Radiofrequency Catheter Ablation of Sustained Intra-atrial Reentrant Tachycardia in Adult Patients

Identification of Electrophysiological Characteristics and Endocardial Mapping Techniques

Shih-Ann Chen, MD; Chern-En Chiang, MD; Chin-Juey Yang, MD; Chen-Chuan Cheng, MD; Tsu-Juey Wu, MD; Shih-Pu Wang, MD; Benjamin N. Chiang, MD; and Mau-Song Chang, MD

Background. Information about electrophysiological characteristics and radiofrequency ablation of intra-atrial reentrant tachycardia has not been reported before. We proposed that induction and termination of intra-atrial reentrant tachycardia by atrial extrastimuli or rapid atrial pacing and resetting the response pattern by atrial extrastimuli during intra-atrial reentrant tachycardia could ensure the mechanism of reentry and that the earliest site of endocardial activation and concealed entrainment pace mapping with the shortest stimulus–P wave interval could localize a critical area responsible for intra-atrial reentrant tachycardia and radiofrequency ablation.

Methods and Results. Seven patients with refractory atrial tachycardia were referred for electrophysiological studies and radiofrequency ablation. Electrophysiological studies and endocardial mapping found (1) 10 atrial foci with atrial tachycardia cycle length of 406±41 ms; (2) atrial tachycardia had induction and termination by atrial extrastimuli (8 of 10) or rapid atrial pacing (10 of 10); (3) atrial tachycardia had increasing (6 of 10) or mixed (flat and increasing, 4 of 10) resetting response pattern, with resetting interval of 57±13 ms (14±4% of atrial tachycardia cycle length); (4) exit sites of atrial tachycardia in right atrial free wall (6 of 10), right atrial septum (3 of 10), and midposterior left atrium (1 of 10); and (5) earliest activation site had timing relative to P wave by −37±7 ms, and concealed entrainment pace mapping had shortest stimulus–P interval 23±3 ms. By the endocardial activation pace-mapping technique, radiofrequency energy (8±1 pulses, 31±3 W, 101±11 seconds) successfully eliminated the 10 atrial foci without recurrence (follow-up, 16±5 months).

Conclusions. Mechanism of intra-atrial reentrant tachycardia could be confirmed by the electrophysiological characteristics, and radiofrequency ablation energy delivered to a critical area in the atrial reentrant circuit is safe and effective for the treatment of intra-atrial reentrant tachycardia. (Circulation 1993;88:578-587)

Key Words • radiofrequency • ablation • tachycardia • atrium
TABLE 1. Clinical Characteristics of Seven Patients with Intra-atrial Reentrant Tachycardia

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (y)/Sex</th>
<th>Pattern of IART</th>
<th>History of IART (y)</th>
<th>Associated cardiovascular disease</th>
<th>Previous antiarrhythmic drugs</th>
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<tr>
<td>1</td>
<td>56/F</td>
<td>Incessant</td>
<td>5</td>
<td>Cardiomyopathy</td>
<td>Dig, Proc, Qui, Ami</td>
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<tr>
<td>2</td>
<td>62/M</td>
<td>Paroxysmal</td>
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<td>Ver, Pro</td>
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<tr>
<td>3</td>
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<td>Dig, Pro, Ver, Ami</td>
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<tr>
<td>4</td>
<td>69/M</td>
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<td>Hypertension, biventricular enlarge</td>
<td>Dig, Pro, Ver</td>
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<tr>
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<td>Pro, Ver</td>
</tr>
<tr>
<td>6</td>
<td>48/M</td>
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<td>None</td>
<td>Dig, Pro, Ver</td>
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<tr>
<td>7</td>
<td>32/M</td>
<td>Paroxysmal</td>
<td>4</td>
<td>None</td>
<td>Dig, Proc, Qui</td>
</tr>
</tbody>
</table>

IART indicates intra-atrial reentrant tachycardia; Ami, amiodarone; Dig, digoxin; Proc, procainamide; Pro, propranolol; Qui, quinidine; and V, verapamil.

critical myocardial focus is responsible for intra-atrial reentrant tachycardia. Furthermore, a successful outcome would help establish the concept about accurate mapping of intra-atrial reentrant tachycardia.

Methods

Patient Population

Seven consecutive patients (one woman and six men; mean age, 57±13 years) with intra-atrial reentrant tachycardia were referred for electrophysiological study and treatment of tachycardia by radiofrequency catheter ablation technique at our institution from November 1990 to November 1992. The procedure was performed under a protocol approved by the Clinical Investigation Committee. None of the seven patients had previously undergone diagnostic electrophysiological study. The history of atrial tachycardia ranged from 2 to 5 years (mean, 3±1 years). All subjects had failed to respond to two to four antiarrhythmic drugs (mean, 3.1±0.9). Tachycardia was incessant in one patient, and intermittent in the remainder. One patient had cardiomyopathy, one patient had biventricular enlargement identifiable by echocardiography, and the other five patients did not have any associated cardiovascular disorders (Table 1). Two patients presented with presyncope, and five patients had palpitations only. All patients had multiple ECG recordings of tachycardia before referral.

Electrophysiological Study and Endocardial Mapping Technique

As described previously, each patient was studied in the postabsorptive nonfasted state. Antiarrhythmic drugs were discontinued for at least five half-lives in all patients. Three multipolar, tip-reflectable, closely spaced (2 mm) electrode catheters (Mansfield, Watertown, Mass) were positioned in the right atrium, Hisbundle area, and right ventricle; two orthogonal electrode catheters (Mansfield) were positioned in the coronary sinus for recording and/or stimulation. Three surface leads (I, II, and V1) were recorded simultaneously with intracavitary electrograms using a VR-13 recorder (Electronics for Medicine, Pleasantville, NY) at a paper speed of 100 to 150 mm/s and filtered between 30 and 500 Hz. Electrical stimulation was delivered by a programmable stimulator (Bloom Associates, Ltd, Narbeth, Pa) with a pulse duration of 2 ms at approximately twice the diastolic threshold. Baseline electrophysiological study consisted of measurement of conduction intervals, followed by determination of atrial and atrioventricular node refractory periods. Refractory periods were determined using a single extrastimulus (S2) during sinus rhythm and during at least two atrial pacing cycle lengths. The S2 was introduced after every eighth paced beat with coupling intervals decreasing by 10-ms intervals. Rapid right atrial stimulation (paced cycle length from 600 ms to 2:1 capture was noted) and right atrial extrastimuli (single and/or double) were used for induction and termination of atrial tachycardia, and they were repeated two to four times to ensure reproducibility of the responses. Electrophysiological criteria used for diagnosis of intra-atrial reentrant tachycardia have included the following: (1) P wave configuration different from that of sinus rhythm and atrial activation sequence supporting a nonsinus origin for initial depolarization, (2) PR interval appropriate to the tachycardia rate, (3) tachycardia reproducibly initiated or terminated with atrial programmed stimuli, (4) tachycardia induction and maintenance independent of atrioventricular node conduction, and (5) elimination of accessory pathways or atrioventricular nodal reentry as a diagnostic possibility.12-15 Detailed attention was given to the 12-lead ECG of any atrial tachycardia to determine similarity to the previously documented clinical tachycardia.

For the assessment of resetting response patterns, a single atrial extrastimulus was synchronized to the atrial activation wave and was delivered at the high lateral right atrium during atrial tachycardia in 10-ms decrements (beginning 10 ms less than the cycle length of atrial tachycardia, until the entire cardiac cycle was scanned, local atrial refractoriness was reached, or atrial tachycardia was terminated). Each coupled single atrial extrastimulus was delivered after every 20th tachycardia beat with stable cycle length, and it was repeated two to four times to ensure reproducibility of the response. Resetting of the atrial tachycardia was said to have occurred if (1) the interval from the last atrial tachycardia beat before each particular mode of stimulation to the first nonpaced atrial tachycardia beat after stimulation was different from that of the atrial tachycardia without tachycardia termination, and (2) the same atrial tachycardia (identical cycle length and morphology) resumed after pacing, suggesting a fixed exit site from the tachycardia circuit. For each atrial tachycardia, the difference between the longest and shortest coupling interval resulting in resetting was defined as the resetting interval. The return cycle was defined as the interval from the last paced electrogram to the next electrogram measured at the pacing site. Measurements were made from the rapid deflection of the local electro-
FIG 1. A mixed resetting response pattern during the resetting of an atrial tachycardia with single extrastimuli (patient 3). A through F, cycle length of atrial tachycardia \( (A_1A_1) \) is 380 ms, and single extrastimuli \( (S) \) are delivered from the lateral aspect of the high right atrium (LHRA) with coupling intervals \( (A_1A_2) \) of 300 to 230 ms. The return cycles \( (A_2A_3) \) remain fixed (430 ms) during longer coupling intervals (300 to 280 ms) and progressively increased during shorter coupling intervals.

grams recorded from the proximal pair of the quadripolar electrode catheter. Resetting response patterns were characterized by plotting the return cycles versus the coupling intervals of the extrastimuli. The responses observed could be grouped into three patterns. A flat pattern was defined by the presence of 10-ms-or-less difference in return cycle occurring over a 30-ms-or-more range of coupling intervals. An increasing pattern was defined as
an increase in the return cycle as the coupling interval of the extrastimulus was decreased. There were tachycardias meeting criteria for a flat response at long coupling intervals, followed by an increasing response at shorter coupling intervals, and such a pattern was defined as mixed, or “flat plus increasing” (Figs 1 and 2).
Endocardial mapping studies were done at the same time as the baseline electrophysiological study. After atrial tachycardia was established with a uniform P wave configuration and stable cycle length, a 7F quadripolar electrode catheter with 2-mm interelectrode spacing (deflectable, 4-mm tipped, Mansfield) was inserted into the right atrium via the right femoral vein for mapping and ablation. Using biplane fluoroscopy (right and left anterior oblique views), the right atrial endocardium was mapped by rotating the mapping catheter in the anteromedial, anterior, anterolateral, lateral, posterolateral, posterior, posteromedial, and septal positions at four levels, beginning high near the superior vena cava and then withdrawing 1 cm at a time to the inferior vena cava (ie, high, −1 cm, −2 cm, −3 cm). Recording from two or three sites near the right atrial appendage, fossa ovalis, and below the fossa around the coronary sinus ostium were carefully made. The left atrium was recorded via the coronary sinus catheter. It was often facilitated by the use of long curved vascular sheaths (Cordis, Miami, Fla), even in patients with a patent foramen. These sheaths ensured left atrial access during catheter changes and improved torque transmission and overall catheter steering ability. Atriograms were performed in three patients to clarify anatomic landmarks. Local activation time, defined as the maximal slope of the endocardial electrogram as it crossed the baseline, was referenced to the earliest onset of the P wave during atrial tachycardia in surface ECG leads I, II, and V1. An activation sequence map was constructed manually to determine the possible location of the reentrant circuit and earliest atrial activation. If P wave onset was indistinct or obscured by T wave activity, a stable intracardiac signal (eg, low septal right atrium in the His-bundle area) was initially used as the reference. Criteria of transient entrainment are in abbreviated form: (1) constant fusion, (2) progressive fusion, (3) localized conduction block, and (4) an abrupt shortening in local electrogram activation time with change in electrogram morphology during progressively faster pacing of tachycardia (according to Waldo and colleagues16). Concealed entrainment was defined as acceleration of the tachycardia to the pacing cycle length without alteration of P wave morphology or endocardial activation sequence and without termination of atrial tachycardia on cessation of pacing.18,19 During entrainment pace mapping, 12-lead ECGs were recorded and compared with baseline atrial tachycardia. An area of slow conduction was presumed to be present at a specific site if a concealed entrainment pace mapping with a long stimulus-to-P wave interval (more than 40 ms) was noted during pacing. The presumed exit site from the area of slow conduction was identified by the presence of a concealed entrainment pace mapping with a short stimulus-to-P wave interval (less than 40 ms).19

Radiofrequency Ablation Technique

Radiofrequency ablation was performed using a radiofrequency generator (Radionics-3C, Radionics, Inc, Mass.) providing 500 kHz, unmodulated sine wave energy, connected to the distal 4-mm tip of the mapping/ablation catheter via a switch box and grounded to the patient’s posterior chest wall using a standard electrosurgical grounding pad. Applied voltage and measured current were displayed continuously, and impedance was monitored by the meter on the radiofrequency generator. Details were described previously elsewhere.16,17 Radiofrequency energy was applied in a power range from 20 to 40 W, and it was commenced with constant rhythm monitoring. If atrial tachycardia terminated within 10 seconds after energy application (test pulse), a full 60-second application (long pulse) was performed at that site. Radiofrequency discharge was terminated immediately on the occurrence of impedance rise, displacement of catheter, or complaint by the patient of severe chest pain. If an ablation attempt at a particular site was unsuccessful in terminating atrial tachycardia within 10 seconds, radiofrequency discharge was terminated. Endocardial mapping was repeated in an attempt to identify a more accurate ablation site by systematically repositioning the ablation catheter several millimeters around the initial position. If a second atrial tachycardia focus was found, procedures including electrophysiological study, endocardial mapping, and radiofrequency ablation were repeated.

After successful ablation of atrial tachycardia, programmed stimulation was performed immediately and after approximately 20 to 30 minutes to determine if atrial tachycardia was inducible. For the prevention of possible recurrent tachycardia, a long pulse (60 seconds) of radiofrequency energy was delivered to the successful ablation site. Isoproterenol (2 to 4 μg/min) was routinely used to facilitate induction after initial success. After completion of the study, patients were monitored in the coronary care unit overnight.

Patient Follow-up

All patients were seen in the outpatient clinic at 1 week, 1 month, and then every 3 months, at which time a history of recent symptoms was taken, and a physical examination, 12-lead ECG, and 24-hour Holter were performed. Patients were followed without antiarrhythmic drugs.

Statistical Analysis

Data were analyzed by the Student’s t test for paired or unpaired data and, where appropriate, the Wilcoxon rank-sum test. Data are expressed as mean±SD. Statistical significance is defined as P<.05.

Results

Baseline Electrophysiological Study

Baseline sinus cycle length (600 to 760; mean, 700±20 ms), sinus node recovery time (680 to 1210; mean, 970±66 ms), AH interval (70 to 140; mean, 90±15 ms), HV interval (40 to 50; mean, 45±2 ms), QRS duration (80 to 105; mean, 90±7 ms), and atrial effective refractory periods (180 to 240; mean, 210±10 ms, measured at a cycle length of 500 ms) were obtained in every patient (Table 2). Sustained atrial tachycardia was induced in all seven patients. The tachycardia was initiated by a single atrial extrastimulus (one patient) or both rapid atrial pacing and a single atrial extrastimulus (six patients). The mean coupling interval of atrial extrastimuli for induction of atrial tachycardia (tachycardia zone) was 57±12 ms (range, 40 to 80 ms). Furthermore, an inverse relation between the prematurity of tachycardia initiating extrastimulus and the coupling interval of the first tachycardia beat was found in the six patients. The tachycardia cycle
length ranged from 330 to 410 ms (mean, 406±41 ms). Intravenous isoproterenol was not necessary for induction of atrial tachycardia in any patient.

It was noted that introduction of a single atrial extrastimulus during tachycardia would reproducibly reset or terminate the tachycardia. The resetting response pattern showed mixed pattern in six atrial tachycardias (Figs 1 and 2, A) and increasing pattern in four atrial tachycardias (Fig 2, B). The mean resetting interval for the entire group was 57±13 ms, and it represented 14.2±3.8% of the tachycardia cycle length. The mean resetting interval was significantly longer in the mixed pattern than it was in the increasing pattern (63±13 versus 47±5 ms; P<.05). There was no significant correlation between the resetting interval and the tachycardia cycle length. Atrial tachycardia cycle length did not differ significantly in the patients with mixed or increasing resetting response pattern (428±19 versus 392±47 ms; P>.05).

**Endocardial Atrial Mapping**

Local electrical activity recorded from the successful ablation site (during tachycardia) preceded the onset of the surface P wave by 25 to 45 ms (mean, 38±7 ms). The signals contained no evidence of early extraprotentials, fractionation, or continuous electrical activity suggesting slow conduction area (Fig 3, A). Concealed entrainment pace mapping at the successful ablation site had a short stimulus-to-P wave interval (20 to 30 ms; mean, 23±3 ms). Three patients had double intra-atrial reentrant tachycardia. The new reentrant circuit was found after successful ablation of the initial tachycardia, and the exit site of the new reentrant circuit was far from the original one with different activation sequence (Table 2).

**Results of Radiofrequency Ablation**

The number of "test pulse" of radiofrequency energy (less than 10 seconds) ranged from 3 to 20 (mean, 8±1 pulses), and the number of full duration radiofrequency applications (long pulse) was two for each tachycardia. Successful radiofrequency energy (long pulse) was delivered for a total of 80 to 118 seconds (mean, 102±11 seconds) with power of 25 to 35 W (mean, 30.5±2.1 W). The initial response of the tachycardia focus to a successful radiofrequency pulse was always rapid and ranged from 0.5 to 3.0 seconds (mean, 1.8±0.8 seconds). On nine occasions, the atrial tachycardia simply stopped abruptly during the initial few seconds of radiofrequency application; in patient 6, the atrial tachycardia slowed over a few beats before termination. The time between onset of radiofrequency output and termination of atrial tachycardia ranged from 1.0 to 5.0 seconds (mean, 2.3±1.2 seconds). Procedure time ranged from 2.0 to 3.8 hours (mean, 3.1±0.3 hours), with fluoroscopy time of 25 to 100 minutes (mean, 42±10 minutes) (Table 3).

**Table 2. Electrophysiological Parameters and Endocardial Mapping Data in Patients With Intra-atrial Reentrant Tachycardia**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Methods of AT induction</th>
<th>Methods of AT termination</th>
<th>Resetting response pattern</th>
<th>Resetting interval (ms)</th>
<th>IART cycle length (ms)</th>
<th>Earliest endocardial activation site</th>
<th>Timing relative to P wave (ms)</th>
<th>Timing stimulus-to-P wave interval (ms)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>1: RAE</td>
<td>RAP, RAE</td>
<td>Mixed (F+I)</td>
<td>60</td>
<td>440</td>
<td>PSRA</td>
<td>−45</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>2: RAE</td>
<td>RAP, RAE</td>
<td>Mixed (F+I)</td>
<td>50</td>
<td>400</td>
<td>LPLRA</td>
<td>−40</td>
<td>25</td>
</tr>
<tr>
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<td>1: RAP, RAE</td>
<td>RAP, RAE</td>
<td>Increase</td>
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<td>360</td>
<td>LPRA</td>
<td>−25</td>
<td>20</td>
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<tr>
<td></td>
<td>2: RAP, RAE</td>
<td>RAP, RAE</td>
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<td>MLRA</td>
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<td>20</td>
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<td>370</td>
<td>HMRA</td>
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<td>RAP, RAE</td>
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<td>50</td>
<td>430</td>
<td>PSRA</td>
<td>−40</td>
<td>25</td>
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<tr>
<td>5</td>
<td>RAP, RAE</td>
<td>RAP, RAE</td>
<td>Increase</td>
<td>40</td>
<td>420</td>
<td>MSRA</td>
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<td>25</td>
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<td>6</td>
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<td>RAP, RAE</td>
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<td>460</td>
<td>MPLA</td>
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<td>RAP, RAE</td>
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<td>PLRA</td>
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<td>406±41</td>
<td>−38±7</td>
<td>23±3</td>
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</table>

IART indicates intra-atrial reentrant tachycardia; AT, atrial tachycardia; RAE, right atrial single extrastimulus; RAP, right atrial rapid pacing; F, flat pattern; I, increasing pattern; PSRA, posteroseptal area of right atrium (RA); LPLRA, low posterolateral area of RA; LPRA, low posterior area of RA; MLRA, midlateral area of RA; HMRA, high medial area of RA; ASRA, anteroseptal area of RA; MSRA, midseptal area of RA; MPLA, midposterior area of left atrium; and PLRA, posterolateral area of RA.
Complications

Complications were not encountered. Postprocedure echocardiograms (M-mode, two-dimension, and Doppler) showed no evidence of pericardial effusion, deterioration in ventricular function, intracardiac thrombus, or new valvular insufficiency. Measurements of serum creatinine phosphokinase (CPK) were available in all the patients, and the maximum values for total CPK varied between 70 and 166 IU/mL with an MB fraction of 5% to 16%. No clear correlation was found between CPK data and total radiofrequency energy.

Postablation Follow-up

All of the patients had regular follow-up (range, 10 to 24 months; mean, 16 ± 5 months) in the outpatient clinic and remained in sinus rhythm without medication. They were free of symptoms, and "new" arrhythmias related to the radiofrequency lesions have not been detected on follow-up Holter monitoring. The patient (patient 1)
with cardiomyopathy had improvement of ventricular function on 3-month follow-up echocardiograms. Left ventricular end-diastolic diameter decreased from 64 to 58 mm, and fractional shortening increased from 14% to 22%. Five patients received 3-month follow-up electrophysiological study, and intra-atrial reentrant tachycardia was not inducible.

**Discussion**

We report the first clinical series of adult patients who have had intra-atrial reentrant tachycardia managed by radiofrequency ablation. This study shows the electrophysiological characteristics of intra-atrial reentrant tachycardia and indicates that a successful result can be expected with the described ablation technique.

**Mechanisms of Atrial Tachycardia**

Atrial tachycardia is an uncommon arrhythmia in adults,13-15 and the exact mechanisms of atrial tachycardia are not well known. Previous studies20-22 have suggested enhanced atrial automaticity or reentrance. Wyndham and colleagues22 reported in vitro electrophysiological studies in atrial tissue from a 41-year-old patient with paroxysmal atrial tachycardia. Transmembrane voltage recordings from the surgically excised atrial tissue demonstrated triggered automaticity as a probable mechanism in that case.

Evidence for a reentrant mechanism was noted in this study. Because our patients had atrial tachycardia reproducibly induced and terminated by programmed electrical stimuli, automaticity is an unlikely mechanism. Two additional findings independently suggest a reentrant mechanism versus triggered activity. First, the behavior of the coupling interval from the last paced beat to the first escape complex of the tachycardia (the first postspacing interval) has been found to be useful in the differentiation between reentrant and triggered arrhythmias.23,24 An inverse relation between the prematurity of the tachycardia initiating extrastimulus and the coupling interval of the first tachycardia beat has been considered to be specific for reentry, whereas a concordant pattern has been thought to be typical for triggered activity.23,24 In this study, a concordant pattern was not found. Second, there was a gradually increasing or mixed resetting response pattern. In experimental preparations of sustained triggered rhythms, single extrastimuli have been shown to result in resetting with a flat or a decreasing pattern followed by increasing responses.25,26 In contrast, our patients did not have the above two patterns. Furthermore, the pattern that showed totally or partially increasing (increasing or mixed pattern) has not been described during triggered rhythms but was observed during atrioventricular bypass tract reentrant tachycardias.27

**Nature of the Reentrant Circuit in Intra-atrial Reentrant Tachycardia**

Several models of reentry have been described. Mines28 proposed an anatomically defined reentrant pathway. Allessie and colleagues29 proposed the so-called "leading circle" model in which the reentrant pathway is the shortest possible pathway and is determined on an instantaneous basis by refractoriness ahead of the activation wave front. Mehra and colleagues30 found that reentry could occur with a "figure-eight model" around two functional areas of block. Because in the leading circle model the reentrant pathway is determined by refractoriness ahead of the activation wave front, the excitable gap has to be composed of partially refractory tissue. The resetting response curve of such a circuit would not be expected to contain a flat zone. Six of the 10 tachycardias that could be analyzed in our study demonstrated resetting response curves with at least some flat zone, therefore militating against the leading circle model as the mechanism of these intra-atrial reentrant tachycardias. This same contention is also supported by the relatively long resetting intervals observed, which exceeded 10% of the tachycardia cycle length in 9 of the 10 atrial tachycardias. We believe that this finding is also evidence against a leading circle type of reentry, in which the excitable gap is expected to be absent or of minimal duration. Furthermore, in a leading circle model, the cycle length is determined only by refractoriness, and the gap is as short as possible, independent of the cycle length. In contrast, in the other two models of reentry, because the circuit is determined by areas of anatomic or functional block, a fully excitable gap of significant duration is conceivable. In an anatomically defined

**Table 3. Biophysical Parameters and Results of Radiofrequency Ablation in Patients With Intra-atrial Reentrant Tachycardia**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Radiofrequency No.</th>
<th>Successful radiofrequency pulse</th>
<th>Response to successful radiofrequency ablation</th>
<th>Follow-up period (mo)</th>
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<tbody>
<tr>
<td></td>
<td>Test pulse</td>
<td>Long pulse</td>
<td>Power (W)</td>
<td>Duration (s)</td>
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<tr>
<td>1</td>
<td>1: 4 2</td>
<td>2</td>
<td>30</td>
<td>80</td>
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<td>2: 8 2</td>
<td>2</td>
<td>32</td>
<td>102</td>
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<td>2</td>
<td>26</td>
<td>101</td>
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<td></td>
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<td>2</td>
<td>25</td>
<td>98</td>
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<td>118</td>
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<td>100</td>
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<td>7</td>
<td>6 2</td>
<td>2</td>
<td>35</td>
<td>92</td>
</tr>
<tr>
<td>Mean ±SD</td>
<td>8±1</td>
<td>2±0</td>
<td>30.5±3.1</td>
<td>102.5±11</td>
</tr>
</tbody>
</table>

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reentrant circuit, the slower the conduction velocity, the longer will be the cycle length and the excitable gap (refractoriness being constant). If the patient population was larger, we probably could have found a significant correlation between resetting interval and the tachycardia cycle length.

Regarding the resetting response pattern, three mechanisms could explain an increasing response pattern or an increasing zone in a mixed response pattern. One mechanism is conduction delay of the paced impulse between the pacing site and the circuit. Although this could be the case in individual instances, no direct evidence was found. Another mechanism is a variable site of entrance determined by the tail of refractoriness in the reentrant circuit or by a functional conduction block in the tissue surrounding the circuit. In these cases, late coupled impulses would enter the reentrant pathway more distally than early coupled ones. Thus, more premature impulses would have to proceed over a longer pathway within the reentrant circuit, resulting in a longer return cycle and an increasing response. A third mechanism is an excitable gap composed entirely of partially refractory tissue. In this case, premature stimuli would encounter increasingly refractory tissue with increasing conduction delays and longer return cycles at shorter coupling intervals. If a zone of fully excitable tissue preceded that of partially refractory tissue, a mixed (flat plus increasing) response would ensue. This study showed that resetting intervals are shorter in patients with an increasing pattern and longer in patients with mixed patterns (Table 2). This finding is consistent with the hypothesis that excitable gaps that are composed totally of partially refractory tissue might be shorter and those composed of fully excitable and partially refractory tissue might be longer.

**Mapping of Intra-atrial Reentrant Tachycardia**

The mapping/ablation features that best predicted successful elimination of intra-atrial reentrant tachycardia included (1) local electrical activity preceding onset of the surface P wave by 20 to 45 ms (mean, 38 ± 7 ms), (2) a concealed entrainment with a relatively short stimulus-to-P wave interval of 20 to 30 ms, suggesting that the site was at the exit from the reentry circuit, and (3) rapid resolution of tachycardia within less than 5 seconds of beginning radiofrequency application. Concealed entrainment with a long stimulus-to-P wave interval was demonstrated during pacing near the earliest activation site, suggesting the existence of an area of slow conduction. Although the intracardiac electrograms suggesting slow conduction were not found in the present study, radiofrequency energy delivered to the exit site could eliminate atrial tachycardia. Because pacing within a region of slow conduction that is connected to the reentrant circuit but is not critical to the maintenance of reentry might also result in concealed entrainment, it is possible that the success rate of radiofrequency catheter ablation would decrease if we used only concealed entrainment as a guide to the ablation site. Furthermore, Feld and colleagues reported that a combination of endocardial activation mapping and entrainment pace mapping could determine a critical area for radiofrequency ablation of common type atrial flutter.

**Management of Atrial Tachycardia**

Poor control of atrial tachycardia may ultimately necessitate aggressive therapy. Results of prospective studies have shown short-term effects of antiarrhythmic drugs, but the long-term outcome has been disappointing as a result of recurrent arrhythmia or drug side effects. Transcatheter ablation is now evolving as a realistic alternative to chronic pharmacological or surgical management of this condition. Catheter ablation of the normal atrioventricular conduction system allowed atrial tachycardia to persist but controlled the ventricular rate. However, it created pacemaker dependence. A more attractive option is direct eradication of the atrial tachycardia focus. Direct-current ablation carried a potential risk of perforation through the relatively thin atrial wall. Radiofrequency lesions at the atrial level have been shown to produce transmural necrosis that heals with a well-organized fibrous scar and have not been associated with significant risk for either early or late perforation. Radiofrequency ablation is also free of the barotrauma and catheter fling seen with direct-current ablation, further decreasing the likelihood of acute atrial trauma. Radiofrequency current would thus appear to be well suited for atrial muscle ablation, including the fragile areas near the atrial appendages. Successful radiofrequency ablation of atrial tachycardia was reported before, but the mechanisms of these atrial tachycardias were due to enhanced automaticity or triggered activity. Some series describing surgical therapy for atrial tachycardia (due to enhanced automaticity or triggered activity) have reported arrhythmia recurrence after excision of atrial foci. Furthermore, some patients have had multiple foci or diffuse atrial disease. In the present study, three of the seven patients had more than one focus responsive for atrial tachycardia, and the mechanism of tachycardia was reentry. Although we did not detect any recurrent atrial tachycardia, it is important to follow-up the long-term results. However, the initial results remain quite encouraging and suggest that the technique is at least as effective as a surgical procedure.

In the present study, fluoroscopy and procedure times were similar to the times reported for radiofrequency ablation of accessory atrioventricular pathways or slow atioventricular nodal pathways in our laboratory. Innovations in mapping catheter design may permit faster simultaneous mapping of multiple atrial sites; more experiences with this ablation technique and the availability of laboratory equipment with the capacity for pulsed fluoroscopy at slow frame rates will shorten procedure times and reduce radiation exposure times to the patient and operator. Although radiofrequency ablation currently is very well established as definitive therapy for tachyarrhythmias due to accessory atrioventricular pathways or dual atrioventricular nodal pathways in patients of all ages, this report represents the first large series to describe its application to intra-atrial reentrant tachycardia. Unlike the accessory pathways and slow atioventricular nodal pathways, accurate localization of an atrial focus must involve a three-dimensional mapping and is further confounded by the absence of reliable electrogram markers (such as accessory pathway or slow atrioventricular nodal pathway activation potentials) apart from local activation time and entrainment pace mapping. Despite these difficulties, the technique was successful in all patients, and
they remained in sinus rhythm without medications. Radiofrequency catheter ablation of atrial tissue could be recommended as an alternative to medication or surgery in patients with intra-atrial reentrant tachycardia.

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