Left Septal Atrial Flutter
Electrophysiology, Anatomy, and Results of Ablation

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Background—We describe the clinical and electrophysiological characteristics of a novel macroreentrant form of left atrial flutter.

Methods and Results—A total of 11 patients were included in the study. The mean tachycardia cycle length was 278±41 ms. Nine of the 11 patients were treated with antiarrhythmic drugs at the time of the study for concomitant atrial fibrillation. With the use of entrainment pacing and either the CARTO Biosense mapping system (9 patients) or conventional mapping (2 patients), the flutter circuit was found to rotate around the left septum primum with a critical isthmus located between the pulmonary veins posteriorly and/or mitral annulus anteriorly and the septum primum. In 5 patients, radiofrequency ablation was performed from the septum primum to the right inferior pulmonary vein (group 1), and in 6 patients, a lesion was made from the septum primum to the mitral annulus (group 2). After a follow-up of 13±6 months, 2 patients in group 1 and all patients in group 2 remained in sinus rhythm without recurrence.

Conclusions—Slowing of electric conduction in the left atrial septum due to antiarrhythmic drugs and/or atrial myopathy seems to promote left septal atrial flutter. Radiofrequency ablation of this arrhythmia is usually effective and safe. A line of block between the septum primum and the mitral annulus proved to be effective for cure of tachycardia. (Circulation. 2004;109:2440-2447.)

Key Words: atrial flutter • ablation • electrophysiology

Isthmus-dependent counterclockwise or clockwise flutter has been well defined and is amenable to ablation of the isthmus between the tricuspid annulus and the inferior vena cava.1–6 More recently, attention has been directed to the elucidation and cure of patients with left atrial (LA) circuits.7–9 Olgin et al9 first described a reentrant circuit involving the musculature of the coronary sinus (CS) that was amenable to ablation of the muscle at the CS ostium. A variety of LA circuits that occurred in patients with organic cardiac disease that were associated with large “silent” areas over the posterior LA have been described.7,8 These circuits were found to revolve around the mitral annulus (MA), between the “silent” areas and pulmonary veins (PVs), or around the PVs. Reentry circuits with a protected isthmus could be identified in 89% of patients with LA flutter.

Embryologically, the LA septum originates from 2 different anatomic and histological regions of the sinus venosus and represents the adhesion of the septum primum (SP) and secundum.10,11 The role of this structure in the maintenance of LA flutter has not been previously characterized. In the present study, we describe the clinical and electrophysiologic characteristics of spontaneous left SP atrial flutter observed in patients without history of cardiac surgery.

Methods

Patients

Between March 1998 and October 2000, 300 patients with atrial flutter were evaluated in our laboratories. Of 67 patients in whom mapping of both atria revealed LA flutter, 11 patients demonstrated left septal flutter in the absence of previous surgery and were included in this study. The study population consisted of 8 men and 3 women with a mean age of 65±9 years. Atrial flutter was paroxysmal in 7 patients and incessant in 4. The median duration of symptoms was 4.6 years (range, 2 to 10 years). Coronary artery disease was present in 3 patients; 1 patient had mild mitral valve regurgitation; 1 had dilated cardiomyopathy, and no structural heart disease was present in the remaining 6 patients. One patient had a pacemaker implanted after atrioventricular node ablation. LA enlargement was present in 4 patients (LA diameter >45 mm). This arrhythmia did not respond to amiodarone in 6 patients or sotalol in 3 patients. Oral anticoagulation was stopped on admission and restarted after the electrophysiology study and ablation.

Electrophysiology Study Protocol

All patients gave written informed consent. Electrode catheters were positioned under fluoroscopic guidance. Four multipolar catheters were inserted percutaneously into the right femoral vein. A 7F duodecapolar catheter (interelectrode spacing 2 mm; Daig Corporation) was placed around the tricuspid annulus, and a 6F octapolar catheter (interelectrode spacing 2 mm; EP Technologies 1000) was
used to record His-bundle potentials. A 6F quadripolar catheter (interelectrode spacing 2.5-2 mm; Daig Corporation) was placed at the right ventricular apex. CS recordings were performed from a 6F decapolar catheter (interelectrode spacing 2.5-2 mm; Daig Corporation) advanced through the right internal jugular vein. In 2 patients, a 7F quadripolar asymmetrical thermocoupled ablation catheter (4-mm tip electrode; EP Technologies 1000) was used for atrial mapping and ablation. In the remaining 9 patients, a 7F deflectable 4-mm tip quadripolar catheter ( Biosense Webster) was used for the same purpose. The LA was approached through a transseptal puncture. Intravenous heparin was given to keep the activated clotting time at >300 seconds.

The 3D electroanatomic mapping system has been described previously. Briefly, it consists of an electromagnetic location pad positioned under the patient’s back, a CARTO processor unit (Biosense Webster), a Silicon Graphics workstation, and an electromagnetic sensor-equipped catheter. This catheter was used for navigation mapping and ablation.

Before the LA was approached, a right atrial (RA) flutter circuit was excluded, with application of the following criteria: RA activation time during tachycardia <40% of arrhythmia cycle length, failure to achieve concealed entrainment by pacing the RA at several sites, including the RA septum; and ability to dissociate the RA from the tachycardia by means of RA overdrive pacing.

Concealed entrainment from a critical isthmus was considered if pacing from that specific site at 10 to 30 ms faster than the tachycardia cycle length and at double the pacing threshold resulted in concealed fusion. This was defined as pacing-induced increase in flutter rate without change in either the endocardial electrograms or surface ECG, when available, free from obstruction of the stimulus artifact. In addition, the postpacing interval (PPI) was <30 ms than the tachycardia cycle length.\textsuperscript{13,14}

**Ablation**

Radiofrequency ablation was performed after we defined the isthmus of the flutter circuit, identified by concealed entrainment. The ablation line was performed by connecting either the SP to the right PV (posterior ablation line; group 1) or the SP to the MA (anterior ablation line; group 2). Successful ablation was defined as termination of atrial flutter during ablation and noninducibility of the same flutter morphology with the use of RA and LA overdrive and programmed pacing after ablation. Six hours after the procedure, all patients were restarted on warfarin for at least 3 months.

**Follow-Up**

All patients were discharged to home the day after the procedure and were either seen in our outpatient clinic or called at 3 monthly intervals. In case of flutter recurrence, antiarrhythmic drug therapy was reintroduced, and a second procedure was advised.

### Atrial Septum Microscopic and Macroscopic Preparation

Eleven autopsy hearts from patients who died with various cardiac and noncardiac conditions were examined. After perfusion with 10% buffered formalin at 20 cm of water, all hearts had been dissected along the lines of flow. In all specimens, the atrial septum was examined carefully from both the RA and LA aspects. In 7 hearts, the atrial septum was sectioned for histology across the midpoint of the oval fossa, including the limbus. These tissues were processed for histology according to standard methods, paraffin-embedded, sectioned at 5 μm, and stained with hematoxylin and eosin or Masson’s trichrome. We also studied left septal structures in those without heart disease for comparison. Four other hearts from patients who died from noncardiac causes were dissected more extensively: the endocardium of the RA over the oval fossa was removed by gentle blunt dissection so that the muscle fiber orientation within the primary atrial septum was apparent. The RA surface of these hearts was marked with black ink before the block of tissue containing the oval fossa and limbus was excised. For 2 of these hearts, this atrial septal block was cut into strips along a plane parallel to the direction of the muscle fibers in the primary atrial septum as seen from the RA. Blocks from the other 2 hearts were cut perpendicular to the muscle fiber orientation of the primary atrial septum. After processing, the 5-μm sections were stained with hematoxylin and eosin.

### Data Analysis

Comparisons between the means were made by paired t test. Data were expressed as mean±SD. A value of P<0.05 was considered statistically significant.

### Results

The clinical and electrophysiological characteristics of left septal atrial flutter in our study patients are demonstrated in the Table. Six patients were taking amiodarone, and 3 were taking sotalol for paroxysmal or persistent atrial fibrillation at the time of study. The mean LA diameter was 44±4 mm. In all patients the RA activation time was <40% of the atrial flutter cycle length. All patients presented with surface ECG showing monomorphic distinct flutter wave morphologies (Figure 1A). We excluded the RA as part of the reentrant circuit by entrainment pacing at lateral, posterior, septal, and

### Clinical and Electrophysiological Characteristics of Left Septal Atrial Flutter

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age, y</th>
<th>Sex</th>
<th>Hx of AF</th>
<th>LA Size, mm</th>
<th>Antiarrhythmic Drugs (Preablation)</th>
<th>Atrial Flutter Cycle Length, ms</th>
<th>CW vs CCW</th>
<th>P Wave in V1</th>
<th>Site of Ablation</th>
<th>Success</th>
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<td>225±16</td>
<td>CW</td>
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<tr>
<td>4</td>
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<td>No</td>
<td>42</td>
<td>Amiodarone</td>
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<tr>
<td>6</td>
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<td>42</td>
<td>Amiodarone</td>
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<td>255±15</td>
<td>CW</td>
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<tr>
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<td>52</td>
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<tr>
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<td>40</td>
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<td>280±10</td>
<td>CW</td>
<td>Negative</td>
<td>Anterior</td>
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</tbody>
</table>

Pt indicates patient; SHD, structural heart disease; Hx of AF, history of atrial fibrillation; LVEF, left ventricular ejection fraction; CW, clockwise circuit; and CCW, counterclockwise circuit.
subeustachian isthmus sites in all patients. We excluded a CS flutter by entrainment mapping of the distal and proximal CS (Figure 2). Entrainment mapping around the whole circumference of the PVs (right or left) and the MA was conducted to exclude a flutter circuit around these structures.

**Flutter Circuit**

After a left-sided origin of the arrhythmia was confirmed, transseptal puncture was performed. Electroanatomic mapping of the LA was performed in 9 patients. The mean flutter cycle length was $278 \pm 39$ ms. A mean of $80 \pm 20$ points was

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**Figure 1.** Variation in monomorphic P-wave morphologies in 2 of the study patients. A, Counterclockwise left septal atrial flutter with positive flutter waves in leads V1, II, III, and aVF; B, clockwise left septal atrial flutter with negative flutter waves in lead V1 and positive flutter waves in leads II, III, and aVF.
needed to complete sequential activation reconstruction of the LA 3D map in 9 patients. The 3D activation and propagation maps of the LA described a macroreentrant circuit rotating around the left SP (Figure 3). Observing the septum from the left lateral view, we could show a clockwise rotation of the flutter wave in 6 patients and counterclockwise circuit in 5 patients. The rest of the LA and the RA were passively activated, as shown in Figure 3. Note that the LA was activated from the septal to lateral wall, and paced mapping of MA showed that it was outside the circuit.

**Entrainment Sites of Septum**

Entrainment was used to confirm the critical left septal regions involved in the flutter circuit (Figure 2). In 5 patients, concealed entrainment could be shown between the SP and the aforementioned ridge (Figure 2). In the remaining 6 patients, concealed entrainment was proven between the MA and the SP. Bipolar recordings along a 2-cm-long ridge in front of the right PV showed double potentials (Figure 4). Pacing from the inferior and the anterolateral aspect of the MA never resulted in concealed entrainment, thus excluding the MA as a critical part of the reentrant circuit (Figure 3B). Manifest entrainment was documented in 5 patients by pacing superiorly and inferiorly to the SP at a cycle length 20 to 30 ms below the flutter cycle length; the PPI was identical to the tachycardia cycle length at these sites. These sites were interpreted as being in the nonisthmus part of the reentrant circuit.

**Surface Flutter Wave Morphology**

Patients presenting with counterclockwise and clockwise septal flutter (defined per 3D mapping) revealed a prominent positive or negative P-wave deflection in lead V1, whereas the limb leads demonstrated flat P-wave morphology (Figure 1A). Clockwise septal flutter reflected a prominent negative deflection in lead V1 and demonstrated the same flat P-wave morphology in the remaining leads (Figure 1B).

**Ablation Results**

Two different ablation strategies, the posterior and the anterior approach, were used to terminate the left septal atrial flutter.

With the use of radiofrequency energy in 5 patients (45%), a line connecting the right inferior PV and the SP was drawn (posterior approach; group 1). In 4 patients, acute termination of the flutter was documented during radiofrequency energy administration. In 1 patient, we were unable to interrupt the flutter circuit. Figure 5 demonstrates the ablation lines.

The anterior approach (group 2) consisted of an ablation line between the SP and the MA and was applied in 6 patients (55%). In all patients treated with the anterior linear lesion, ablation was successful in terminating flutter.

During ablation and before termination of flutter, a 30±20-ms prolongation of the flutter cycle length was observed in all patients. After termination of the flutter circuit, the continuity of the ablation line was demonstrated by the presence of double potentials along the ablation line, separated by an isoelectric interval of ≥50 ms (Figure 5).

**Outcome and Follow-Up**

After a mean follow-up time of 13±6 months, 73% of patients (8 of 11) were still in sinus rhythm. By connecting the right inferior PV to the SP, we failed to terminate the left septal flutter in 1 patient (9%) because of difficulty in completion of the ablation line. Two patients (18%) experienced recurrence of atrial flutter after 13±6 months of follow-up time, including the patient who failed the initial ablation. Two patients experienced recurrence of atrial fibrillation and were still in sinus rhythm on antiarrhythmic drug treatment with amiodarone. The anterior approach was successful in terminating all flutter circuits acutely (P<0.05). Moreover, all patients treated with this approach were still in sinus rhythm after a mean follow-up time of 13±6 months.

**Histological Findings**

In all 11 hearts, the primary atrial septum was 1 to 2 mm thick. In histological cross section, the endocardium was much thicker on the left side of the primary atrial septum than on the right. Sandwiched between the endocardia was a narrow band of cardiac muscle (Figure 6). This muscle was continuous with the robust myocardium of the limbus at either end of the section. In the 4 hearts from which the entire atrial septum was sectioned in known orientation, a similar band of cardiac muscle was present in every slice, indicative of a sheet of myocardial tissue throughout the primary atrial septum. In all 4 hearts, there was fibrofatty tissue subdividing the sheet of muscle into leftward and rightward sublayers within the primary atrial septum. Sections in which the muscle fibers of 1 sublayer were cut in longitudinal section showed the other sublayer to be composed of fibers cut in short-axis cross section. This fiber orientation suggested that the 2 sublayers of muscle within the primary atrial septum are orthogonal. In 3 of the 4 hearts, it appeared that the muscle fibers subjacent to the endocardium on the RA aspect coursed toward the LA muscle at the periphery of the primary atrial septum (Figure 7).
**Discussion**

**Main Findings**
To our knowledge, this is the first report of patients with macroreentrant left septal atrial flutter. Most of the patients in our study developed left septal atrial flutter after initiation of antiarrhythmic therapy for atrial fibrillation. Slowing of atrial conduction by antiarrhythmic drugs may be a factor that allows left septal flutter. The zone of double potentials anterior to the right PVs, corresponding to the limbus of the fossa ovalis for the RA, and the MA represent functional and anatomic barriers for left septal atrial flutter, respectively. In addition, our series appeared to suggest that the anterior ablation approach is more likely to provide a long-term cure.

**Use of Entrainment to Define a Left Septal Atrial Flutter Circuit**
Concealed entrainment has been used to identify a protected isthmus between barriers, which are critical for the maintenance of reentrant circuits. In our study, we first excluded a RA flutter circuit. Concealed entrainment was documented by pacing between the zone of double potentials between the right PVs and the SP and between the SP and MA. Thus, the left septal atrial flutter circuit appeared to revolve around the SP with the use of 2 protected isthmi.

The zone of double potential seems to play a role similar to that of the crista terminalis during typical atrial flutter. The zone of double potentials recorded in our patients corresponds to the limbus of the fossa ovalis on the right septum, which seems to complete the embryological separation of the 2 atria. Sun et al described a similar zone of double potentials in the inferior left septum in a canine model. The region between the SP and the MA defines another critical isthmus for the left septal atrial flutter circuit. A similar situation has been described by Kalman et al in the RA, where the tricuspid annulus forms a continuous anterior barrier in patients with typical flutter. In the present study, we...
demonstrate a similar role of the septal part of the MA in forming an anterior anatomic barrier for the left septal flutter circuit. Moreover, Jais et al.\(^8\) also recognized the MA as a critical anatomic barrier in 36% of patients with LA flutter.

**Surface P-Wave Morphology**

The morphology of the surface P waves in canine flutter is determined primarily by LA activation forces.\(^{18}\) In our patient population, counterclockwise circuit and clockwise circuit septal flutter revealed a flat P wave in the all but the V\(_1\) lead. This is potentially explained by simultaneous rapid activation of the RA from both Bachmann’s bundle and the CS musculature, resulting in cancellation of the superior and inferior forces.

**Slowing of Left Intra-Atrial Conduction and Left Septal Atrial Flutter**

It is possible that the flutter described in our patients may be dependent on the intra-atrial conduction time. Indeed, 6 of 11 patients were taking amiodarone, which prolongs the action potential duration and/or conduction time.\(^{19,20}\) In addition, 4 patients had atrial dilatation, which could also lead to impaired atrial conduction.\(^{21,22}\)

Class III as well as class IC antiarrhythmic drugs have been implicated in the development of cavotricuspid isthmus–dependent atrial flutter for those with atrial fibrillation.\(^{19,23–25}\) In our study, 6 patients were taking amiodarone for treatment of atrial fibrillation. Antiarrhythmic drugs may slow conduction velocity and thus encourage a stable flutter circuit.

**Catheter Ablation**

Recognition of a critical isthmus in the left septal flutter circuit has important therapeutic implications. The presence

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**Figure 4.** 3D electroanatomic activation map of flutter circuit in left lateral view rotating around SP with early activation (red) meeting late activation (violet) in front of MA. Corridor of double potentials is plotted on 3D map. RUPV indicates right upper pulmonary vein; RLPV, right lower pulmonary vein.

**Figure 5.** Anatomic picture of left atrial septum demonstrating the flutter circuit with the 2 different defined isthmuses. Right PVs form the posterior boundary, and MA forms the anterior boundary. Red lines represent the anterior and posterior ablation lines. Green points demonstrate location of split potentials across the anterior ablation line between SP and MA.

**Figure 6.** Histology of primary atrial septum. In a cross section of the thinnest portion of the atrial septum, a band of non-muscle tissue (NM) containing small vessels and collagen separates 2 layers of cardiac muscle. In the layer closer to the RA cavity (denoted by black ink), the muscle fibers are cut in longitudinal axis, confirming the orientation seen in the gross specimen. In the layer closer to the left atrium, the fibers are cut in short axis, suggesting an orthogonal arrangement between the muscle fibers constituting the 2 layers. Hematoxylin and eosin stain; bar=40 μm.
of a posterior isthmus between the right inferior PV and the SP and an anterior corridor between the SP and the MA suggested that both counterclockwise and clockwise left septal flutter can be eliminated by creating a linear lesion along the aforementioned regions. In our series, the SP was connected to the right inferior PV in 5 patients (group 2) and to the MA in 6 patients (group 1). The anterior approach proved to be more efficacious in interrupting the macroreentrant flutter circuit compared with the posterior approach. This could reflect either a different thickness of the posterior isthmus or simply a region that anatomically is more difficult to reach. It is possible that ablation technologies able to achieve deeper lesions may increase cure regardless of the approach utilized by the operator.

**Histological Findings**

The present study documents a thin layer of muscle sandwiched between the endocardial surfaces of the primary atrial septum. That this thin membrane-like wall between the atrial cavities contains muscle has largely been ignored. In his detailed study of atrial musculature in mammalian hearts, Papez described 2 components of the SP: (1) a right component appearing as a bundle of muscle fibers caudal to the oval fossa, coursing from the sinus ridge to the inferior vena cava, and (2) a left component coursing from the posteromedial atrioventricular ring to the orifice of the LA appendage anteriorly. To our knowledge, our study is the first to demonstrate the bilaminar structure of the muscle constituting the thin membranous primary atrial septum in the human heart. The embryological basis for this architecture remains unclear; perhaps the primary atrial septum starts as an infolding, similar to the secondary atrial septum. This bilaminar structure could have contributed to the protection and maintenance of the left septal flutter circuit.

**Limitations**

Ablation success in this study was determined by symptomatic recurrences on routine surveillance. Although asymptomatic recurrences could have occurred, all patients included in this study were symptomatic and failed multiple trials of antiarrhythmic drug therapy before ablation. In 2 patients undergoing ablation with the posterior approach, no 3D electroanatomic map was performed, which could have negatively influenced the success rate in this group. Although we did not prove the bilaminar structure in our patient population with left septal atrial flutter, the consistency of the histology in the studied hearts strongly suggests a similar septal morphology in this group of patients.

**Conclusion**

Slowing of electric atrial conduction due to antiarrhythmic drugs or atrial myopathy may prompt LA flutter in the absence of incisions or scars. Radiofrequency linear lesions between the membranous septum and the MA seem to be an effective and safe therapy for this arrhythmia. The left atrium septum possesses a unique muscular architecture that would allow the maintenance of left septal atrial flutter.

**References**


