Catheter Ablation of the Atrial Myocardium in Human Type I Atrial Flutter

N. Saoudi, G. Atallah, G. Kirkorian, and P. Touboul

To avoid atrioventricular node–His bundle ablation, catheter ablation of the atrial myocardium was attempted in eight patients with drug refractory type I atrial flutter. In seven of eight patients, a zone of prolongation and fragmentation of the endocardial electrogram was found in the low posterior part of the right atrium. Entrainment of the atrial flutter by high right atrial pacing was accompanied by local recording of second-degree regional block in several atrial sectors but never in the low septal area. We, therefore, hypothesized that the latter represented the critical slow conduction zone of the reentrant flutter circuit. One or two cathodal DC shocks were locally delivered without immediate or late complications. One single ablation attempt was performed in five patients, whereas three patients underwent a second attempt because of early flutter recurrence. Patients were initially discharged without (and after a second session with) antiarrhythmic drugs. After a mean follow-up of 15.5 months (range, 10–23 months), five patients are free of arrhythmias without antiarrhythmic drug therapy. Two patients did not experience atrial arrhythmias while on a drug regimen that was previously found to be ineffective, and a third patient had flutter recurrences. This study suggests that patients with type I atrial flutter referred for atrioventricular node–His bundle ablation may be successfully managed by delivering the ablative shock directly on the atrial arrhythmia substrate. (Circulation 1990;81:762–771)

The goals of therapy in human atrial flutter are to restore sinus rhythm and prevent tachycardia recurrences. While the former is usually achieved, the latter is often difficult to obtain despite numerous antiarrhythmic drug trials, pacing therapy, and recently, direct atrial surgery. This is the reason why recurrent atrial flutter, along with atrial fibrillation, accounts for as much as 60% of the indications for His bundle ablation. The chief drawback of this procedure is the development of pacemaker dependency, which may be undesirable in young and otherwise healthy patients. After the early work of Puech in the late 1950s, recent studies have confirmed that type I atrial flutter is a reentrant arrhythmia that is confined to the right atrium. In almost all patients, endocardial mapping during tachycardia allows recording of low voltage prolonged and fragmented potentials in the area of the low posterior right atrial septum, which is suspected as being the critical slow conduction area of the reentrant circuit. This fact led to an attempt at direct ablation of this atrial area in 1987. The procedure is described, and the clinical characteristics of eight patients in whom it was performed are reported.

Methods

All pertinent clinical patient data are detailed in Table 1. All patients were referred for His bundle ablation because of extremely symptomatic type I atrial flutter. The latter could be incessant with several daily (patients 6 and 7) or monthly (patients 3, 4, and 8) attacks. One patient (2) experienced pulmonary edema with each recurrent episode of atrial flutter, one of which led to cardiac arrest. In another patient (5), episodes of 1:1 atrioventricular conduction were accompanied by hemodynamic collapse that required emergency cardioversion. No structural heart disease could be found in five patients. Two patients had a dilated cardiomyopathy, one had a moderately elevated high blood pressure, and two had a chronic obstructive lung disease. Patients were resistant to a mean of four (range, one to six) antiarrhythmic drugs, including quinidine, digitalis, flecainide, β-blocking agents, disopyramide, sotalol, and amiodarone used alone or in combination. At the time of ablation, seven of eight patients were on long-term oral amiodarone therapy.

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Table 1. Clinical Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Associated disease</th>
<th>AF (yr)</th>
<th>AA (n)</th>
<th>Symptoms</th>
<th>Drug regimen</th>
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<tr>
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<td>F</td>
<td>HBP</td>
<td>6</td>
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<td>Palpitations</td>
<td>Amiodarone, flecainide, digoxin</td>
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<td>62</td>
<td>M</td>
<td>Dil card</td>
<td>2</td>
<td>1</td>
<td>Palpitations, pulmonary edema, cardiac arrest</td>
<td>Amiodarone</td>
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<td>COPD, Dil card</td>
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<td>Amiodarone</td>
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<tr>
<td>4</td>
<td>62</td>
<td>M</td>
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<tr>
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<td>6</td>
<td>Palpitations</td>
<td>Amiodarone, sotalol</td>
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<tr>
<td>7</td>
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<td>5</td>
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<td>Palpitations</td>
<td>Amiodarone</td>
</tr>
<tr>
<td>8</td>
<td>56</td>
<td>F</td>
<td>None</td>
<td>2</td>
<td>4</td>
<td>Palpitations, presyncope</td>
<td>Amiodarone</td>
</tr>
</tbody>
</table>

AF, atrial flutter; AA, antiarrhythmic agents; HBP, high blood pressure; Dil card, dilated cardiomyopathy; COPD, chronic obstructive pulmonary disease.

In all patients, ablation was performed after careful explanation of the experimental nature of the procedure and procurement of informed consent. Baseline electrophysiological evaluation was performed with four standard 6F USCI tetrapolar (or tripolar) pacing catheters with 10-mm interelectrode spacing, percutaneously introduced via the conventional femoral approach. Catheters were positioned respectively in the high right atrium, atroventricular node–His bundle area, and right ventricular apex, with the fourth catheter being used for atrial mapping. A fifth bipolar catheter was introduced via a subclavian vein when the femoral approach was not considered sufficient for adequate atrial mapping. Mapping catheters were a priori tested to assess suitability for ablation. The latter was done according to the following protocol. With reference to the abrupt loss of local recording, which identified the entrance of the venae cavae, recording sites were defined as high, mid, or low in the septal, posterior, lateral, or anterior right atrial wall. The low posteroseptal area of the right atrium was approached by first placing the mapping catheter in the His bundle region and then withdrawing it with a slight clockwise rotation in such a way that the catheter tip is located approximately midway between the latter area and the os of the coronary sinus in the anteroposterior plane. Further adjustments in catheter positioning were then made by slightly displacing it in a search for local fragmentation. A complete mapping of the remainder of the right atrium was also performed in an attempt to record other fragmentation areas. Bipolar endocardial electrograms were filtered (band pass, 50–500 Hz), amplified at 5–20 mm/mV, and recorded with an ink-jet Siemens recorder (Mingograf 7 or T16) at a paper speed of 100 mm/sec. We deliberately chose to maintain our technique of endocardial electrogram filtering and gain whenever possible at constant settings to allow comparison between the different recording sites. At least two surface electrocardiographic leads (including lead II) were simultaneously recorded. Prolonged electrograms were arbitrarily defined as local potentials extending more than 80 msec in duration, a value that is consistent with previously published data for atrial flutter. Low-voltage electrograms were also arbitrarily defined as a local recording that required an increase in gain of more than 10 mm/mV to record clear-cut atrial activity. At times, the gain was increased up to 40 mm/mV to correctly identify all local electrograms. Electrical artifacts caused by catheter movement were excluded when the same phenomenon was reproducibly found in a given area and when no catheter displacement was apparent on multiple fluoroscopic views. In two patients, several areas of fragmented local potentials were found. The behavior of fragmentation was systematically studied during fixed-rate high right atrial pacing. Two-millisecond pulses were delivered at twice-diastolic threshold by a programmable stimulator (Savita, France, or Janssen, Belgium). The initial pacing cycle length was 10 msec shorter than that of tachycardia and was further decreased by 10-msec steps. According to previously published criteria, transient entrainment of atrial flutter occurred when at least one of the following phenomena were observed: constant fusion beats between flutter and paced impulses at a given pacing rate (except for the last beat, which is entrained but not fused) (criterion 1), different degrees of stable fusion at different fixed pacing rates (criterion 2), or interruption of tachycardia associated with localized conduction block to a site for one beat with subsequent activation of that site from a different direction and with a shorter conduction time. The end point of the pacing protocol was either tachycardia termination, the attainment of a pacing cycle of 150 msec (shorter cycles were not used to avoid pacing induced atrial fibrillation), or recording of local electrogram dissociation during tachycardia entrainment.

When an area of localized fragmentation was found and believed to be part of the reentrant circuit, a short-acting general anesthetic (sodium pentothal, 150–300 mg) was administered, and one or two shocks (2–2.5 J/kg body wt) were delivered using the tip of the ablation catheter as the cathode and a back paddle as the anode. Immediate inducibility of atrial flutter was tested 30 minutes after the shocks using programmed atrial stimulation and high rate atrial pacing. The procedure was then stopped indepen-
dently of the result at that point, and patients were monitored in the intensive care unit for 24 hours. Two-dimensional echocardiograms as well as serial creatine phosphokinase–MB assays were performed during 24 hours. All patients were discharged without antiarrhythmic drugs and were carefully followed up by repeated telephone conversations with the patient or the referring physician or on an outpatient basis by one of the authors (N.S. or P.T.). Holter recordings were scheduled at the end of the first and three months after the procedure, and a repeat electrophysiologic study was performed after a 3-month period in all patients. Additional Holter recordings were performed at least every 4 months.

**Results**

Electrophysiological data at the time of ablation are shown in Table 2. In four of eight patients, clinical atrial flutter was present at the beginning of the session, and in two of these, it was reproducibly terminated and reinduced by atrial stimulation. In the remaining four, it was easily induced with one or two premature atrial extrastimuli or by atrial burst pacing. In all cases, the typical sawtooth pattern of type 1 atrial flutter was observed in the inferior leads, with slight morphological variations from one patient to another. In most of the cases, the atrial flutter cycle lengths were longer than the usually observed 200 msec for this arrhythmia because of ongoing amiodarone therapy. Two patients (2 and 5) did not undergo the complete pacing protocol. This occurred at the beginning of our experience at a time when we were studying how to exclude standby dead-end pathways using the tachycardia entrainment concept (but did not systematically do it). In the six patients who underwent the complete protocol, entrainment criterion 1 was always fulfilled, criterion 2 was filled three times, and criterion 3 was filled once (Table 2).

**The Fragmented Electrogram**

In seven of eight patients, a fragmented electrogram was recorded in the area of the low posterior right atrial septum. Its beginning was always synchronous with the plateau phase, and it extended up to the negative portion of the F wave. Figure 1 depicts such an abnormal electrogram. The flutter cycle length is 280 msec, and electrical activity in this area is recorded for 135 msec. The stability of the isoelectric baseline and the position of the catheter tip that was firmly applied against the atrial wall almost certainly excluded the possibility of recording electrical artifacts. During all pacing runs, the 1:1 relation between the low posteroseptal electrogram, the stimulus artifact, and the other atrial electrogram was always maintained as long as the entrainment criteria were fulfilled. Moreover, a long delay between the pacing artifact and the beginning of the local fragmented electrogram (whose morphology was unchanged) was also always noted during transient entrainment suggesting orthodromic capture of this zone through the flutter circuit (Table 2). In patients 1 and 8, the delay between the pacing artifact and the beginning of the local electrogram was not particularly long (50 and 80 msec, respectively). However, the duration of the local electrogram itself in the posteroseptal area in these patients was rather long (130 msec), suggesting that an area of slow conduction may be localized in a relatively small area since the end of the fragmented electrogram was captured with a long interval of 180 (50+130) and 210 (80+130) msec, respectively.

It should be noted that if abnormal electrograms were quasiconstantly recorded in this area, they could at times also be recorded in very distant areas such as the sinus node region (patients 1 and 3) or a more lateral portion of the right atrial free wall in the same area (patient 3). In both cases, the local fragmented electrical activity was seen as a well-delineated, double-spike electrogram that had a duration of no more than 100 msec (see Figure 2 in Reference 11) Figures 2 and 3 depict such electrograms that were recorded in patient 3. When the electrode was positioned in the posterior portion of the high right atrium (Figure 2), local 6:5 conduction

**Table 2. Electrophysiological Data**

<table>
<thead>
<tr>
<th>Patient</th>
<th>AF cycle length (msec)</th>
<th>Site</th>
<th>Length (msec)</th>
<th>Entrainment Criteria (n)</th>
<th>Delay S-LPRAS</th>
<th>Shocks n</th>
<th>Total energy (J)</th>
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<tbody>
<tr>
<td>1</td>
<td>270</td>
<td>LPRAS,</td>
<td>130</td>
<td>1</td>
<td>50</td>
<td>2</td>
<td>250</td>
</tr>
<tr>
<td>2</td>
<td>200</td>
<td>...</td>
<td>...</td>
<td>Not done</td>
<td></td>
<td>2</td>
<td>440</td>
</tr>
<tr>
<td>3</td>
<td>270</td>
<td>LPRAS,</td>
<td>90</td>
<td>1</td>
<td>170</td>
<td>1</td>
<td>150</td>
</tr>
<tr>
<td>4</td>
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<td>145</td>
<td>1</td>
<td>180</td>
<td>1</td>
<td>150</td>
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<tr>
<td>5</td>
<td>260</td>
<td>LPRAS</td>
<td>80</td>
<td>Not done</td>
<td></td>
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<td>260</td>
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<tr>
<td>6</td>
<td>250</td>
<td>LPRAS</td>
<td>90</td>
<td>1,2</td>
<td>140</td>
<td>2</td>
<td>320</td>
</tr>
<tr>
<td>7</td>
<td>280</td>
<td>LPRAS</td>
<td>110</td>
<td>1,2,3</td>
<td>200</td>
<td>2</td>
<td>360</td>
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<tr>
<td>8</td>
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<td>130</td>
<td>1,2</td>
<td>80</td>
<td>1</td>
<td>150</td>
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</table>

AF, atrial flutter; LPRAS, beginning of the low posterior right atrial septum electrogram; S, stimulus artifact; HRA, high right atrium.
block of the Wenckebach type was recorded. When the catheter tip was positioned an added 1 cm laterally (Figure 3), the local electrical activity was found to be highly prolonged and fragmented. However, when pacing was started at a rate slightly faster than tachycardia rate (here, 210 msec for a spontaneous flutter cycle length of 270 msec), the surface electrocardiogram showed acceleration of the flutter rate with slight changes in the flutter wave pattern that suggested fusion between the tachycardia and paced impulses. During this pacing run, the low posteroseptal area of the right atrium (which also showed a local fragmented electrogram) accelerated to the pacing rate after a long delay (as shown by the last beat that is entrained but not fused) and without any change in electrogram morphology, suggesting orthodromic entrainment of this site through the flutter circuit (Table 2). Concomitant dissociation occurred in the high lateral area where local electrogram exhibited second-degree block of the 2:1 type. Similar behavior of two localized areas of fragmentation was recorded in our first patient. We interpreted the dissociation of two components of a double-spike electrogram that occurred either spontaneously (patient 3) or during tachycardia entrainment (patients 1 and 3) as the manifestation of a local dead-end pathway of activation. In a selected case (patient 7), high right atrial pacing induced termination of atrial flutter. As depicted in the upper portion of Figure 4, an 8:7 pattern of second-degree block occurred at a paced cycle length of 200 msec. This was preceded by a local prolongation of the fragmented electrogram that ended with a missing beat in the low posterior right atrial septal area. The eighth paced beat was blocked in the right atrium between the pacing (HRA) and the recording site (LPRA) after which the atrial surface electrocardiographic pattern changed, suggesting atrial flutter interruption. The local increase in duration of the fragmentation leading to a dropped beat and to tachycardia termination can be interpreted as recording of an area of slow conduction that is critical for perpetuation of the circus movement. Moreover, at the end of this sequence, the same area exhibited narrow electrograms that were occurring after the high right atrial free wall activity with a much shorter conduction time than during atrial flutter. This phenomenon accounts for the third criterion of transient entrainment of reentrant tachycardias as defined by Waldo et al. The singularity of this case is that conduction block leading to tachycardia interruption is not only deduced from a change in surface electrocardiographic morphology and intracardiac electrogram timing but also directly recorded as a dynamic change preceding the disappearance of local fragmentation.

The Ablation Procedure

In all eight patients, at least one shock was delivered in the low posterior right atrial septal area using the tip of the mapping catheter as the cathode and a back paddle as the anode. In seven patients, the
shock was delivered in this area after the above-mentioned fragmentation was recorded. In the remaining patient, we were not able to record such an abnormal electrogram. Careful mapping during atrial flutter revealed that the low posteroseptal area exhibited the earliest local electrogram in reference to the beginning of the negative portion of the flutter wave. Therefore, the shock was given in this zone because (as was commonly done in ventricular tachycardia ablation) we considered it to be the tachycardia exit point, preceding the start of a new flutter cycle.

In four patients, a brief period of complete atrioventricular block occurred after the shocks and required ventricular pacing for a maximum of 3 minutes. In the other patients (in whom no obvious atrioventricular nodal effect of the first shock was seen), a second shock was delivered during the same anesthesia 1 minute after the first. Both shocks were 2–2.5 J/kg.

The only immediate complication observed was in one patient in whom transient ventricular fibrillation occurred and who was treated successfully via external DC cardioversion. No patient experienced hemodynamic collapse; pericardial effusion was ruled out due to lack of a postshock murmur and a normal control two-dimensional echocardiogram performed the same day (n=5). After ablation, rapid pacing and programmed atrial stimulation were performed. Atrial flutter was not inducible in seven of eight patients. A different atrial tachycardia was induced in the remaining patient and converted by rapid overdrive pacing. Such arrhythmia had never been recorded previously and did not recur at a long-term follow-up of 23 months.

The mean follow-up time was 18.8 months and ranged from 13 to 27 months (Table 3). Three patients experienced recurrences of atrial flutter: two (6 and 7) at day 3 and one (4) 1 week before the repeat electrophysiological study. All three underwent a second noncomplicated ablation procedure at the end of which atrial flutter remained inducible in one patient (4). We arbitrarily decided to discharge these patients on the preablation antiarrhythmic drug regimen that was previously ineffective (flecainide, 100 mg b.i.d.; sotalol, 80 mg t.i.d.; amiodarone, 300 mg, respectively). We followed this course because we considered that these patients could constitute a subgroup where the chances of success may be lower and we did not want to attempt direct atrial ablation more than twice before doing His bundle ablation.

Two patients (4 and 6) did not have atrial flutter recurrences, whereas in one (7) atrial flutter resumed on the third day after ablation.

Late postablation electrophysiological studies were performed in the five patients who remained asymptomatic. Atrial flutter was still inducible in two patients (1 and 3) and not inducible in two (2 and 8), and atrial fibrillation was induced in the remaining patient (5).

Holter recordings showed that in addition to case 7, three patients had episodes of intermittent atrial fibrillation that were not seen before the ablation.
Atrial flutter is confined to the right atrial chamber. The classic studies of Lewis et al.,12 Rosenblueth and Garcia Ramos,13 and Kimura et al14 conclusively showed that a circulating wave can be forced to propagate around the natural or artificial obstacles of the right atrium. At the same period, focal automaticity was suspected because either local injection of aconitine or high-rate pacing of the atrial area surrounding the mouth of the coronary sinus was able to reproduce rapid atrial flutter–like activity on the surface electrocardiogram.15,16 Nevertheless, the latter theory ignored the fundamental intra-atrial conduction defect underlying atrial flutter, which is frequently suggested by the P wave morphology in sinus rhythm. Reentry was exemplified by the discovery by Boineau et al17 of a dog with spontaneous atrial flutter that closely mimicked human type I atrial flutter. In this case, the surface electrocardiographic pattern of the arrhythmia was characterized by broad positive undulations separated by brief nadirs, and epicardial mapping was associated with a counterclockwise rotating wave front when the atria were viewed anteriorly. The basis of atrial flutter

procedure and required drug therapy in two (Table 3). No other previously unknown arrhythmia was seen up to the end of follow-up.

Discussion

The present study suggests that patients presenting with drug-refractory recurrent atrial flutter may undergo direct catheter ablation without the need for His bundle interruption and permanent pacemaker implantation. This new therapeutic approach intends to be curative, that is, directs the fulguration shock on the arrhythmia substrate rather than prevents its consequences as is currently done with His bundle ablation. It is based on several a priori premises, including the right atrial reentrant nature of the pathological process, the ability to identify the critical slow conduction area of this reentrant circuit by catheter techniques, and the feasibility and safety of DC shocks in the human right atrium.

Right Atrial Reentrant Phenomenon

The great bulk of evidence points to a reentrant mechanism that in the common (type 1) form of atrial flutter
circuit in this animal was atrial hypoplasia and myocardial discontinuity leading to localized areas of slow conduction. Further convincing evidence for the reentrant mechanism of atrial flutter was later provided by Waldo et al,\textsuperscript{18} who used fixed rate right atrial pacing to show how each wave front from the pacing impulses was likely to enter the excitable gap of the reentrant circuit and travel both antidromically (where it collides with the preceding tachycardia or paced impulse) and orthodromically (where it resets the tachycardia to the pacing rate). These authors found that pacing the high portion of the right atrium or of the left atrium always results in stable atrial fusion at a given pacing rate and in various degrees of fusion at different pacing rates.\textsuperscript{19} Additionally, in our experience as well as in others,\textsuperscript{5-7} pacing the atrial

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure4.png}
\caption{Patient 7: Atrial flutter termination during fixed rate atrial pacing. Upper panel, left: Each paced impulse is conducted from the high to the low posterior right atrium with a long conduction time (155 msec). Note the progressive disappearance of the local fragmentation until the eighth paced beat for which there is local conduction block at that site. The latter is accompanied by a change in surface atrial electrogram, which no longer exhibits fusion identifying tachycardia termination. Thereafter, each paced impulse is conducted from the high to the low atrial recording site with a short conduction time (90 msec), and local electrogram is no longer fragmented despite continuation of pacing at the same cycle length.}
\end{figure}

<table>
<thead>
<tr>
<th>Patient</th>
<th>Duration of follow-up (mon)</th>
<th>Flutter inducibility at EPS control</th>
<th>Clinical flutter recurrences</th>
<th>Control Holters</th>
<th>Postabl. AA drugs</th>
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</tr>
<tr>
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</tr>
<tr>
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<td>No</td>
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EPS, electrophysiologic study; Postabl. AA drugs, postablation antiarrhythmic drugs; AFib, atrial fibrillation; AF, atrial flutter.
area that surrounds the mouth of the coronary sinus during atrial flutter does not result in atrial fusion beats but reproduces the spontaneous tachycardia beats. Such responses, which are hardly understandable on the basis of focal automaticity, are currently interpreted as being the result of pacing at various sites of a reentry loop, with the location of the pacing site relative to the area of slow conduction being critical for the demonstration of the entrainment criteria.20,21 If the pacing site is proximal to the area of slow conduction, the surface electrocardiogram will exhibit atrial fusion complexes in the paced chamber; therefore, transient tachycardia entrainment will be patent. On the other hand, if the pacing site is distal to the slow conduction zone, full paced beats are seen on surface electrocardiogram and transient entrainment (if occurring) is concealed. Pacing induced spontaneous tachycardia acceleration without evidence of surface electrocardiographic fusion suggests complete orthodromic capture of the chamber being paced.23 In this setting (as shown in computer simulation), the antidromic wave front is either blocked or collides with the preceding orthodromic wave front inside the slow conduction zone.24 The use of transient entrainment of tachycardia was proposed as a guide for localizing the "site of origin" and, therefore, as an aid to the ablation of reentrant arrhythmias.20

**Area Surrounding the Coronary Sinus Os**

The low-to-high spread of activation within the interatrial septum, along with the persistent lack of recording of a permanent electrical activity throughout the flutter cycle, initially led many researchers to conclude that atrial flutter was more likely due to an automatic focus located between the inferior vena cava, the coronary sinus ostium, and the tricuspid orifice. The ability to completely cover the atrial flutter cycle by local recordings during catheterization in humans was first provided by Puech et al.25 who convincingly showed that prolonged micropotentials could be recorded in the right side of the lower portion of the interatrial septum, which suggests this area could be a zone of localized slowing of conduction preceding the start of a new atrial flutter cycle. Studies on the pattern of resetting with single extrastimuli were also consistent with a reentrant circuit associated with slow conduction in the low posterior right atrial septum.5,23 Additionally, in a recent study where two patients underwent attempted direct atrial surgical therapy for type I atrial flutter, preoperative endocardial mapping showed "early potentials" in reference with the negative portion of the F wave in the region of the coronary sinus orifice.1 Premature atrial beats delivered in this area advanced the subsequent F wave without any detectable fusion of the atrial surface electrogram and with a poststimulation cycle identical to the flutter cycle length. In the same patients, perioperative epicardial mapping yielded a clustering of isochrones in the posteroseptal region that further suggested this area as critical to the circuit, and local cryosurgical ablation prevented long-term atrial flutter recurrences. These data prompted us to look specifically at the postero-septal area where we consistently found arguments for a critical localized slowing of conduction.6

**Fragmentation of the Local Electrograms**

It is generally accepted that reentry requires a circuit with unidirectional block for its initiation and an area of slow conduction for its initiation and perpetuation. With the currently available catheter techniques, it is usually possible to record in vivo low-voltage and multiphasic prolonged electrograms that are usually interpreted as recording of a pathway of excitation with prolonged conduction time. Because it has been suggested that they may also represent recordings of artifacts caused by movement at the electrode-myocardium interface or by activity occurring far away from the recording electrodes, the significance of such electrograms has been a source of controversy.26 In fact, localized slow conduction is most likely to occur in our cases as well as in similar previously published studies because 1) the relevant electrogram was selectively recorded in certain areas of the endocardial right atrium without any associated catheter motion, 2) slight displacements of the catheter were consistently associated with immediate reappearance of local narrow electrograms, 3) reproducible patterns of second-degree block were locally recorded either spontaneously or during tachycardia entrainment—the striking regularity with which these patterns were reproduced (especially the Wenckebach patterns or the 2:1 during irregular atrioventricular nodal transmission [Figure 3]) allowed us for exclusion of transient loss of contact with the endocardial wall, and 4) similar electrograms were previously recorded in atrial flutter.4,6,7 Considering the specific forms of fragmentation that are the double-spike electrograms, if they indeed represent slow yet linear conduction between two myocardial areas,7 a proposed alternative interpretation is that they represent recording of two nonlinearly linked wave fronts of excitation. In a series of interesting studies by Cosio et al with a recording technique similar to ours, the investigators found a high prevalence of fragmented or multiple-spike electrograms in the low septal and posterior right atrium. They were not able to determine whether these electrograms represented an intracircuit conduction delay (in which case, the double spike should be the image of the entrance and exit of an activation front crossing a zone of slow conduction) or an anatomical barrier enlarging the central anatomical obstacle made by the inferior vena cava (in which case they may represent two wave fronts of activation traveling in opposite directions).4,7 Intracardiac recording techniques do not seem to be refined enough to make such a distinction. We believe that the clinical relevance of such electrograms (i.e., the ability to demonstrate that one area is the critical slow conduction area of a given reentrant circuit)
may be approached indirectly. The demonstration of dissociation of these potentials from tachycardia must identify standby pathways of excitation. This is obvious in Figure 2, in which every sixth potential is lacking without affecting atrial flutter cycle length. This can be further demonstrated by straightforward pacing when entrainment criteria are fulfilled, whereas concomitant endocardial potentials dissociate (Figure 3). Indeed, dissociation of the fragmented potentials in the low posteroseptal area at the site of ablation was never observed either spontaneously or during tachycardia entrainment. Conversely, we were always able to dissociate the fragmented electrogram that was outside the posteroseptal region, but this may not necessarily always be the case. In a previous mapping study of seven patients with type I atrial flutter, all had fragmented potentials in the posteroseptal region, and three had an additional zone of fragmentation in the sinus node area. In one out of three patients, we were not able to dissociate this area from the tachycardia.6 A similar approach has been used by Fitzgerald et al27 in a search of the critical slow conduction zone of reentry in patients suffering from ventricular tachycardia. Shocks delivered in this area proved to be highly successful in preventing the recurrences of this arrhythmia, suggesting that these potentials may indeed be clinically relevant.

**Catheter Ablation of the Human Right Atrium**

With the development of catheter ablation techniques, direct ablation of tachycardia pathways or foci has grown as a challenging possibility to cure arrhythmias without resorting to open heart surgery or permanent cardiac pacing. The crucial question of the safety of such approaches arises, especially when applying DC shock to the relatively thin atrial wall. Animal studies28–30 have shown a direct relation between the strength of the electrical shocks and the extent and severity of the myocardial injury, but perforation of the atrial wall did not occur when energies of less than 200 J were delivered in puppies with 3 kg body wt. These authors, therefore, recommended this amount of energy as a safe upper limit in small subjects. Atrial ablation has already been performed in humans for other types of atrial tachycardias without complication and with apparently good long-term results using delivered energy up to 400 J in adults.31–36 We chose to limit the energy to 2.5 J/kg because we believe it yields a good safety margin for this procedure in the relatively thick area surrounding the coronary sinus ostium. Cardiac tamponade has been encountered in ablation of posteroseptal accessory pathways when ablation was performed via the proximal part of a catheter electrode introduced inside the coronary sinus os.37 Such complications, in fact, did not occur in larger series of patients if the ablation shocks were delivered at the coronary sinus os without direct catheterization of this structure.38

**Mechanism of Action of Atrial Ablation**

The precise mechanism by which atrial flutter recurrences were prevented in this series is unknown. We never observed significant short-term or long-term modifications of P wave morphology. Limited but significant areas of atrial myocardium necrosis probably occurred in four patients in whom creatine kinase (CK) levels rose slightly after the shocks, thereby supporting true ablation as the intimate mechanism of this intervention. For now and in this small series, we were not able to find any set of clinical or electrophysiological findings that were predictive of short-term or long-term success. Additionally, neither the amount of delivered energy nor the magnitude of CK rise correlated with lack of flutter recurrences. Conversely, at the time of post-ABLation electrophysiological study, in the patients with inducible flutter (1 and 3), no significant change in electrogram morphology or stimulation protocol could be found between ablative and postablation study. However, patient 1 was already in flutter at the beginning of the first session, and amiodarone was withdrawn between ablation and repeat study, therefore rendering any electrophysiological comparison difficult. These as well as those with spontaneous atrial flutter at the beginning of the second procedure (patients 4 and 7) were not different than before ablation with regard to the presence of a zone of low posteroseptal fragmentation and the ability to demonstrate slow conduction in this area. The fact that atrial flutter remained inducible in two of five patients at late electrophysiological study despite the absence of any spontaneous tachycardia recurrence suggests that catheter-induced modification of the arrhythmia substrate may be minor yet sufficient to prevent relapses. In addition to catheter-induced atrial myocardium necrosis, other mechanisms may have been operative in this case. In a recent report, Levy et al37 delivered intracavitary atrial DC shocks in an attempt to restore normal sinus rhythm in external DC shock-resistant patients with chronic atrial fibrillation. Surprisingly, it was found that in addition to the high success rate of atrial defibrillation, such an intervention prevented further long-term arrhythmic recurrences.31 A sudden increase in intracavitary atrial pressure was believed to be responsible for this effect. Part of a similar mechanism could be invoked in some of our patients.

**Conclusions**

These results suggest that direct atrial ablation may be useful in patients with symptomatic and drug-resistant type I atrial flutter. Low voltage, fragmentation, and prolongation of local electrograms are consistently found in the area of the low posteroseptal right atrium, and ablation shocks delivered in this area seem to prevent atrial flutter recurrences without the need for definite His bundle ablation. Pacemaker therapy may, therefore, be avoided, and the normal atrioventricular sequence may be pre-
served. Although based on a limited number of patients, these preliminary results are encouraging. Further studies are required to confirm these results.

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References

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