Radiofrequency Catheter Ablation for the Treatment of Common Atrial Flutter in Humans

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Endocardial catheter ablation has been recently been proposed for the treatment of arrhythmias originating in the right atrium. In this study, we used this technique in 5 patients with paroxysmal common atrial flutter and 3 patients with paroxysmal uncommon atrial flutter. Various antiarrhythmic agents had failed to prevent the recurrence of these episodes. In each procedure, we used a large-tip 7 F quadripolar catheter electrode that was introduced via the femoral vein into the lower part of the right atrium. The two distal electrodes were used to record double-spike potentials or fragmented electrograms. A unipolar electrogram recording from a distal electrode of the same catheter was also used to identify local activation. The targets for ablation were sites which showed double-spike potential and fragmented electrogram 40–60 msec earlier than the onset of the F wave. Application of radiofrequency (RF) energy (25–40 watts) (3–17 applications) terminated atrial flutter and prevented reinduction of atrial flutter in the 5 patients with common atrial flutter. However, atrial flutter could not be terminated with the application of RF energy in the 3 patients with uncommon atrial flutter. The sites at which ablation was successful were located inferior or posterior to the coronary sinus ostium between the inferior vena cava and the tricuspid valve annulus, and were characterized by double-spike potentials and fragmented electrograms with activation times ≥40 msec before the onset of the F wave. This area may represent the exit site from the area of slow conduction, since pacing from this site showed concealed entrainment of the F wave, and a local electrogram to the onset of the F wave coincided with the pacing spike to the onset of the F wave. Follow-up of these 5 patients (19.4 ± 10.4 weeks) revealed recurrence of the original atrial flutter in 1 patient and a new type of atrial flutter in 1 patient. The other 3 patients have been episode-free, although an antiarrhythmic agent was given for the treatment of paroxysmal atrial fibrillation in 2 patients. We conclude that the application of RF energy to the presumed critical area in the atrial flutter reentrant circuit seems to be effective in terminating and preventing common atrial flutter. Long-term follow-up is required for the recurrence of atrial flutter.

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Atrial flutter is a common type of supraventricular tachycardia. Treatment for common atrial flutter includes transesophageal or endocardial overdrive stimulation, antiarrhythmic drugs and direct current (DC) cardioversion. Transesophageal

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Radiofrequency Catheter Ablation of Atrial Flutter

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![ECG traces](image)

Fig.1. 12-lead ECG from patient 7 demonstrating typical sawtooth type I atrial flutter (common flutter) at a cycle length of 217 msec, with F waves inverted in leads II, III, and aVF, biphasic in leads I and V6, and upright in lead VI.

al and endocardial overdrive stimulation are usually used during electrophysiologic study in the electrophysiology laboratory. DC cardioversion is highly successful in restoring sinus rhythm, however, recurrence is common. Drug therapy is usually required to prevent recurrence. Although a growing number of pharmacological agents are available for the treatment of arrhythmia, some patients are either intolerant of drug treatment or achieve only inadequate relief from their symptoms.

Recently, catheter ablation has become an attractive nonpharmacological therapy for selected patients with drug-refractory arrhythmias.\(^1\)\(^2\)

Electrical ablation of the atroioventricular junction, accessory atroioventricular pathway, selected slow pathway and fast pathway of atroioventricular node reciprocating tachycardia, and ventricular arrhythmogenic substrates are now performed routinely in clinical electrophysiology laboratories. However, the role of ablation in atrial flutter remains to be elucidated.\(^3\)\(^-\)\(^10\)

Atroioventricular node/His bundle ablation has been used to interrupt atroioventricular conduction to treat patients with drug-refractory atrial flutter. However, this is not an ideal solution, since it does not treat the flutter itself and requires the implantation of a permanent pacemaker. Based on the improved understanding of the mechanisms of flutter gained from animal models of this arrhythmia and from human intracardiac electrophysiologic studies, typical atrial flutter (ie, common atrial flutter, or type I atrial flutter) has been shown to consist of macroreentry in the right atrium, with the direction of the circuit being caudocranial in the septum and craniocaudal in the anterolateral wall, and with an area of slow conduction posterior-inferiorly in the triangle of Koch.\(^1\)\(^1\)\(^-\)\(^16\)

More recently, endocardial activation sequence mapping and pacing techniques have suggested that a large right atrial reentry circuit during atrial flutter surrounds a central obstacle formed by the inferior vena cava (IVC) and adjacent areas of functional conduction block.\(^17\)\(^-\)\(^20\) These findings encouraged researchers to attempt direct catheter ablation of this atrial area using DC or radiofrequency (RF) energy as a mode of treatment for drug-refractory common type atrial flutter.\(^3\)\(^-\)\(^10\) This report describes the results of RF ablation for atrial flutter.

METHODS

Patient Characteristics

The study population consisted of 8 consecutive patients (7 men and 1 woman, aged 16–68 years, mean ± SD, 47 ± 17 years) who were referred for evaluation of atrial flutter and underwent endocardial atrial mapping and RF catheter ablation at Nihon University School of Medicine Itabashi Hospital from July, 1992 to May, 1993. Seven patients had a documented history of common atrial flutter with inverted F waves in ECG leads
<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Cardiovascular diagnosis</th>
<th>ECG</th>
<th>AFL CL (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>65</td>
<td>M</td>
<td>None</td>
<td>Af Common AFL</td>
<td>238</td>
</tr>
<tr>
<td>2</td>
<td>16</td>
<td>M</td>
<td>None</td>
<td>Common AFL</td>
<td>235</td>
</tr>
<tr>
<td>3</td>
<td>47</td>
<td>M</td>
<td>None</td>
<td>Af Common AFL</td>
<td>216</td>
</tr>
<tr>
<td>4</td>
<td>68</td>
<td>M</td>
<td>Hypertension</td>
<td>Uncommon AFL</td>
<td>276</td>
</tr>
<tr>
<td>5</td>
<td>68</td>
<td>M</td>
<td>None</td>
<td>Af</td>
<td>221</td>
</tr>
<tr>
<td>6</td>
<td>32</td>
<td>F</td>
<td>Post VSD operation</td>
<td>Uncommon AFL</td>
<td>222</td>
</tr>
<tr>
<td>7</td>
<td>38</td>
<td>M</td>
<td>None</td>
<td>Af</td>
<td>214</td>
</tr>
<tr>
<td>8</td>
<td>42</td>
<td>M</td>
<td>WPW</td>
<td>Common AFL</td>
<td>212</td>
</tr>
</tbody>
</table>

AFL: atrial flutter, Af: atrial fibrillation, VSD: ventricular septal defect, AFL CL: atrial flutter cycle length

II, III, and aVF (Fig. 1) Only 1 patient (patient 4) had a history of uncommon atrial flutter with the F wave upright in ECG leads II, III and aVF. All of the patients were symptomatic, and complained of either palpitation, syncope, or symptoms of heart failure due to episodes of 1:1 atrioventricular conduction. None of the patients presented with persistent atrial flutter. No structural heart disease could be found in 6 patients. One patient had systemic hypertension, 1 patient was associated with B type Wolf-Parkinson-White syndrome, and 1 patient had been accompanied with ventricular septum defect which had undergone surgery 23 years previously. Paroxysmal atrial fibrillation accompanied in 4 patients (patients 1, 3, 5 and 7) (Table I). Patients were resistant to three or more antiarrhythmic drugs, including class 1a (quinidine, procainamide, and disopyramide), class 1c (flecainide and propafenone), beta-blocking agents, calcium blockers (verapamil), and digitalis. Amiodarone was also used either alone or in combination.

Electrophysiologic Study Procedure
The study protocol was approved by the Nihon University School of Medicine, Itabashi Hospital. After providing their written informed consent, each patient was studied in the postabsorptive state. All antiarrhythmic medications were discontinued at least 36 h before the procedure. All patients were studied under heavy sedation with fentanyl (50 to 150 μg per hour) and midazolam (2 to 6 mg per hour). Two standard 6F quadripolar electrode catheters (Bard Electrophysiology, Tewksbury, Mass) were inserted percutaneously into the right and left femoral veins and advanced to right ventricular apex and high right atrium. A 6F hexapolar electrode catheter was placed across the tricuspid valve to record His bundle activation. A 7F quadripolar deflectable-tip catheter with a 4 mm distal electrode (EP Technologies Inc., or Webster Laboratories, Inc.) was positioned in the low septal right atrium and used for mapping and delivery of RF energy. Another 7F hexapolar electrode catheter with a lumen was introduced into the right internal jugular vein and placed through the coronary sinus. Radiopaque contrast was injected into this catheter to locate the coronary sinus ostium.

Electrocardiographic leads II and VI and intracardiac electrograms were displayed on an oscilloscope and recorded on an 8-channel recorder (MIC-9800, CA-202, Fukuda Denshi Corp., Tokyo, Japan) or on a 12-channel recorder (RCM-2000, Nihon Koden Corp., Tokyo, Japan) at a paper speed of 100 to 200 mm per second. Pacing was performed with a programmable stimulator.
(BC-02A, Fukuda Denshi corp., Tokyo, Japan) using stimuli of 2 msec duration at twice the diastolic threshold from the high right atrium and coronary sinus catheters.

Refractory periods were determined at two cycle lengths (600 and 400 msec). If atrial flutter was not induced by these maneuvers, programmed stimulation was performed with double premature stimuli or burst rapid pacing. Pacing was repeated during the infusion of isoproterenol at a rate of 1 to 2 μg per minute if atrial flutter could not be induced in the baseline state. In 3 patients, programmed stimulation was performed only after ablation of atrial flutter because they presented with atrial flutter when they came to the electrophysiology laboratory. The end-point of the pacing protocol was the attainment of a pacing cycle length of 180 msec (shorter cycles were not used to avoid pacing-induced atrial fibrillation).

**Endocardial Mapping**

Endocardial mapping was then performed during either spontaneous or induced atrial flutter using the distal pair of electrodes of the 7F steerable, 4-mm tip quadrupolar electrode catheter at the area surrounding the coronary sinus ostium between the IVC and the tricuspid valve annulus to record double-spike potentials and fragmented electrograms. We also recorded unipolar electrograms (0.3–500 Hz) from distal electrode (Fig. 2). Endocardial entrainment pacing during atrial flutter was then performed from high right atrium and the site at which double-spike potentials, or fragmented electrograms were recorded in some patients (patients 1, 2, 4, 6, 7 and 8). Entrainment pacing was not performed in 2 patients (patients 3 and 5) since it was easy to induce paroxysmal atrial fibrillation.

Transient entrainment of atrial flutter was judged to have occurred when at least one of the following phenomena was observed: constant fusion beats between flutter and 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 short conduction time, or a change in conduction time and electrogram morphology. Concealed entrainment was defined as 1) acceleration of the tachycardia to the pacing length without alteration of F wave morphology and without termination of atrial flutter upon cessation of pacing and 2) the pacing spike to the onset of the F wave coincides with the first component of the local electrogram to the onset of the F wave. During entrainment pacing, 12-lead ECGs were recorded and compared with baseline atrial flutter in 3 patients (patients 1, 7 and 8) (Fig. 3).

An area of slow conduction was defined as a site at which concealed entrainment was observed. The presumed exit site from the area of slow conduction was defined as an area which showed atrial activation 40–60 msec before the onset of the F wave. The onset of the F wave was identified from surface electrogram II or III as the onset of negative deflection.

**Radiofrequency Ablation Procedure**

RF ablation was performed using radiofrequency generator which provided 520 KHz, continuous, and unmodulated sine wave energy (HAT 200S, Osypka; NL-50-I, Japan Life Line; or Nova Frame RA-50, Internova). A standard adhesive pad was positioned on the back. RF energy was applied in a range of 25 to 40 watts for 30 to 60 sec or until an impedance rise was observed. In the event of an impedance rise, the catheter was withdrawn and the blood clot on the electrode (if present) was removed. If atrial flutter terminated during energy application, RF application was added at that site for another 60 sec. The target for ablation was the site where the earliest activation with double-spike potentials or fragmented electrograms was recorded, and, in patients 1, 7 and 8, where concealed entrainment was observed. If an ablation attempt at a particular site did not terminate atrial flutter, catheter mapping of local activation and/or entrainment pacing was repeated in an attempt to identify an area with an earlier activation by repositioning the ablation catheter several millimeters superior or inferior and then anterior or posterior to the initial position under guidance of fluoroscopy.
Fig. 2. Panel A (Upper): Electrograms from the high right atrium, coronary sinus, His bundle (distal electrodes), and posteroseptum of the right atrium (near the coronary sinus ostium between the IVC and the tricuspid valve annulus) by ablation catheter in patient 7. Note that the earliest atrial activation referenced from the onset of the F wave is recorded at the ablation catheter electrode. Fragmented electrogram is recorded at these electrodes. The amplitude of the initial component is five times larger than that of the last component. The earliest activation time is 40 msec before the onset of the F wave, which suggests that this is the exit from the area of slow conduction. Pacing entrainment from this site is shown in Fig. 3. Panel B (Lower): The surface ECG (II, VI) and intracardiac electrograms from patient 3. The ablation catheter has been positioned just inferior to the coronary sinus ostium. Note the early activation at the ablation catheter with fragmented and double-spike potentials recorded by bipolar electrodes. The first component of the double-spike potentials is recorded at the same timing of negative slope of the unipolar electrogram recorded by the distal electrode of the same catheter. The earliest activation time is 40 msec before the onset of the F wave, which suggests that this occurs near the exit from the area of slow conduction. I, II, III, VI, surface ECG leads; HRA, high right atrium; CSprox., dist., electrograms from the coronary sinus proximal and distal electrode pairs, respectively; ABLunipolar, ABL bipolar, electrograms from the ablation catheter just posterior and inferior to the coronary sinus ostium in unipolar and bipolar modes; HBE, electrogram from the distal (patient 7) or proximal (patient 3) electrode pair of the hexapolar His bundle catheter.
Ablation was then reattempted at these sites. The procedure was limited by design to a maximum of 20 deliveries of energy.

After successful termination of atrial flutter, programmed stimulation was repeated after 30 min to determine if atrial flutter could be induced.

All of the patients were followed in our outpatient clinic at 1 week after discharge and then each month, at which time any history of recent symptoms was noted and an ECG was recorded. Patients were instructed to report any symptoms suggestive of recurrence of arrhythmia, at which time an ECG and Holter ECG would be performed to evaluate the symptoms. Aspirin (81 mg per day) was prescribed for anticoagulation therapy for the first 2 months after RF ablation.

RESULTS

Results of Electrophysiologic Study and Endocardial Mapping

Clinically documented atrial flutter was induced by programmed stimulation or rapid pacing in all patients, except one in whom isoproterenol infusion was necessary to induce sustained atrial flutter. In 3 patients, uncommon atrial flutter with positive deflection in lead II was also induced.

The mean atrial flutter cycle length was 230.0 ± 20.7 msec (range 212–276 msec) for common atrial flutter and 211.5 ± 7.6 msec for uncommon atrial flutter (range 200–221 msec) at the electrophysiology study (Table I).

Double-spike potentials or fragmented electrograms were recorded from sites (low posteroseptal area) near the coronary sinus ostium between the IVC and the tricuspid valve annulus in all patients during common atrial flutter, and were prolonged up to 130 msec (ranged 80–130 msec) and spanned the onset of the F wave on the surface ECG (Fig. 2).

Constant fusion of the F wave was noted during entrainment pacing from the high right atrium, in both common and uncom-
mon atrial flutter (ie, overt entrainment). Concealed entrainment (with F waves similar to those during atrial flutter) was observed during pacing near the coronary sinus ostium (Figs. 3, 4) where the double-spike potential or fragmented electrogram was recorded.

The earliest atrial activation, typically 40–60 msec before the of the F wave onset, was recorded posterior and inferior to the coronary sinus ostium between the IVC and tricuspid valve annulus (which suggests that this is the exit site for the area of slow conduction) in all patients in whom ablation terminated atrial flutter (Table II). RF energy, applied at earliest atrial activation of <40 msec (ranged 0–30 msec) before the onset of the F wave, did not terminate atrial flutter.

**Results of RF Ablation of Atrial Flutter**

RF ablation at the sites of earliest activation with double-spike potentials or fragmented electrograms (Fig. 2) successfully terminated atrial flutter in 4 patients with common atrial flutter (patients 1, 2, 3 and 7) and in one with common atrial flutter accompanied by inducible uncommon atrial flutter (patient 4) after 3 to 17 applications of energy, applied at earliest atrial activation of <40 msec (ranged 0–30 msec) before the onset of the F wave, did not terminate atrial flutter.

**TABLE II  OUTCOME OF RADIOFREQUENCY ABLATION FOR ATRIAL FLUTTER**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>RF energy applications (No.)</th>
<th>Response of AFL to RF ABL</th>
<th>Atrial activation to F wave onset in success site</th>
<th>Follow-up in weeks</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>5</td>
<td>Terminate</td>
<td>-50</td>
<td>8</td>
</tr>
<tr>
<td>2</td>
<td>12</td>
<td>Terminate</td>
<td>-60</td>
<td>28</td>
</tr>
<tr>
<td>3</td>
<td>14</td>
<td>Terminate</td>
<td>-48</td>
<td>30 (22)</td>
</tr>
<tr>
<td>4</td>
<td>17</td>
<td>Terminate</td>
<td>-60</td>
<td>27</td>
</tr>
<tr>
<td>5</td>
<td>20*</td>
<td>None</td>
<td>-</td>
<td>22</td>
</tr>
<tr>
<td>6</td>
<td>20</td>
<td>None</td>
<td>-</td>
<td>29</td>
</tr>
<tr>
<td>7</td>
<td>11</td>
<td>Terminate</td>
<td>-40</td>
<td>8 (4)</td>
</tr>
<tr>
<td>8</td>
<td>6</td>
<td>None</td>
<td>-</td>
<td>3</td>
</tr>
</tbody>
</table>


( )<sup>a</sup>: time to recurrence of original atrial flutter

( )<sup>b</sup>: time to recurrence of uncommon atrial flutter

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*Fig. 4. Panel A (Left): 30° right anterior oblique fluoroscopic view of the heart showing the position of the ablation catheter in patient 7. Panel B (Right): 60° left anterior oblique fluoroscopic views showing the position of the ablation catheter in the same patient. Note that the tip of the ablation catheter is located just inferior to the coronary sinus ostium. Early activation and concealed entrainment with fragmented and double-spike potentials were obtained at this site (Fig. 2A). Application of RF energy at this site terminated atrial flutter (Fig. 2A). CS: coronary sinus catheter, RV: right ventricle catheter, HIS: His bundle catheter, ABL: 4-mm tip ablation catheter. HRA: high right atrium catheter.*
Fig. 5. Panel A (Upper): Radiofrequency energy (40 watts) was applied through the ablation catheter (Fig. 2A), just inferior to the coronary sinus ostium in patient 7. Note the termination of atrial flutter after approximately 5 sec of RF delivery, with immediate resumption of sinus rhythm. An additional RF application of a full 60 sec was added at this site after the preceding 30-sec application. Atrial flutter could not be induced thereafter. Panel B (Lower): RF energy (40 watts) was applied through the ablation catheter just inferior to the coronary sinus ostium between the IVC and the tricuspid valve annulus in patient 3. This site demonstrated similar electrogram and entrainment pacing characteristics to those obtained in patient 7 (Fig. 2B). The earliest activation time recorded at the ablation catheter is 46 msec before the onset of the F wave. Note the sudden termination of atrial flutter after approximately 12 sec of RF energy, and the return to sinus rhythm. An additional application of a full 60 sec was added to this site when the preceding application ended. Atrial flutter could not be induced thereafter.

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sequence mapping and pacing entrainment during atrial flutter. The problem is to identify the weak link in the circuit where ablation should be performed. An analysis of data recorded from sites where ablation was successful revealed an activation time 40–60 msec before the onset of the F wave with double-spike potentials or fragmented electrograms in which the amplitude of the first potential was two or more times greater than that of the second potential. In addition, the first potential was recorded simultaneously with negative slope of unipolar electrogram recorded by the same catheter (Fig. 2). At these sites, concealed entrainment was observed in one patient. The stimulus-to-F wave interval of 46 msec was similar to the duration from local activation to the onset of the F wave (40 msec) (Fig. 3). RF ablation was unsuccessful when RF energy was applied at the sites with double-spike potentials or fragmented electrograms and earliest atrial activation of <40 msec before the onset of the F wave.

**Fragmented Electrogram and Double-spike Potential in Atrial Flutter**

Fragmented electrogram indicates an area of slow conduction and this area of slow conduction seems to represent a crucial component of the macroreentrant circuit. Klein et al. reported that coronary sinus pacing did not change the sequence of flutter wave morphology in electrogams, which is consistent with concealed entrainment, in which the flutter circuit is entrained even though fusion cannot be demonstrated. These phenomena can be explained by the presence of a large right atrial reentrant circuit beginning in the region of the coronary sinus and proceeding superiorly to activate the high right atrium, with the return limb returning to the coronary sinus via the right atrial free wall.

Recent studies of common atrial flutter have demonstrated that double potentials are recorded by endocardial catheter placed at specific locations in the right atrium. One proposed mechanism of double potentials is that they represent sequential activation at the center of a reentrant circuit as the reentrant wave front passes on either side of the recording electrode. The experimental results

**Follow-up of Patients**

All 8 patients were followed in outpatient clinic for a mean of 19.4 ± 10.4 weeks (range, 3–30 weeks) after their procedures. There were no acute complications. Among 5 patients in whom common atrial flutter could not be induced after ablation, 3 patients (patients 1, 3 and 7) were prescribed class 1a (disopyramide) or 1b (aprinidine) antiarrhythmic medications because of paroxysmal atrial fibrillation. One patient (patient 3) experienced recurrence of his original atrial flutter at 22 weeks after ablation, and another patient (patient 7) experienced uncommon atrial flutter within 4 weeks after ablation (patient 7).

**DISCUSSION**

Our present results of ablation for common atrial flutter agree with those of other groups and support the idea that common atrial flutter in humans is caused by reentry in the right atrium and demonstrate that a critical area of the reentry circuit can be identified and modified with RF energy.

A circus movement in the right atrium during common atrial flutter is now accepted because of the development of activation se-
of Schoels et al\textsuperscript{20} suggested that the two potentials could be interpreted as local activation and as electronic potential reflecting from an adjacent recording site on the opposite side of the arc of the block. Allessie et al\textsuperscript{13} reported that double potentials are indicative of functional conduction in the center of the reentry circuit caused by a centripetal wave coming from the opposite direction. Oshansky et al\textsuperscript{22} demonstrated that double potentials are related to reentry and represent the collision of activation wave fronts in the functional center of a reentrant circuit around which the reentrant wave front circulates. An alternative explanation is that double potentials, like fragmented electrograms, may represent electrical activity in an area of slow conduction within the reentrant circuit\textsuperscript{14,18,24,25} Cosio et al\textsuperscript{18} demonstrated that double-spike electrograms represent activation on both sides of a conduction delay zone. The two spikes represent the entry and exit of the activation front in the slow conduction area. Coronary sinus pacing resulted in an increased separation between the double spikes, whereas high right atrium pacing caused a decrease. This finding was interpreted as the result of orthodromic (during coronary sinus pacing) or antidromic (during high right atrium pacing) penetration of an area of slow conduction which spreads into the posterior wall and the IVC orifice to form a functional and anatomical obstacle, which supports the reentry pathway. Brachmann et al\textsuperscript{24} also suggested that double deflection may represent the entry and exit of an activation front in an area of slow conduction within an activation circuit.

In all of our patients, double-spike potentials and fragmented electrograms recorded near the coronary sinus ostium between the IVC and the tricuspid valve annulus, and concealed entrainment observed during pacing in this area, suggest that this site is an area of slow conduction of the reentrant circuit in which unidirectional block of the antidromic paced wave front probably occurs. Our results are consistent with this second explanation for double potentials. It is likely that double potentials separated by an isoelectric interval represent the functional block (the first explanation), while double potentials with fragmented electrograms (without an isoelectric interval, ie, double-spike potentials), represent slow conduction.

\textbf{RF ablation of Common and Uncommon Atrial Flutter}

Previous studies\textsuperscript{3,5,26} have shown that catheter ablation in the area of slow conduction terminated atrial flutter. The endocardial region in the area of slow conduction with the earliest activation may be the exit site of the area of slow conduction. An activation time of $\geq 40$ msec before the onset of the F wave in this area was obtained in all of the patients in whom ablation terminated atrial flutter. This suggests that the critical area for ablation of common atrial flutter is the exit site of the area of slow conduction, which is located near the coronary sinus ostium between the IVC and the tricuspid valve annulus, or between the coronary sinus ostium and the tricuspid valve annulus. The large first potential suggests that the catheter is located near the exit site of the area of slow conduction and far from the entrance site of this area. To improve the resolution of local activation, unipolar electrogram was recorded from distal electrode during the ablation procedure, since it more accurately reflects the electrical activity immediately beneath the electrode used for ablation.

In patients with common atrial flutter accompanied by uncommon atrial flutter, ablation did not terminate atrial flutter although double-spike potentials and fragmented electrograms were recorded and entrainment was demonstrated in patients in whom entrainment pacing was performed during uncommon atrial flutter (patients 6 and 8). This may be explained by a small lesion created by RF ablation. Previous reports\textsuperscript{27,28} have indicated that, in some patients with common atrial flutter, atrial stimulation induces atrial flutter with positive waves on the inferior leads, ie, uncommon atrial flutter. In these patients, uncommon atrial flutter shows the same activation pathway as common atrial flutter, with areas of functional block and slow conduction in the same locations. However, the rotation is reversed (clockwise). More recently, Cosio et al\textsuperscript{6} demonstrated that the production of a continuous line of necrosis by RF ablation across the isthmus from the IVC to the tricuspid ring terminated atrial flutter in
patients with common atrial flutter either alone or accompanied with by uncommon atrial flutter. This technique may be suitable for patients who show both common and uncommon atrial flutter since it can create a relatively large lesion. However, this technique may require several applications of energy. Other investigators have used DC current to treat atrial flutter with favorable results, but with some risk of atrioventricular block. Moreover, ablation of atrial flutter using DC energy obscures significance of the local effect. In 1993, Nakagawa et al. and Fisher et al. reported that RF ablation between tricuspid annulus and CSos or inferior vena cava orifice was efficient in more than 70% of the cases with common atrial flutter.

**Limitations**

In this study, RF ablation appeared to be effective for terminating and preventing the recurrence of common atrial flutter, as in previous studies. However, RF ablation does not affect all myocardial abnormalities which could cause atrial flutter. In this small group of patients, one patient (patient 3) experienced delayed recurrence, while another (patient 7) developed a new type of atrial flutter (uncommon atrial flutter). Patients treated with RF ablation should be followed with caution, since a new type of atrial flutter, atrial fibrillation, or perhaps other types of atrial arrhythmia may develop if these myocardial abnormalities progress. Another limitation is that RF ablation for a critical site which may be the exit from an area of slow conduction may not be suitable for patients with uncommon atrial flutter because only a small lesion is created by this procedure.

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