A Tailored Approach to Catheter Ablation of Paroxysmal Atrial Fibrillation

Hakan Oral, MD; Aman Chugh, MD; Eric Good, DO; Sundar Sankaran, MD; Stephen S. Reich, MD; Petar Igic, MD; Darryl Elmouchi, MD; David Tschopp, MD; Thomas Crawford, MD; Sujoya Dey, MD; Alan Wimmer, MD; Kristina Lemola, MD; Krit Jongnarangsin, MD; Frank Bogun, MD; Frank Pelosi, Jr, MD; Fred Morady, MD

Background—Because the genesis of atrial fibrillation (AF) is multifactorial and variable, an ablation strategy that involves pulmonary vein isolation and/or a particular set of ablation lines may not be equally effective or efficient in all patients with AF. A tailored strategy that targets initiators and drivers of AF is a possible alternative to a standardized lesion set.

Methods and Results—Catheter ablation was performed in 153 consecutive patients (mean age, 56 ± 11 years) with symptomatic paroxysmal AF with the use of an 8-mm tip radiofrequency ablation catheter. The esophagus was visualized with barium. The pulmonary veins and left atrium were mapped during spontaneous or induced AF. Arrhythmogenic pulmonary veins were isolated or encircled. If AF was still present or inducible, complex electrograms in the left atrium, coronary sinus, and superior vena cava were targeted for ablation. The end point of ablation was absence of frequent atrial ectopy and spontaneous AF during isoproterenol infusion and noninducibility of AF. Routine energy applications near the esophagus were avoided. During follow-up, left atrial flutter developed in 19% of patients and was still present in 10% at >12 weeks of follow-up. A repeat ablation procedure was performed in 18% of patients. During a mean follow-up of 11.2 ± 4 months, 77% of patients were free from AF and/or atrial flutter without antiarrhythmic drug therapy. Pericardial tamponade or transient neurological events occurred in 2% of procedures.

Conclusions—A tailored ablation strategy that only targets triggers and drivers of AF is feasible and eliminates paroxysmal AF in ∼80% of patients. (Circulation. 2006;113:1824-1831.)

Key Words: ablation ▪ arrhythmia ▪ atrium ▪ catheter ablation ▪ fibrillation

Most ablation strategies for atrial fibrillation (AF) consist of a standardized approach of pulmonary vein isolation and/or encirclement with or without additional ablation lines.1–7 However, because the pathogenesis of AF is multifactorial,8 a standardized ablation approach may not be equally effective or efficient in all patients with AF. A predefined, standardized lesion may involve more ablation than necessary in some patients and may be insufficient in others. Furthermore, ablation along the posterior left atrial wall is a component of most standardized ablation strategies and is associated with a risk of atrioesophageal fistula formation.9–11 For example, during wide area circumferential ablation, encircling lesions around the pulmonary veins often intersect the esophageal borders adjacent to the posterior left atrium.9

Clinical Perspective p 1831

Recently, it has been suggested that AF also can be eliminated by targeting complex electrograms characterized by a short cycle length, fractionation, and/or continuous electric activity.12 This strategy, however, is aimed at the left and right atrial substrate and does not specifically target the triggers and drivers that originate in the pulmonary veins and that often play a critical role in the genesis of AF.2,13,14 The hypothesis underlying the present study was that the triggers and drivers of AF vary from patient to patient and that it is possible to identify the specific generators of AF by mapping the pulmonary veins, left atrium, coronary sinus, and superior vena cava during AF. This study determined the feasibility of a tailored catheter ablation strategy guided by the electrophysiological characteristics of AF, without the use of a standardized lesion set.

Methods

Study Subjects

The subjects of this study were 153 consecutive patients with symptomatic, paroxysmal AF who underwent a left atrial ablation procedure to eliminate AF. Patients who had undergone a prior ablation procedure for AF were excluded from this study. The clinical characteristics of study subjects and their prior antiarrhythmic drug therapy are described in the Table.
Clinical Characteristics of Study Group

<table>
<thead>
<tr>
<th>Clinical Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>153</td>
</tr>
<tr>
<td>Age, y</td>
<td>56.1 ± 11</td>
</tr>
<tr>
<td>Gender, M/F</td>
<td>110/43</td>
</tr>
<tr>
<td>Duration of AF, y</td>
<td>7 ± 6</td>
</tr>
<tr>
<td>No. of episodes of AF (per month)</td>
<td>12 ± 14</td>
</tr>
<tr>
<td>Left atrial diameter, mm</td>
<td>41 ± 6</td>
</tr>
<tr>
<td>Left ventricular ejection fraction</td>
<td>0.57 ± 0.08</td>
</tr>
<tr>
<td>Structural heart disease</td>
<td>61 (40%)</td>
</tr>
<tr>
<td>Hypertensive heart disease</td>
<td>43 (28%)</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>14 (9%)</td>
</tr>
<tr>
<td>Nonischemic cardiomyopathy</td>
<td>4 (3%)</td>
</tr>
<tr>
<td>No. of prior antiarrhythmic drugs</td>
<td>1 ± 0.8 (range, 0-4)</td>
</tr>
<tr>
<td>0 antiarrhythmic drugs</td>
<td>29 (19%)</td>
</tr>
<tr>
<td>1 antiarrhythmic drug</td>
<td>87 (57%)</td>
</tr>
<tr>
<td>≥2 antiarrhythmic drugs</td>
<td>37 (24%)</td>
</tr>
<tr>
<td>Previously ineffective antiarrhythmic drugs</td>
<td>43 (28%)</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>9 (6%)</td>
</tr>
<tr>
<td>Sotalol</td>
<td>36 (24%)</td>
</tr>
<tr>
<td>≥1 class I antiarrhythmic drug</td>
<td>74 (48%)</td>
</tr>
</tbody>
</table>

Continuous variables are shown as mean ± SD.

Electrophysiological Study

After informed written consent was obtained, an electrophysiological study was performed in the fasting state. All antiarrhythmic medications, except for amiodarone in 17 patients (11%), were discontinued 4 to 5 half-life periods before the procedure. Amiodarone therapy was discontinued 4 to 6 weeks before the procedure. A quadripolar catheter (EP Technologies, Sunnyvale, Calif) was positioned within the coronary sinus and used for pacing. After transseptal catheterization, systemic anticoagulation was achieved with intravenous heparin to maintain an activated clotting time of 300 to 350 seconds. Mapping of the pulmonary veins and superior vena cava was performed with a decapolar ring catheter (Lasso, Biosense-Webster, Diamond Bar, Calif). Mapping of the left atrium and coronary sinus and ablation were performed with a temperature-controlled, 8-mm tip quadripolar catheter (Navistar, Biosense-Webster).

Bipolar electrograms were recorded at a bandpass of 30 to 500 Hz (EPMedSystems, West Berlin, NJ). Atrial pacing was performed at an output of 10 mA and a pulse width of 2 ms (EPMedSystems, Clinical Stimulator model EP-3).

A 3-dimensional depiction of the left atrium and pulmonary veins was created with an electroanatomic mapping system (Carto, Biosense-Webster). Patients then were asked to swallow 5 mL of a barium sulfate esophageal cream (E-Z-EM, Lake Success, NY), and the location of the esophagus was noted by fluoroscopy. Because the esophagus is a mobile structure, the position of the esophagus relative to the posterior left atrium was monitored throughout the procedure by fluoroscopy. After the barium swallow, conscious sedation was achieved with midazolam and fentanyl.

Ablation at the pulmonary vein ostia and within the coronary sinus was performed at a power of 35 W and temperature of 50°C. Ablation in the left atrium was performed at a power of 70 W and temperature of 50°C. Applications generally were 15 to 30 seconds in duration. Ablation near the esophagus was avoided whenever possible. If a critical target site was located near the esophagus, ablation was performed at a maximum power of 35 W and temperature of 45°C for ≤10 seconds.

Study Protocol

The study protocol was approved by the institutional review board. AF was induced by rapid atrial pacing in patients who were in sinus rhythm. Pacing was performed within the coronary sinus for 10 seconds at the shortest cycle length that yielded 1:1 atrial capture. If pacing alone was insufficient to induce sustained AF, rapid pacing was repeated during isoproterenol infusion. Sustained AF was inducible in all patients.

The pulmonary veins were mapped with the decapolar ring catheter. Pulmonary veins that harbored pulmonary vein tachycardias were identified (Figure 1). As described previously, a pulmonary vein tachycardia was said to exist when the cycle length of the electrograms recorded within a pulmonary vein was shorter than the cycle length of the electrograms recorded within the coronary sinus (Figure 1).

All pulmonary vein tachycardias were eliminated by either pulmonary vein isolation, targeted ablation of an arrhythmogenic fascicle, or pulmonary vein encirclement. If pulmonary vein tachycardias were present within the ipsilateral pulmonary veins, the veins were encircled as described previously. If only 1 pulmonary vein was present, conscious sedation was achieved with midazolam and fentanyl.

Ablation at the pulmonary vein ostia and within the coronary sinus was performed at a power of 35 W and temperature of 50°C. Ablation in the left atrium was performed at a power of 70 W and temperature of 50°C. Applications generally were 15 to 30 seconds in duration. Ablation near the esophagus was avoided whenever possible. If a critical target site was located near the esophagus, ablation was performed at a maximum power of 35 W and temperature of 45°C for ≤10 seconds.

Study Protocol

The study protocol was approved by the institutional review board. AF was induced by rapid atrial pacing in patients who were in sinus rhythm. Pacing was performed within the coronary sinus for 10 seconds at the shortest cycle length that yielded 1:1 atrial capture. If pacing alone was insufficient to induce sustained AF, rapid pacing was repeated during isoproterenol infusion. Sustained AF was inducible in all patients.

The pulmonary veins were mapped with the decapolar ring catheter. Pulmonary veins that harbored pulmonary vein tachycardias were identified (Figure 1). As described previously, a pulmonary vein tachycardia was said to exist when the cycle length of the electrograms recorded within a pulmonary vein was shorter than the cycle length of the electrograms recorded within the coronary sinus (Figure 1).

All pulmonary vein tachycardias were eliminated by either pulmonary vein isolation, targeted ablation of an arrhythmogenic fascicle, or pulmonary vein encirclement. If pulmonary vein tachycardias were present within the ipsilateral pulmonary veins, the veins were encircled as described previously. If only 1 pulmonary vein was present, conscious sedation was achieved with midazolam and fentanyl.

Ablation at the pulmonary vein ostia and within the coronary sinus was performed at a power of 35 W and temperature of 50°C. Ablation in the left atrium was performed at a power of 70 W and temperature of 50°C. Applications generally were 15 to 30 seconds in duration. Ablation near the esophagus was avoided whenever possible. If a critical target site was located near the esophagus, ablation was performed at a maximum power of 35 W and temperature of 45°C for ≤10 seconds.

Study Protocol

The study protocol was approved by the institutional review board. AF was induced by rapid atrial pacing in patients who were in sinus rhythm. Pacing was performed within the coronary sinus for 10 seconds at the shortest cycle length that yielded 1:1 atrial capture. If pacing alone was insufficient to induce sustained AF, rapid pacing was repeated during isoproterenol infusion. Sustained AF was inducible in all patients.

The pulmonary veins were mapped with the decapolar ring catheter. Pulmonary veins that harbored pulmonary vein tachycardias were identified (Figure 1). As described previously, a pulmonary vein tachycardia was said to exist when the cycle length of the electrograms recorded within a pulmonary vein was shorter than the cycle length of the electrograms recorded within the coronary sinus (Figure 1).

All pulmonary vein tachycardias were eliminated by either pulmonary vein isolation, targeted ablation of an arrhythmogenic fascicle, or pulmonary vein encirclement. If pulmonary vein tachycardias were present within the ipsilateral pulmonary veins, the veins were encircled as described previously. If only 1 pulmonary vein was present, conscious sedation was achieved with midazolam and fentanyl.

Ablation at the pulmonary vein ostia and within the coronary sinus was performed at a power of 35 W and temperature of 50°C. Ablation in the left atrium was performed at a power of 70 W and temperature of 50°C. Applications generally were 15 to 30 seconds in duration. Ablation near the esophagus was avoided whenever possible. If a critical target site was located near the esophagus, ablation was performed at a maximum power of 35 W and temperature of 45°C for ≤10 seconds.
isolation was performed, depending on the location of the arrhyth-
mogenic fascicle(s) and the location of the esophagus. Although
complete isolation of the pulmonary veins was not a required end
point,18 elimination of all pulmonary vein tachycardias was required.

AF was reinduced by rapid atrial pacing whenever AF terminated
during ablation. After elimination of all pulmonary vein tachycardias, if AF still was present or inducible, left atrial mapping
was performed to identify and ablate sites of complex electrograms
or electrograms that had a shorter cycle length than in the coronary
sinus (Figure 2). Mapping of the left atrium was performed system-
atically, starting with the septum, then the roof, anterior wall, base of
the appendage, and posterior left atrial wall. After elimination of all
target sites in the left atrium, additional ablation was performed
within the coronary sinus and/or the superior vena cava to target
complex or rapid electrograms (relative to the coronary sinus) until
AF became noninducible or until all target sites were eliminated.

When a repeat ablation procedure was performed for recurrent AF
during follow-up, the same tailored ablation strategy was used.

End Points of Catheter Ablation

The end point of ablation was termination and noninducibility of AF.
Reinduction was attempted 5 times by pacing in the coronary sinus
for 10 seconds at the shortest cycle length, resulting in 1:1 atrial
capture. AF was considered inducible if it lasted >1 minute. When
AF became noninducible, isoproterenol was infused at rates of 10 to
20 μg/min, and further mapping and ablation were performed if
frequent atrial ectopy or AF was provoked. If AF had not terminated
or still was inducible after ablation of all target sites, sinusrhythm
was restored by pharmacological (ibutilide) or transhoracic cardio-
version. If there was an immediate recurrence of AF, additional
mapping and ablation were performed, with a focus on the pulmo-
nary veins.19

Postablation Management

Patients received intravenous heparin and warfarin during an over-
night hospital stay. They were discharged from the hospital the next
day and were treated with low-molecular-weight heparin until the
international normalized ratio was >2.0. Warfarin was administered
for 3 months. Class I or III antiarrhythmic drugs were used for 8
weeks after the procedure in 90 patients (59%) who had been treated
with an antiarrhythmic drug before the procedure or who had an
early recurrence of AF or atrial flutter. Among these 90 patients, 25
(16%) received amiodarone, 4 (3%) received dofetilide, 14 (9%)
received sotalol, and 47 (31%) received a class I antiarrhythmic drug.

Follow-up

The patients were seen in an outpatient clinic at 3 months after the
procedure and every 3 months thereafter. Patients were instructed to
call whenever they experienced symptoms and were provided with
an autotriggered event monitor to document the nature of their
symptoms. A dedicated nurse practitioner contacted each patient
every 6 to 8 weeks after the procedure for follow-up. No patient was
lost to follow-up. The absence of AF and atrial flutter was confirmed
in all asymptomatic patients with serial ECGs and/or 24-hour Holter
monitor recordings. A 30-day autotriggered event monitor was
ordered for all patients. However, only 30% of patients wore the
event monitor. The remaining patients declined the device because of
lack of insurance coverage or because they did not think it was
necessary. Because early recurrences of AF may be transient,20 a
blanking period of 8 weeks was used. Freedom from AF and atrial
flutter was defined as the absence of recurrent AF or atrial flutter
(starting at 8 weeks of follow-up) in the absence of antiarrhythmic
drug therapy. The mean duration of follow-up was 11±4 months
after the last ablation procedure.

Statistical Analysis

Continuous variables are expressed as mean±1 SD and were
compared by Student’s t test. Categorical variables were compared
by χ² analysis or by Fisher exact test when appropriate. P<0.05
indicated statistical significance.

The authors had full access to the data and take full responsibility
for its integrity. All authors have read and agree to the manuscript as
written.

Results

Target Sites and Catheter Ablation

Among the 153 patients, only the right-sided pulmonary veins
were encircled in 15 patients (10%), only the left-sided pulmonary
veins were encircled in 5 patients (3%), and both the right- and left-sided pulmonary veins were encircled in 50
patients (33%). In 69 of the 153 patients (45%), ≥1 pulmo-
nary vein was encircled (Figure 3). Focal applications of
radiofrequency energy to eliminate pulmonary vein
tachycardias were delivered at the ostium of a left superior
pulmonary vein in 36 patients (24%), right superior pulmo-
nary vein in 29 patients (19%), left inferior pulmonary vein in
30 patients (20%), and right inferior pulmonary vein in 19 patients (12%).

After elimination of pulmonary vein tachycardias, left atrial ablation to target driver tachycardias and/or complex electrograms was performed in 106 of the 153 patients (69%; Figure 4). Radiofrequency energy was also delivered in the coronary sinus in 71 patients (46%), and tachycardias originating in the superior vena cava were ablated in 9 patients (6%; Figure 5).

The mean duration of radiofrequency energy application was $32 \pm 11$ minutes. The mean fluoroscopy and procedure times were $48 \pm 18$ and $201 \pm 41$ minutes, respectively.

**Termination and Noninducibility**

AF converted to sinus rhythm (45%) or to atrial tachycardia/flutter (27%) in 110 of 153 patients (72%; Figure 6). In the 110 patients who converted to sinus rhythm or atrial tachycardia/flutter during ablation, AF could be reinduced in 20%.

Overall, AF was rendered noninducible in 58% of the 153 patients (Figure 6). In the remaining 42% of the patients, sinus rhythm was restored by cardioversion.

**Proarrhythmia**

Left atrial flutter developed after ablation in 29 of 153 patients (19%). The left atrial flutter resolved spontaneously in 50% of these patients by 12 weeks of follow-up. In 15 patients (10%), left atrial flutter was persistent beyond 12 weeks after ablation.

**Repeat Ablation**

At a mean follow-up of $8 \pm 2$ months after the first procedure, a repeat ablation procedure was performed in 28 patients (18%) for recurrent AF in 20 (13%) or for atypical atrial flutter in 8 (5%).

During the repeat ablation procedure in 20 patients with recurrent AF, arrhythmogenic activity in pulmonary veins that were not initially targeted was identified in 12 (60%); recovery of conduction into previously targeted pulmonary veins was observed in 3 (16%); and arrhythmogenic activity in the superior vena cava that was not previously recognized, in addition to the pulmonary veins, was observed in 2 (11%). In 1 patient, no arrhythmogenic activity in the pulmonary veins or superior vena cava was observed, and additional ablation was performed to target residual complex electrograms. In the other 2 patients, there were immediate recurrences of AF originating in a previously targeted left-sided pulmonary vein. However, because of close proximity to the esophagus, effective applications of energy were not delivered at these sites during the first procedure. Because the

---

**Figure 3.** Tailored ablation. After encircling of the right-sided pulmonary veins to eliminate pulmonary vein tachycardias, left atrial ablation was performed to target high-frequency and/or complex electrograms. Shown are the right anterior oblique (A) and posteroanterior (B) projections of the 3D left atrial shell. Red tags indicate sites of radiofrequency energy applications. RS indicates right superior; PV, pulmonary vein; LS, left superior; RI, right inferior; and LI, left inferior.

**Figure 4.** An example of tailored ablation. Shown are the right anterior oblique (A) and left anterior oblique (B) projections of the left atrial shell. Radiofrequency energy was delivered at the ostia of the left and right superior pulmonary veins and also in the left atrium. Abbreviations are as in Figure 3.
esophagus had moved, these sites were effectively targeted during the repeat ablation procedure.

Freedom From Recurrent AF and Atrial Flutter
During a mean follow-up of 11±4 months after the last ablation procedure, 118 of the 153 patients with paroxysmal AF (77%) were free from recurrent AF or atrial flutter in the absence of antiarrhythmic drug therapy. Seven patients (5%) had atypical atrial flutter, and 28 patients (18%) still had paroxysmal AF. The mean time to the first recurrent episode of AF after the blanking period was 27±35 days (range, 1 to 124 days; median, 9 days). Four of the 28 patients with paroxysmal AF had >50% reduction in the duration and frequency of their symptoms in the absence of antiarrhythmic drug therapy.

Noninducibility and Freedom From AF
Eighty-eight percent of the 88 patients whose AF was rendered noninducible were free of AF or atrial flutter at a mean of 11±4 months of follow-up, compared with 66% of the 65 patients who acutely remained in AF or still had inducible AF after ablation (P=0.003).

Complications
During 180 left atrial procedures in 153 patients, pericardial tamponade developed in 2 patients (1%). This was managed by percutaneous drainage in 1 patient and by surgical repair in the other. Transient neurological events occurred in 2 patients (1%). All patients were discharged home without any sequelae.
Main Findings

In this study, a tailored ablation strategy that targeted driver tachycardias and complex electrograms without a predetermined anatomic lesion set or routine pulmonary vein isolation resulted in freedom from recurrent AF/flutter in ~80% of patients with paroxysmal AF. An electrophysiological end point for ablation was used and was achieved in a majority of, but not all, patients. In patients whose AF was rendered noninducible, the long-term efficacy of the ablation procedure was significantly higher than when this electrophysiological end point was not achieved.

Tailored Ablation Versus Prior Ablation Techniques

The tailored ablation strategy in this study differs from prior ablation strategies that used a standardized, predetermined lesion set in 2 major ways, as follows.

First, tailored ablation avoids unnecessary applications of radiofrequency energy. Because noninducibility of AF is the procedural end point, ablation is no longer required once AF terminates and is noninducible. In contrast, during pulmonary vein isolation or circumferential pulmonary vein ablation, radiofrequency energy applications are continued until the lesion set is complete and/or all veins are isolated. With a tailored approach, radiofrequency energy is not routinely delivered at sites where the risk of fistula development is high, whereas during circumferential pulmonary vein ablation, part of the lesion set often impinges on the esophagus. Furthermore, the total amount of radiofrequency energy delivered may be less during tailored ablation. In this study, the mean total duration of radiofrequency energy applications was 32 minutes. This compares favorably with the amount of radiofrequency ablation reported in prior studies. For example, the mean duration of radiofrequency energy applications with circumferential pulmonary vein ablation was 42 minutes.17

Second, the tailored approach seeks to identify mechanisms of AF that are not within the pulmonary veins or included in the encircling lesions of circumferential pulmonary vein ablation. The genesis of AF is multifactorial, and a majority of the triggers and drivers that initiate and perpetuate AF may originate from the pulmonary veins in patients with paroxysmal AF. However, the left atrium and other sites may also be important in some patients with paroxysmal AF. By seeking noninducibility as the procedural end point, it may be possible to identify these other mechanisms of AF.

Tailored Ablation: Mechanisms of Action

The efficacy of tailored ablation depends on the accurate identification of driver mechanisms of AF. On the basis of prior studies that demonstrated the critical role that pulmonary vein tachycardias play in the initiation and perpetuation of AF,13,14,19 pulmonary vein tachycardias, if present, were targeted as the first step in the tailored approach. In the course of targeting pulmonary vein tachycardia, complete pulmonary vein isolation was achieved for some pulmonary veins. However, complete pulmonary vein isolation was not a required end point because pulmonary vein tachycardias often can be eliminated in the face of residual pulmonary vein conduction.18

Although pulmonary vein arrhythmogenicity plays an important role in the initiation and perpetuation of AF, it is not the only mechanism of AF. Ablation techniques that target only the pulmonary vein ostia have had modest efficacy.3,7,21 Consistent with the multifactorial nature of AF,8 autonomic innervation of the left atrium,22,23 nonpulmonary venous arrhythmogenicity,24 multiple reentrant wavelets,25 and anisotropic reentry leading to rotors26,27 all have been implicated in the genesis of AF. Therefore, elimination of only the pulmonary vein input may not be sufficient in all patients with AF.

In the tailored approach to AF ablation, attempts are made to identify all potential drivers of AF, not just the ones originating in the pulmonary veins. Left atrial sites where high-frequency and/or complex electrograms are recorded are ablated. Animal studies demonstrated that drivers rotate AF in the left atrium.26-27 The central core of these rotors may have high-frequency electric activity, whereas the periphery of the drivers may display complex electrograms because of wave break and fibrillatory conduction.28,29 Complex fractionated electrograms may also indicate sites of wave collision, slow conduction, or pivot points for reentrant circuits30 and may facilitate multiple wavelet reentry. It is also possible that complex electrograms reflect underlying ganglionic plexi, providing another potential reason for ablating these sites.31

Inducibility of AF

Consistent with the results of prior studies, the acute noninducibility of AF after ablation was associated with a better clinical outcome than in patients left with inducible AF.14,32,33 Unlike prior reports, however, the noninducibility of AF was the primary end point of the procedure. The findings of this study suggest that the noninducibility of AF is a clinically useful end point that usually is accurate in indicating when a sufficient number of AF drivers have been ablated.

In this study, >60% of patients who still had inducible AF acutely after ablation remained free from recurrent AF. It is possible that progressive fibrosis and lesion maturation during follow-up accounts for a favorable long-term clinical outcome in these patients. It also is possible that AF did not recur during follow-up because the triggers of AF were eliminated.

The Esophagus

With the tailored ablation strategy, clinical efficacy comparable to that of other techniques was achieved with few or no applications of radiofrequency energy near the esophagus. This suggests that ablation of a critical number of drivers may have been sufficient to eliminate AF even when drivers near the esophagus were not ablated. However, in 2 patients, recurrent AF clearly was caused by arrhythmogenic pulmonary veins that could not be effectively isolated because of close proximity to the esophagus. Fortunately, because the esophagus was not near these pulmonary veins during the repeated procedure, effective ablation was achieved safely.

Prior Studies

A prior study described ablation of AF by targeting complex fractionated electrograms in 121 patients with paroxysmal
and chronic AF using a 4-mm tip ablation catheter.\textsuperscript{12} Although the induction protocol for AF was not described in detail in that study, a similar proportion of patients with paroxysmal AF were rendered noninducible in both studies. In contrast to the prior study, in this study the pulmonary veins were systematically mapped and arrhythmogenic fascicles were ablated before left atrial complex electrograms were targeted. Another difference is that ablation was also performed in the right atrium in the prior study, whereas right atrial ablation was limited to the superior vena cava in this study. In the prior study, a larger proportion of patients with paroxysmal AF (89\%) were reported to have a successful outcome. It is possible that the failure to ablate complex fractionated electrograms in the right atrium accounts for the lower success rate in the present study.

Limitations
Because of poor patient compliance, extended ECG monitoring to detect asymptomatic recurrences of AF was not performed in all patients. This study included only patients with paroxysmal AF. Therefore, the findings may not be applicable to patients with chronic AF. Additional studies are needed to determine the feasibility of tailored ablation in patients with chronic AF. However, our preliminary clinical experience suggests that the noninducibility of AF is not a useful acute end point for ablation of chronic AF.

Conclusions
A variety of mechanisms may play a role in the genesis of AF, and different mechanisms may be operative in different patients. With the use of a tailored ablation strategy, it may be possible to identify and eliminate the specific mechanism(s) of AF and at the same time minimize risk to the esophagus. By using noninducibility as an end point, unnecessary applications of radiofrequency energy can be avoided. However, it is noteworthy that the end point of noninducibility could not be achieved in 42\% of patients. It is clear that further studies are needed to better understand and identify the drivers of AF.

Disclosures
Drs Oral and Morady are founders and major stockholders of Ablation Frontiers, Inc, and have consulted for Ablation Frontiers and Biosense-Webster. The other authors report no conflicts.

References
CLINICAL PERSPECTIVE

Because the mechanisms of atrial fibrillation (AF) are multifactorial and may not be the same in different patients, a standardized ablation strategy may be more necessary in some patients and insufficient in others. Furthermore, ablation in the posterior left atrium should be minimized because of the risk of esophageal injury. This study investigated the feasibility of a tailored ablation strategy that targeted only the initiators and drivers of AF in 153 patients with paroxysmal AF. Ablation was guided by local electrograms that had a short cycle length and/or were complex. Among the target sites were the ostium and antrum of the pulmonary veins, left atrium, coronary sinus, and superior vena cava. Ablation was continued until AF terminated and became noninducible or until all target sites were ablated. The mean procedure time was <3.5 hours. During a mean follow-up of 11 months, 77% of the patients remained free from recurrent AF and atrial flutter. A repeat ablation was performed in 18% of the patients. This study demonstrates that the drivers of AF can be identified and ablated in a large proportion of patients with paroxysmal AF. Because the end point is reached as soon as AF is rendered noninducible, unnecessary applications of radiofrequency energy are avoided, and the risk of atrioesophageal fistula is minimized.
A Tailored Approach to Catheter Ablation of Paroxysmal Atrial Fibrillation
Hakan Oral, Aman Chugh, Eric Good, Sundar Sankaran, Stephen S. Reich, Petar Igic, Darryl Elmouchi, David Tschopp, Thomas Crawford, Sujoya Dey, Alan Wimmer, Kristina Lemola, Krit Jongnarangsin, Frank Bogun, Frank Pelosi, Jr and Fred Morady

_Circulation_. 2006;113:1824-1831; originally published online April 10, 2006; doi: 10.1161/CIRCULATIONAHA.105.601898

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2006 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/113/15/1824

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation_ is online at:
http://circ.ahajournals.org/subscriptions/