±81</td>
<td>81</td>
</tr>
</tbody>
</table>

SHD indicates structural heart disease; ATCL, atrial tachycardia cycle length; Isop, isoproterenol infusion; and CAD, coronary artery disease.

Figure 1. Radiographs showing a multielectrode array balloon catheter (Balloon) in the right atrium, a decapolar catheter in the coronary sinus (CS), and an ablation catheter (ABL) around crista terminals. A and B. Right and left anterior oblique views, respectively.
site for geometric annotation of location and for generation of virtual electrograms that can be compared with the associated contact electrograms. Simultaneous recording of the bipolar and unipolar electrogram from the distal tip of the contact catheter was performed. Signals for both contact and noncontact electrograms were filtered with a bandwidth of 2 to 300 Hz. Electrogram morphologies, activation time difference, and electrogram voltage between contact and noncontact electrograms that were taken from the same endocardial sites were compared by use of a well-described template comparison algorithm.8,11,12

Catheter Ablation and Follow-Up
Catheter ablation (40 to 50 W, 50° C to 60° C, 40 seconds) was performed using a 4-mm electrode-tipped ablation catheter connected to an EPT-1000 generator (Boston Scientific Co). We first delivered RF energy on the origin or the proximal portion of preferential conduction from the origin. After catheter ablation, the same stimulation protocols used to induce AT before ablation were performed to make sure AT was noninducible. Successful catheter ablation was defined as inability to reinduce focal ATs. After hospital discharge, the patients were followed up closely (every 1 to 3 months) in the outpatient clinic. Long-term efficacy was assessed clinically on the basis of the resting surface ECG, 24-hour Holter monitoring, event recorder, and clinical symptoms.

Statistical Analysis
Continuous data were expressed as mean±SD. For validation of the mapping accuracy, correlation between contact and noncontact electrogram was explored by calculating Pearson’s correlation coefficients and using Bland-Altman technique for agreement. Differences were considered significant at P<0.05.

Results
Electrophysiological Characteristics
Fourteen focal ATs were demonstrated. Four ATs were incessant. Six ATs occurred spontaneously at the laboratory with or without isoproterenol, 2 ATs were initiated after right atrial stimuli, and 2 ATs were initiated after isoproterenol infusion plus atrial stimuli. Mean AT cycle length was 394±81 ms, and the mean earliest activation time was 51±22 ms before the onset of the P wave (Table 1). Furthermore, 11 of 14 ATs originated from inside the LVZ or border of LVZ.

Validation Data
The correlation between contact and noncontact unipolar electrograms showed the correlation coefficient of electrogram morphology was 0.88 (P<0.001), time difference was 2.25±2.79 ms, and correlation value of peak negative voltage (PNV) was 0.77 (P<0.001). These correlations showed the similar results in the area outside LVZ (correlation coefficient of electrogram morphology, 0.86; P<0.001; time difference, 3.1±3.0 ms; correlation value of PNV, 0.77; P<0.001) and inside LVZ (correlation coefficient of electrogram morphology, 0.89; P<0.001; time difference, 1.7±2.5 ms; correlation value of PNV, 0.93; P<0.001). The agreement analysis of voltage for all validation points and inside LVZ showed only 3%, 5%, and 3% of validation points were outside 2 SD, respectively (Figure 2). For the timing difference, 7% of validation points were outside 2 SD.

The mean absolute value of the contact unipolar electrogram PNV was −0.94±0.54 mV (sinus rhythm, −1.03±0.62; AT, −0.93±0.54 mV) in the area outside LVZ and −0.35±0.19 mV (sinus rhythm, −0.48±0.17; AT, −0.28±0.16 mV) inside LVZ. The mean absolute value of the noncontact virtual unipolar electrogram PNV was −1.02±0.41 mV (sinus rhythm, −1.61±0.69; AT, −0.96±0.32 mV) in the area outside LVZ and −0.42±0.20 mV (sinus rhythm, −0.52±0.12; AT, −0.37±0.21 mV) inside LVZ.

Response to Adenosine
Adenosine could not terminate AT in patient No. 2 with incessant AT. However, adenosine (3 to 12 mg) terminated ATs in the other 5 patients (Nos. 3, 8, 11, 12, and 13). Noncontact mapping demonstrated 2 types of adenosine-induced termination of AT. The first type (n=1) showed shifting of AT origin before termination. The second type (n=4) showed no change of AT origin. All AT termination episodes showed disappearance of focal activation at origin,
not attributable to block at the area of preferential conduction or exit site.

Anatomical Relation Between Origin, Preferential Conduction, and Breakout Site

Noncontact mapping clearly showed the anatomic locations and activation wavefronts from origins with preferential conduction and activated the myocardium surrounding the origin (Figures 3 through 5). The distance from the balloon center to AT origins was 21.6 ± 6.2 mm (Table 2).

Catheter Ablation and Follow-Up

For case No. 2, RF energy was applied on the proximal portion of preferential conduction from the origin because of continuous shifting of the origin within a small area (≈1.5 × 1.0 cm²). For case No. 3, RF energy was applied on the proximal portion of preferential conduction, because this patient had severe chest pain when we applied RF energy on the origin. For cases Nos. 1 and 7, RF energy was applied on the origin only. For the other 9 patients, RF energy was applied on the origin and proximal portion of preferential conduction. A mean of 11 ± 7 RF applications were required for eliminating AT, and 7 ± 9 RF applications were used for insurance ablation in the origins or preferential conduction. For case No. 3, AT cycle length increased to approximately 500 ms; because of the long procedure time, patient preferred discontinuation of the procedure, and had regular follow-up of the clinical symptoms. There was no complication during the ablation procedure. During a follow-up of 8 ± 5 months, only case No. 1 had recurrent AT originating from crista terminalis (CT), and the second procedure had successful ablation of AT from the old focus. The other 11 patients did not have recurrence of focal AT.

Discussion

Major Findings

To the best of our knowledge, this is the first study demonstrating the anatomical relation and electrophysiologic characteristics of origin with preferential conduction of focal AT.
using the noncontact mapping system. Catheter ablation of the origin or proximal portion of preferential conduction is effective in eliminating focal AT.

**Electrophysiological Characteristics**

In this study, 8 (57%) AT origins were located at CT. Kalman et al.\(^1\) have demonstrated the predominant origin of focal AT along the CT. CT showed an area of prominent anisotropy with slow conduction property and might play an important role in development of microreentry.\(^1\) Furthermore, cardiomyocytes in CT may have pacemaking activity or abnormal automaticity,\(^1\) and thus focal AT may originate from CT. The preferential conduction of 9 ATs was located along or across the CT; this finding suggests CT can be the part of preferential conduction that AT activation wavefronts pass through.

Information about the relationship between substrate property and AT origins is limited. Previous studies on the atrial specimen resected from the area with atrial arrhythmias showed a slow-response action potential with spontaneous depolarization.\(^1\) Josephson et al.\(^1\) also showed the slow response or depressed fast response action potential from the atrial specimen resected from human AT. These findings suggest focal AT may originate from diseased atria and explain the possible mechanism of 11 (78.6%) ATs originating from LVZ or border zone around the LVZ. The noncontact unipolar electrograms in the LVZ demonstrated wide, low-amplitude, and fractionated electrograms, suggesting a delayed and nonuniform anisotropic conduction through the diseased right atrium. This may be related to atrial fibrosis resulting from proliferation of smooth muscle cells and collagen fibers beneath the endocardial lining.\(^1\)

In previous myocardial infarction models that were used for validation of Carto system, the mean reduction of unipolar electrogram voltage in the infarcted area was 40.4% to 53.2%.\(^1\) When we selected the 30% of maximum peak negative voltage as the criteria for low voltage area, we found the mean reduction of contact unipolar electrogram voltage in the low voltage zone was 49% of the contact unipolar electrogram in the region outside the low voltage area. This is compatible with the previous study of myocardial infarction model.\(^1\)

**Figure 4.** Noncontact mapping of focal AT (No. 5) in the posterolateral view. Isopotential map shows overlapping of the activation wavefront located at origin, preferential conduction, and breakout site. Isopotential maps showing the activation sequence of AT. Color scale for each isopotential map has been set so that white indicates most negative potential and purple indicates least negative potential. The focal activation originates from the middle portion of CT, and the wavefront propagates up to the upper portion of CT through the preferential conduction area, then reaches the breakout site, and spreads to the whole atrium. The noncontact unipolar electrogram reveals a QS pattern at origin. The spread from the breakout site activates the myocardium surrounding the AT origin. Noncontact electrograms at the origin and proximal site of the preferential conduction reveal multicomponents of activation. Origin indicates origin of AT; BO, breakout site of AT; and RAA, right atrial appendage.
account for functional variation in unipolar peak negative voltage and low voltage zone of both beat to beat and patient to patient would make voltage data more comparable.

**New Insight of Focal AT From RF Catheter Ablation**

Conventionally, intracardiac mapping of focal AT using 1 or 2 roving catheters was used to localize the origin of AT. A QS pattern in the unipolar recording is highly predictive of the successful ablation site. Recently, noncontact mapping systems have provided an accurate guide for mapping and ablation of focal tachycardia.

Several investigators have demonstrated and validated the accuracy of noncontact mapping system in mapping of atrial and ventricular arrhythmias if the distance from noncontact mapping system balloon center was <4 cm. This study showed that the distance from balloon center to origin, area of preferential conduction, or breakout site was within 4 cm in 13 of the 14 ATs. Validation of the origin, area of preferential conduction, and breakout site showed the significant correlation between contact and noncontact electrogram morphology, time difference, and voltage. These correlations also were demonstrated inside and outside the LVZ.

The present study also showed that applications of RF energy on the origin or proximal portion of preferential conduction could eliminate focal AT. This finding raises the issues of selecting appropriate sites for RF ablation. Previous studies showed impulses generated within the sinus node could not propagate to the atrium when conduction in the zone of perinodal fibers becomes depressed (attributable to pathological conditions). In case No. 2, the noncontact mapping showed continuous shifting of focal activation site within a small area, and we observed disappearance of AT after application of RF energy on the proximal portion of preferential conduction only. In case No. 3, who had severe chest pain and vagal reflex during ablation of the AT origin, we changed the ablation target from origin to preferential conduction and could decrease the AT rate.

Marchlinski et al have reported focal AT with preferential conduction using magnetic electroanatomical mapping. The concepts from previous studies may explain that ablation of specific fiber connected from the origin can create the exit
block from origin.\textsuperscript{27,28} These findings suggest that preferential conduction may play a critical role in ablation of focal AT, and we can choose the ablation target according to the concept of origin and preferential conduction of AT. Although the true mechanism of preferential conduction was unknown, this conduction may be preferential in 1 direction because of anisotropic conduction, anatomic obstacles, or conduction through islands of scar or poor conduction. Such conduction may not be electrically protected either permanently or functionally.

**Study Limitations**

Although we have demonstrated the accuracy of voltage correlation inside and outside the LVZ, future research using the experimental model with pathologic study is still necessary.

**Conclusions**

Noncontact mapping successfully demonstrated precise locations and electrophysiologic characteristics of origin and preferential conduction of focal AT. Focal AT originates from a small area, conducts through a preferential area, and spreads out to the whole atrium. Application of RF energy on origin or proximal portion of preferential conduction was effective in eliminating focal AT.

**Acknowledgments**

This work was supported by grants NSC92-2314-B-010-052, NSC92-2314-B-038-050, VGH92-37, 238, RFCM92-01-009, and SKH-TMU-92-17.

**References**


distributed atrial pacemaker complex in the human heart. Circulation.

16. Ten Eick RE, Singer DH. Electrophysiological properties of diseased
human atrium. I: low diastolic potential and altered cellular response to

cellular electrophysiology to clinical function and ultrastructure. Circ Res.
1983;52:188–199.

response to pharmacologic agents of fibers from diseased human atria.

automatic atrial tachycardia: anatomic and electrophysiologic correlates.


clinical experiences using an electromechanical endocardial mapping
procedure to distinguish infarcted from healthy myocardium. Circulation.

mapping in the porcine model of healed anterior myocardial infarction:
correlation with intracardiac echocardiography and pathological analysis.

of chronic myocardial infarction in the canine coronary occlusion model.


on the electrical properties of isolated rabbit and canine atria. Circ Res.

mapping for ablation of focal atrial tachycardias. Pacing Clin Electrophysiol.

27. Strauss HC, Bigger JT. Electrophysiological properties of the rabbit

28. Kirchhof CJ, Allessie M. Sinus node automaticity during atrial fibrillation
Focal Atrial Tachycardia: New Insight From Noncontact Mapping and Catheter Ablation
Satoshi Higa, Ching-Tai Tai, Yenn-Jiang Lin, Tu-Ying Liu, Pi-Chang Lee, Jin-Long Huang,
Ming-Hsiung Hsieh, Yoga Yuniadi, Bien-Hsien Huang, Shih-Huang Lee, Kwo-Chang Ueng,
Yu-An Ding and Shih-Ann Chen

Circulation. 2004;109:84-91; originally published online December 22, 2003;
doi: 10.1161/01.CIR.0000109481.73788.2E
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2003 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/109/1/84

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/