Characterization of Reentrant Circuits in Left Atrial
Macroreentrant Tachycardia

Critical Isthmus Block Can Prevent Atrial Tachycardia Recurrence

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Background—Left atrial macroreentrant tachycardia (LAMRT) has not been characterized in detail.

Methods and Results—Twenty-eight patients with LAMRT, including 4 patients with ablated typical atrial flutter (AFL), underwent electroanatomic mapping of the left atrium (LA) between February 1999 and October 2001. LA maps were performed during LAMRT in 26 patients and during sinus rhythm in 2 patients. Electrically silent areas or continuous lines of double potentials were identified as acquired anatomic barriers in all patients. In 23 of 26 patients with LAMRT mapping, 42 reentry circuits with a protected isthmus were identified. The isthmus was 11.8±5.9 mm wide, with the maximal amplitude of 0.07 to 3.61 mV. Radiofrequency pulses terminated all LAMRTs in 23 patients and resulted in conduction block across the isthmus in 20 patients. In 2 patients with sinus mapping, all identified isthmuses were ablated. Additionally, AFL was induced and ablated in 6 patients. Atrial tachycardia recurred in 4 patients: 3 patients without validated block across the isthmus presented with recurrence of the same LAMRT, and 1 patient without ablated cavotricuspid isthmus presented with AFL. All tachycardias were abolished during a second procedure. Of 25 patients with identified isthmuses, 20 patients were without atrial arrhythmia and 5 had only atrial fibrillation during a median follow-up of 14 months.

Conclusion—The reentry circuit with a protected isthmus can be identified in 89% patients with LAMRT by electroanatomic mapping. The isthmuses were amenable to radiofrequency applications in most patients. No atrial tachycardia recurred in any patients with isthmus block. (Circulation. 2002;105:1934-1942.)

Key Words: catheterization • ablation • electrophysiology • mapping • tachycardia

Catheter ablation guided by 3D electroanatomic mapping has been used to manage incisional atrial reentrant tachycardia in patients with repaired congenital heart disease.1–7 A recent study has reported that left atrial macroreentrant tachycardia (LAMRT) can be successfully abolished by radiofrequency (RF) linear lesion in most patients,7 but the critical isthmus was not delineated in detail. The aims of this study were (1) to describe the electroanatomic characteristics of the reentry circuit in patients with LAMRT, (2) to assess the effect of RF lesions on the critical isthmus and the conduction block across the isthmus, and (3) to investigate atrial tachycardia recurrence in patients after successful ablation.

Methods
Between February 1999 and October 2001, 28 consecutive patients (16 male; age, 64±10 years; range, 18 to 81 years) were studied. Twenty-six patients had permanent and 2 patients had paroxysmal atrial tachycardia for 2.6±2.5 years (range, 1 month to 10 years), despite the use of 2.2±1.0 (range, 1 to 5) antiarrhythmic drugs. Twenty-two of the 28 patients had structural heart disease. Of these, 9 had mitral valve surgery (MVS), as a result of rheumatic valvular disease in 5 patients and of nonrheumatic valvular disease in 4 patients. The left atrium (LA) size measured by transthoracic echocardiography was 51.6±6.0 mm (40 to 62 mm). Four patients had previous cavotricuspid isthmus ablation for typical atrial flutter (AFL). All patients had been on oral anticoagulants before ablation. Transesophageal echocardiography was performed to rule out LA thrombi in all patients. Oral anticoagulants were stopped at admission and replaced by intravenous heparin to maintain partial thromboplastin time at 2 to 3 times the control value. This was stopped 6 to 8 hours before ablation for transseptal puncture. All patients gave written informed consent.

Electrophysiological Study
After the withdrawal of antiarrhythmic drugs except amiodarone (9 patients), all patients underwent electrophysiological evaluation. Multipolar catheters were introduced to the His bundle and the coronary sinus. Mapping of the right atrium (RA) was initially performed to exclude RA tachycardia on the basis of the following criteria: (1) the site of the earliest RA activation was located in the...
Bachmann bundle area, as demonstrated by electroanatomic mapping in the initial 6 patients, and (2) the postspacing interval in the RA was longer than the tachycardia cycle length by >40 ms in at least 6 different sites in the RA, including the cavitricuspid isthmus and RA free wall.

Electroanatomic Mapping
LA mapping was performed with a 4-mm electrode tip (Navi-Star, Biosense Webster Inc) via a transseptal approach. Bipolar electrograms were recorded on the electroanatomic mapping system (filtered at 10 to 400 Hz) and a separate Quinton EP system (filtered at 30 to 400 Hz). Validation of this technology has been previously reported. Activity mapping was performed relative to the timing reference from the catheter inside the coronary sinus. Detailed mapping was performed in electrically silent areas (ESAs), which presented with no atrial potential distinguishable from noise and were displayed in gray, or along continuous lines of double potentials (LDPs) separated by an isoelectric interval of ≥50 ms. Anatomic location of ESAs and LDPs in the LA was arbitrarily defined as anterior, posterior, septal, lateral wall, and roof. A new map was performed to identify a different reentrant circuit if P wave morphology or tachycardia cycle length changed during RF ablation. Sinus mapping was performed to identify the tachycardia isthmus in case of unstable hemodynamics or spontaneous tachycardia change.

Definitions of LAMRT and Reentrant Circuit
LAMRT was defined as follows: (1) continuous sequence of atrial activation, with earliest activation adjacent to latest activation, and (2) range of activation times >90% of the tachycardia cycle length. Furthermore, entrainment mapping was performed to identify the reentrant circuit and its critical isthmus. The tachycardia reentrant circuit was defined as a single or dual loop with an isthmus constrained between nonconductive tissues. This nonconductive tissue can be either an acquired ESA or LDP. It can also encompass a natural anatomic barrier such as the mitral annulus (MA) or the pulmonary venous ostia (PVO).

RF Ablation
RF energy was delivered at the critical isthmus. It was initiated at 20 W, titrated to reach a target temperature of 50°C to 55°C with a limit of 50 W, and maintained until the bipolar atrial potential recorded from the ablation electrode decreased by 80% or split into double potentials, indicating local conduction block, with a time limit of 120 seconds. The catheter was held stationary or dragged across the isthmus.

If conventional RF energy was delivered without amplitude reduction of the atrial potential and termination of LAMRT, an irrigated-tip catheter (Irrigated Navi-Star, Biosense-Webster) was used. RF energy was delivered according to a safe protocol found in experimental studies. This consisted of temperature-controlled RF delivery with a power limit of 50 W, a target temperature of 45°C, and an infusion rate of 30 mL/min during RF delivery. Procedural success was defined as (1) termination and noninducibility of LAMRT and (2) conduction block across the tachycardia isthmus.

Validation of Conduction Block Across the Tachycardia Isthmus
Conduction block across the isthmus was validated by (1) pacing close to the ablation line and demonstration of marked delay and reversal in the direction of activation on the opposite side of the linear lesion when RF application closed the isthmus between the posterior wall and the lateral MA; (2) counterclockwise activation of the LA and/or coronary sinus around the MA during sinus rhythm when RF application closed the anterior isthmus near the MA; and (3) absence of any atrial activation within isthmuses far from the MA.

Postablation Management
Transesophageal echocardiography was performed within 48 hours after ablation. All patients were maintained on anticoagulation. Patients without mechanical valve replacement or atrial fibrillation were then discharged under oral anticoagulation with warfarin for 3 months. Follow-up was obtained from the referring physician or our outpatient clinic. Additional ablation procedures were performed in case of any atrial tachycardia recurrence.

Statistical Analysis
Values are given as mean±SD or median as appropriate.

Results
Patients With MVS
Mapping of the LA was performed with 240±34 points (range, 158 to 291 points) to reconstruct the LA in 9 patients. The mapping data are shown in the Table. One ESA or LDP was constantly found anterior to the septal PVO in all patients, which indicated the left atriotomy (Figure 1A and 1B). One to 4 ESAs or LDPs were found in the LA. In 7 patients with 17 mapped LAMRTs, the tachycardia circuits presented as dual loop through an isthmus between 2 posterior ESAs in 3 (Figure 2A and 2B), between the LA atriotomy and a posterior ESA in 3 (Figure 2C and 2D), or between a posterior ESA and a PV in 6 (Figure 2E and 2F), or presented as single loop around the MA with an isthmus between a posterior ESA and the lateral MA in 3 (Figure 3A) and with an isthmus between an anterior ESA and the anterior MA in 2. The tachycardia was terminated by 1 to 7 RF applications across the isthmus in 5 patients. In patients 4 and 9, during RF application, the clinical tachycardia changed to a different tachycardia, which propagated through a different isthmus and was terminated by RF applications. In patient 3, after block of the critical isthmus, 3 linear lesions were applied at different isthmuses to prevent potential new tachycardias (Figure 2C and 2D).

In 2 patients, mapping was not feasible during induced tachycardia (patient 6 with mitral valve replacement after endocarditis because of 1:1 atrioventricular conduction with cycle length of 260 ms, and patient 8 with mitral commissurotomy after rheumatic mitral stenosis because of cycle length variation from 270 to 400 ms). The ESAs and LDPs were identified during sinus rhythm. The isthmuses were located between an anterior ESA and the atriotomy (21.7 mm; amplitude, 0.12 to 0.27 mV), between an anterior ESA and the left superior PVO (14.1 mm; amplitude, 0.13 to 0.50 mV), and between an anterior ESA and the MA (16.9 mm; amplitude, 0.09 to 0.23 mV) in 1 patient, and between the atriotomy and the right PVO (14.1 mm; amplitude, 0.53 to 1.45 mV) in the other patient (Figure 1B). Four to 6 RF pulses were delivered at each isthmus during sinus rhythm.

Conduction block across all targeted isthmuses was achieved by 4.0±1.8 conventional RF pulses, except in 2 isthmuses between the posterior ESA and the lateral MA in patients 1 and 4. These isthmuses could not be blocked by 10 and 14 conventional RF applications after tachycardia termination. LAMRT recurred with the same reentry circuit after the initial ablation in both patients (Figure 3A). Irrigated RF application terminated tachycardia and produced permanent conduction block in patient 1 (Figure 3B), and temporary conduction block in patient 4, in whom a permanent conduction block was achieved by epicardial ablation from the coronary sinus (Figure 4).
Mapping and Ablation Data During Tachycardia

<table>
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<th>Patient</th>
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<th>AT-CL, ms</th>
<th>Reentry Circuit</th>
<th>Location</th>
<th>Width, mm</th>
<th>Amplitude, mV</th>
<th>Potential</th>
<th>Conduction Block</th>
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RHD indicates rheumatic heart disease; MS/MI, mitral stenosis/regurgitation; MVR, mitral valve replacement; AVR, aortic valve replacement due to aortic regurgitation; MC, mitral commissurotomy; CAD, coronary artery disease; HCM, hypertrophic cardiomyopathy; LHV, left ventricular hypertrophy due to primary hypertension; DCM, dilated cardiomyopathy; LDP, line of double potentials; Ant, anterior; Post, posterior; RPV, right pulmonary venous ostia; LPV, left pulmonary venous ostia; CC-MA/C-MA, counterclockwise/clockwise reentry around the mitral annulus; D, diastolic potential; S, systolic potential.

*Patients with amiodarone.
Patients Without MVS

A map of the LA was obtained during sustained LAMRT in 19 patients without MVR; 223/11006 (range, 115 to 345 points) were acquired to reconstruct the LA. In 16 patients with 25 identified reentry circuits, 1 to 4 ESAs or LDPs were found (Table). The maps showed reentry circuits with dual loop in 19 and single loop in 6. The reentry circuits propagated through an isthmus between a posterior ESA/LDP and a PVO in 7, between an anterior ESA/LDP and the MA in 5 (Figure 5, A through C), between 2 anterior ESA/LDPs in 6 (Figures 6 and 7), between an anterior ESA and a PVO in 4, between a roof LDP and the left PVO in 1, between an posterior and a roof ESA in 1, and between 2 roof ESAs in 1.

One to 6 RF applications terminated the tachycardia in all patients, except in patients 18 and 28. In these 2 patients, RF delivery resulted in different LAMRTs, which propagated through a different isthmus (Figure 8, A through C) and required 1 to 3 RF applications for termination.

In 3 patients (patient 12, with structurally normal heart; patient 14, with mitral regurgitation; and patient 21, with coronary artery disease on amiodarone), the reentry circuit could not be identified during LAMRT with cycle lengths of 205, 220, and 230 ms. Also, the mapping showed multiple ESAs and/or LDPs. The tachycardias were not terminated with 15/11006 conventional RF pulses.10–13

In patients with identified reentry circuits, conduction block across the targeted isthmuses was achieved by a mean of 3.3±1.8 conventional RF pulses in 15 patients.1–6 In patient 17, conduction block could not be validated. LAMRT recurred with the same reentry circuit in this patient. A single RF delivery at the isthmus terminated tachycardia with conduction block during a second procedure.

Characteristics of the Isthmus Responsible for LAMRT

The anatomic location, width, and amplitude of the isthmuses during tachycardia are shown in the Table. The local electrogram during LAMRT within the targeted isthmus exhibited diastolic activity in 36 tachycardias and systolic activity in 6 tachycardias relative to the P wave in V1 on the surface ECG with single, double, triple, or fragmented atrial potentials. The targeted isthmus was 11.8±5.9 mm (4.0 to 31.4 mm) in width. The maximal amplitude of the bipolar atrial potential at the targeted isthmus ranged from 0.07 to 3.61 mV. The maximal amplitude of bipolar atrial potential was <0.5 mV in 31 tachycardias and extremely low (<0.1 mV) in 3 tachycardias. In patients 1 and 4, the maximal amplitude within the isthmus between a posterior ESA and the lateral MA was 1.81 mV and 3.61 mV, respectively. Both isthmuses were successfully ablated only with irrigated RF applications or epicardial RF delivery from the coronary sinus during a subsequent procedure.

Other Atrial Arrhythmias

AFL had been previously ablated in 4 patients (patients 2, 4, 10, and 11). During the LAMRT procedure, sustained AFL was induced in 6 patients (patients 9, 16, 19, 22, 24, and 28). During follow-up, AFL occurred in patient 25. AFL was successfully ablated in those 7 patients. Also, right atrial macroreentrant tachycardia and atrio-ventricular nodal reentry tachycardia were induced in patient 6. Both arrhythmias were successfully ablated.

Complications and Follow-Up

Groin hematomas occurred in 2 patients. The procedure time was 384±145 minutes, with fluoroscopic time of 18.4±8.8 minutes. Patient 2, who had stable sinus rhythm, died from progressive heart failure 1 month later. Patients with MVS and
patients without MVS were followed up 1 to 27 months (median, 14 months) and 1 to 31 months (median, 15 months), respectively. Twenty-five patients with validation of the isthmus block had no atrial arrhythmia recurrences, except 5 patients (patients 4, 5, 19, 20, and 25) with only atrial fibrillation. In 3 patients without identified reentry circuit, 2 patients had LAMRT recurrence and 1 patient developed chronic atrial fibrillation.

Discussion

Anatomic Barriers for LAMRT

A previous study described ESAs or LDPs as anatomic barriers for LAMRT. In 26 patients or during sinus rhythm in 2 patients. Such acquired anatomic barriers confirmed the previous studies that atrial fibrosis could be found in patients with rheumatic and nonrheumatic heart disease, such as hypertrophic or dilated cardiomyopathy and coronary artery disease, and even in aging without structural heart disease. However, the ESAs or LDPs were variably located in those patients, even in patients with the same disease. Our findings suggest that the atrial fibrosis may be related not only to mechanic overload but also to some unknown atrial myopathy.

Isthmus Characteristics and RF Ablation

In 23 patients with identified reentry circuits during tachycardia, the reentrant circuit presented either as dual loop in 21 patients with 31 tachycardias or as single loop in 10
patients with 11 tachycardias, and propagated through a protected isthmus. The isthmus was located between 2 anatomic barriers, which consisted either of the natural MA or PV or of acquired ESAs or LDPs. Atrial activation at the targeted sites occurred during diastole in 36 and during systole in 6, which was caused by these electrograms’ being recorded from the entrance or exit of the isthmus (Figure 8B). This finding was similar to the study of Nakagawa et al in right atrial macroreentrant tachycardia.

The isthmuses were relatively narrow, with low amplitude in most patients. Such isthmuses were amenable to focal or short RF linear lesions. Our data showed that conventional RF applications could terminate LAMRT and produce conduction block, except in case of the isthmuses between a posterior ESA and the lateral MA in patients 1 and 4. Irrigated catheter ablation was used to block the isthmus permanently in 1 patient; in the other patient, the isthmus conduction block was permanently achieved by epicardial RF delivery from the coronary sinus. Tachycardia transformation during RF delivery was noted in 4 patients. In all patients, absence of atrial activity within the ablated isthmus was noted, suggesting isthmus block. Remapping of the new tachycardia showed that an isthmus,
which had been a bystander in the previous tachycardia, had become a critical part in the new circuit.

In 2 patients with mapping during sinus rhythm, the isthmus was identified in the anterior wall and successfully ablated. This may provide an alternative strategy for catheter ablation of LAMRT by electroanatomic mapping.

Atrial Tachycardia Recurrence in Patients With LAMRT Without Block

Our experience suggests that conduction block of all isthmuses can prevent atrial tachycardia recurrences. This is demonstrated by the following: (1) The same LAMRT only recurred in 3 patients without demonstrated conduction block across the

**Figure 5.** Illustration of a single-loop LAMRT circuit in patient 19. A, Map shows a protected isthmus between an anterior ESA and the MA. RF pulses (brown tags) terminated the LAMRT. B, Remap shows validation of conduction block across the isthmus during sinus rhythm. Note that the left atrial activation propagates counterclockwise around the MA. Blue tags indicate double potentials along the ablation line. C, Fluoroscopic LAO and RAO views (left panel) of the mapping catheter position, a 20-polar catheter inside the coronary sinus (CS) with distal electrode near the anterior MA, and a catheter at the His bundle region in patient 19. In the right panel, the atrial activation sequence showing only counterclockwise activation in the CS and map catheters during LAMRT is the same as during sinus rhythm, indicating clockwise conduction block across the isthmus (also illustrated in Figure 5B). Abbreviations as indicated in Figure 1. LAO and RAO indicate left and right anterior oblique, respectively.
tachycardia isthmus, and (2) AFL was not observed in any of the 10 patients with documented block across the cavotricuspid isthmus, as opposed to being observed in 1 out of 18 patients without documented block across the cavotricuspid isthmus.

Study Limitations
This study has some limitations. Because of the availability of 1 single transeptal access, (1) the assessment of conduction block across isthmuses far from MA was not performed by
pacing the site close to the isthmus but by the absence of atrial activity, and (2) functional conduction block was not distinguishable from fixed conduction block. However, the lack of LAMRT recurrence during follow-up seems to confirm that permanent conduction block had been achieved.

Conclusions
Using electroanatomic mapping, the reentry circuit of LAMRT can be identified in 89% patients. It presents as dual loop in 74% tachycardias and single loop in 26% tachycardias, with a protected isthmus between 2 anatomic barriers. These isthmuses are relatively narrow with low amplitude and are amenable to RF application in most patients. Conduction block across the isthmus can prevent atrial tachycardia recurrence.

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References

Figure 8. Illustration of the complex substrate of a dual-loop LAMRT in patient 18. A, Dual-loop LAMRT with a protected isthmus between the left pulmonary venous ostia (LIPV, LSPV) and a posterior ESA. Note that a collision of 2 activation wavefronts occurred at the area (marked with parallel lines) between the posterior ESA and the right inferior pulmonary venous ostium (RIPV), indicating bystander activation of the isthmus between the posterior and roof ESAs. Brown tags indicate RF applications. B, Tachycardia cycle length suddenly prolonged from 320 to 510 ms during the second RF application connecting the posterior ESA and LIPV, indicating a different LAMRT. C, Remap shows the slow LAMRT with a protected long isthmus between the posterior ESA and a roof ESA, which was activated as a bystander during the previous LAMRT. A single RF application terminated tachycardia. Note the absence of electrical activity in the isthmus of the previous tachycardia, which indicates conduction block. D, Sinus map shows absence of atrial activity in the former isthmuses after RF delivery. Abbreviations as indicated in Figure 2 and Figure 4.
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