Radiofrequency Catheter Ablation of Atrial Arrhythmias
Results and Mechanisms

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Background Radio frequency catheter ablation is accepted therapy for patients with paroxysmal supraventricular tachycardia and has a low rate of complications. For patients with atrial arrhythmias, catheter ablation of the His bundle has been an option when drugs fail or produce untoward side effects. Although preventing rapid ventricular response, this procedure requires a permanent pacemaker and does not restore the atrium to normal rhythm. Therefore, we evaluated the safety and efficacy of radiofrequency ablation directed at the atrial substrate.

Methods and Results Thirty-seven patients with 42 atrial arrhythmias (mean±SD age, 41±24 years) who had failed a median of three drugs were enrolled. Diagnoses were automatic atrial tachycardia in 12, atypical atrial flutter in 1, typical atrial flutter in 18, reentrant atrial tachycardia in 8, and sinus node reentry in 3 patients. Sites for atrial flutter ablation were based on anatomic barriers in the floor of the right atrium. For automatic atrial tachycardia, the site of earliest activation before the P wave was sought. All with reentrant atrial tachycardia had previous surgery for congenital heart disease and reentry around a surgical scar, anatomic defect, or atrioventricular incision and our goal was to identify a site of early activation in a zone of slow conduction. At target sites, 20 to 50 W of radiofrequency energy was delivered during tachycardia between the 4- or 5-mm catheter tip and a skin patch, except in 4 patients with atrial flutter, in whom a catheter with a 10-mm thermistor-embedded tip was used. Procedure end point was inability to induce tachycardia. Acute success was achieved in 11 of 12 (92%) with automatic atrial tachycardia, 17 of 18 (94%) with typical atrial flutter, 7 of 8 (88%) with reentrant atrial tachycardia, and 3 of 3 (100%) with sinus node reentry but not in the patient with atypical atrial flutter. For tachycardia involving reentry (reentrant atrial tachycardia and atrial flutter), successful ablation required severing an isthmus of slow conduction. For those with atrial flutter, this was between the tricuspid annulus and the coronary sinus os (10) or posterior (4) or posterolateral (3) between the inferior vena cava (2) or an atriotomy scar (1) and the tricuspid annulus. Deep venous thrombosis occurred in 1 patient. At mean follow-up of 290±40 days, the ablated arrhythmia recurred in 1 (9%) with automatic atrial tachycardia, 5 (29%) with atrial flutter, and 1 (14%) with reentrant atrial tachycardia, all of whom had successful repeat ablation. Previously undetected arrhythmias occurred in 2 patients who are either asymptomatic or controlled with medication.

Conclusions Ablation of automatic and reentrant atrial tachycardia and atrial flutter had a high success rate and caused no complications from energy application. Repeat procedures may be required for long-term success, especially in patients with atrial flutter. The mechanism by which ablation is successful is similar for atrial flutter and other forms of atrial reentry and involves severing a critical isthmus of slow conduction bounded by anatomic or structural obstacles. Automatic arrhythmias are abolished by directing lesions at the focus of abnormal impulse formation. (Circulation. 1994;89:1074-1089.)

Key Words • catheterization • ablation • atrial flutter • radiofrequency • tachycardia • congenital heart disease

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has been reported on the use of ablation to treat patients with reentrant atrial tachycardia complicating atrial surgery for congenital heart disease.

The purpose of the present study was to evaluate the safety and efficacy of radiofrequency ablation directed at the atrial substrate in a consecutive series of patients with drug-refractory atrial arrhythmias. All were being considered for complete atrioventricular junctional ablation and pacemaker implantation to achieve rate control, but they were offered the option of primary arrhythmia cure. Included in this cohort were patients with atrial flutter, automatic atrial tachycardia, sinus node reentry, and reentrant atrial tachycardia associated with surgically repaired congenital heart disease. In addition to evaluating safety and outcome, we made observations regarding the electrophysiological and anatomic substrate to compare and contrast the mechanism by which ablation successfully abolished tachycardia for each type of arrhythmia.

Methods

Patient Population

Thirty-seven consecutive patients were treated between January 1991 and April 1993. Patients were enrolled when diagnosed with automatic atrial tachycardia, reentrant atrial tachycardia (including sinus node reentry), or atrial flutter. Those whose predominant arrhythmia was atrial fibrillation were excluded, except if atrial fibrillation occurred briefly after periods of rapid, regular atrial tachycardia.

All patients underwent an initial evaluation that included history, physical examination, ECG, 24-hour ambulatory monitoring, and echocardiogram with color Doppler flow mapping. For each patient, the atrial arrhythmia at which catheter ablation was to be directed, that is, the arrhythmias forming the basis of this report, had been documented by either 12-lead ECG or two-lead ambulatory monitoring to occur spontaneously before invasive electrophysiological testing and therefore was not considered an “artifact” of such testing or incidental findings. Although the purpose of the present study was to address ablative therapy of atrial tachycardia, several patients also had other tachycardias (eg, atrioventricular nodal reentry), which were induced at electrophysiology testing. In most cases, these were also suspected clinically before invasive testing and were treated with ablation. No patient in this series had ablation of an arrhythmia that was not clinically documented.

Definition of Tachycardia Mechanisms

Automatic atrial tachycardia was diagnosed using accepted criteria, including (1) prolonged or incessant episodes of rapid atrial rhythm exhibiting variation in rate and “warm-up” and/or “cool-down” during some paroxysms, (2) abnormal P-wave axis and/or morphology during tachycardia and endocardial activation sequence consistent with sinus origin, (3) inability to initiate or terminate tachycardia using programmed electrical stimulation with premature beats, and (4) exclusion of an atrioventricular accessory pathway and atrioventricular nodal reentry by standard techniques (eg, persistence of atrial tachycardia despite spontaneous or pharmacologic atrioventricular block, a premature ventricular beat at the time of His bundle refractoriness failing to advance or delay the next atrial depolarization, and an atrial sequence inconsistent with retrograde activation from the atrioventricular node or an accessory atrioventricular connection).

Reentrant atrial tachycardia (other than atrial flutter) was diagnosed if the following were present: (1) an atrial cycle length between 250 and 600 milliseconds in the absence of antiarrhythmic medications, (2) abnormal P-wave axis and/or morphology during tachycardia and endocardial activation sequence consistent with sinus origin, (3) initiation and termination or resetting of tachycardia with appropriately timed premature atrial extrastimuli, and (4) exclusion of an atrioventricular accessory pathway and atrioventricular nodal reentry.

Sinus node reentry was diagnosed when all of the following criteria were observed: (1) P-wave axis and morphology during tachycardia and endocardial activation sequence identical to that during normal sinus rhythm, (2) initiation and termination of tachycardia with appropriately timed premature atrial extrastimuli, (3) onset and termination occurring with an abrupt change in rate from normal sinus rhythm, and (4) exclusion of an atrioventricular accessory pathway and atrioventricular nodal reentry. Although not included as part of this formal definition based on prior reports, all patients with suspected sinus node reentry were administered intravenous adenosine, which consistently resulted in slowing followed by termination of tachycardia (see “Results”).

Typical atrial flutter, for the present study, was diagnosed when the atrial cycle length, in the absence of antiarrhythmic medications, was less than or equal to approximately 250 milliseconds and when the surface ECG exhibited the typical “sawtooth” pattern with inverted F waves in the inferior leads. In several cases, atrial cycle length was slightly more than 250 milliseconds, but the surface morphology and endocardial activation sequence were consistent with typical atrial flutter and are included in that group.

Atypical atrial flutter was diagnosed when the atrial cycle length, in the absence of antiarrhythmic medications, was less than or equal to 250 milliseconds and the surface ECG exhibited broad flutter waves with an inferiorly directed axis.

Electrophysiological Testing

All were referred for definitive therapy, so that the diagnostic and ablation procedure were performed in the same laboratory session, except in 3 patients who had electrophysiology studies at other institutions. Our protocol was approved by the Committee on Human Research, and informed written consent was obtained from the patient or their parent.

Antiarrhythmic medications were discontinued at least five half-lives before the procedure, which was performed with the patient in the postabsorptive state under mild intravenous sedation with fentanyl and midazolam. Quadripolar or hexapolar catheters (6F or 7F) were positioned in the high right atrium, low septal right atrium to record a His bundle electrogram, right ventricular apex, and coronary sinus. In three cases of typical atrial flutter and one case of reentrant atrial tachycardia, a “halo” catheter consisting of 10 bipolar pairs (2-mm intracathode distance, 5-mm interbipole distance) (Webster Laboratory) was positioned along the circumference of the tricuspid annulus. When left atrial recordings were required other than those provided by a catheter in the coronary sinus, a transseptal puncture was performed using a Brockenbrough needle and a Mullins sheath, or a patent foramen ovale was crossed. Surface leads V1, I, II, and aVF and intracardiac electrograms, filtered from 30 to 250 Hz, were recorded on an electrostatic paper recorder (PPG) at a paper speed of 100 mm/s or a computer-based digital amplifier/recorder system with external disk storage (ART Inc). In addition, we were able to obtain full 12-lead ECGs at any time during electrophysiology testing, such as during attempts at entrainment (see below).

For patients whose arrhythmia was not present at the time of electrophysiology study, single and double programmed extrastimuli were delivered after an 8-beat drive of at least two cycle lengths and two atrial sites and atrial decremental burst pacing down to the cycle length at which two-to-one capture occurred. For all arrhythmia mechanisms, if tachycardia was not induced or was not sustained, isoproterenol was infused starting at a dose of 0.5 µg/min (or 0.025 µg·kg⁻¹·min in children), with dose increases every 5 minutes until the heart rate increased by 40% or tachycardia became sustained either spontaneously or with pacing.
Intracardiac Mapping and Radiofrequency Ablation

Detailed mapping and ablation were performed using a 7F or an 8F catheter with deflectable tip and a 4- or 5-mm distal electrode (Mansfield/Webster or EP Technology), except in 4 patients with atrial flutter in whom a custom deflectable-tip catheter with a 10-mm thermistor-embedded distal electrode was used. At target sites during tachycardia, 20- to 30-W test applications of unmodulated radiofrequency energy (500 kHz; EP Technology or Radionics) were delivered for 10 to 15 seconds between the tip of the ablation catheter and a large surface area skin patch. If tachycardia terminated, energy application was continued at up to 50 W for 60 seconds. If a sudden rise in impedance (>250 Ω) occurred during energy application, energy delivery was automatically discontinued, the catheter was removed, and any adherent coagulum was cleaned from its tip. In the four cases in which a thermistor-tip catheter was used, power was adjusted to achieve a tip-tissue interface temperature of 65 to 70°C.

Automatic Atrial Tachycardia

For patients with automatic atrial tachycardia, initial regional- ization was accomplished by analysis of the surface P wave and of multiple simultaneous atrial sites recorded from catheters in the high and low septal right atria and coronary sinuses. For each location, the activation time in relation to the onset of the P wave on the surface was recorded. Detailed mapping with the ablation catheter was then used to select a target for ablation, searching for the earliest local activity before the P wave. This detailed mapping began in the right atrium. The right atrium was conceptually divided into approximately five levels 1 cm apart from the roof of the right atrium near the superior vena cava–right atrium junction to the floor of the right atrium at the inferior vena cava–right atrium junction. At each level, 5 to 10 recordings were made around the circumference of the atrium, and the activation time taken as the onset of the local atrial electrogram with respect to the surface P wave. If no recording site in the right atrium appeared early (ie, less than 0 milliseconds before the onset of the P wave) or the surface P wave in tachycardia strongly indicated a left atrial focus (ie, inverted P wave in leads aVL or I), then the left atrium was mapped in a similar fashion, after transseptal catheterization. In multiple simultaneous mapping in an attempt to match the paced P wave with the P wave during tachycardia was not routinely used because of the difficulty in clearly discerning P-wave morphology because of superimposed QRS and T waves.

When a site was identified that had an earlier activation time with respect to the surface P wave than all surrounding sites, radiofrequency energy was applied. If we were not successful in terminating tachycardia, the catheter tip was moved several millimeters, and energy delivery was repeated.

Reentrant Atrial Tachycardia

For patients with reentrant atrial tachycardia, the same type of systematic mapping using a deflectable-tip ablation catheter was used as described above for patients with automatic tachycardia. In addition, when a natural or postsurgical structural abnormality was known to be present in a patient with reentrant atrial tachycardia (eg, atrial septal defect repair or Fontan conduit), the region near this defect was mapped in greater detail, including regions around atriotomy sites. Reference to older catheter reports was an essential part of procedure planning. Our goal was to identify a protected zone of slow conduction using entrainment techniques. Pacing from the high right atrium and the coronary sinus was performed during tachycardia at cycle lengths 10 to 50 milliseconds shorter than the tachycardia to ensure that criteria for overt entrainment were met. Potential target sites were identified that preceded the surface P wave by at least 20 milliseconds. Once identified, we again entrained the tachycardia by pacing from the high right atrium and/or coronary sinus to determine whether the potential target site was activated in constant and early relation to the subsequent P wave. Pacing was then performed from the potential target site to see whether entrainment without surface fusion, that is, concealed entrainment,23-25 was present. Concealed entrainment is defined, for purposes of the present study, to be continuous resetting of the atrial tachycardia without alteration in P-wave morphology on 12-lead ECG and with a delay between the stimulus and the surface P wave. To be sure that the potential target site was within a critical zone of slow conduction, and not a bystander site merely connected to the critical slow zone, we required that the first postspacing interval at the potential target site be within 10 milliseconds of the tachycardia cycle length and that the time between the pacing stimulus and the surface P wave (pacing to P) was similar to the activation time at that site. Similar techniques have been described for identification of the slow zone during ablation of ventricular tachycardia.24,25

Sinus Node Reentry

For patients with sinus node reentry, we mapped the region near the superior vena cava–right atrium junction down to the crista terminalis in detail using the deflectable-tip ablation catheter. Our aim was to identify a site that had the earliest activation with respect to the P wave.

Atrial Flutter

Before we attempted ablation, pacing from the high right atrium and/or coronary sinus was performed during tachycardia at cycle lengths 10 to 40 milliseconds shorter than the tachycardia to entrain atrial flutter to confirm an activation sequence ascending from the low septal right atrium. This was done by examining the first beat after cessation of pacing, which was entrained but not fused, and noting the order of activation of endocardial recordings with respect to the surface flutter wave.26 Having proven that the tachycardia could be entrained, we then turned our attention to positioning the tip of the ablation catheter in the low right atrium at potential target sites. Sites for atrial flutter ablation were based on anatomic barriers in the floor of the right atrium, either between the inferior vena cava–right atrium junction and the tricuspid ring or between the coronary sinus and tricuspid ring. Mapping was begun in the low right atrium adjacent to the tricuspid ring in a directly posterior position. Our goal was to find sites in a protected zone of slow conduction bounded by anatomic obstacles such that local activation occurred during the isoelectric interval between successive flutter waves and preceded the onset of the flutter wave by at least 50 milliseconds.27-29 We attempted to pace from potential target sites to determine whether criteria for concealed entrainment (see above) were met.23-25 However, we were often unable to capture atrial myocardium despite a pacing current of up to 10 mA. In any case, when capture was achieved, concealed entrainment occurred over a wide area in the low right atrium. If radiofrequency energy application at the directly posterior site failed to terminate atrial flutter, the catheter tip was moved—first several millimeters more medial (posteroseptal) and then more lateral (posterolateral)—and the technique was repeated.

For all arrhythmias, biplane cine images were obtained of the catheter at the site of successful ablation, and the relation of this site to anatomic structures was carefully noted. The electrogram at the successful site was examined for fractionation, defined for the present study as having more than five inflections and being at least twice as long as electrograms obtained nearby.

After apparently successful ablation, aggressive testing for inducibility, including the administration of isoproterenol, was performed again as described above and was repeated periodically for at least 30 minutes after the last lesion.

Follow-up

Echocardiograms were obtained on the day after the ablation procedure. Patients were monitored for 24 hours after the
procedure. A 24-hour ambulatory ECG was obtained approxi-
mately 1 month later, and all patients had close clinical
follow-up at periodic intervals. Follow-up electrophysiology
studies were not routinely recommended in asymptomatic
patients.

Results

Patient Population

The study group consisted of 37 patients with a total of
42 atrial tachyarrhythmias. Clinical and demographic
characteristics are listed in Table 1. There were 16
females and 21 males with an average age of 41±24
years (range, 5 to 81 years). The arrhythmia diagnoses
were atrial tachycardia in 12, atypical atrial flutter in 1,
typical atrial flutter in 18, reentrant atrial tachycardia
in 8, and sinus node reentry in 3 patients. Multiple
distinct atrial arrhythmias were present in 3 patients.
A 29-year-old woman (patient 27) who had
had an atrial septal defect repair in the past had two
distinct forms of reentrant atrial tachycardia and atrial
flutter. Another (patient 23) who had had an atrial
septal defect repair also had two forms of reentrant
atrial tachycardia and atrial flutter. Patient 28 had two
distinct morphologies of automatic atrial tachycardia.
In addition, 4 patients had atrioventricular nodal reentry
(typical in 3 and atypical in 1) successfully treated with
slow pathway ablation in the same session during which
atrial tachycardia was ablated. In 2 of 4, this was evident
clinically before ablation. In the other 2, atrioventricular
nodal reentry was easily inducible and sustained, al-
though it had not been recorded before the electrophys-
iology study, whereas the atrial tachycardia had. It
was judged, therefore, that atrioventricular nodal reentry
would ultimately present as a clinical tachycardia, and
ablative therapy was administered.

Symptoms, including palpitations, fatigue, congestive
heart failure, and syncope, had been present from
several months to 30 years, and all patients had failed
at least one drug trial to control the ventricular response
and/or convert to sinus rhythm (median, 3; range, 1 to
6). Structural heart disease was present in 7 of 9
patients with automatic atrial tachycardia, in 5 of whom
the diagnosis was tachycardia-related cardiomyopathy.
Structural heart disease was present in 12 of 19 patients
with atrial flutter. All with intra-atrial reentrant tachy-
cardias had had previous atrial surgery. Two each had
undergone closure of secundum atrial septal defects, a
Fontan procedure, or a Mustard atrial baffle. One
patient with sinus node reentry had chronic pulmonary
disease and an enlarged right atrium

Observations During Mapping and Ablation

The electrophysiological characteristics and ablation
results are listed in Table 2.

Automatic Atrial Tachycardia

All patients with automatic atrial tachycardia had
incessant tachycardia either in the baseline state or after
isoproterenol infusion. Premature beats neither initi-
ated nor terminated the arrhythmia. The average atrial
cycle lengths are given in Table 2, but in any individual
patient the atrial rate varied depending on catechol-
amine dose. In 5 of 12 tachycardias, the focus was
mapped to the left atrium through transseptal catheter-
ization. Local activation preceded the surface P wave by
15 to 60 milliseconds (mean, 45±5 milliseconds). Fra-
tionated electrograms were recorded from a small sur-
rounding region before successful ablation in 7 of 11
tachycardias. Fig 1 shows intracardiac and surface trac-
ings and cineangiographic images for a patient with
successful ablation of a left atrial automatic tachycardia
localized to the mouth of the left upper pulmonary vein.

Acute success defined by absence of tachycardia
spontaneously or after the infusion of isoproterenol was
achieved in 11 of 12 tachycardias, including 1 patient
(28) with two distinct foci. Success was achieved using a
median of five applications of radiofrequency energy at
a mean power of 30±5.5 W. Sites of successful ablation
for the 11 tachycardias are notable for a clustering
around pulmonary veins and the mouths of the atrial
appendages (see Fig 2). All except 1 patient with
successful ablation had termination during radiofre-
quency energy application, and the other patient had
tachycardia termination from catheter placement onto
the endocardium, where radiofrequency was subse-
wntly applied. In three cases, there was an increase in
tachycardia rate with radiofrequency application just
before termination.

Atrial Flutter

Atrial flutter was present spontaneously in 11 of 19
patients and was induced with rapid atrial pacing or
programmed extrastimuli in 8. The mean flutter cycle
length was 220±6 milliseconds. Ablation successfully
terminated typical atrial flutter in 17 of 18 and was
unsuccessful in the patient with atypical atrial flutter.
Success was achieved using a median of six applications
of radiofrequency energy at a mean power of 38±8.5 W.
In those with typical flutter, mapping confirmed an
activation sequence up the intra-atrial septum and down
along the right atrial free wall. A typical patient with
atrial flutter is illustrated in Fig 3. With the flutter wave
in aVf serving as a reference, the low septum near the
coronary sinus os was activated at the time of its initial
downward deflection, followed by the high septum and
then the superior atrial free wall, lateral atrial free wall,
and low postero lateral atrial free wall. The left atrium
appeared passively activated as determined by the vari-
able timing of coronary sinus sites with respect to the
flutter wave onset during entrainment at various sites
and rates.

A remarkably consistent finding in patients with
typical atrial flutter was a region of long conduction
time from the low postero lateral right atrial free wall
to the low postero medial (posterosetal) right atrium,
bounded by the inferior vena cava–atrium junction
laterally and the coronary sinus os medially. This region
of 2 to 3 cm accounted for 100 to 150 milliseconds of
activation time during the isoelectric portion of the
surface flutter wave, and it was here that ablative energy
was directed. In 10 patients, the site of success was low
postero medial in the region between the tricuspid annu-
ulus and the coronary sinus os. In 4 patients, the
successful site was at the low posterior, and in 3, it was
at the low postero lateral right atrium. These catheter
positions are illustrated in Fig 4, in which the floor of
the right atrium is depicted. The electrogram at the
successful site was fractionated in 12 of 15 patients in
whom it was recorded. In 4 patients, flutter cycle length
prolonged during radiofrequency energy application,
with the identical surface morphology and endocardial
activation sequence. An example of this is shown in Fig
3C.
**TABLE 1.** Characteristics of 37 Patients Who Received Radiofrequency Catheter Ablation

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Arrhythmia*</th>
<th>No. of Drugs That Failed</th>
<th>Symptom Duration, y</th>
<th>Structural Heart Disease</th>
<th>LV EF, %</th>
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<td>2</td>
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<tr>
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<td>10</td>
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<td>11</td>
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<td>5</td>
<td>10</td>
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<td>12</td>
<td>55</td>
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<td>3</td>
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<td>3</td>
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<td>0.2</td>
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<td>30</td>
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<td>16</td>
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<td>4</td>
<td>0.2</td>
<td>Coronary artery disease, myocardial infarction</td>
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<td>3</td>
<td>0.5</td>
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<td>62</td>
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<td>3</td>
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<td>Dilated cardiomyopathy, COPD, CABG</td>
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<td>Afl-T</td>
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<td>5</td>
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<td>9</td>
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<tr>
<td>24</td>
<td>17</td>
<td>RAT</td>
<td>5</td>
<td>15</td>
<td>Double-inlet single LV, D-TGA, Mustard procedure</td>
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<tr>
<td>25</td>
<td>8</td>
<td>RAT</td>
<td>1</td>
<td>0.5</td>
<td>Hypoplastic TV and RV, functional single V, Fontan repair</td>
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<tr>
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<td>66</td>
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<td>3</td>
<td>0.5</td>
<td>COPD</td>
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<tr>
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<td>29</td>
<td>RAT</td>
<td>4</td>
<td>8</td>
<td>Atrial septal defect repair at age 5</td>
<td>50</td>
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<td>None</td>
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LV EF indicates left ventricular ejection fraction; AAT, automatic atrial tachycardia; RAT, reentrant atrial tachycardia; Afl-T, typical atrial flutter; Afl-A, atypical atrial flutter; SNRT, sinus node reentry; AVNRT, atioventricular nodal reentry; COPD, chronic obstructive pulmonary disease; CABG, coronary artery bypass grafting; TGA, transposition of the great arteries; nl, normal; and NA, not applicable.

*Other arrhythmia (in parentheses) also was successfully treated with RF ablation but not tabulated as part of this series of primary atrial tachycardias.
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<td>13 S 150 63</td>
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<td>15 S 255 45</td>
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<td>Slowed</td>
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<td>Slowed</td>
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<td>Mean±SEM*</td>
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<td>−53±7.4</td>
<td>−82±6.7</td>
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</table>

**SVT CL** indicates supraventricular tachycardia cycle length; **RF** number of applications of radiofrequency current, including "test" applications; **S/F** success/failure; **Proc. T** procedure time; **Fluoro. T** fluoroscopy exposure time; **Act. T** activation; **LA** left atrium; **RA** right atrium; **CS** coronary sinus; **TA** tricuspid annulus; **NR** not recorded; and **SP** slow atrioventricular nodal pathway.

*Mean±SEM, except RF applications are given as median.
†Additional arrhythmia ablated during same session is another entry.
‡Tachycardia terminated during an RF application at a site with these characteristics but was still inducible and classified as an acute failure.

**Table 2. Electrophysiological Characteristics of Tachycardias and Results of Ablation**

**Atrial flutter**

**Reentrant atrial tachycardia**

**Sinus node reentry**
In 2 patients, both with prior atrial surgery, atrial flutter was no longer inducible after successful ablation of another arrhythmia, implying a common zone of slow conduction. In patient 23, both atrial flutter (cycle length, 230 milliseconds) and reentrant atrial tachycardia (cycle length, 425 milliseconds) were abolished by ablation along the posterolateral right atrium between the inferior vena cava and an atriotomy scar. In patient 33, both atrioventricular nodal reentrant tachycardia and atrial flutter were rendered noninducible with a lesion placed between the coronary sinus os and the tricuspid annulus in a patient with a Mustard repair in whom this location was approached through retrograde transaortic catheterization.

In 4 patients, multiple applications of radiofrequency energy to the low right atrium using a 4-mm distal electrode failed to disrupt the tachycardia. Substitution of a catheter with a 10-mm thermistor-embedded tip resulted in successful ablation in a single application of radiofrequency energy in all cases. For these 4, a mean of 55±10 W was required to achieve a tip temperature of 60°C.

In the 1 patient with atypical atrial flutter, the surface flutter waves were less well defined than those during typical flutter. There appeared to be a site of early activation near the right atrial appendage, but ablation here, as well as below the coronary sinus os, failed to disrupt the arrhythmia.

**Reentrant Atrial Tachycardia**

Electrophysiological characteristics of the 8 reentrant atrial tachycardias present in 6 patients is given in Table 2. All were reproducibly induced and terminated with programmed atrial extrastimuli before ablation. The mean tachycardia cycle length was 340±30 milliseconds. All except 1 had successful catheter ablation, defined as termination of tachycardia during radiofrequency energy application and inability to induce the arrhythmia despite aggressive stimulation and catecholamine infusion. Success was achieved using a median of seven applications of radiofrequency energy at a mean power of 33±6.5 W.

In the patients with reentrant atrial tachycardia (23, 24, 25, 27, 34, and 36), the substrate for tachycardia was previous atrial surgery for congenital heart disease (Table 1). Successful ablation was achieved at a site of early activation near a surgical anatomic defect or scar. The atriotomy incision on the lateral right atrial wall provided a barrier that appeared to be involved in sustaining reentry in 4 tachycardias. In these 4 arrhythmias, a site of early activation and fractionated electrograms was found in the low lateral right atrium at the inferolateral extent of the surgical atriotomy scar, which typically ends just cephalad to the inferior vena cava and lateral tricuspid ring. In most cases, the atriotomy scar could be palpated with the roving ablation catheter as a distinct “step off,” or ridge. That is, as the catheter tip was dragged down along the lateral right atrial wall, a movement of the catheter over a ridge could be felt in the operator’s hands through the shaft of the catheter, which could also be seen on fluoroscopy. Markedly disparate activation times on either side of this ridge and the recording of double potentials in its vicinity suggested the atriotomy scar as a line of block extending from the inferolateral to the anteromedial right atrial free wall. The difference in activation times, or of the two components of the double potential, was greatest near the superior portion on the right atrium and decreased progressively as the catheter tip was drawn inferiorly. A set of recordings for a typical patient is shown in Fig 5. A radiofrequency lesion extending from the inferior margin of the atriotomy to the inferior vena cava...
cava in 3 and to the posterolateral tricuspid annulus in 1 successfully abolished atrial reentry. An artist’s rendition of where the catheter tip lay with respect to the lower edge of the atriotomy and the tricuspid annulus is provided in Fig 5E.

Patient 27 had a second tachycardia with early activation and fractionated electrograms in the region of the atrial septal defect patch. This patient also had typical atrial flutter (included in that group above) and atrioventricular nodal reentry that was eliminated by slow-pathway ablation. In patient 24 with double-inlet, single left ventricle and transposition of the great arteries, who had had a palliative Mustard procedure, early atrial electrograms with fractionation were found at the posteroseptal pulmonary venous atrium between the baffle and the mouth of the coronary sinus, which had been left to drain to the pulmonary venous atrium. In patient 25, who had undergone a Fontan procedure, successful ablation was achieved at a site of early atrial activation and fractionation during tachycardia, which was found at the anterolateral right atrioventricular groove in an isthmus formed by the hypoplastic tricuspid annulus and the origin of the conduit connecting the right atrium to the pulmonary artery.

Sinus Node Reentry

In the 3 patients with sinus node reentry, ablation was successfully directed near the junction of the superior vena cava and anterolateral right atrium where activation preceded the surface P wave during tachycardia by between 40 and 45 milliseconds. Although adenosine was not routinely administered to all patients in our study, it was used in all three cases of sinus node reentry. Interestingly, in all three cases, there was slowing of the tachycardia followed by termination. There was no disruption of the normal sinus mechanism after ablation.

Complications

The only complication was femoral venous thrombosis, which developed the day after the procedure in patient 18, a 77-year-old woman who had successful ablation of atrial...
flutter. She was treated with anticoagulant medication with no further sequelae. There were no complications from application of radiofrequency energy to the endocardium or any of the five transseptal procedures performed for ablation of left atrial tachycardias.

Procedure and fluoroscopy times are given in Table 2; the averages for the group were 208±80 and 44±20 minutes, respectively.

Follow-up
The patients have been followed for 290±40 days (range, 30 to 840 days). Of 10 patients with initially successful ablation of 11 automatic atrial tachycardias, 1 (10%) had recurrence of the original arrhythmia 6 weeks after initially successful ablation and underwent repeat ablation.

Of 17 patients with initially successful ablation of atrial flutter, 5 (29%) had recurrence after 1, 2, 7, 14, and 88 days. All had successful repeat ablation. Two patients developed atrial fibrillation that has been controlled with propafenone in 1 and quinidine in the other, agents previously unsuccessful in preventing atrial flutter.

Of those with reentrant atrial tachycardia, 1 patient (25) developed an arrhythmia with the same P-wave axis but markedly longer cycle length, and repeat ablation was successful. The patients with sinus node reentry remain in normal sinus rhythm.

All 5 patients with automatic atrial tachycardia and reduced ejection fraction before ablation had return to normal function at follow-up echocardiographic examination.

Discussion
Main Findings
This report contains the largest series to date in which radiofrequency catheter ablation has been safely applied in the atrium to treat patients with several forms of drug-refractory atrial tachycardia, restoring sinus rhythm and avoiding the need for His bundle ablation and permanent pacing. We included patients with a variety of arrhythmia mechanisms to compare and contrast the methods for mapping and successful ablation of each, to show that radiofrequency energy has a low risk of perforation of the relatively thin-walled atrium, because more than one type of arrhythmia may coexist in a given patient and because the precise electrophysiological mechanism may not be apparent at the time of clinical presentation.

Acute success was achieved in 17 of 18 patients (94%) with typical atrial flutter, 11 of 12 (92%) of those with automatic atrial tachycardia, all 7 of 8 (88%) reentrant atrial tachycardias, and all 3 patients (100%) with sinus node reentry. At follow-up, all those with successful ablation are free of their targeted arrhythmia, although 5 with atrial flutter, 1 with reentrant atrial tachycardia, and 1 with automatic atrial tachycardia had recurrence requiring repeat ablation.

Ours is the first report to include extensive results of catheter ablation in 7 patients with atrial arrhythmias complicating the late postoperative course of surgery for congenital heart disease. These patients presented a particular technical challenge because of distorted postsurgical anatomy and the presence of multiple arrhythmias in 4. We were successful in our attempts to ablate reentrant arrhythmias by analogy to lessons learned from ablation of atrial flutter. As discussed further below, the essential steps in abolishing all forms of atrial arrhythmias resulting from reentry are the identification of the barriers that constrain impulse propagation through a region of slow conduction and the production of a lesion that cuts the isthmus from barrier to barrier. Automatic foci, on the other hand, were ablated by identifying the site of earliest activation from which impulse propagation proceeded.

Comparison With Previous Studies
Our study confirms the results of several recent reports. Walsh et al.14 and Kay et al.15 reported a success rate similar to ours for children and adults with automatic atrial tachycardia. Tracy et al.16 achieved success in 80% of 10 patients using the interesting technique of paced endocardial activation-sequence mapping. In our series, we used activation mapping for patients with automatic atrial tachycardia, although a direct comparison of these methods in terms of procedure and fluoroscopy times would be of value. Unlike previous reports, our study included a larger percentage of patients with left atrial foci approached via transseptal catheterization.

For ablation of atrial flutter, our success rate is comparable to that of Feld et al.13 However, our technique for choosing target sites differs somewhat in that we did not always achieve concealed entrainment to prove that we were in a protected isthmus of slow conduction.27,32-34 In our experience, entrainment of the tachycardia that resulted from pacing outside the zone of slow conduction (overt entrainment) was helpful to verify a counterclockwise activation sequence during atrial flutter. It was also helpful to confirm that the electrogram recorded from the ostium of the coronary sinus occurred at the onset of the rapid negative deflection of the surface flutter wave, allowing us to substitute this more easily identified electrogram as the reference point to find sites of earlier activation in the putative zone of slow conduction. At anatomically based target sites in the slow conduction zone (see Fig 4), we attempted to pace during tachycardia (concealed entrainment23) but were sometimes unable to capture despite high pacing current, perhaps because of dis-
eased tissue. In any case, no site chosen based on anatomic landmarks and exhibiting an early activation time was ever rejected because it did not meet criteria for concealed entrainment. In this way, our method is similar to the anatomically based approach of Cosio et al. However, unlike that group, we found that ablation between the tricuspid ring and either the inferior vena cava or the coronary sinus os was sufficient, possibly implying that the reentrant circuit encompassed both structures.

In all reports of ablation for atrial flutter, including ours, there is an unfortunate high rate of recurrence. We could find no predictors of recurrence, including number of applications or delivered power of radiofrequency energy, location of successful ablation, age, or presence of structural heart disease. This should be interpreted with caution, however, because the small numbers in each group make statistical inference difficult. Whether recurrence is the result of disease progression with the development of a new circuit; inadequate aggressiveness of the stimulation protocol after acute, ablation-induced tachycardia termination; or reversible effects of radiofrequency application is unknown.

Our report of patients with successful radiofrequency ablation of a reentrant circuit arising from previous atrial surgery points to the possibility of treating such patients who have a high incidence of atrial arrhythmias after repair of congenital heart disease. Unlike typical atrial flutter, where observing concealed entrainment from a potential target site was merely confirmatory, we found this technique to be crucial in identifying a protected zone of slow conduction in those with reentrant atrial tachycardia. Arrhythmias are quite common after extensive atrial surgery and may and be partially responsible for the occurrence of sudden death in such patients. Therapeutic options may be limited in these patients; sinus node disease and ventricular dysfunction may increase the risk of antiarrhythmic medication. Antibradycardia and antitachycardia pacing may be problematic in patients with residual intracardiac shunting. Finally, loss of atrioventricular synchrony, which may accompany atrioventricular node ablation, may not be well tolerated in such patients. Ablation of the substrate for atrial tachycardia avoids many or all of these problems and may be the preferred approach.

**Observations on Arrhythmia Substrate and Mechanism of Successful Ablation**

The electrophysiological and anatomic characteristics at the site of successful ablation and the response during application of radiofrequency energy allow insights into the substrate of these atrial arrhythmias.

**Reentry**

Several investigators have commented on the requirement of barriers to define a reentrant path. Rosenbleuth and Garcia-Ramos noted reentry around barriers consisting of an intracaval crash lesion and orifices of the great veins. More recently, Frame et al provided experimental evidence and a cogent theoretical argument of the need for two barriers to stabilize reentry. For successful ablation of atrial flutter and reentrant atrial tachycardia in the present series, lesions were placed so that they spanned a conducting isthmus bounded by two barriers, in fulfillment of the hypothesized criteria of Frame et al and Mines.

In patients with atrial flutter, we were able to ablate an isthmus of slow conduction in the low right atrium as far lateral as the posterolateral inferior vena caval/right atrial junction and as far medial as the space between the coronary sinus os and the tricuspid ring (Fig 4), suggesting these locations as the entrance and exit, respectively, of the protected isthmus. Also of note is that in four cases, ablation through a 4-mm tip was not successful, whereas switching to ablation through a 10-mm tip resulted in successful flutter termination. Ours is the first report of the use of such a large tip for ablation of atrial flutter. This indicates that portions of the isthmus may be wider than the lesion created with a 10-mm catheter tip. Clearly, for a lesion to be curative, it must sever the entire width of the isthmus, leaving no gaps through which the excitation wave can continue to propagate.

In the one patient with atypical atrial flutter, we were unable to identify the two barriers responsible for creating a protected isthmus. We created lesions in the low right atrium between the coronary sinus and the tricuspid annulus that were not successful, suggesting that in this patient atypical flutter was not simply a reentrant wave front traveling in the opposite direction over the same path as typical flutter, despite alternate speculation.

For patients with reentrant atrial tachycardia in the setting of prior atrial surgery, the site of successful ablation also bridged two constraining barriers. The isthmus included the space between a Fontan conduit and the tricuspid annulus in 1 patient, a corridor between a Mustard atrial baffle and the coronary sinus os in another, and a corridor between an atrial septal patch and the tricuspid annulus in a third. The atriotomy scar on the lateral right atrium played an important role in the genesis of tachycardia in 4 patients. The surgeon gains access to the interior of the right atrium and atrial septum through an oblique incision from the right atrial appendage down to the inferoposterolateral right atrial free wall. The incision typically stops short of the atrioventricular groove (and inferior vena cava) to avoid damaging coronary vasculature. However, this narrow isthmus between the inferolateral extent of the atriotomy and either the tricuspid annulus or the inferior vena cava can act as a protected isthmus, with the extensive atriotomy serving as an obstacle around which reentrant excitation can circulate. Extending the scar from the atriotomy to the inferior vena cava or tricuspid annulus using radiofrequency energy, as shown in Fig 5E, is curative in these patients. Perhaps future atrial surgery might include an incision or cryoablation extending the atriotomy inferiorly in a similar fashion, preventing some forms of postoperative atrial arrhythmia.

Although some canine models do not necessarily require a zone of slow conduction if the central barrier is sufficiently large, there was evidence for slow conduction in all our patients, based on activation mapping, and the presence, in almost all arrhythmias involving reentry, of low-amplitude fractionated electrograms at the site of success. Although the exact etiology of slow conduction cannot be determined, one can speculate that for those with atrial surgery, the operation, in addition to creating fixed anatomic barriers, may have damaged atrial myocardium, producing patchy fibrosis and consequent conduction slowing. Propagation through nonuniformly anisotropic atrial
tissue would then account for the fractionated electrograms recorded from the slow zone.42

Anisotropic propagation may also have played a role in patients with atrial flutter. The atrium in the region of the tricuspid ring contains two fiber layers: radially oriented fibers and those running circumferentially around the annulus.43,44 A paucity of circumferential fibers might develop in some patients because of atrial stretch or disease. Propagation through the corridor between the coronary sinus os or the inferior vena cava and the tricuspid ring would then be forced to encounter the remaining radial fibers transversely with a velocity less than one third that of longitudinal propagation.45,46 Fractionated electrograms, such as those seen in our patients, would also be expected. What combination of anatomic and functional conditions causes the unidirectional block required for tachycardia initiation and for the impulse to then be forced through this isthmus is not known. Nevertheless, this hypothesis is consistent with the canine model of congenital atrial
flutter described by Boineau et al.\textsuperscript{47} that demonstrated the pathological role of fiber thinning in the low right atrium.

The relation of a region of slowly conducting tissue to a pair of anatomic (or functional) barriers is important. It is certainly possible that multiple areas of tissue damage or fiber separation exist in the atria that have the potential for slow conduction. However, it is only when such areas are juxtaposed between barriers that there is no alternate path and the barriers oblige the propagating wave front to fully confront a zone of slow conduction.

![Diagram](image_url)

**Fig 4.** Catheter positions for ablation of atrial flutter are shown on a cut-open view of floor of right atrium (RA). Wave of reentrant excitation is indicated by curved arrow. In 10 patients, site of success was low posteromedial (PM) in region between tricuspid annulus (TA, dashed white curve) and coronary sinus os (CS). In 4 patients, the successful site was at low posterior RA (P), and in 3, it was at low posterolateral RA (PL) between inferior vena cava (IVC) and TA.
Automaticity

For automatic tachycardias, successful ablation was achieved at focal atrial sites at which local activation preceded the P wave by an average of 46 milliseconds. Interestingly, we found fractionated electrograms at most (7 of 11) successful sites (see, for example, Fig 1A), which is at variance with data from other studies, perhaps because of the different ages of study groups. Atrial fractionation in our patients may imply a region of more generally dysfunctional atrial tissue with abnormal automaticity occurring in the midst of relatively poorly coupled fibers. In fact, such relative uncoupling between normal surrounding atrium and a focus of automaticity may in some cases be a required element of the arrhythmia mechanism that prevents normal atrium from electrotonically clamping the automatic cells and inhibiting their phase 4 depolarization. The requirement of relative uncoupling for automatic cells to drive normal cells has been shown in computer modeling and in vitro studies.

In 3 patients with automatic tachycardia, there was speeding of the tachycardia just before termination with radiofrequency application, possibly the result of an increased rate of firing in heated cells. This is in contrast to those resulting from reentry, in which radiofrequency application caused slowing just before termination, probably because of progressive damage in the critical zone of slow conduction.
Conclusions

This study demonstrates that by severing corridors of slow conduction or abolishing foci of abnormal automaticity, radiofrequency catheter ablation can safely treat atrial arrhythmias in humans. Recurrence is not uncommon in patients with structural heart disease and atrial flutter, the reasons for which require further investigation. However, repeat ablation is usually successful. Tachycardia-related cardiomyopathy reverses after successful ablation of automatic atrial tachycardia.

Long-term follow-up will be required to determine the incidence of recurrence or the emergence of new
arrhythmias, given that many of these patients have progressive atrial disease. Nevertheless, radiofrequency catheter ablation offers the potential for curing patients with atrial arrhythmias who cannot be treated by other methods.

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