Mapping and Ablation of Left Atrial Flutters

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Background—Typical right atrial isthmus–dependent flutters have been described in detail, but very little is known about left atrial (LA) flutters.

Methods and Results—We performed conventional and 3D mapping of the LA for 22 patients with atypical flutters. Complete maps in 17 patients demonstrated macroreentrant circuits (n=15) with 1 to 3 loops rotating around the mitral annulus, the pulmonary veins, and a zone of block or a silent area. In 2 patients, a small reentry circuit with a zone of markedly slow conduction was identified. Linear ablation performed across the most accessible part of the circuit cured 16 patients (73%) with a follow-up of 15±7 months.

Conclusions—LA reentrant tachycardias are related to individually varying circuits and are amenable to mapping guided radiofrequency ablation. (Circulation. 2000;101:2928-2934.)

Key Words: atrial flutter ■ mapping ■ ablation

Macreorentrant arrhythmias designated as flutters are divided into typical flutter (with counterclockwise or clockwise pericitricuspidian rotation) and atypical flutters. Pericitricuspid flutters have been studied extensively with conventional or 3D computerized mapping, and their ablation in the cavitricuspid isthmus is remarkably effective and safe.1 A few cases of atypical flutters have been reported, usually after surgical incisions of the right atrium (RA).2 Few data are available about left atrial (LA) flutters.3 This study describes the electrophysiological characteristics and results of ablation in a series of 22 patients presenting with spontaneous LA flutter.

Methods

Patients

Between March 1997 and March 1999, 22 patients (7 women, 15 men, 60±14 years old) were studied. They had been suffering from persistent atrial flutter for 5±7 years despite the use of 4±1 ineffective antiarrhythmic drugs. Amiodarone failed in 18 and was interrupted because of side effects in 2. Seventeen of the 22 (77%) had structural heart disease, including 3 who had undergone mitral valve replacement or repair (Table 1). Two patients had common flutter ablation before being included in this study. In all patients, the surface ECG showed a regular monomorphic persistent atrial arrhythmia with a P wave predominantly positive in lead V1, and distinct in the limb leads from the pattern described for pericitricuspidian flutters (Figure 1). In 4 patients, however, the surface ECG morphology was compatible with a common counterclockwise RA flutter.

Oral informed consent was obtained in all cases after discussion of the risks and benefits of the procedure. Patients had been under effective oral anticoagulants for ≥1 month before the ablation, and transthoracic and transesophageal echocardiography were performed to rule out LA thrombi. Oral anticoagulants were stopped at admission and replaced by subcutaneous heparin to maintain a partial thromboplastin time of 2 to 3 times the control value. This was stopped 6 to 8 hours before ablation for possible transseptal catheterization.

Electrophysiological Study

All antiarrhythmic drugs except amiodarone were discontinued ≥3 to 4 days before the study. After 6 hours of fasting, 2 to 3 multipolar catheters (Cordis-Webster and Bard or ELA Medical) were used to record bipolar electrograms filtered at 30 to 500 Hz and amplified at 0.1 mV/cm on a PPG Midas polygraph with a paper speed of 100 mm/s. An LA flutter was defined as an atrial arrhythmia with a regular and monomorphic ECG pattern demonstrated by intracardiac mapping to be due to a reentrant circuit in the LA.

The first step was to exclude an RA atypical flutter circuit based on ≥1 of the following 3 RA mapping criteria: (1) RA activation time as determined by sequential conventional mapping (with ≥8 evenly distributed points) accounting for <50% of the arrhythmia cycle length; (2) pacing interval (PP) in the RA longer than the cycle length by ≥40 ms in ≥3 different points in the RA, including the cavitricuspid isthmus and RA free wall but excluding the septum and coronary sinus; and (3) spontaneous variations of >100 ms in the RA with concomitant variations of <20 ms in the LA.

The second step was direct LA mapping to demonstrate the reentrant circuit during tachycardia with a 3D electroanatomic mapping system (Biosense Cordis-Webster). A transseptal puncture (Brockenbrough needle and Daig sheath) was required in 20 cases, whereas 2 patients had a patent foramen ovale. The mapping system has been described previously.4 Briefly, it includes a location pad, a processor (Carto), and a monitor and workstation (Silicon graphics), as well as 2 sensor-equipped catheters. One of these sensor-equipped catheters was used as a reference and placed in a stable position (RA appendage or coronary sinus in the event of spontaneous RA flutter).
variations) to record a stable high-amplitude atrial signal. For the last 4 patients, an external reference was placed on the patient’s back, with a diagnostic quadripolar catheter used as the electrophysiological reference. A sensor-equipped rove catheter was used for mapping and identification of anatomic landmarks. Local activation time was automatically determined by the computer, according to the maximum negative slope $dV/dt$ of bipolar electrograms filtered at 30 to 400 Hz. After sequential construction of the map, all points were manually checked and corrected if necessary. Bipolar voltage maps were analyzed in relation to activation maps. Mapping was complete when a sufficient density (>80 points) was achieved to allow understanding of the LA circuit. Some maps could not be completed because of premature termination of tachycardia, irregular varying cycle length (despite a monomorphic ECG aspect of the flutter), or change to another morphology and/or cycle length and/or atrial fibrillation (AF) or technical dysfunction. In these patients, conventional mapping, including entrainment maneuvers and PPI analysis, was performed after reinduction of the clinical tachycardia. Pacing sites with a PPI not exceeding the cycle length by $\pm 20$ ms were considered to be part of the circuit. Pacing for entrainment resulted in transformation to another morphology or to AF in 3 of 6 initial patients. Subsequently, it was not performed systematically.

**Definitions**

Electrically silent areas were defined as no recordable activity or amplitude $<0.05$ mV (which is the baseline noise in the Biosense system), accompanied by inability to capture the atria from these areas at 20 mA. Such areas were tagged as “scar” and therefore appear in gray on the 3D maps (Figures 2, 3, and 4). With conventional mapping, slow conduction was arbitrarily defined as complex and fractionated activity of long duration ($>50$ ms). A zone of block was defined by double potentials separated by an isoelectric interval of $>50$ ms. With the Biosense system, isochronal crowding indicating a conduction velocity of $<0.033$ cm/ms (slower than $0.05$ cm/ms)$^4$ was considered a zone of slow conduction, whereas a collision of 2 wave fronts traveling in different directions separated

### TABLE 1. Clinical and Electrophysiological Data

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<th>Patient</th>
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</table>

Mean $\pm$ SD 60 $\pm$ 14 135 $\pm$ 33

**Median** 63 128

SHD indicates structural heart disease; M, mitral; MR, mitral regurgitation; MS, mitral stenosis; DCM, dilated cardiomyopathy; ICM, ischemic cardiomyopathy; HCM, hypertrophic cardiomyopathy; VR, valve replacement; Ao, aortic; sr, small reentry; ZB, zone of block; SR, sinus rhythm; FL, flutter; RPV, right PV; LPV, left PV; SA, silent area; CW, clockwise; CCW, counterclockwise; OK, PPI $\leq$ cycle length $+20$ ms, and NA, not available.

![Figure 1. Twelve-lead ECGs of 4 patients (1, 7, 9, and 13). All are “atypical” in frontal limbs, and P waves are predominantly positive in lead V1. Note very low amplitude of atrial activity recorded in patient 9. Corresponding Biosense maps for patients 9, 7, and 13 are shown in Figures 2, 3, and 4, respectively.](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.84.14.2929)
temporally by 50 ms was defined as a region of local block (Figures 2, 3, and 4).

A macroreentrant circuit was defined as a circuit propagating in a large part of the cavity with a minimum diameter of >3 cm. Conversely, small reentrant circuits were defined as circuits with a diameter of <3 cm along with activation covering the entire cycle length and centrifugal activation of the remaining LA (Figure 5).

Ablation

After each successful mapping procedure, radiofrequency (RF) ablation was performed by targeting either the narrowest and/or the most accessible part of the circuit (allowing the best electrode-tissue contact along the desired line). The ablation line was chosen to transect an area critical for the circuit and connected 2 anatomic areas of block or an electrically silent area to an anatomic zone of block (pulmonary vein [PV], mitral annulus). In patients with incomplete maps, the ablation was guided by conventional mapping targeting a critical isthmus and/or a zone of slow conduction shown to be part of the circuit by pacing maneuvers. After ablation, the catheter was used to retrace the same line (during sinus rhythm), showing either the absence of electrograms or a complete line of block demonstrated by parallel double potentials recorded all along the line. Finally, high-rate pacing with a stimulus strength of 20 mA was performed on the RA and/or the LA after successful termination to assess flutter or fibrillation inducibility. Any arrhythmia lasting ≥3 minutes was considered sustained. The procedural end point was defined as interruption and noninducibility of the targeted flutter morphology.

A Stockert Cordis generator was used to deliver RF for 60 seconds at each site with a target temperature of 50°C to 55°C and a power limit of 70 W, except when there was an impedance rise. The catheter was progressively pulled back during RF delivery to produce coalescent lesions from one part to the other of the predetermined line.

An irrigated-tip catheter was used for the initial cases in which the flutter was not interrupted because of resistant gaps in the ablation line and systematically for the last 15 patients with a protocol previously shown to be safe. Heparin was administered after transseptal puncture to maintain a partial thromboplastin time of 2 to 3 times the control value.

Postablation Management

Patients were maintained on anticoagulation and monitored by telemetry and ambulatory ECG recordings. Transthoracic echocardiography was performed 1 to 3 days after the ablation. A previously ineffective antiarrhythmic drug was used in case of AF during days 6 to 10 of in-hospital telemetry surveillance after ablation. Patients were then discharged under oral anticoagulation for 6 months to 1 year. Late follow-up consisted of visits to the hospital or the referring physician and ambulatory recordings. The outcome of the procedure was considered a success in case of persistent sinus rhythm or success with drug if previously ineffective antiarrhythmic drugs were required for persistent flutter or associated AF. A second procedure was performed in case of a recurrence of organized atrial arrhythmia.

Results

Mapping Results

The results of mapping in the RA are shown in Table 2. The RA activation time accounted for <50% of the cycle length.
in all but 3 patients in whom spontaneous local variations or local PPI interval ruled out an RA arrhythmia.

In 17 patients, complete maps were obtained for 18 LA flutter morphologies. Varying macroreentrant circuits were identified in 15 patients and a small reentry circuit in 2 (Figure 6). The mean LA volume was 134±33 mL (median, 127 mL) versus 34±10 mL in normal LA in our laboratory. A mean of 112±28 points covering 95±3% of the arrhythmia cycle length were recorded. In 9 patients, LA pacing showed a PPI not exceeding the cycle length by ≥20 ms. In 5 patients, a complete map could not be achieved.

Electrically Silent Areas and/or Zone of Block
Eleven electrically silent areas were noted in 8 patients with complete maps (Figure 6). They were located in the posterior LA (n=5) and were associated with an anterior silent area in 2 patients. The posterior silent area was of varying dimensions, extending to the roof in 3 or to the septum in 1. An isolated silent area in the roof was observed in 2 patients and associated with an anterior area in 1. Three patients had 2 different silent areas: roof and anterior LA (n=1) and anterior and posterior LA (n=2). In 3 patients with incomplete maps, 2 posterior and 1 anterior silent areas were noted.

Thirty-two zones of block (≥1 per patient) were identified in various locations, as shown in Figure 7. These zones of block were anchored at the ostium of the left PV (35%), right PV (15%), or base of the appendage (22%). They coincided with regions of significant voltage gradient (0.1±0.7 versus 1.6±0.6 mV) in 11 of 17 patients (65%) (Figure 5).

Characteristics of Circuits
The complete map results are summarized in Table 1.

Single-Loop Reentrant Circuits
A single-loop reentrant circuit was identified in 13 patients (Figure 2). It was rotating around the mitral annulus in 7 (counterclockwise rotation [left lateral view] in 1, clockwise

**Figure 5.** 3D Biosense activation (left) and voltage (right) map of LA acquired in patient 5 during clinical arrhythmia. This left posterior view of LA (left) shows small reentrant circuit on lateral and inferior aspect of mitral annulus (M). Spontaneous line of block is visible at base of LA appendage descending toward mitral annulus. It matched perfectly with significant voltage gradient. Mean amplitude of 5 points (stars) in A was 0.1±0.04, vs 0.9±0.4 in B. Most inferior aspect of this spontaneous line is incomplete and is responsible for very slow conduction area of circuit, exhibiting fractionated and complex potentials. PPI analysis was poor in entire LA except on represented circuit. RF ablation of slow conduction area resulted in flutter termination and in noninducibility. CI indicates cycle length.

**Figure 6.** Schematic of locations of silent areas and their percentage with circuits encountered in LA. See Table 1 for relative frequency. Note that nearly half of 11 silent areas were posteriorly located. Abbreviations as in previous figures.
in 5, both in 1) and was bounded by a silent area in 4 (posterior, n=1; anterior plus posterior, n=2; roof, n=1).

In 2 patients (patients 12 and 16), the core center of the circuit was a posterior or a roof silent area bounded by a zone of block anchored in the left PV ostium (n=1) or at the left appendage base (n=1).

Multiple-Loop Circuits
One patient had a figure-8 reentrant circuit with 2 loops rotating in opposite directions: one around a posterior silent area and the other around a vertical zone of block anchored in the right PVs (Figure 3). The common channel was in the posterior LA, with the activation proceeding upward, dividing itself into 2 waves in the LA roof, one going downward in the anterior LA around the right PVs and the other proceeding posteriorly around the silent area. In another patient, 3 loops were coexisting during the same flutter (Figure 4). One was rotating around an anterior silent area, with another one around a posterior silent area. The third loop was ascending in the anterior LA, propagating downward in the lateral LA through a channel common for the 3 circuits between the base of the appendage and the left PVs.

Small Reentry
Patient 5 had a wide vertical zone of block extending from the lower part of the lateral LA to the roof through the left PVs and then to the LA appendage base (Figure 5). A gap at the base of this line of block constituted a critical zone of slow conduction which permitted the maintenance of a small reentrant circuit in the lateral LA. The entire cycle length was recorded in this small area, with the (successful) ablation site displaying a continuous activity of 206 ms for a cycle length of 286 ms. The rest of the LA was passively activated, as well as the RA. Another small reentrant circuit was identified in patient 22 in the left septum, rotating around the right superior PV and fossa ovalis, with a slow-conduction area at the junction between right superior PV and LA roof.

Ablation Results
One, 2, or 3 sessions were required in 14, 7, and 1 patients, respectively (mean, 1.4±0.5; median, 1). The mean cumulative procedure and fluoroscopy durations were 339±113 and 95±42 minutes per patient.

Macroreentrant Circuits
A total of 39±33 minutes of RF application resulted in the interruption of the 18 mapped flutter morphologies in 17 patients. The 7 perimitral flutter circuits were ablated by a line connecting the mitral annulus to the left superior PV (n=2), the right superior PV (n=2), or a silent area located anteriorly (n=2) or in the LA roof (n=1). The circuits propagating around a silent area were interrupted when the silent area was connected to either the right or left superior PV. The peri-PV circuits were ablated by joining of the PV to the mitral annulus (n=3) or to the contralateral superior PV across the LA roof (n=1).

The double-loop figure-8 circuit (patient 7) was ablated by connection of the posterior silent area to the right inferior PV (Figure 3). The 3-loop reentrant circuit (patient 13) was ablated targeting the common channel in the roof from the left superior to the right superior PV ostium (Figure 4).

For patient 11, with a perimitral circuit, the right superior PV mitral line could not be completed. As a result, cycle length increased from 280 to 450 ms without interruption.

A vertical zone of block anchored in the right (n=3) or left (n=1) PVs was the center of the circuit in 4 patients.

**TABLE 2. RA Mapping Data During LA Flutter**

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<tr>
<th>Patient</th>
<th>Flutter CL, ms</th>
<th>RA Activation Time, ms</th>
<th>RA Activation/CL Ratio, %</th>
<th>RA PPI, ms</th>
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The RA activation time/cycle length (CL) ratio demonstrates that the RA is usually activated within 50% of the cycle length except in a few patients in whom some bystander slow conduction areas prolong the activation time.

**Figure 7.** Four views of LA shell. Double white lines are spontaneous zones of block noted in our patients and their respective frequencies. Abbreviations as in previous figures.
Another line joining the mitral annulus to the left PV was again incomplete and did not modify the arrhythmia. For patient 15, the ablation line joining the mitral annulus to the left PV resulted in interruption of the clinical arrhythmia, which, however, remained inducible despite multiple attempts to complete the line of block.

**Small Reentrant Circuits**
Both small reentrant circuits were successfully ablated by local discrete lesions delivered on the slow conduction area, with flutter interruption obtained within 1 minute of RF delivery.

**Incomplete Maps**
In the 5 patients without complete Biosense maps, the ablation was guided by conventional mapping data for 2 patients. In patient 10, 2 different morphologies of flutter were ablated, the first one by connection of a zone of double potentials present in the roof to the right superior PV and the second by connection of the right superior PV to an area of complex and fractionated electrograms of the fossa ovalis. In patient 4, flutter was interrupted with an ablation line joining the right superior PV to the mitral annulus. In 3 other patients (patients 2, 3, and 17), multiple flutter morphologies were observed, and we therefore performed the 3 LA ablation lines used for AF ablation with a successful outcome in 1, an increase of the cycle length without conversion to sinus rhythm in a second (patient 17), and no change in the third.

**Postablation Flutters**
Another flutter morphology not previously documented occurred after the initial ablation of a perimitrinal circuit in 4 patients. Mapping showed that the initial ablation line between PV and mitral annulus was complete and that the new circuit was rotating around the opposite PV. They were successfully ablated by a line connecting the left superior to the right superior PV (across the roof of the LA).

**Safety**
The first patient had a reversible ischemic neurological deficit at the end of the procedure. No other significant side effects were observed.

**Outcome**

**Acute**
Twenty patients were in sinus rhythm after ablation, but the clinical arrhythmia persisted in 2. Despite high-rate pacing, 10 of them were noninducible; atypical sustained flutter was inducible in 5, atypical nonsustained in 3, and AF in 2.

**Midterm Follow-Up**
With a mean follow-up of 15±7 months, 16 patients (73%) were successfully treated, with no antiarrhythmic drugs in 15; 1 was taking amiodarone for paroxysmal AF. LA flutter persisted in 2 and reoccurred in 3 patients 1 day to 2 months after the last ablation. One woman had chronic AF.

**Discussion**
This study demonstrates the varying characteristics of reentrant tachycardia in the human LA (LA flutter), occurring mainly in patients with underlying structural heart disease and the feasibility and safety of curative catheter ablation.

**Diagnosis of LA Flutters**
The diagnosis of LA flutter was established by comprehensive mapping, including in 17 patients with a 3D electroanatomic system, and was confirmed by the results of catheter ablation achieving sinus rhythm for 20 patients or prolonging the tachycardia cycle length in 2.

Various circuits were demonstrated. In most cases, the arrhythmia rotated around the mitral annulus, a zone of block including the PVs, or a silent area. Lines of block and silent areas also acted as lateral barriers, probably allowing the stabilization of the circuit and preventing short circuiting. In a few patients, the circuits were more complex, with 2 or 3 loops rotating concomitantly. There was no marked area of slow conduction in these macroreentrant circuits, in contrast to the 2 cases of small reentrant circuit, in which a zone of very slow conduction was found, accounting for \( >\frac{2}{3} \) of the cycle length. Slow-conduction areas were frequently reported in animal models, usually being the center of the circuit, either alone or in association with anatomic obstacles.

In contrast, a silent area has not previously been reported clinically. It seems to be a distinctive and relatively common feature of human LA flutter, present in 50% of the patients in this series. This is probably related to severe atrial fibrosis (and atrial myocardial cell modification/disappearance), a common phenomenon in patients with structural heart disease. It may also be possible that in those patients who had suffered from atrial arrhythmias for many years, histological changes have occurred as a result of atrial arrhythmia.

**Review of the Literature**
Various studies have described atypical RA reentrant arrhythmias, frequently related to surgical incisions. A flutter circuit involving the coronary sinus has also been reported with conventional mapping. However, there is no previous study on LA arrhythmia circuits and on their mechanisms in humans. Schools et al and Uno et al demonstrated some right atypical circuits with single- or double-loop reentry in the canine sterile pericarditis model. The LA was rarely involved.

Surgical lines of block placed in the LA (to simulate unilateral or bilateral lung transplantation) provided an electrophysiological substrate for LA flutter in a dog model. Again, the circuit was large enough to sustain reentry in the absence of marked slow conduction.

In a dog model, Schuessler et al also demonstrated atrial flutters, usually rotating around anatomic and functional zones of block. However, in dogs with enlarged and/or hypertrophied LA, most of the circuits were located in the RA. Pure LA circuits were rarely found, usually rotating around the PV (4 dogs in Schuessler’s experience). Therefore, both human and animal data suggest that the RA is more frequently involved in flutters than the LA. However, the exact incidence of LA flutters in humans is currently unknown.
Catheter Ablation
The ablation procedure duration and x-ray exposure involving the mapping part were relatively long, but the long-term results clearly indicate the feasibility of successful ablation. There was a 73% short-term success rate for patients resistant to antiarrhythmic drugs, and in 70%, sinus rhythm persisted with a follow-up of >1 year without drug. This success rate is smaller than the one achieved for common flutter ablation but can probably be improved. This is likely to be due to the greater thickness of the LA and to the longer ablation line required.

It is also interesting to note that there was a limited incidence of AF after ablation despite the underlying structural heart disease. This may be related to the presence of silent areas and lines of block (spontaneous and created by RF), which could reduce the electrically active atrial mass below a critical threshold for fibrillation.

Limitations
Several limitations in this study were inherent to the mapping system used.

The arrhythmia must be perfectly stable, with cycle length variations of <10% to provide exploitable maps.

It is impossible to differentiate very slow conduction from complete block in the present clinical conditions. Isochronal mapping is limited by interpolation and by the selection of local activation time. Moreover, the absence of systematic pacing and entrainment because of the risk of inducing AF is a limitation in this study.

Conclusions
LA flutters sustained by highly variable macroreentrant or small-reentrant circuits can occur in humans, usually with structural heart disease affecting the LA, mainly mitral disease. These arrhythmias require detailed mapping to guide successful ablation.

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Mapping and Ablation of Left Atrial Flutters
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