Background and objective. We are reporting the characteristics of 9 patients with left atrial macroreentrant tachycardia, an arrhythmia not well studied in man.

Patients and method. Mean age was 60 years and 7 were men. Tachycardia was spontaneous in 6 and induced in 3. Two had no heart disease, 2 sick sinus syndrome, 3 aortic prosthesis, 2 hypertension, 1 cardiomyopathy and 1 chronic bronchitis. Simultaneous recordings from right atrial, coronary sinus and right pulmonary artery were obtained at baseline and with atrial pacing. Macroreentrant tachycardia was diagnosed when entrainment with fusion was documented.

Results. Cycle length was 230-440 ms (287 ± 67). The ECG showed atypical flutter in 3 patients and P waves with flat baseline in 6. Coronary sinus activation was distal to proximal in 7. Right atrial activation was circular in 3 with previous typical flutter ablation. Entrainment from the right atrium produced long return cycles in the right atrial recordings, but equal to basal tachycardic cycle in coronary sinus recordings. Entrainment from the coronary sinus produced local return cycles equal to basal cycle in 8 and prolonged in 1. After stimulation, 4 recovered sinus rhythm, 4 went to atrial fibrillation and 1 had no change. After a follow-up of 9-19 months 5 remain in sinus rhythm treated with antiarrhythmic drugs and/or atrial pacing.

Conclusions. Left atrial macroreentrant tachycardia is associated with organic heart disease. The ECG most frequent pattern tends to show P waves with flat baseline at a relatively slow rate. Most circuits turn clockwise in anterior view. Atrial stimulation is not very effective for cardioversion to sinus rhythm. The prognosis of long term rhythm is uncertain.

Key words: Atrial flutter. Arrhythmia. Electrophysiology. Mapping. Reentry.
Flutter has been characterized as a macroreentrant tachycardia (MRT) around anatomic and functional obstacles in the right atrium (RA) with a counterclockwise circuit (oblique left anterior view). Reverse flutter (clockwise) has been described, which uses the same circuit with activation in the opposite direction.\(^1\)\(^-\)\(^4\) Other right atrial MRT are caused by surgical scars, generally after surgery for congenital heart disease.\(^5\)\(^,\)\(^6\) Tachycardia with presumably functional obstacles (areas of low-voltage potentials) has been described in patients without previous surgery.\(^7\) The localization and description of these MRT circuits have made it possible to perform ablation of the critical isthmuses, thus permanently interrupting the mechanism of the arrhythmia.

MRTs of the left atrium (LA) have not been studied systematically until very recently. The only publication on the topic is that of Jaïs et al.\(^8\) who used cardiac electrophysiological studies with electromagnetic localization of the catheter position in the LA to describe various types of circuit. These circuits are generally several centimeters in diameter and turn around anatomic structures and low-voltage zones, possibly related with scar tissue. These left atrial MRTs can be interrupted, although the presence of large low-voltage zones could denote the presence of serious, possibly irreversible, myocardial damage, and the prognosis for the long-term recovery of contractile function is dubious.

Given the risk of left intra-atrial electrophysiological studies, it would be useful to define methods for diagnosing left atrial MRT without having to resort to transseptal puncture. We report our experience with 9 cases of left atrial MRT diagnosed by electrophysiological studies of the RA, coronary sinus (CS), and right pulmonary artery (RPA) and the study of the fusion patterns and return cycles after transitory entrainment in multiple simultaneous recordings.

**PATIENTS AND METHODS**

**Patients**

We studied 9 patients (7 males) ranging in age from 40 to 81 years (mean ± SD = 60 ± 16) with left atrial MRT that was spontaneous (6 cases) or induced in the electrophysiological study (3 cases). Seven of the 9 patients (77.7%) had cardiovascular disease, including 3 with previous aortic valve surgery, 2 sinus node dysfunction, 2 hypertension, 1 chronic obstructive pulmonary disease, and 1 dilated cardiomyopathy of unclear origin. Two patients showed no appreciable structural heart disease.

In 6 patients the electrocardiogram (ECG) revealed regular atrial tachycardia with P waves separated by an isoelectric baseline (Figure 1) and in the other 3 pa-
tients, atypical flutter defined as a continuous ondulation without an isoelectric baseline (Figure 2). In 4 of the 6 patients analyzed in sinus rhythm, signs of advanced interatrial conduction disorder were observed, with a pattern of blockade of the Bachmann bundle in the surface ECG (Figure 3). Seven of the 9 patients had other documented atrial arrhythmias (Table 1): 2 had a history of atrial fibrillation, 2 right atrial MRT, 2 common flutter, and one focal atrial tachycardia of the septal RA.

**Electrophysiological study**

The electrophysiological study was made in fasting conditions and mild sedation with midazolam after obtaining informed consent from the patient.
Antiarrhythmic drugs were discontinued 24 to 36 h before the study, except in 3 patients who received amiodarone (1 g per week), 2 digoxin, and 1 who received beta blockers.

Intra-atrial electrophysiological study

The cardiac electrophysiological study was made using an electrode catheter with 12 pairs of poles (pole separation 2 mm and pair separation 8 mm, Bard Electrophysiology Inc., MA, USA) as the stable reference in the RA. The electrode catheter was inserted through the femoral vein and placed against the anterolateral and septal walls (Figure 4). LA activity generally was recorded with a decapolar electrode catheter (pole separation 2 mm and pair separation 5 mm) in the coronary sinus and an electrode catheter with deflectable tip in the RPA. Six to 10 simultaneous recordings were made of the RA and 2 to 5 of the coronary sinus. In 3 patients, electrograms of the cephalad left atrium were recorded from the RPA (Figure 5).

Bipolar recordings (30-500 Hz) were stored on magnetic tape (FM analog recording) or optical disk (digitized at 1 KHz) for later analysis. Measurements were made at a recording speed of 100 mm/s.

Transitory entrainment

Changes in activation during RA or CS stimulation were studied in all the recordings together. Atrial stimulation began at a cycle length (CL) 10 ms shorter than the CL of the MRT to avoid modifying circuit

### TABLE 1. Clinical and electrophysiological characteristics of 9 patients with left atrial macroreentrant tachycardia

<table>
<thead>
<tr>
<th>Patients</th>
<th>Age (years)</th>
<th>Organic disease</th>
<th>Cycle length</th>
<th>Anterior/ septal RA activation</th>
<th>LAMRT</th>
<th>Coronary sinus activation</th>
<th>Fusion</th>
<th>Other arrhythmias</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>77</td>
<td>Sinus node dysfunction</td>
<td>255</td>
<td>Simultaneous/ ascending</td>
<td>Induced</td>
<td>Distal to proximal</td>
<td>Constant</td>
<td>Atrial fibrillation</td>
<td>Atenolol</td>
</tr>
<tr>
<td>2</td>
<td>78</td>
<td>Aortic valve replacement</td>
<td>310</td>
<td>Simultaneous/ simultaneous</td>
<td>Clinical manifestations</td>
<td>Distal to proximal</td>
<td>Progressive</td>
<td>No</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>3</td>
<td>58</td>
<td>AHT</td>
<td>330</td>
<td>Ascending</td>
<td>Clinical manifestations</td>
<td>Distal to proximal</td>
<td>Progressive</td>
<td>No</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>4</td>
<td>65</td>
<td>DCM sinus node dysfunction</td>
<td>250</td>
<td>Simultaneous/ simultaneous</td>
<td>Clinical manifestations</td>
<td>Distal to proximal</td>
<td>Progressive</td>
<td>Free wall RAMRT</td>
<td>Digoxin</td>
</tr>
<tr>
<td>5</td>
<td>81</td>
<td>AHT, COPD</td>
<td>235</td>
<td>Descending</td>
<td>Clinical manifestations</td>
<td>Distal to proximal</td>
<td>Progressive</td>
<td>No</td>
<td>Atrial flutter</td>
</tr>
<tr>
<td>6</td>
<td>40</td>
<td>Aortic valve replacement</td>
<td>280</td>
<td>Simultaneous/ simultaneous</td>
<td>Clinical manifestations</td>
<td>Distal to proximal</td>
<td>Progressive</td>
<td>No</td>
<td>Atrial flutter</td>
</tr>
<tr>
<td>7</td>
<td>40</td>
<td>No</td>
<td>440</td>
<td>Descending</td>
<td>Induced</td>
<td>Distal to proximal</td>
<td>Constant</td>
<td>RAMRT</td>
<td>No</td>
</tr>
<tr>
<td>8</td>
<td>55</td>
<td>No</td>
<td>250</td>
<td>Descending</td>
<td>Induced</td>
<td>Distal to proximal</td>
<td>Progressive</td>
<td>Atrial flutter</td>
<td>No</td>
</tr>
<tr>
<td>9</td>
<td>49</td>
<td>Aortic valve replacement</td>
<td>230</td>
<td>Ascending</td>
<td>Clinical manifestations</td>
<td>Distal to proximal</td>
<td>Constant</td>
<td>Focal AT</td>
<td>Amiodarone</td>
</tr>
</tbody>
</table>

AHT indicates arterial hypertension; AT, atrial tachycardia; COPD, chronic obstructive pulmonary disease; CS, coronary sinus; DCM, dilated cardiomyopathy of origin unknown; LAMRT, left atrial macroreentrant tachycardia; RA, right atrium; RAMRT, right atrial macroreentrant tachycardia.
properties, such as induction of delayed conduction dependent on increased frequency. In the recordings made at a distance from the stimulation point, the return cycle was measured after the last entrained electrogram.

**Definition of terms**

The diagnosis of left atrial MRT required demonstration of entrainment with progressive and/or constant fusion and return cycles (20 ms difference) equal to the baseline cycle of the tachycardia in the CS and/or RPA recordings. A diagram of the correlation between the changes in activation and the recordings is shown in figure 6 with the aid of a helicoidal representation of reentry. The existence of entrainment with constant and/or progressive fusion indicates the presence of a large circuit (MRT) because small circuits (microreentry) can be entrained but do not exhibit fusion. The return cycle inside and outside the MRT circuit is also shown in Figure 6.

Transitory entrainment was considered to be established when all electrograms in 2 trains of stimulation increased in frequency to that of stimulation in all the recording points, and the tachycardia immediately returned to the baseline CL when stimulation stopped, in at least one recording, even if not at the point of stimulation. Constant fusion was considered to be present when a partial change occurred in the sequence of electrograms during entrainment, with recovery of the baseli-
ne sequence when stimulation ended. No attempt was made to analyze the possible fusion pattern in the ECG because of the difficulty of analyzing P waves or flutter waves due to interference by QRS complexes and T waves.

Progressive fusion was defined as a change in the degree of fusion upon increasing the frequency of entrainment, with displacement of the point of collision of the activation wavefronts (the reentrant orthodromic and stimulated antidromic wavefronts) near the site of the circuit.

RESULTS

The electrophysiological findings are summarized in Table 1. The cycle length of left atrial MRT in the 9 patients was 230-440 ms (287 ± 67), and 236-440 ms (285 ± 77) in 6 patients not treated with amiodarone. Activation of the septal RA was ascending in 7 patients and simultaneously cephalocaudad in 2 patients. Activation of the anterior face was simultaneously cephalocaudad in 4 cases, descending in 3 and ascending in 2. In 3 patients with previous ablation of the cavotricuspid isthmus (2 for typical flutter and 1 for right atrial TMR dependent on the isthmus), RA activation was descending in the anterior wall and ascending in the septum, simulating typical flutter. The CS activation was distal to proximal in 7 patients, proximal to distal in 1, and simultaneous throughout its length in 1. The recorded RA electrograms covered 100% of the CL in 2 patients with circular activation of the RA and a CL of 250 and 235 ms; in the rest they covered 27-50% of the CL. The combination of the RA, CS, and RPA electrograms covered 48-100% (mean, 80 ± 18%) of the tachycardia cycle.

Entrainment from the anterior and septal RA revealed return cycles 30 ms longer than the baseline CL throughout the RA, while the return cycle was equal to the baseline CL in the CS and/or RPA in all cases (Figure 7). During entrainment, constant fusion was demonstrated in all patients (Figures 7 and 8) and progressive fusion in 6 patients (Figure 9). In 4 patients with progressive fusion, the point of collision of the orthodromic (entrained) and antidromic (stimulated) wavefronts could be identified in the CS. The displacement of the point of collision in the CS with shortening of the stimulation CL was proximal to distal in 3 patients who had distal to proximal activation and RA stimulation, and distal to proximal in 1 patient with...
Fig. 8. Entrainment from the lower right atrium of a left atrial macroreentrant tachycardia (stimulation cycle length 260 ms). The first two recordings show leads I and V1 of the surface ECG. The other recordings show bipolar electrograms of the anterior face (A cephalad to A4) and septal face (PS4 to P cephalad) of the right atrium (RA), and of the proximal to distal coronary sinus (CS1 to CS5). A slight change in the activation of the septal face of the RA and the collision of two activation wavefronts in CS3-CS4 of the coronary sinus (constant fusion) are appreciated. 
A cephalad indicates anterior cephalad RA; A1, upper anterior RA; A2-A3, middle anterior RA; A4, lower anterior RA; CS, coronary sinus; P cephalad, posterior cephalad RA; PS1, upper septal RA; PS2-PS3, middle septal RA; PS4, lower septal RA; RPA, right pulmonary artery; S, stimulus. Recording speed 100 mm/s.

Fig. 9. Entrainment from the distal coronary sinus of a left atrial macroreentrant tachycardia at different stimulation cycle lengths (left panel 220 ms, central panel 190 ms, and right panel 185 ms). The first two recordings show leads I and V1 of the surface ECG. The other recordings show bipolar electrograms of the anterior face (A cephalad to A4) and septal face (PS4 to P cephalad) of the right atrium (RA), proximal to distal coronary sinus (CS1 to CS5), and right pulmonary artery (RPA). The displacement of the zone of collision of stimulated and reentrant wavefronts (progressive fusion) toward the proximal coronary sinus is evident. A cephalad indicates anterior cephalad RA; A1, upper anterior RA; A2-A3, middle anterior RA; A4, lower anterior RA; CS, coronary sinus; P cephalad, posterior cephalad RA; PS1, upper septal RA; PS2-PS3, middle septal RA; PS4, lower septal RA; RPA, right pulmonary artery; S, stimulus. Recording speed 100 mm/s.
proximal to distal activation during distal CS stimulation (Figure 9).

After atrial stimulation, 4 patients went to sinus rhythm, 4 developed atrial fibrillation and 1 remained in left atrial MRT without change. Follow-up data for 9 to 25 months was available for 8 patients. Three remained tachycardic after the study and were treated with frequency control and anticoagulation. Two patients were implanted a DDD pacemaker for sinus node dysfunction. One has not suffered any recurrence in 16 months without antiarrhythmic drugs and the other relapsed at 18 months, but recovered sinus rhythm with oral propafenone. Of 3 patients who received amiodarone (1 g per week), 1 maintained sinus rhythm for 9 months, 1 for 13 months until the patient died in the postoperative period of cardiac valve replacement, and the third relapsed at 19 months and is awaiting a new study or cardioversion.

DISCUSSION

Left atrial MRT has been little studied in human clinical practice. The need for transseptal catheterization makes it difficult to justify a direct cardiac electrophysiological study without a clear therapeutic indication, so in most cases the diagnosis must be established using indirect techniques, such as observation of the response to stimulation (entrainment). Only very recently, Jaïs et al. published a relatively extensive experience with direct cardiac electrophysiological studies of left atrial MRT. They demonstrated that in most cases circuits are large and activation is routed around combinations of natural obstacles, particularly the mitral valve, and obstacles of unknown nature, in the form of lines of conduction block or zones of low-voltage electrical activity that suggest the absence of functional myocardium. These authors used entrainment techniques to confirm the location of circuits that could not be fully delimited by cardiac electrophysiological study. Only summaries have been published of other experiences, which gives us little information about the anatomic and functional details.

The diagnosis of right atrial MRT is easier because recordings of circular activity throughout the entire MRT cycle is very suggestive of macroreentry, as opposed to focal activity. However, if transseptal catheterization is not performed, this finding will not be of much help in left atrial MRT because RA activity covers much of the CL, especially when added conduction disorders force activation to be circular.
Entrainment is necessary to distinguish between reentrant activation and «passive» circular activation 15 because, in the case of a RA circuit, return cycles at this point are always equal to the baseline CL.9,16 This study applied techniques used in the study of right atrial MRT, including typical flutter, to the diagnosis of left atrial MRT. However, entrainment was supported by multiple recordings in both atria, which, as we have demonstrated in the RA,9 make it possible to more easily detect return cycles equal to the baseline CL and signs of fusion than isolated recordings17 because they provide information from within and around the circuit. In the absence of fusion and/or a complete cardiac electrophysiological study of the LA, the mere detection of concealed entrainment, a criterion used by other authors, does not allow MRT to be distinguished from a focal tachycardia with a microreentrant mechanism.18

In Figure 6 a schema illustrates how a stimulated wavefront from outside the circuit captures a point in the circuit and initiates an antidromic wavefront that collides with, and interrupts, the circuit, and then an orthodromic wavefront reinitiates it, according to the classic description of the entrainment.15 In this Figure, the inversion of the direction of activation in the part of the circuit captured antidromically can be seen clearly, as reflected by the inversion in the sequence of electrograms recorded in this zone. The point of return is equal to the baseline CL at the point where the stimulated wavefront captures the circuit, but in order to measure the return cycle in orthodromically entrained recordings, one must await the cycle after the first non-fusion cycle because it is still entrained.

However, a return cycle equal to the MRT cycle can occur outside the circuit, if it is activated orthodromically from the circuit during entrainment at a distance (Figure 6). Consequently, this finding does not serve to locate the circuit precisely, although it allows it to be situated in the LA when the return cycles are long in all RA recordings and equal to the CL of the left atrial MRT. Case 5 is an example of this situation, with a return cycle in the distal CS equal to the MRT cycle when entrainment was from the RA, but prolonged when entrainment was from the distal CS (Figure 10).

It is interesting that activation tended to turn «clockwise» around the mitral valve in our cases and those of Jaïs et al.8 It is likely that the tendency toward ascending activation of the septal RA is due to the emergence of the activation wavefront around the ostium of the CS. The detection of distal to proximal CS activation during flutter is a sign suggestive of the left-side origin of activation and would contribute to the diagnosis of left atrial MRT when RA activation is counterclockwise, as in common flutter, which may suggest typical flutter in the ECG.

The ECG pattern is equivocal because 6 patients had a well-delimited P wave with isoelectric lines and relatively low frequencies, even in the absence of treatment with antiarrhythmic drugs, suggestive of “focal” atrial tachycardia. Only 3 patients presented the pattern of continuous waves (flutter) generally associated with macroreentrant tachycardia. The inscription of the P wave generally coincided with RA recordings, indicating that most of the activity in the LA reentrant circuit was low-voltage and not clearly manifested in the ECG. This concurs with the observations of Schoels et al in an experimental model of pericarditis in the dog.19 where they found this type of situation in the LA reentrant circuits. Therefore, the image of tachycardia «with P waves» in the ECG should not be taken as indicative of focal tachycardia without first performing cardiac electrophysiological studies and entrainment to exclude MRT.

The clinical characteristics of the patients were interesting, suggesting the idea that left atrial MRT is a manifestation of underlying disease of the atrial myocardium. This coincides with the findings of other authors,8,20 who also observed an association with heart valve disease and previous surgery. The low-voltage zones in the atrial myocardium and lines of functional blockade could indicate the presence of myocardial lesions, which brings into question the long-term prognosis of the atrial mechanical function and rhythm, although the tachycardia circuit can be interrupted acutely. Only 5 of our patients passed to a stable sinus rhythm during admission, 2 had relapses in a follow-up of 1-2 years and required antiarrhythmic drugs, and 2 required chronic atrial stimulation for sinus node dysfunction.

REFERENCES
5. Van Hare GF, Lesh MD, Ross BA, Perry JC, Dorostkar PC. Mapping and radiofrequency ablation of intraatrial reentrant tachycardia after the Senning or Mustard procedure for transposition of the great arteries. Am J Cardiol 1996; 77: 985-991.


