Radiofrequency Catheter Ablation for the Treatment of Human Type 1 Atrial Flutter
Identification of a Critical Zone in the Reentrant Circuit by Endocardial Mapping Techniques

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Background. Recent studies of human type 1 atrial flutter demonstrated reentry in the right atrium and an area of slow conduction in the low posteroseptal right atrium. Direct-current catheter ablation of this area has been only moderately successful in preventing recurrence. Therefore, we performed endocardial activation mapping and entrainment pace mapping during atrial flutter to determine the critical site for radiofrequency ablation of this arrhythmia. Methods and Results: Twelve consecutive patients (seven men and five women; age, 21-73 years) with type 1 atrial flutter (mean cycle length, 253±39 msec) underwent right atrial endocardial activation and entrainment pace mapping using standard transvenous catheter techniques to localize the atrial flutter reentrant circuit, the area of slow conduction, and the exit site from the area of slow conduction. Upon identifying appropriate sites, radiofrequency energy (16-29 W) was applied via a 4-mm tipped catheter. Activation mapping of atrial flutter revealed a counterclockwise reentrant wave front originating just inferior or posterior to the coronary sinus ostium, proceeding superiorly in the atrial septum to the right atrial free wall, then inferiorly toward the tricuspid annulus and finally medially between the inferior vena cava and the tricuspid annulus, where low-amplitude fragmented electrical activity was noted. Entrainment pace mapping from this area produced an exact P wave match to atrial flutter on 12-lead ECG with a long (>40 msec) stimulus-to-P interval indicating slow conduction, whereas pacing just inferior or posterior to the coronary sinus ostium produced an exact P wave match with a short stimulus-to-P interval (<40 msec), presumably identifying the exit site from the area of slow conduction. Radiofrequency energy (one to 14 applications) was effective in terminating and preventing reinduction of atrial flutter in 10 patients. In two patients, atrial flutter was not terminated during radiofrequency energy application but during subsequent pacing attempts. Sites where ablation was successful, located just inferior or posterior to the coronary sinus ostium, were characterized by discrete electrograms with activation times of -20 to -50 msec before P wave onset and exact entrainment pace maps with a stimulus-to-P interval of 20 to 40 msec, consistent with the exit site from the area of slow conduction. Follow-up (mean, 16±9 weeks; range, 2–31 weeks) revealed recurrence of the original atrial flutter in two patients, one of whom underwent repeat ablation without further recurrence, self-limited infrequent recurrence of a new atrial flutter or atrial fibrillation in three suppressed by β-blocker or digoxin, and no recurrence in seven.

Conclusions. 1) Radiofrequency energy applied to a critical area in the atrial flutter reentrant circuit, inferior or posterior to the coronary sinus ostium, will terminate and prevent arrhythmia reinduction. 2) Long-term follow-up in a larger series of patients will be required to confirm efficacy of this technique, although short-term results look promising. (Circulation 1992;86:1233–1240)

Key Words • atrial flutter • radiofrequency ablation

Recent studies in humans have demonstrated that type 1 atrial flutter is due to reentry in the right atrium and that an area of slow conduction is present in the reentry circuit.1-6 This area of slow conduction, identified by prolonged low-amplitude fragmented electrical activity and long stimulus-to-P wave intervals during pacing entrainment, is located in the low posteroseptal right atrium.5-6 Studies in a limited number of patients have shown that direct-current (DC) catheter ablation near this region of slow conduction may prevent recurrence of atrial flutter.7,8 However,
TABLE 1. Clinical Characteristics of Patients With AFL

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Cardiovascular diagnosis</th>
<th>AFCL (msec)</th>
</tr>
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<tr>
<td>1</td>
<td>52</td>
<td>M</td>
<td>DCM</td>
<td>240</td>
</tr>
<tr>
<td>2</td>
<td>73</td>
<td>F</td>
<td>None</td>
<td>220</td>
</tr>
<tr>
<td>3</td>
<td>38</td>
<td>M</td>
<td>DCM</td>
<td>260</td>
</tr>
<tr>
<td>4</td>
<td>69</td>
<td>M</td>
<td>None</td>
<td>220</td>
</tr>
<tr>
<td>5</td>
<td>66</td>
<td>F</td>
<td>ATP</td>
<td>270</td>
</tr>
<tr>
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<td>260</td>
</tr>
<tr>
<td>7</td>
<td>70</td>
<td>F</td>
<td>CABG</td>
<td>250</td>
</tr>
<tr>
<td>8</td>
<td>71</td>
<td>F</td>
<td>None</td>
<td>300</td>
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<td>240</td>
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<td>11</td>
<td>61</td>
<td>M</td>
<td>PHTN</td>
<td>350</td>
</tr>
<tr>
<td>12</td>
<td>38</td>
<td>M</td>
<td>None</td>
<td>200</td>
</tr>
</tbody>
</table>

AFCL, atrial flutter cycle length in msec; ATP, antitachycardia pacemaker; CABG, history of coronary bypass surgery; DCM, dilated cardiomyopathy; PHTN, idiopathic pulmonary hypertension.

because endocardial DC shock produces distant damage due to barotrauma, in addition to a large local area of ablation and can immediately convert atrial flutter to sinus rhythm, this technique does not allow precise localization of the critical zone of the reentrant circuit.\(^7,8\) Therefore, we postulated that a combination of atrial endocardial activation and entrainment pace mapping in patients with type 1 atrial flutter would allow localization of a critical area of slow conduction in the right atrium. If application of radiofrequency energy near this area would result in termination of atrial flutter and prevention of its recurrence, this would confirm that a discrete area of tissue is critical to the reentrant mechanism.

Methods

Patient Characteristics

Twelve consecutive patients referred for evaluation of type 1 atrial flutter underwent endocardial atrial mapping and radiofrequency catheter ablation at UCSF Medical Center from October 1, 1991 to May 6, 1992 (see Table 1). Entry into the study required that patients have a documented history of type 1 atrial flutter, with P waves inverted in a typical "saw-tooth" pattern in ECG leads II, III, and aVF, biphasic in I and V, and upright in V. Seven men and five women, age 21–73 years (mean, 56±17 years), entered the study. Eight patients presented with persistent atrial flutter; two were unresponsive to antiarrhythmic drugs (patients 1 and 11), one had received no therapy (patient 2), and five had received after electrical or pharmacological cardioversion despite chronic antiarrhythmic therapy (patients 3, 4, 7, 9, 12). Four patients (patients 5, 6, 8, 10) presented with paroxysmal atrial flutter that recurred daily to several times weekly despite chronic antiarrhythmic therapy. Three patients (patients 5, 6, 8) also had episodes of paroxysmal atrial fibrillation previously documented, although atrial flutter was their predominant arrhythmia. Patient 5 had an antitachycardia pacemaker but underwent attempted ablation because of the large number of episodes of atrial flutter requiring pace termination per day. Patient 7, who developed symptomatic palpitations after recurrence of sustained atrial flutter despite previous AV node ablation and decreased exercise tolerance presumably caused by VVIR pacemaker implantation, underwent attempted atrial flutter ablation with the intent to upgrade the pacemaker to a dual chamber system.

Endocardial Mapping Technique

Patients were studied under light sedation and local anesthesia. Antiarrhythmic drugs were withdrawn 24–48 hours before study. Standard 6F quadripolar electrode catheters (Bard Electrophysiology, Tewksbury, MA) were positioned in the right atrium, and a bipolar pacing electrode was placed in the right ventricle. The atrial activation time was determined by pacing from the coronary sinus ostium; follow-up in weeks, time since initial ablation (time to recurrence of original atrial flutter); Inf, inferior; Post, posterior; *AFL terminated during pacing. Note that patient 6 had a successful second RF ablation without further recurrence, whereas patient 8 opted for AV node ablation and VVIR pacemaker implantation since.
bury, Mass.) were positioned in the right ventricle and His bundle region via the left or right femoral veins and the coronary sinus via the right internal jugular vein using Seldinger technique. A 7F steerable, 4-mm tipped, quadrupolar electrode catheter with 2-mm inter-electrode spacing (Mansfield, Inc., Watertown, Mass.), was used for mapping and radiofrequency ablation, was inserted into the right atrium via the right femoral vein.

Eight patients (patients 1, 2, 3, 4, 7, 9, 11, 12) were in sustained atrial flutter at the time of the study. In the four patients (patients 5, 6, 8, 10) who were in sinus rhythm, sustained atrial flutter was reproducibly induced by programmed stimulation before mapping and ablation (see method of induction described below). Using biplane fluoroscopy, the right atrial endocardium was mapped by rotating the steerable catheter in the anterolateral, anterior, anterolateral, lateral, posterolateral, posterior, posteromedial, and septal positions at four levels, beginning high near the superior vena cava and then withdrawing 1 cm at a time to the inferior vena cava (i.e., high, −1, −2, −3 cm). The left atrium was recorded via the coronary sinus catheter. Local activation time, defined as the maximal slope of the endocardial electrogram as it crossed the baseline, was referenced to the earliest onset of the P wave during atrial flutter in surface ECG leads I, aVF and V1. An activation sequence map was constructed manually to determine the apparent location of the reentrant circuit and earliest atrial activation as well as any area of low-amplitude fragmented electrical activity or double potentials.

Endocardial entrainment pace mapping was then performed during atrial flutter via the distal electrode pair of the mapping catheter at areas of early activation or apparent slow conduction by pacing at cycle lengths 30–50 msec shorter than the atrial flutter cycle length. During entrainment pace mapping, 12-lead ECGs were recorded and compared with baseline atrial flutter. An exact entrainment pace map was defined as one in which the P wave morphology was unchanged during pacing compared with baseline in all 12 leads. Overt entrainment of atrial flutter was defined as acceleration

**FIGURE 2.** Panel A: Electrograms from the coronary sinus, high right atrium, His bundle (proximal electrodes), and right ventricular catheters in patient 1. Note that the earliest atrial activation referenced from P wave onset is at the proximal coronary sinus electrode. Panel B: Prolonged, low-amplitude, fragmented electrograms are recorded from the isthmus between the inferior vena cava and tricuspid annulus in the low posterior right atrium. Note the continuous electrical activity for 100 msec spanning the onset of the P wave, suggesting that this is in the area of slow conduction. Panel C: For greater stability, the ablation catheter has been repositioned via the right internal jugular vein after removal of coronary sinus catheter, with the distal 4-mm tip positioned just inferior to ostium of coronary sinus. Note the early timing of this electrogram relative to P wave onset and its narrowly split second component. I, II, III, aVF (F), V1; surface ECG leads; HRA, high right atrium; CS, cSp,m,d; electrograms from coronary sinus proximal, middle, and distal electrode pairs respectively; CSw, electrogram from radiofrequency ablation catheter just posterior and inferior to coronary sinus ostium; HIS, electrogram from proximal electrode pair of His bundle catheter; RV, right ventricular electrogram; LPRA, low posterior right atrial electrogram.
of the tachycardia to the pacing length with constant fusion of P waves and without termination of atrial flutter upon cessation of pacing. Concealed entrainment was defined as acceleration of the tachycardia to the pacing length without alteration of P wave morphology or endocardial activation sequence and without termination of atrial flutter upon cessation of pacing. An area of slow conduction was presumed to be present at a
specific site if an exact entrainment pace map with a long stimulus-to-P wave interval (>40 msec) was noted during pacing. The presumed exit site from the area of slow conduction was identified by the presence of an exact entrainment pace map with a short stimulus-to-P wave interval (<40 msec).

Radiofrequency Ablation Technique

Radiofrequency ablation was performed using a radiofrequency generator providing 550 kHz, unmodulated sine wave energy (American Cardiac Ablation Corp., Inc., Taunton, Mass.) connected to the distal 4-mm tip of the mapping/ablation catheter via a switch box and grounded to the patient's skin using a standard surgical electrosurgery grounding pad. Applied voltage and measured current were displayed continuously on a strip-chart recorder (EVR-18, PPG, Inc., Pleasantville, N.Y.), and impedance was monitored by meter on the radiofrequency generator. Radiofrequency energy was applied in a range from 40 to 60 V, producing 16 to 29 W power for 30 to 60 seconds or until an impedance rise was observed. If atrial flutter terminated during energy application, a full 60-second application was performed at that site. Because ablation within the area of slow conduction was not effective in terminating atrial flutter in the first two patients, attempts were made to initially target the exit site from the area of slow conduction near the ostium of the coronary sinus, in patients 3–11. If an ablation attempt at a particular site was unsuccessful in terminating atrial flutter, local activation and entrainment pace mapping was repeated in an attempt to identify an area with earlier atrial activation and a more exact entrainment pace map by systematically repositioning the ablation catheter several millimeters superior or inferior and then anterior or posterior to the initial position. Ablation was then reattempted at sites demonstrating earlier activation and a more exact entrainment pace map than the previous site. The duration of the procedure was limited by design to a maximum of 4 hours or 90 minutes of fluoroscopy time in order to minimize risk to the patient and physicians.

After successful ablation of atrial flutter, programmed stimulation was performed immediately and after approximately 15–30 minutes to determine if atrial flutter was inducible. Pacing was performed at twice diastolic threshold from the high right atrium and coronary sinus catheters, with single and double premature stimuli (S1S2 and S2S3) introduced after a 10-beat drive (S1S3) at three cycle lengths (600, 500, and 400 msec) to refractoriness. Burst pacing was also performed at two sites (10–20 beats) at progressively shorter cycle lengths until 2:1 capture was noted. After completion of the study, patients were monitored on telemetry overnight and discharged home the following day.

Patient Follow-up

All patients were seen in the outpatient clinic at 1 week, 1 month, and then every 3 months, at which time a history of recent symptoms was taken and an ECG and physical examination were performed. Patients were instructed to report any symptoms suggestive of recurrent arrhythmia, at which time an ECG and Holter monitor would be performed and a cardiofrequency monitor would be issued for evaluation of further symptoms. Patients were followed on no antiarrhythmic medications. Aspirin 325 mg per day was prescribed for the first 3 months after ablation in the event of recurrence of atrial flutter to reduce the risk for systemic embolism.

This study was approved by the Human Subjects Committee at the University of California, San Diego, School of Medicine. All patients gave written informed consent.

Results

Results of Endocardial Mapping and Entrainment Pace Mapping

Mean atrial flutter cycle length was 253 ± 39 msec (range, 200–350 msec) at the time of electrophysiology study (Table 1). In each patient, endocardial mapping revealed a reentrant circuit in the right atrium, rotating counterclockwise when viewed from anterior to the patient (Figure 1). Earliest atrial activation (Figures 2A and 2B), typically 20–50 msec before P wave onset, was recorded near the ostium of the coronary sinus in all patients (Table 2). The activation wave front then proceeded superiorly around the foramen ovale in the interatrial septum, posteriorly and laterally to the high right atrial free wall, inferiorly toward the tricuspid annulus, medially through the isthmus between the inferior vena cava and tricuspid annulus where prolonged low-amplitude fragmented electrical activity was observed, returning finally to the region of the coronary sinus ostium. Low-amplitude fragmented electrical activity recorded near the isthmus between the inferior vena cava and tricuspid annulus was prolonged up to 100 msec and spanned the onset of the surface ECG P wave (Figure 2C).

Multiple entrainment pace maps were obtained at sites of early activation near the coronary sinus ostium and at sites of low amplitude fragmented electrical activity near the isthmus between the inferior vena cava and tricuspid annulus. In each patient, one or more sites could be identified at which pacing entrainment produced an exact or nearly exact pace map (Figures 3A and 3B), with P waves unchanged from those during atrial flutter (i.e., concealed entrainment). These sites were always near the coronary sinus ostium or between the coronary sinus ostium and the isthmus between the inferior vena cava and tricuspid annulus. The farther laterally in the isthmus the pacing site was, the longer the stimulus-to-P wave interval, up to 80 msec. At other sites in the atrium, constant fusion of the P wave was always noted during entrainment pace mapping (i.e., overt entrainment).

Results of Radiofrequency Catheter Ablation of Atrial Flutter

Radiofrequency ablation, attempted at sites of early activation with exact or near exact entrainment pace
maps, was successful in terminating atrial flutter in 10 of 12 patients after one to 14 applications of energy (Figures 4A and 4B). (Also see Table 2.) After ablation, sustained atrial flutter was no longer inducible by programmed stimulation in these 10 patients. In two patients, atrial flutter was not terminated by multiple energy applications but terminated during subsequent pacing attempts and was no longer inducible. Analysis of recordings from sites where ablation was successful revealed discrete or narrowly split electrograms with activation times between −20 to −50 msec before P wave onset. At these sites exact entrainment pace maps with a stimulus-to-P wave interval of 20–40 msec were obtained. These sites were always located just inferior
or posterior to the ostium of the coronary sinus (Figures 1, 5A, and 5B), at the exit of the isthmus between the inferior vena cava and tricuspid annulus into the interatrial septum (i.e., the exit site from the area of slow conduction). Radiofrequency ablation was not successful when applied at sites with prolonged, low-amplitude, fragmented electrical activity, and an exact entrainment pace map with a long stimulus-to-P wave interval (i.e., in or proximal to the area of slow conduction). There were no acute complications noted in any patient during or immediately after the procedure.

Follow-up of Patients Undergoing Successful Radiofrequency Catheter Ablation of Atrial Flutter

All 12 patients have been observed in the outpatient clinic for a mean of 16±9 weeks (range, 2–31 weeks) since their procedures. There have been no late complications. Two patients (patients 6 and 8) had recurrence of their original atrial flutter within approximately 2 weeks after ablation. Patient 6 underwent repeat ablation without further recurrence. Patient 8 opted for AV node ablation and VVIR pacemaker implantation. Two patients (patients 1 and 4) had a single self-limited episode of a new morphology of atrial flutter after ablation, which has not recurred since on therapy with digoxin (patient 1) or a β-blocker (patient 4), respectively. Patient 5 continues to have daily self-limited episodes of atrial fibrillation, but she is asymptomatic on β-blocker therapy. Seven patients (patients 2, 3, 7, 9, 10, 11, 12), including those two (patients 2 and 7) in whom atrial flutter was not terminated abruptly during radiofrequency ablation, have had no recurrences of atrial flutter.

Discussion

This study supports the findings of others that type 1 atrial flutter in humans is caused by reentry in the right atrium and demonstrates that a critical area in the reentrant circuit can be identified and modified with radiofrequency energy to terminate and prevent recurrence of the arrhythmia. This critical area, probably the exit site from the area of slow conduction, is located inferior and posterior to the coronary sinus ostium and

FIGURE 5. Panel A: 30° right anterior oblique and panel B: 45° left anterior oblique fluoroscopic views of the heart showing positions of the coronary sinus (CS), right ventricular (RV), His bundle (HIS) and 4-mm tipped ablation catheter (RF) in patient 10. Note that the tip of the ablation catheter is located just inferior to the coronary sinus ostium. At this site, early activation and concealed entrainment with an exact P wave match and short stimulus-to–P wave interval were noted. Radiofrequency energy application at this site terminated atrial flutter.
just anterior to the narrow isthmus of tissue between the inferior vena cava and the tricuspid annulus where slow conduction is observed.

**Activation and Entrainment Pace Mapping of Atrial Flutter**

In each patient, P wave morphology on 12-lead ECG suggested a posteroseptal origin of earliest activation, with P waves inverted in inferior leads II, III and aVF, biphasic in I and V6, and upright in V1. Activation mapping confirmed that a reentrant circuit was located in the right atrium and that earliest activation was always located inferior or posterior to the coronary sinus ostium. Concealed entrainment with a long stimulus-to-P wave interval was demonstrated during pacing near the isthmus between the inferior vena cava and the tricuspid annulus in all patients, suggesting that this is an area of slow conduction in which unidirectional block of the antidromic paced wave front probably occurs. Furthermore, concealed entrainment with a short stimulus-to-P wave interval was observed just anterior to the area of slow conduction, suggesting that this may be the exit site from the area of slow conduction. These observations are consistent with previous endocardial mapping studies in atrial flutter, particularly with respect to the presence of an area of slow conduction in the low posteroseptal right atrium.

These findings also suggested that the area of slow conduction might be critical to sustaining reentry and that the area of slow conduction or the exit site from the area of slow conduction might be potential targets for radiofrequency catheter ablation.

**Radiofrequency Ablation of Atrial Flutter**

This study has demonstrated that radiofrequency energy applied to a discrete area in the low posteroseptal right atrium can terminate atrial flutter and prevent its immediate reinduction by programmed stimulation. Radiofrequency ablation was successful at sites demonstrating earliest activation with a discrete electrogram and an exact entrainment pace map with a relatively short stimulus-to-P wave interval of 20–40 msec, suggesting that these sites were at the exit from the area of slow conduction.

Successful ablation of atrial flutter using DC shock applied to a similar location has also been reported. However, the number of patients was small, and late recurrence rates have been significant. Furthermore, because DC shock immediately cardioverts atrial flutter and because its electrical and barotraumatic effects may not be as localized as those of radiofrequency energy, questions have remained as to where the critical site for ablation of atrial flutter is located. This study confirms that ablation of a relatively small area of endocardium will terminate and prevent recurrence of atrial flutter. This study also suggests that a critical portion of the reentrant circuit is anatomically defined, which may account for the consistent morphology and rate of atrial flutter among all patients.

**Clinical Implications and Limitations of the Study**

The duration of follow-up in this study is relatively short, but the results are encouraging. Eleven of 12 patients ultimately had elimination of symptomatic recurrences of atrial flutter after radiofrequency ablation, whereas one patient opted for AV node ablation and VVI pacemaker implantation after recurrence. Longer follow-up will clearly be required to confirm that this form of therapy is an effective alternative to currently available treatment, because late recurrences of new atrial flutter morphologies or atrial fibrillation may limit its usefulness.

**References**

Radiofrequency catheter ablation for the treatment of human type 1 atrial flutter. Identification of a critical zone in the reentrant circuit by endocardial mapping techniques.

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